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Alternatives to pesticides and paint

Insect pests are an intimate part of every home.

In the air, carpet, counter or cupboard, every home shares it's resources with these tiny, often unseen invaders. Pesticides are available for most common household insect pests, but these potent chemical compounds may be more harmful to you and the environment than the pests.

Here are some natural, non-toxic ways to control household insect pests.

Ants

The first line of defense is to remove the attractants: keep counters free of crumbs and sticky spots. Cover the sugar and put the honey jar in a plastic baggie. Cut off water sources such as drips or dishes left soaking overnight. If the ant invaders persist, try these simple measures:

- **Keep a small spray bottle handy, and spray the ants with a bit of soapy water.**
- **Set out cucumber peels or slices in the kitchen or at the ants' point of entry. Many ants have a natural aversion to cucumber. Bitter cucumbers work best.**
- **Leave a few tea bags of mint tea near areas where the ants seem most active. Dry, crushed mint leaves or cloves also work as ant deterrents.**
- **Trace the ant column back to their point of entry. Set any of the following items at the entry area in a small line, which ants will not cross: cayenne pepper, citrus oil (can be soaked into a piece of string), lemon juice, cinnamon or coffee grounds.**
- **Mix one liter of water, one teaspoon of Borax and a cup of sugar. Soak cotton balls in the solution and place them in a small yogurt container with holes punched in the lids to allow ants access. Place container in a location where ants are present. Ants will carry the bait back to their colonies where it will eventually kill the colony. Important: use indoors only; must be kept away from pets and children.**
- **Leave a small, low wattage night light on for a few nights in the area of most ant activity. The change in light can disrupt and discourage their foraging patterns.**
- **Ants on the deck? Slip a few cut up cloves of garlic between the cracks.**
- **Clove oil-based commercial ant deterrents are available** online

Dust Mites

Microscopic dust mites are everywhere in the home - in our beds, clothing, furniture, book shelves and stuffed animals. For people with allergies or asthma, dust mites are a problem.

Here's how to reduce the dust mite population in your home:

- **Vacuum mattresses and pillows. For people with sensitivities to dust mite allergens, dust mite bedding is available with zippered, allergen-impermeable encasings designed to block dust mites.**
- **Wash bedding at 55 degrees Celsius (130F) or higher. Detergents and commercial laundry products have no effect on mites unless the water temperature is high.**
- **Keep books, stuffed animals, throw rugs and laundry hampers out of the bedroom of allergy sufferers. Wash stuffed animals occasionally in hot water.**
- **Tannic acid neutralizes the allergens in dust mite and animal dander. Dust problem areas with tannic acid powder, available at health food stores and pet centers.**
- **Cover mattress and pillows with laminated covers which prevent penetration by dust mites. Avoid fabric-covered headboards.**
- **Cover heating ducts with a filter which can trap tiny dust particles smaller than 10 microns.**
- **Avoid using humidifiers. Dust mites thrive on warmth and humidity.**

Cockroaches

The best defense against cockroaches is a clean kitchen and bathroom. If roaches are a problem in your home or apartment, vacuum well and wash the area with a strong soap. Dispose of the vacuum cleaner bag in a sealed container.

Also try:

- **It is a little known fact that roaches like high places. If you put boric acid on TOP of your kitchen cabinets (not inside), if space allows between ceiling and cabinets, the roaches will take the boric acid to their nests, killing all of them. Boric acid is toxic by mouth - keep away from children and pets.**
- **Diatomaceous earth is a safe alternative which can be sprinkled in areas where roaches congregate, especially hidden areas such a cabinet tops and behind appliances. Harmless to people, the tiny particles cut the waxy exoskeleton and kills the insect within 48 hours. For a week or so after the treatment, the dehydrating insects will search more actively for water. Therefore, do not be surprised if you see roaches more often after the treatment. Most roaches should be killed within two weeks of application.** more info or to purchase
- **Catnip is a natural repellent to cockroaches. The active ingredient is nepetalactone, which is non-toxic to humans and pets. Small sachets of catnip can be left in areas of cockroach activity. Catnip can also be simmered in a small amount of water to make a "catnip tea" which can be used as a spray to apply around baseboards and behind counters. This natural repellent should only be used in homes without cats!**
  (A site visitor who has tried this sends the following comments)
- **Keep a spray bottle of soapy water on hand. Spraying roaches directly with soapy water will kill them.**
- **In an empty one pound coffee can, place 1 or 2 pieces of bread which have been soaked thoroughly with beer. Place in areas known to have roach infestations.**
- **Leave bay leaves, cucumber slices or garlic in the affected area as deterrents.**
- **The fruit of the Osage orange tree, the hedgeapple, is a natural roach repellent. Leave one hedgeapple per room for effective deterrence up to two months. You can learn more about hedgeapples for pest control at hedgeapple.com.**
- **Non-toxic roach traps are commercially available.** more info or to purchase

Fleas

Fleas usually gain entry to your home through your pet or visitors' pets. For every flea on your pet, there may be as many as 30 more in the pet's environment.

Before reaching for pesticides, try these safer choices:

- **Bathe and comb your pet regularly. Use mild soap, not insecticides. If fleas are found on the comb, dip the comb in a glass of soapy water.**
- **Citrus is a natural flea deterrent. Pour a cup of boiling water over a sliced lemon. Include the lemon skin, scored to release more citrus oil. Let this mixture soak overnight, and sponge on your dog to kill fleas instantly.**
- **Add brewer's yeast and garlic, or apple cider vinegar, to your pets' food. However, it is not advisable to use raw garlic as a food supplement for cats.**
- **Cedar shampoo, cedar oil and cedar-filled sleeping mats are commercially available. Cedar repels many insects including fleas.**
- **Fleas in the carpet? The carpet should be thoroughly vacuumed especially in low traffic areas, under furniture, etc. Put flea powder in the vacuum cleaner bag to kill any fleas that you vacuum up, and put the bag in an outdoor garbage bin.**
- **Trap fleas in your home using a wide, shallow pan half-filled with soapy water. Place it on the floor and shine a lamp over the water. Fleas will jump to the heat of the lamp and land in the water. The detergent breaks the surface tension, preventing the flea from bouncing out.**
- **In the yard or garden, plant fleabane (Fleabane Daisy Erigeron speciosus) to repel fleas. This is an annual growing 16-24" tall with violet, daisy like flowers.**
- Nontoxic flea traps **are available commercially.**
- Flea Control Nematodes **can be used to control fleas in outdoor areas your pets frequent.**
- **For more information, read our article:** Natural Flea Control

Mosquitos

The first line of defense against mosquitos is to seal their point of entry. Mosquitos are most active in the early morning and early evening. They seek areas of still air because they are hampered by breezes. Close windows and doors on the side of your house which are opposite the breeze.

Then try:

- **The most important measure you can take is to remove standing water sources. Change birdbaths, wading pools and pet's water bowl twice a week. Keep your eavestroughs clean and well-draining. Remove yard items that collect water.**
- **In a New England Journal of Medicine study, oil of eucalyptus at 30% concentration prevented mosquito bites for 120.1 minutes, while Bite Blocker with 2% soybean oil kept bites away for 96.4 minutes. (the eucalyptus oil must have a minimum of 70% cineole content, the active therapeutic ingredient.) Citronella, a common alternative to DEET, performed poorly, warding off bugs for only 20 minutes.**
- **If you're using the barbeque, throw a bit of sage or rosemary on the coals to repel mosquitos.**
- **An effective natural bug repellent, mix one part garlic juice with 5 parts water in a small spray bottle. Shake well before using. Spray lightly on exposed body parts for an effective repellent lasting up to 5 - 6 hours. Strips of cotton cloth can also be dipped in this mixture and hung in areas, such as patios, as a localized deterrent.**
- **Neem oil is a natural vegetable oil extracted from the Neem tree in India. The leaves, seeds and seed oil of the Neem tree contain sallanin, a compound which has effective mosquito repelling properties. Neem oil is a natural product and is safe to use. Look for new Neem Oil-based commercial products on the market.**
- **Planting marigolds around your yard works as a natural bug repellent because the flowers give off a fragrance bugs and flying insects do not like.**
- **Campers often report that the very best mosquito repellent is Avon Skin-So-Soft® bath oil mixed half and half with rubbing alcohol.**
- **Safe, nontoxic** pheromone-based mosquito traps **are now commercially available.**
- **Commercial** DEET- free mosquito repellents **are now available on the market.**

Is DEET safe?

The active ingredient in most chemical-based mosquito repellents is DEET (N,N-diethyl-meta-toluamide), developed by the US military in the 1940s. This powerful chemical is absorbed readily into the skin, and should be used with caution. Common side-effects to DEET-based products include rash, swelling, itching and eye-irritation. often due to over-application. For safer use, consider the following:

~ The American Academy of Pediatrics recommends that repellents used on children contain no more than 10% DEET. Parents should assist children in applying DEET-based products.
~ Lotions can be applied more effectively than sprays. Only a thin layer should be used.
~ Be careful to avoid areas near the eyes or mouth.
~ Wash skin exposed to DEET after coming in from mosquito areas.
~ Minimize exposed skin areas by wearing long-sleeved shirts and long pants, if possible.

Recent research suggests that DEET products, used sparingly for brief periods, are relatively safe. Other research points to toxic encephalopathy associated with use of DEET insect repellents. Experts warn that DEET shouldn't be used in combination with sun-screen because DEET shouldn't be reapplied often.

Thai lemon grass

(Cymbopogon citratus) is a natural and effective mosquito repellent. It contains the natural oil, citronella, which is safe and effective; in fact, lemon grass citronella is considered more effective than true citronella as an insect repellent.

You can buy Thai lemon grass at garden centers and supermarkets, and it grows readily into a clump about 15" across and about 2ft tall. To use as a mosquito repellent, break a stalk off from the clump, peel off the outer leaves, until you find the scallion-like stem at the base. Bend the stem between your fingers, loosening it, then rub it vigorously between your palms - it will soon become a pulpy, juicy mass. Rub this over all exposed skin, covering thoroughly at least once. You can also make a tincture using alcohol, for spray applications. Plantings around the patio will also help repel mosquitoes.

Flies

- **Use mint as a fly repellent. Small sachets of crushed mint can be placed around the home to discourage flies.**
- **Bay leaves, cloves and eucalyptus wrapped in small cheesecloth squares can be hung by open windows or doors.**
- **Place a small, open container of sweet basil and clover near pet food or any open food in the house.**
- **A few drops of eucalyptus oil on a scrap of absorbant cloth will deter flies. Leave in areas where flies are a problem.**
- **You can make your own flypaper with this simple recipe: Mix 1/4 cup syrup, 1 tbsp. granulated sugar and 1 tbsp. brown sugar in a small bowl. Cut strips of brown kraft paper and soak in this mixture. Let dry overnight. To hang, poke a small hole at the top of each strip and hang with string or thread.**
- **A site visitor suggests a light spray of Pine Sol on affected areas will deter flies.**
- **Safe, nontoxic, pheromone-based** outdoor **and** indoor fly traps **are available.**
- **For outdoor infestations, the best trap you can use is the Flies Be Gone trap, now available in our online store.**

Other Home Insect Pests

Wasps
Eartheasy has a separate page about natural wasp control. See Natural Wasp Control

Moths
Cedar chips in a cheesecloth square, or cedar oil in an absorbant cloth will repel moths. The cedar should be 'aromatic cedar', also referred to as juniper in some areas.
Homemade moth-repelling sachets can also be made using any of the following: bay leaves, cinnamon sticks, cloves, eucalyptus leaves, lavender, pepper corns or wormwood.
Dried lemon peels are also a natural moth deterrent - simply toss into clothes chest, or tie in cheesecloth and hang in the closet.
Natural attractant pheromones have been developed for controlling moths, and are now available as clothes moth traps **and** pantry moth traps**.**

Earwigs
Diatomaceous earth is a safe and effective way to control earwigs in the home. One application in key spots (bathroom, baseboards, window frames) can be a long-term repellent.
To trap earwigs, spray a newspaper lightly with water, roll it up loosely and secure with a string or rubber band. Place on the ground near earwig activity. The next morning pick up and discard the paper in a sealed container. Another method to trap earwigs is to take a shallow, straight-sided container and fill it half full with vegetable oil. Clean the trap daily; the oil can be re-used.

Silverfish
Silverfish prefer damp, warm conditions such as those found around kitchen and bathroom plumbing. Start by vacuuming the area to remove food particles and insect eggs. Silverfish can be easily trapped in small glass containers. Wrap the outside with tape so they can climb up and fall in. They will be trapped inside because they cannot climb smooth surfaces. Drown them in soapy water. The best preventive control is to remedy the damp conditions.
Nontoxic silverfish traps **are also commercially available.**

Making A Difference With Non Toxic Paint and Wood Finish.

Source: http://www.earthpaint.net/

It has never been more vital to make a difference with non toxic paint and wood finish. Clearly, the planet is heading in a dangerous direction, threatening everyone on earth. Dangerous petrochemicals inundated every facet of our life [baby clothes, food, diapers, deodorant, fuel, heat, sunscreen, blankets, computers, toothpaste, candy, paint, deck stain, wood finish, floor finish and scariest of all is medicine!].

From Bhopal to Alaska to the Gulf of Mexico, chemicals are everywhere in America and the world. Weaning off these petro poisons is not easy for any industry that is dependent on them to put food on the table. It's kind of like burning down the house to cook dinner! They seem to make living better, cheaper, more affordable, more convenient but they eventually make living worse, more difficult and painful to watch as they kill the people and the planet in front of our eyes.

The paint and wood finish industry has historically been dominated by petrochemicals. Some chemicals are safe but many are dangerous petrochemicals. These toxins have been overused and minimized. The dangers have been shirked off and sometimes disregarded altogether. Two of the worst offenders are Deck Stain and Floor Finish but Wood Finish and Paint of all types have been loaded with dangerous petrochemicals. It has gotten so bad that even the EPA put regulations on petrochemical solvents in paint and wood Finish. They call this VOC regulation. Most people have heard of VOC or volatile organic compounds (primarily petrochemical solvents). VOC's have become well known but many other dangerous chemicals lurk beyond the scope of the VOC measurements. Low Voc deck stain and wood finish simply is not enough. Non toxic paint and wood finish cannot be measured by VOC claims on a fancy label. It requires more.

Finally! Beautiful Paint & Wood Finish without the Poisons!!

Earthpaint produces only Healthy Sustainable Finish! Earthpaint was founded by Tom Rioux, a professional painter for 25 years. Tom was poisoned by the paints and wood finish he was told were safe. He narrowly survived. This is the basis for Earthpaint's deep, life affirming commitment to make strong, safe paint and wood finish. Earthpaint now offers beautiful floor finish, deck stain, wood finish and non-toxic paint products that are as good or better than conventional paint and wood finish. Earthpaint was the first, and may still be the only, USA based Healthy Sustainable Finish manufacturer. Earthpaint is proving that there are better, kinder alternatives. We do not use toxic petrochemical solvents in deck stain. We do not use mineral spirits in our oil wood finish. No naphtha, toluene, xylene, vinyl or benzene in our wood finish. No Poison! We are confident that Earthpaint is providing the safest alternative on the planet!

Our biodegradable paint and wood finish contain non-toxic and natural ingredients derived from plants, vegetables, trees, minerals and elements. Nearly all of these finishing components are gathered locally (within a day's drive of Asheville, NC) and are domestically produced and harvested. We avoid using anything in our paint and wood finish that gets shipped in from overseas. Embodied energy and the entire ecological footprint is examined throughout the paint and wood finish formulation process. It is then reexamined on a yearly basis because things change and we need to be responsive to that.

The Safest Paint and Wood Finish On The Planet!

Earthpaint won't compromise our non toxic paint and wood finish quality by adding harmful ingredients, and we don't have to! We are making non-toxic, natural finish products that are as good as, or better than conventionally made paint and wood finish products. Did you know that paint and wood finish can emit or release poisonous gases for many years? Contrary to common belief, even with some of the newer acrylic latex or wood finish, toxic emissions do not stop when the paint dries and the "smell' goes away. This is why we offer high quality paint and wood finish without toxic petro-chemical solvents, without the toxic smell and other common hazards.

With Earthpaint you get the best non toxic paint and natural wood finish. Beautiful wood finish makes a home wonderful to be in. Clean smelling walls make you want to be there. You don't need to worry about how long the poisons will seep into grandma's room, or the new baby's room, or the children's room because there are NO POISONS in our non toxic paint and wood finish products. If there isn't any poison in wood finish to begin with, there's no need to worry at all. **Better, natural non-toxic paint and wood finish, *without* the poisons!** Nice!

We also consider the impact of a paint and wood finish product from it's inception to disposal, asking questions such as "How much energy do we use to make this wood finish?", "What resources are used in this wood finish or deck stain? Are they renewable? Sustainable?", "How does this Lime Clean mold eradicator decompose after its lifespan has ended?" If the answers aren't satisfactory we change the process until they are right. If an ingredient isn't safe for a painter it doesn't go in our paint or wood finish. If it's not safe for the planet...the animals...people -it does not go in!!!

Envirosocial Responsibility – Deck Stain and Wood Finish Made The Sustainable Way, Without Chemical Poisons!

We are determined to change the way paint, wood finish, deck stain and other wood finishing products are made. It takes millions of people to **HELP THE KIDS HAVE A LONG, HAPPY LIFE!** This is why we use terms like enviro-social responsibility and petro-poisons. There are millions of people who want colorful deck stain; Ultra Tough Wood finish; and decorative surfaces. But millions of people poison the planet when using toxic paint and wood finish. What about the kids? It's our environmental and social responsibility to help. By calling things what they are ***__poisons__*** the choice becomes more obvious. Buying healthy sustainable paint, deck stain and wood finish sends the message to those who are manufacturing petro poisons on a large scale. We want a healthy, strong world to live in and pass on to the kids!

Creating natural wood finish, deck stain and non toxic paint that can truly replace and reinvent the old toxic paint and wood finish standards requires new thought directives, new formulary, creative sustainable manufacturing processes and innovation. At Earthpaint, we combined these attributes with innovative thinking and expertise to reinvent wood finish with non-toxic, sustainable practices and the utilization of natural ingredient production capabilities. We have a true dedication to the environment and all things living and breathing on our planet!

When using Earthpaint natural wood finish products you are supporting responsible business practices that were developed with sustainable processes to ensure NO HARM comes to the planet or its inhabitants.

Community Based Non-Toxic Paint and Wood finish

Asheville, NC. based Earthpaint Inc., works closely with the community to provide beautiful, satisfying non-toxic paint, deck stain and wood finish without the dangers common to most wood finish. You will not find more beautiful paint and wood finish anywhere. We want to show that business and responsible eco-practices can co-exist and that paint, deck stain and wood finish are not synonymous with poison! Earthpaint wood finish and deck stains will highlight the dramatic beauty of all the floors throughout your home or office. The paint will look and smell great!

Presently, our innovative sustainable processes have shown us that we can produce safe paint, non-toxic wood finish, and other safe coatings competitively. Additionally, every geographic area has a unique set of natural resources that could create a uniquely suited *local* paint and wood finish. We can help identify these materials and use them to make non-toxic paint and wood finish locally and dramatically reduce the amount of transportation and processing fuel required for the production and distribution of paint, deck stain and wood finish.

Earthpaint uses earth friendly recycled plastic containers for some wood finish and deck stains. And quickly biodegradable tin cans for certain deck stain, wood finish and non toxic paint.

Use Essential Oils to Prevent Mosquito Bites: Catnip, Clove, Neem and Lemon Oil

Tuesday, June 08, 2010 by: Melanie Grimes, citizen journalist

(NaturalNews) Essential oils such as clove, catnip and lemon oil prevent mosquito bites. These oils repel mosquitoes, along with citronella and patchouli. Commercial insect repellants contain many harmful ingredients, are irritating to the skin and the eyes, and are potentially damaging to the immune system; therefore, natural plant oils are a preferable source for insect repellants.

Ingredients in Common Insect Repellants
Common ingredients in insect repellants include Picaridin, IR3535, and N-diethyl-3-methylbenzamide, also known as DEET. The U.S. government invented DEET during World War II. In use commercially since 1957, there are only 50 published cases of severe side effects, but many people have experienced irritated skin and eyes. Picaridin is less irritating to eyes, as is IR3535. These ingredients were tested at 20% concentrations, but products may not contain that percentage or active ingredients.

Catnip Oil Prevents Mosquito Bites
Undiluted catnip oil can provide up to two hours of insect repellant properties when applied directly to the skin. Research in 2001 showed that catnip oil repels mosquitoes ten times better than DEET. Further research showed that depending on the species of mosquito, protection lasted for up to four hours. The active ingredient in catnip oil is called nepetalactone. It can cause skin irritations to those with sensitivities, so a patch test is recommended.

Lemon Oil Repels Insects
Lemon oil, also known as Zanthoxylum limonella, is a powerful insect repellent. Used in a 10 to 30% concentration and added to coconut oil, lemon oil showed high protection against the bites of the Aedes albopictus mosquito in research conducted in 2003.

Clove Oil as Mosquito Repellent
Clove oil has been shown to be active as a mosquito repellent; however, it can be irritating to the skin unless used in dilution. The recommended dilution is below 24%. It can be added to olive oil or coconut oil to soothe the skin.

Neem Oil
Neem oil comes from a tree with remarkable healing properties. Because of its many uses in Ayurvedic medicine, it has been called the "village pharmacy." Neem contains azadirachtins, which are known insecticides. The plant is also an antiseptic, antifungal and antiviral.

Other Mosquito Bite Prevention Tips
To prevent mosquito bites, do not drink alcohol. Alcoholic drinks are thought to attract mosquitoes because of the increased sugar content added to perspiration. Some mosquito species, including the Aedes and Ochlerotatus, are attracted to dark colors, so wearing white, green or yellow is recommended; however, the mosquito species Anopheles may prefer light colors. Eating garlic and vitamin B has been purported to prevent mosquitoes from biting, but research has not shown this to be true.

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http://melanie-grimes.blogspot.com/...
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5 Ways to Reduce Children's Pesticide Exposure

Posted By Dr. Mercola | June 05 2010 | 36,321 views

U.S. News & World Report recommends the following steps:

1. **Buy organic fruits and vegetables.** Fruits and vegetables most likely to be grown using pesticides, if they are not organic, include celery, peaches, strawberries, apples, and blueberries.
2. **Detoxify your lawn.** If you have a lawn care service, make sure they are not using the organophosphate pesticide trichlorfon.
3. **Clean out your shed.** The pesticide diazinon (sold under the brand names Diazinon or Spectracide) has been banned from residential, but there might be some left in your old garden shed.
4. **Use natural cures for a lice infection.** Malathion is used for treatment of head lice. Don't put a neurotoxin on your child's head.
5. **Check your school's pest control policy.** If they have not already done so, encourage your school district to move to Integrated Pest Management, which uses less toxic alternatives.

Sources:

- U.S. News & World Report May 17, 2010
- MSNBC May 16, 2010

Dr. Mercola's Comments:

Most of us take an immediate negative reaction to mean that something is dangerous. But few experience any immediate reactions when they're exposed to a pesticide, which makes them all the more insidious.

The problem with pesticide exposure is that the majority of the negative reactions occur sometime in the future.

The scientific literature has already established convincing associations between pesticide exposure and neurological diseases like Parkinson's and muscular sclerosis. Now, a new study in the journal *Pediatrics* has found disturbing links between some of the most commonly used pesticides and a significantly increased risk of ADHD symptoms in children.

Organophosphate pesticides are the most common, accounting for as much as 70 percent of the pesticides used in the U.S. Unfortunately, they may be particularly detrimental to human health.

Their mode of action is to interfere with the nervous systems of insects, but they have a similar effect in mammals as well, including humans. For this reason, although diet is believed to be the number one source of pesticide exposure, you cannot underestimate the potential detrimental impact of *pre-birth* exposure to these toxins.

Clearly, the nervous system of a growing fetus will be far more susceptible to damage than that of an adult or even a child being exposed through the food he ingests.

So if you are pregnant or planning a pregnancy, I strongly urge you to take special precautions to avoid as many toxic exposures as you possibly can, especially pesticide exposure from your own diet before and during pregnancy.

Common Pesticides Linked to ADHD

In this study, the urine of 1,139 children between the ages of 8 and 15 were tested for six pesticide metabolites. One hundred and nineteen of the children were diagnosed with ADHD.

Children with a ten-fold increase in metabolites from the pesticide malathion (found in head lice treatments) were 55 percent more likely to be diagnosed with ADHD, and those with higher than average levels of metabolites from dimethyl thiophosphate were 93 percent more likely to have ADHD compared to those with undetectable levels of this marker.

Be Especially Aware of "Healthy" Berries

Berries, a perennial "healthy" favorite among many children, could actually be some of the worst offenders when it comes to pesticide exposure through your diet.

Conventionally-grown blueberries and strawberries in particular, tend to be frequently contaminated. According to MSNBC, one government report found malathion residue in:

- 28 percent of frozen blueberry samples
- 25 percent of fresh strawberry samples

Likewise, a 2007 EU report on hazardous pesticides found that strawberries in particular contained "a poisonous blend" of no less than 14 different pesticides! Five of them known carcinogens, and three suspected endocrine disruptors.

So what can you do to protect your child from these toxic pesticides?

The US News & World Report above offers several helpful tips, and I recommend you implement them all. However, since your child's diet is the number one source of pesticides, cleaning up in this area is clearly a must.

The most obvious course of action is to seek out organic foods whenever possible.

An added bonus of eating organic is that organic foods also typically contain far more nutrients than their conventionally-grown counterparts!

The biggest study ever into organic food – a four-year EU funded project called the Quality Low Input Food (QLIF) project – found that organic food is definitely about much more than a simple lifestyle choice. It's about getting more nutrition and better health.

For example, this study found that:

- Organic fruit and vegetables contain up to **40 percent more antioxidants**
- Organic produce had **higher levels of beneficial minerals like iron and zinc**
- Milk from organic herds contained up to **90 percent more antioxidants**

The researchers even went so far as to say that eating organic foods can help to increase the nutrient intake of people who don't eat the recommended five servings of fruits and vegetables a day.

Additionally, a 2003 study in the *Journal of Agricultural Food Chemistry* found that organic foods are better for fighting cancer. And in 2005, scientists found that, compared to rats that ate conventional diets, organically fed rats experienced various health benefits, including:

- Improved immune system status
- Better sleeping habits
- Less weight and were slimmer than rats fed other diets
- Higher vitamin E content in their blood (for organically fed rats)

Does Everything Have to Be Organic?

Unfortunately, many people believe they can't afford to buy organic. And although this is not necessarily true in many cases, there are ways you can get the most bang for your buck when buying organic.

Certain fruits and vegetables tend to be far more contaminated than others, simply because they're more susceptible to various infestations and therefore sprayed more heavily. Some foods are also more "absorbent," with thin, tender skins. Such foods would be high on your list for buying organic.

Fortunately, the Environmental Working Group (EWG) has performed the arduous task of conducting nearly 43,000 pesticide tests to determine which fruits and vegetables pose the highest and lowest risk for pesticide exposure.

Their *Shoppers' Guide to Pesticides in Produce* is well worth printing out for future shopping trips.

Of the 43 different fruit and vegetable categories tested, which are all listed in their guide, the following 12 fruits and vegetables had the **highest pesticide load**, making them the **most important to buy or grow organic**:

Peaches	Cherries
Apples	Lettuce
Sweet bell peppers	Grapes (imported)
Celery	Pears
Nectarines	Spinach
Strawberries	Potatoes

Additional Recommendations

Remember that processed foods are in fact processed with a variety of chemicals, and should therefore be avoided as much as possible. Children already diagnosed with ADHD in most cases need to eliminate processed foods entirely.

I also recommend you use only natural cleaning products in your home, especially if you have young children, or are pregnant. Most health food stores now carry natural cleaning products, or you can search online for them. I will also be carrying my own line of highly effective, non-toxic cleaning products shortly.

Switching over to natural brands of toiletries is also a good idea. You'd be surprised to find just how many well-known brands of baby products are loaded with harmful chemicals. The SkinDeep website is an excellent source to determine which brands are the safest for your baby.

Also avoid using insect repellants that contain DEET. It can be tempting to douse your child in order to help her avoid getting bitten, but please be very mindful of what you're putting on her skin. There are safe, effective and natural alternatives out there, like Neem-Based Botanical Outdoor Gel.

Healthier Homes and Gardens - February 2008

Children's Exposure to Pesticides: Diet vs. Home Pesticide Use

Children's exposure to pesticides has been documented in several "snapshot" studies that tested for a range of pesticide residues in single urine or blood samples. Using a novel approach, a recent study of Seattle area children tested two daily urine samples over a period of days looking for residues of two types of insecticides — organophosphates and pyrethroids. By alternating children's diet between conventionally and organically grown food, researchers were able to see a bigger picture that pointed toward food or other factors as the source of their exposure.

Results reported so far boiled down to this: Organophosphate insecticides were not used by any families in their homes, but the children were still exposed through their food except when they switched to organic produce. In the case of pyrethroids, children continued to be exposed, even when organic produce was substituted. Children whose families used pyrethroid insecticides in and around the home had the highest levels of pyrethroids and analysis showed that this home pesticide use was their primary source of exposure.

The Study

To learn more about the contribution of diet and residential pesticide use to children's pesticide exposure, researchers did a longitudinal study of 23 children ages 3 to 11 from the Seattle urban and suburban area. The children's families normally ate only conventionally grown food. During four seasonal study periods parents collected their child's urine twice a day and also kept food logs. The urine was tested for indicators (called metabolites) of both organophosphate and pyrethroid insecticides. Parents also reported on pesticides that were used in and around the home.

In the summer of 2003, researchers substituted organic food items for five of the fifteen day study period. Organic food samples were tested to confirm that they were free of any pesticide residues. In 2006, analyses of both organophosphate and pyrethroid data from this first study phase were published in two articles in the journal Environmental Health Perspectives.

The study continued with three more testing periods during the following fall, winter and spring. The report on organophosphates over this extended period was published online in January 2008 on the Environmental Health Perspectives web site.

Organophosphates

The first phase of the study clearly showed that children were exposed to organophosphates through their diet. All parents reported that they had not used organophosphate products in and around their home. However organophosphates were detected in children's urine throughout the study period. This changed dramatically during the five days when organic food items were substituted for matching items that they normally ate.

Focusing on two chemicals, chlorpyrifos and malathion, testing showed that median concentrations of the chemical indicators decreased to nondetectable levels within the 5 days. Reintroduction of conventional food items — primarily fruits and vegetables — brought pesticide residues back up to previous levels. The authors stated that "an organic diet provides a dramatic and immediate protective effect against exposures to organophosphate pesticides..."

Studies done during fall, winter and spring brought new insights. Organic food substitution in the fall study confirmed that food was the source of children's exposure. Looking at all four seasons, researchers concluded that eating fresh produce year round affected the residue levels of chlorpyrifos and malathion. Researchers suspected that imported food eaten during winter and spring might have been a contributing factor. Compared to domestically grown food, evidence from other sources shows that these two chemicals contaminate certain imported items more frequently and at higher levels.

By 2000, the Environmental Protection Agency had begun restricting uses of organophosphates because of their ability to harm the nervous system. At the time of this study, chlorpyrifos was already banned from use in homes and schools. Malathion products were (and still are) available for gardeners. These two chemicals and other organophosphates continue to be widely used in agriculture.

Pyrethroids

During the same 2003 summer study period described above, researchers also tested for indicators of pyrethroid insecticides that became more widely used in both urban and agricultural settings after some restrictions on organophosphates were implemented.

In the case of pyrethroids, eating organic foods instead of conventional foods did not produce any dramatic effects. Researchers detected pyrethroids throughout the study and many urine samples continued to show pyrethroid contamination during the organic period without indicating a downward trend.

However, differences came out when the seven children whose families reported using pyrethroid products in or around the home were compared to the other 16 children. Some of these seven children had the highest levels of some pyrethroid indicators. In the most extreme case, the parents had sprayed furniture, including beds. All seven children were exposed continuously during the 15 day study indicating that residues from home treatments were still present in the environment.

While analysis showed that children's exposure to pyrethroids was partially attributable to residues on food, use of home use of pesticides was the primary source of exposure. The parents reported using products that contained pyrethroids such as permethrin, deltamethrin and pyrethrins and were used for both garden pests or indoor pests such as fleas and carpenter ants.

Researchers were somewhat surprised to find that older children (8-11 years) in the study had higher levels of pyrethroids than younger children (3-7 years). Other studies have suggested that younger children are more exposed to pesticides, likely due to hand-to-mouth behavior and playing on the floor. In the Seattle study, families reported that older children were more involved in sports and other activities away from home than younger children. Researchers speculated that the older children may have been exposed to pyrethroid products used in recreation sites.

The researchers concluded that children's exposure to pyrethroid insecticides could be reduced by minimizing the use of these products around the home or preventing children from coming into contact with areas or objects that had been treated with these pesticides.

Corrected version posted March 6, 2008.

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NEWS STORY

Andrew Schneider. Seattle Post-Intelligencer. January 30, 2008.
Harmful pesticides found in everyday food products: Mercer Island children tested in yearlong study.
http://seattlepi.nwsource.com/local/349263_pesticide30.html

Health Effects of Pesticides on Children: evidence to policy

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September 27, 2010 - Portland, Oregon

Outline – the big picture

What do we know? What do we wish we knew?

- Vulnerability of children
- Sources and pathways of exposure for children
- An abbreviated review of the evidence base: Health endpoints
- Strengths/Limitations of available data

Moving forward to understand and reduce risk of pesticide use

- Research priorities and policy approaches

Child Vulnerability to Toxicity

Unique endpoints - Developmental toxicity

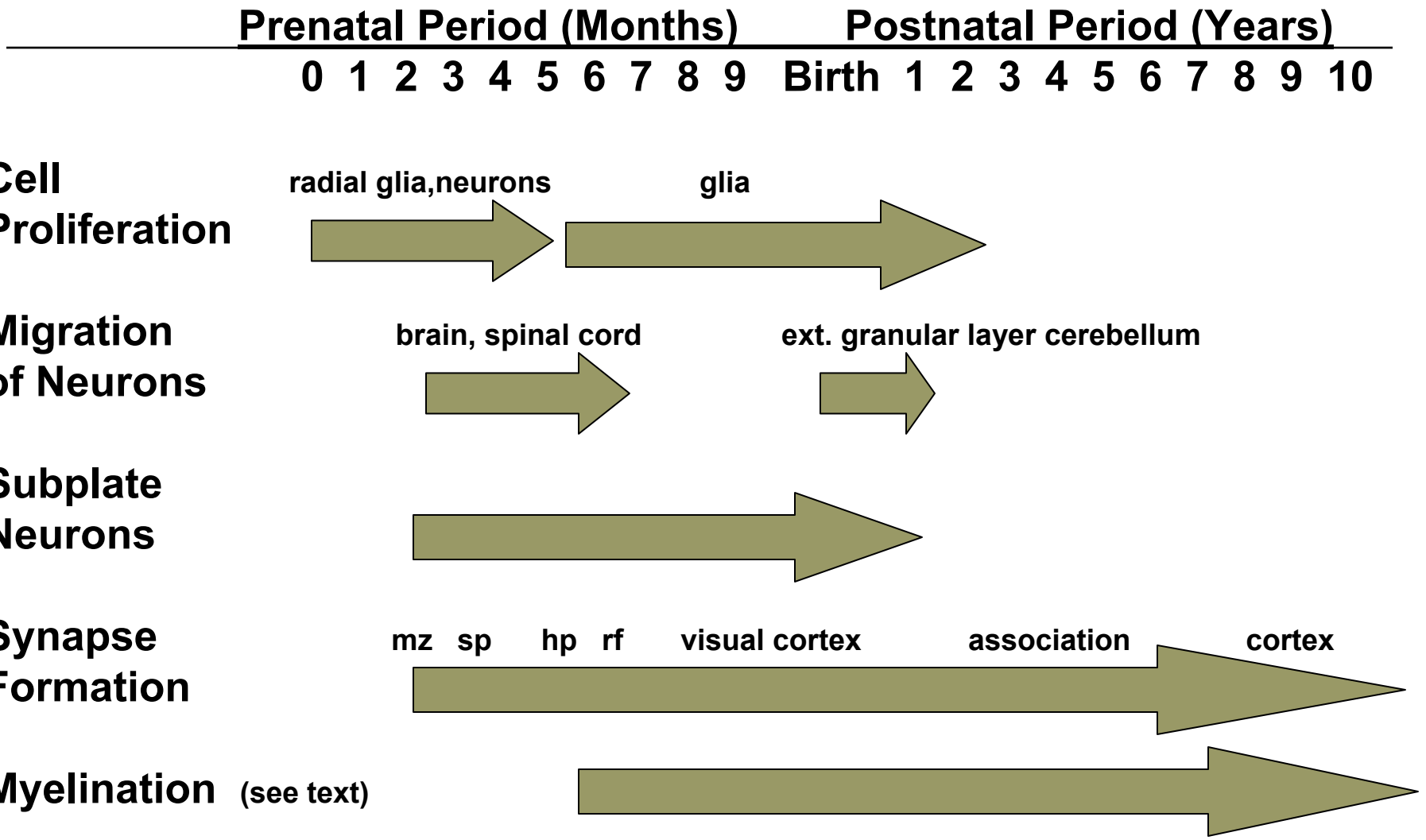
- Neurodevelopment
- Carcinogenicity
- Birth defects/fetal growth
- Immune/Pulmonary function/development
- Reproductive system function/development

Behavioral and physiological influences of childhood that increase exposure

Nervous system development

- "A little kid goes from a single cell to a laughing, sociable, intelligent, friendly human being over the course of two years. That's dramatic growth and development!"
  - Kenneth Olden, PhD, former Director, National Institute of Environmental Health Sciences

Time Lines of Brain Developmental Processes in Humans



Specific processes disrupted depends on **timing** and **toxic effect** of the particular exposure

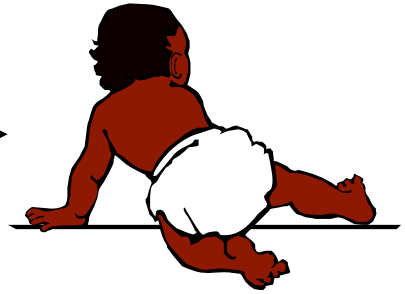
proliferation	**radiation, ethanol, mercury, organophosphate pesticides**
migration	**radiation, mercury, ethanol**
differentiation	**ethanol, nicotine, mercury, lead**
synaptogenesis	**radiation, ethanol, lead, triethyl tin, organophosphate pesticides, PCBs**
gliogenesis & myelinization	**low thyroid, ethanol, lead**
apoptosis	**ethanol, lead, mercury**
signaling	**ethanol, organophosphate pesticides, mercury, lead, PCBs**

Sources – Child/Pesticide Encounters

Residential Use – indoor/outdoor

School/Daycare – indoor/outdoor

Dietary



Drinking water

Rx – lice/scabies

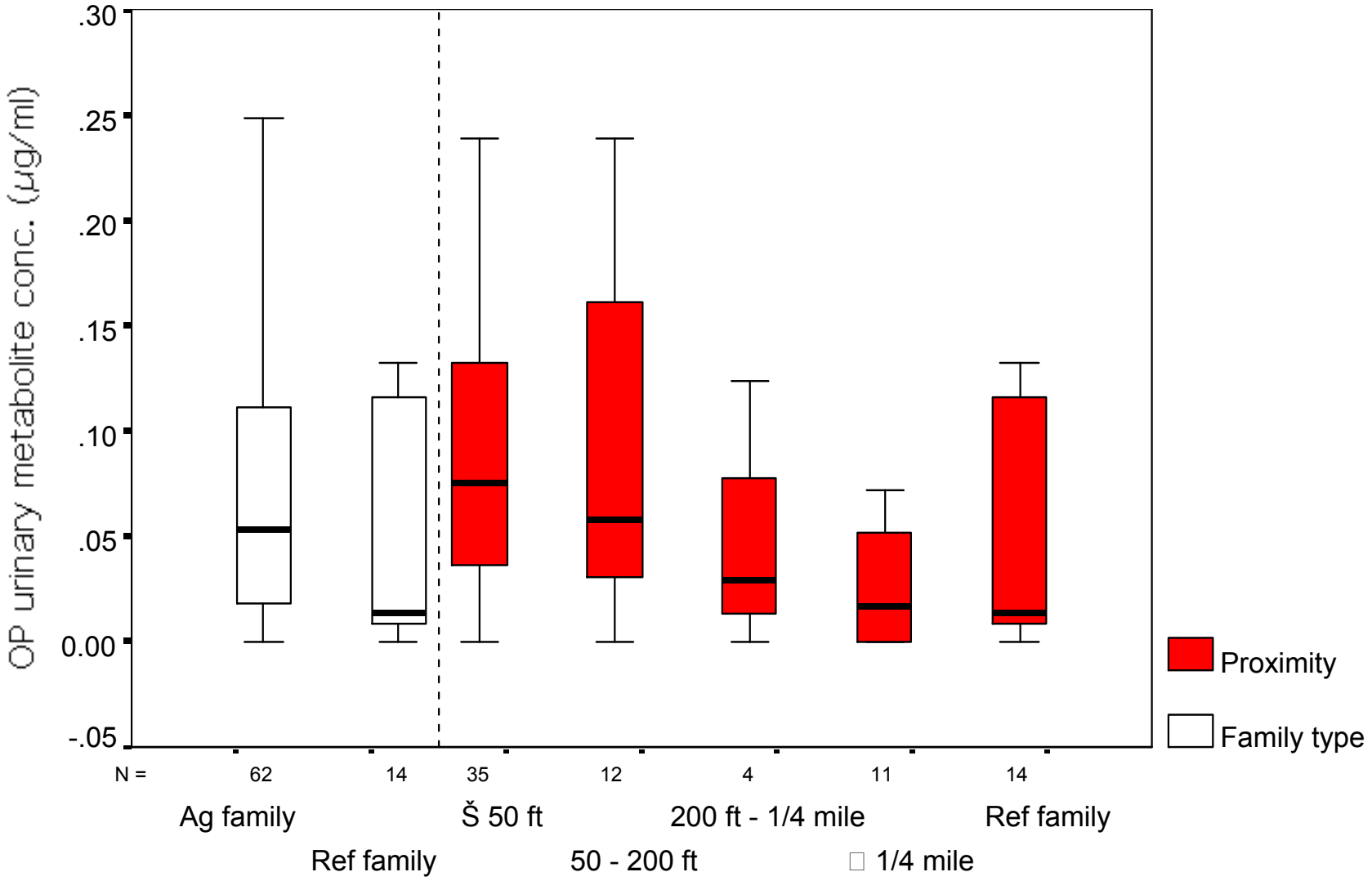
Parks/ neighborhood

Variability based on geography, cultural/social factors, policies

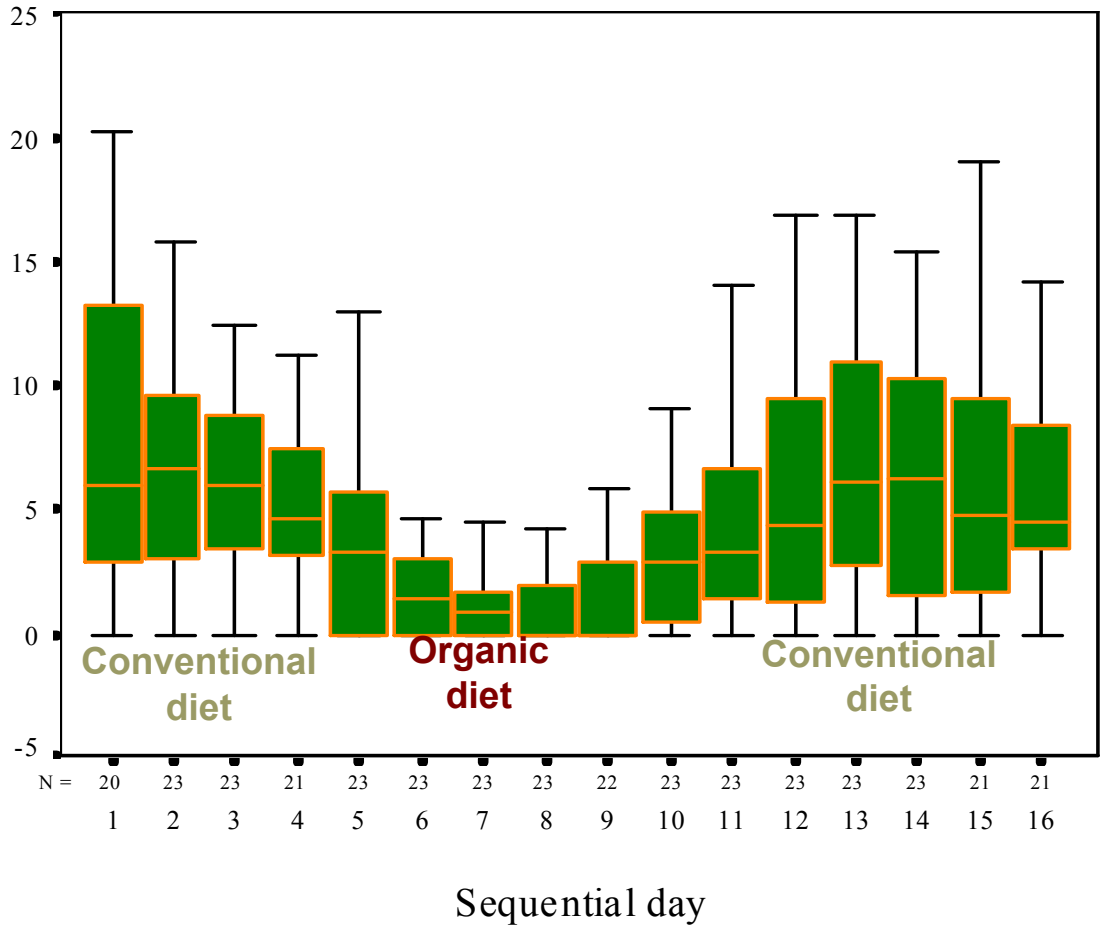
How much/what types from each source?

Parental Take-Home

Proximity to agricultural fields



TCPY Concentrations in the Urine of 22 Children Before, During, and After Organic Diet Intervention



Lu et al. 2005 Environ Health Perspect on-line

Indoor Exposure studies

Indoor application methods influences exposure magnitude

- Broadcast/"bomb" applications with semivolatile products >> crack and crevice treatments

Residues can linger in air, carpet, toys, and housedust

Typical exploratory behavior - playing on and crawling across the floor ↑ risk of dermal, inhalation and oral exposure to residues on surfaces or air as it settles

Lewis 2001, Hore 2005, Curwin 2005, Lu 2000, Fenske 1990

Incidental ingestion: soil-dust

	2.5 year old	Adult
Soil ingestion (mg/day)		
Indoor	50	20
Outdoor	60	0.4

	0-2 year	2-6 years	6-7 years	Adult
GI absorption				
% lead	42-53	30-40	18-24	7-15
% pesticide	?	?	?	?

Selevan. 2000

Exposures: child vs adult

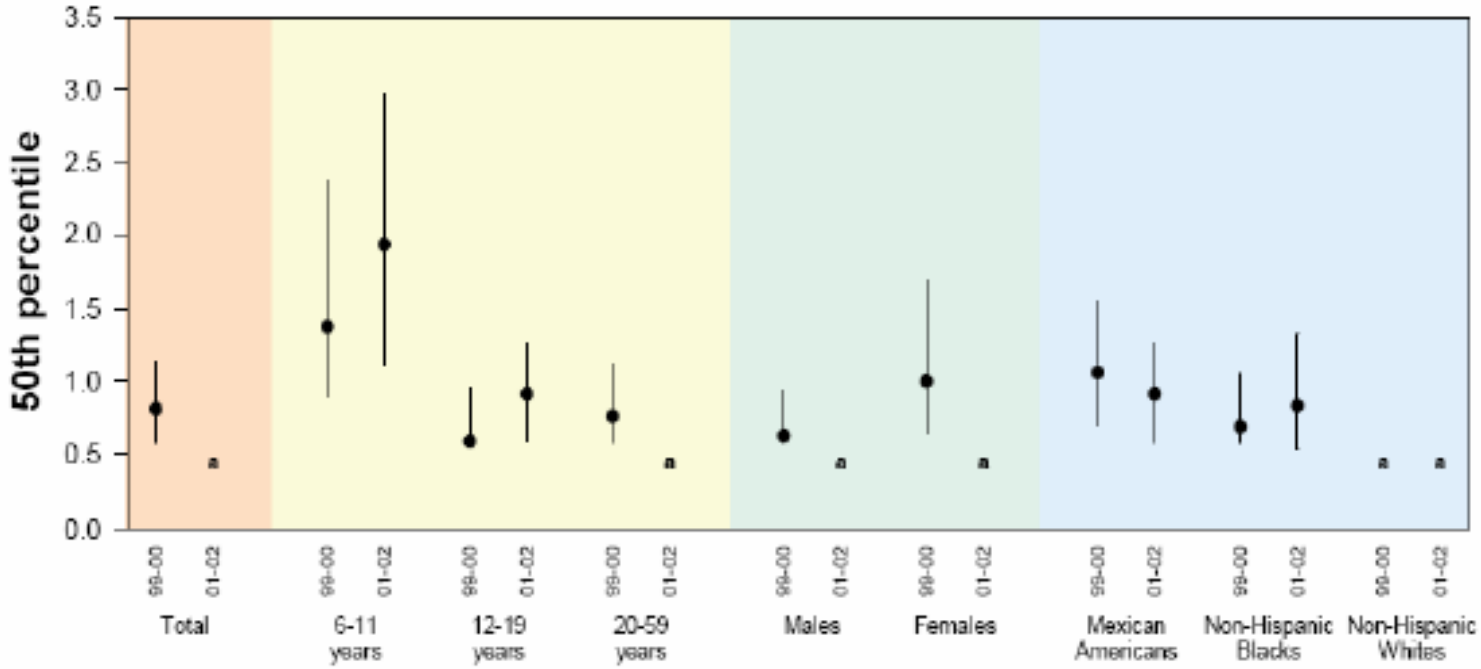
Age (years)	Apple (g/kg/day)	Tap Water (ml/kg/day)	Air (m^3 per kg per day)
<1	5.0	43.5	0.6
3-5	3.8	35.5	0.5
Adolescent/ Adult	0.4	18.2	0.3

Adapted from Selevan 2000

Body Burden of Organophosphate Metabolite

Figure 32. Dimethylphosphate (creatinine corrected)

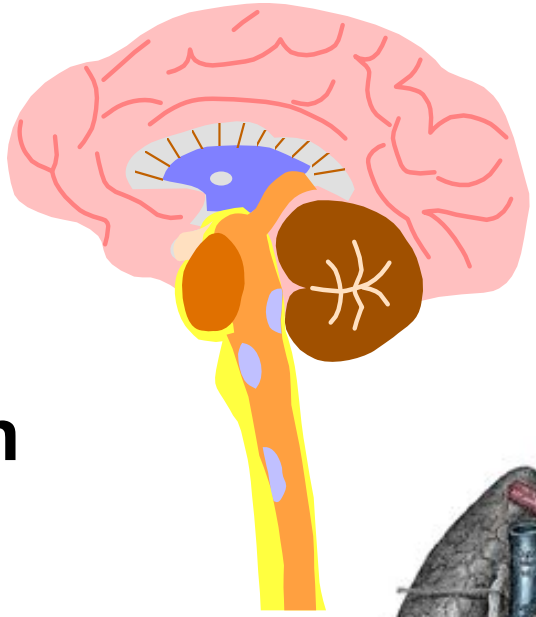
Selected percentiles with 95% confidence intervals of urine concentrations (in µg/g of creatinine) for the U.S. population aged 6-59 years, National Health and Nutrition Examination Survey, 1999-2002.

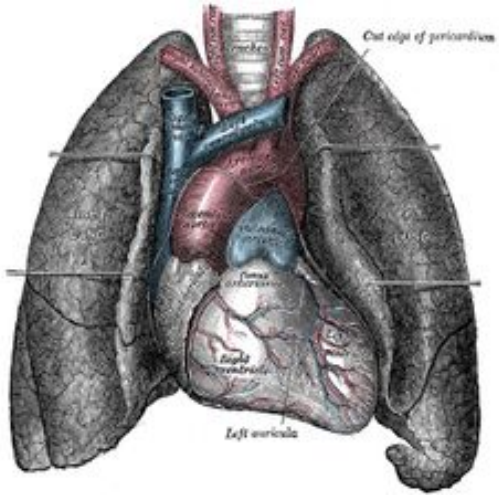


Source: Third *National Report on Human Exposure to Environmental Chemicals*, Centers for Disease Control, http://www.cdc.gov/exposurereport/.

Summary: Development, Behavior & Vulnerability

- **Critical sequence**
- **Metabolic rate-dose**
- **Behavior -dose**
- **Vulnerable to disruption**
- **Size, timing, duration influence impact**
- **Susceptible throughout adolescence**
- **Latent effects into adulthood**





Pesticides & Child Health concerns

- Acute Toxicity (poisoning)
  - Toxicological/Experimental data
  - Case reports
- Chronic sequelae from acute poisoning
  - Animal models
  - Observation epidemiology
- **Chronic Toxicity - disease or disability from low level, chronic exposure**
  - Animal/Epidemiological data

Focus on epidemiological evidence

Humans are a good model for human effects: complex multifactorial disorders of childhood (ADHD, cancer, asthma)

Can represent real life situations (range of exposures, combined exposures, active ingredients plus "inerts")

Can examine subtle, subclinical effects that might not otherwise be recognized in animal models or individuals

Acute poisoning

Overall – acute poisoning in U.S. children is rare

- Improper application
- Improper storage
- Unintentional ingestion

Acute poisoning

No national surveillance/no rates available

Poison center data summaries:

Approximately 45% of all pesticide poisoning reports occurred in children

8th most common substance encountered in children < 5 years (43,526 = 3.4% of young child NPDS reports)

Bronstein 2008

Poison Control Center Data 2007

Pesticide	<6 Years	6–19 Years
Anticoagulant rodenticides	11,592	360
Pyrethroids	5468	1801
Insect repellents	6,738	1,625
Organophosphates	1,096	429
Borates/boric acid	3,447	131
Glyphosate	1,133	321
Carbamates	1,062	235
Naphthalene	1,042	106

Bronstein 2008

Poison Center data

- Rates of reported pesticide poisonings described as moderate/ major / and fatal have declined from 1995-2004 by approximately 42%.

- Sharpest declines in poisonings were from organophosphate and carbamate insecticides (reflective of policy change)

Blondell 2008

Chronic Health Implications

Most focus in recent years has been on **neurodevelopmental effects, childhood cancer**

Also, some data for **birth outcomes** including growth and gestational deficits.

Data describing other outcomes - **birth defects**, **immunological function effects**, respiratory disease including **asthma,** and **endocrine/pubertal** are more limited.

Pediatric cancer and pesticides

- Some pesticides have undergone cancer classification by EPA

  Malathion (possible), Dichlorvos (probable), permethrin (likely human)

- Substantial observational epidemiological data demonstrating a link between pesticide exposure and childhood cancers

- Challenges/limitations: Exposure assessment is generally crude, recall bias, specificity of cancer type

Pediatric leukemia and pesticides

- Most consistently associated tumor type = Acute Lymphocytic Leukemia (ALL)
- Associations with household insecticide use (lawn/garden herbicides, insecticides)
- Maternal pre-conceptional and prenatal exposures

Infante-Rivard 2007

Pediatric brain tumors and pesticides

- 2nd most commonly associated cancer - Brain tumors

- Prenatal exposure to insecticides, particularly in the household, as well as both maternal and paternal occupational exposure before conception though birth

Schuz 2001, Wijngaarden 2003, Cordier 2001, Flower 2004, McKinney 2003, Feychting 2001, Heacock 2000, Rodvall 2003, and Schreinemarchers 2000

Neurodevelopment & Pesticides

- Organochlorines, organophosphates – accumulating and consistent support for adverse impacts

  Biological plausibility and toxicological mechanisms

  Multiple epidemiological studies

- Functional deficits (mental, motor) -- symptoms and behaviors (inattention, hyperactivity, PDD) -- diagnosed conditions (ADHD)

Rosas 2008

Chronic neuropsychological sequelae post OP poisoning

- healthy school-age children
- acute OP poisoning ≤ 3 years
- subtle but significant deficits
  - ↓ restrain and control their motor behaviors
  - compared to both children who had no history of poisoning and children who had a history of early life poisoning with kerosene

Kofman 2006

OP effects on Neurodevelopment: U.S. Birth Cohort Studies

Studies	Neurodevelopmental effects (prenatal exposure)
NYC cohort Mt. Sinai	↓ head circum with low PON1 ↓ Neonatal reflexes
NYC cohort Columbia	↓ **36 month mental** ↓ **36 month motor** ↑ **5 y measures of ADHD, PDD**
Salinas Valley cohort Berkeley	↓ Neonatal reflexes ↓ 24 month mental ↑ 24 month measures of PDD ↑ 5 y measures of ADHD

Rosas 2008. Marks 2010. Rauh 2006

Odds of behavioral disorders on CBCL at age 3 years among infants with high versus low umbilical cord blood chlorpyrifos levels (n=228)

	Odds Ratio	**95% CI**
Attention Syndrome	11.26	1.79, 70.99
ADHD Problems	6.50	1.09, 38.69
Pervasive Developmental Disorder Problems	5.39	1.21, 24.11

Logistic regression controlling for race, gender, gestational age, maternal education, maternal IQ, ETS, and home environment (Home Scale)

Rauh et al., Pediatrics, 118:e1845-59, 2006

OP Pesticides and ADHD

- Cross section study using U.S. NHANES – Are children with higher concentrations of OP metabolites in urine more likely to meet diagnosis of ADHD based on structured interview?

- DMAP 10 fold increase - OR_{adj} 1.55, 95% CI 1.14-2.10
  hyperactive/impulsive subtype - OR_{adj} 2.13; 95% CI 1.08-4.20

- Strengths – Large sample size, valid case definition, biomarker of exposure, representative sample of US kids 8-15 y, some covariates (ses, lead, prem/lbw)

- Limitations – Cross-sectional, potential confounders not addressed (parental neurobehavioral status, stress, etc)

Bouchard 2010

Birth defects and pesticides

- Available studies are heterogeneous in design, conflicting in results, and they often have an insufficient exposure assessment/ecological designs
- paternal or maternal *occupational* exposures
- OC and OP insecticides, phenoxy and triazine herbicides
- cryptorchidism, orofacial clefts, limb reduction defects, and heart defects
- Bottom line, a small risk elevation is noted for birth defects and pesticide exposure, but the findings are not robust and data specific to pesticide subtypes are not adequate

Bell 2001, Arbuckle 1999, Weselek 2007, Nurminen 1995, Garry 1996/2002, Romitti 2007

Fetal growth/pre-term birth

- Several cohort studies associate maternal DDT/DDE preterm birth, IUGR, LBW
- OPs - NYC longitudinal cohorts (Rauh presentation)
- Ecological studies link triazine herbicide exposure and fetal growth

Longnecker 2001, Ribas-Fito 2002, Weisskopf MG 2005, Wolff 2007, Siddiqui 2003, Villanueva 2005

Asthma & pesticides: emerging hypothesis

Indirect evidence that pesticides skew the immune response toward the T-helper 2 (TH-2) phenotype associated with atopic disease

- maternal agricultural work (in a high OP use region) → 26% increase in proportion of TH-2 cells in their 24-month-old infants' blood samples
- % TH-2 cells was associated physician diagnosed asthma and maternal report of wheeze

Duramaud 2006

Asthma & pesticides: emerging hypothesis

- Iowa rural kids - any pesticide use indoors or any outdoor use in the previous year ≠ asthma symptoms and prevalence (Merchant 2005)

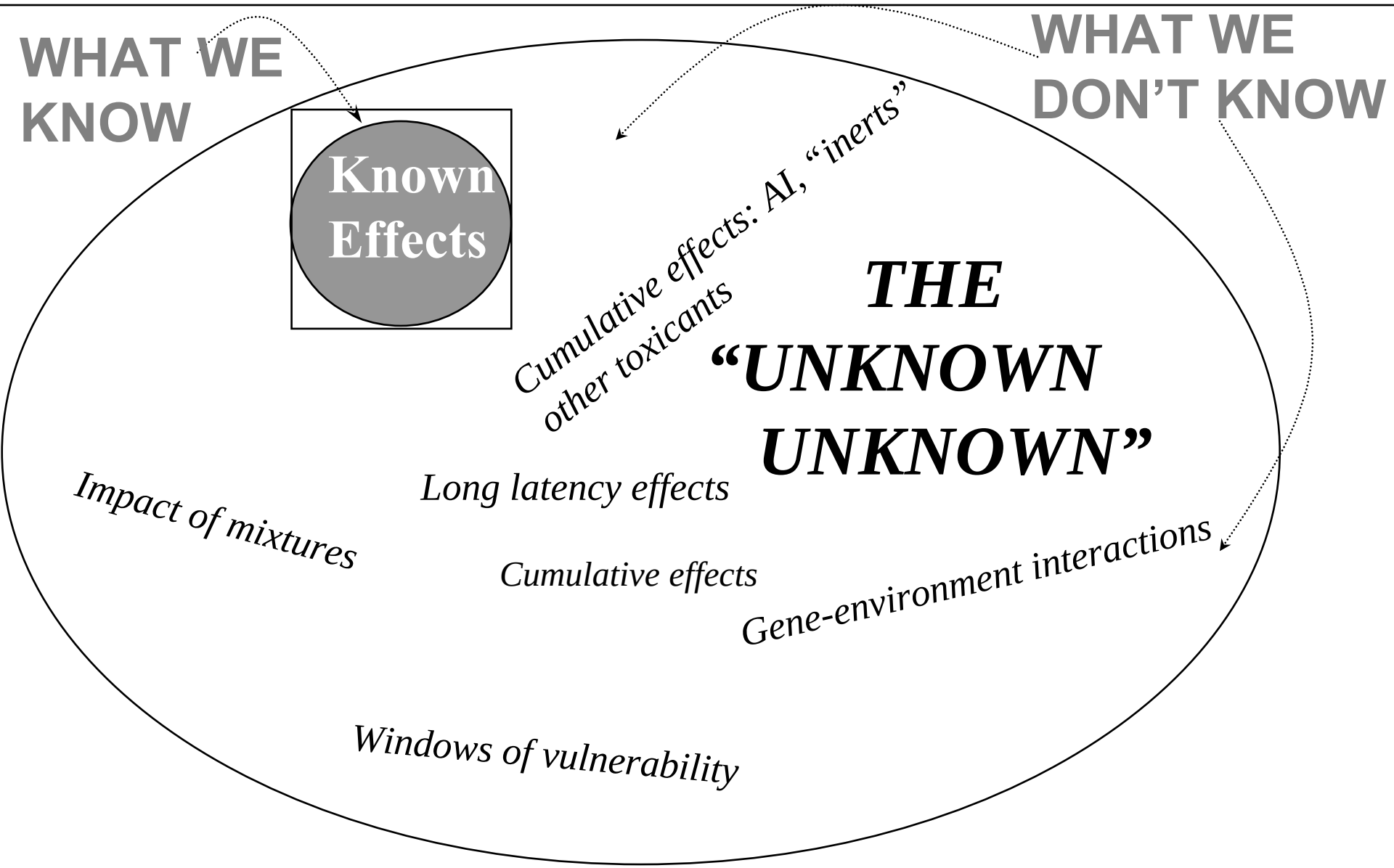
- Lebanese kids - ↑ chronic respiratory symptoms, including wheeze, with any pesticide exposure in the home, exposure related to parent's occupation, and use outside the home (Salameh 2003)

- California Children's Health Study (So.CA) - herbicides and pesticides/insecticides strong association with asthma diagnosis before age 5 years (OR 4.58, 95% C.I. 1.36-15.43 and OR 2.39, 95% C.I. 1.17-4.89, respectively) (Salam 2004)

Pesticide Child Health Studies: Key Points Summary

- Most focus on insecticides (organochlorines/organphophosphates)
  Or simply & often non-specific "pesticide exposure"
- Animal models + well designed cohort studies demonstrate OP exposures that are being experienced by U.S. children may have adverse neurodevelopmental consequences
- Prenatal and very early life exposure are of high concern
- Toxicological and epidemiological investigations of other chronic health endpoints raise concern but are less robust and better characterization is needed (cancer, birth outcomes, asthma, endocrine disruption)

Evidence base: the big picture



Research priorities

- **Exposure focus**
  - ??? chronic health implications of repeat/cumulative exposure to pyrethroid and carbamate insecticides and common herbicides encountered in urban setting
  - Human health effects from low environmental doses or at biomonitored levels are unknown
  - Characterizing exposure time windows, cumulative effects, combined effects

Research priorities

- **Health endpoints focus**

Human data are not without limitations but have provided tremendous insight – continue to support epidemiological approaches

Going beyond OP/neurodevelopment studies

Other common urban pesticides and ndv? (e.g. known neurotoxicants - pyrethroids, carbamates)

Consider influences of commonly encountered pesticides on pulmonary development/disease, immune function, endocrine effects/reproductive health

Research priorities

- More wholistic/sophisticated evaluation – consider pesticides/toxicants with similar health outcome and influence of modifiers that ↑ or ↓ impact

e.g. pyrethroids + OPs + metals + nutritional state + genetic status = ?? neurodevelopmental impact

Evidence to policy: challenges and considerations

- Extrapolating low dose human risks from high dose animal data is challenging and uncertain
- Actions to reduce one risk may substitute a different risk
- Addressing multiple exposures, mixtures/inerts, and differing subpopulation vulnerabilities
- Dealing with uncertainty- ‘Precautionary Principle’ , ‘Uncertainty Factors’?

Policies to reduce exposure and impact

- Promote IPM
- Promote clinician education – risk identification and prevention
- Establish surveillance programs
- Promote safe use/practices
  - Effective labeling
  - Public education

Thank you

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Variation in Organophosphate Pesticide Metabolites in Urine of Children Living in Agricultural Communities

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Children of migrant farmworkers are at increased risk of exposure to organophosphate pesticides because of "carry-home" transport processes and residential location. Although this at-risk status is generally recognized, few available reports describe the extent of this exposure among agricultural communities. We quantified dialkyl phosphate (DAP) levels in serial samples of urine from 176 children, 2–6 years of age, in three Oregon communities hosting differing agricultural industries: pears, cherries, and fruit berries. Up to three spot samples of urine were collected from children at the beginning, mid-point, and end of their parents' work seasons. The median levels of dimethylthiophosphate (DMTP), the most commonly detected metabolite, was significantly higher in urine samples from children in each of the three agricultural communities (17.5, 19.0, and 41.0 ng/mL) relative to a reference group of children who lived in an urban community and whose parents did not work in agriculture (6.5 ng/mL; Kruskal-Wallis, $p < 0.001$). After controlling for age, sex, and weight, the median level of DMTP in children in the pear community was 1.92 times higher than the level in children of the berry community [95% confidence interval (CI), 1.14–3.23] and 1.75 times higher than the level in children of the cherry community (95% CI, 0.95–3.23). We observed increasing levels of DMTP across the work season only within the berry community. Levels decreased in the cherry community and remained constant in the pear community. Substantial temporal variation within the children followed demonstrates the need for multiple urine samples to most accurately characterize longer term and/or cumulative exposure. The observed variability in urinary DAP levels, between communities and over time, could be attributed to the types and amounts of organophosphate pesticides used, the timing of applications and degradation of residues in the environment, work operations and hygiene practices, the proximity of housing to orchards and fields, or the movement of these working families. Additional studies of variation in pesticide exposure across agricultural regions are needed.
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Measurement of dialkyl phosphate (DAP) compounds in urine has been used to assess exposure to organophosphate pesticides (OPs) in children living in rural agricultural settings (Azaroff 1999; Curl et al. 2002; Koch et al. 2002; Loewenherz et al. 1997; Lu et al. 2000; Shalat et al. 2003) and more recently in urban communities (Curl et al. 2003; Lu et al. 2001). These biomarkers provide an integrated estimate of exposure received through ingestion, inhalation, and dermal absorption during the 24–48 hr preceding testing (Feldman and Maibach 1974; Loewenherz et al. 1997). Because the analytical method used to quantify urinary DAPs is relatively new and technically difficult (Moate et al. 1999), data on the extent of OP exposure in various types of communities are limited.

Children have been the focus of many exposure assessments because their activity patterns, behavior, and diet lead to increased risk of exposure relative to adults (Eskenazi et al. 1999). The sensitivity of developing organ systems, specifically the brain and central nervous system, and immature detoxification and elimination capacities further increase children's risk for adverse health effects (Faustman et al. 2000). In the United States, most children are probably exposed to OPs to some extent. Household surveys indicate extensive residential use of these compounds (Whitemore et al. 1994), and the potential for widespread low-level chronic exposure exists (Adgate et al. 2001). Children who live in agricultural communities are regarded to be at particularly high risk for exposure because of their proximity to fields and orchards where these chemicals are applied in high volume (Simcox et al. 1995). Additionally, children whose parents work in agriculture receive "carry-home" exposure via transport on their parents' work clothing and shoes (Lu et al. 2000). Our studies in Oregon have characterized these exposure pathways in children of migrant farmworkers and have demonstrated elevated levels of residues in their residences (McCauley et al. 2001).

In this report, we describe the occurrence of DAP compounds in urine samples collected from children of migrant farmworkers in three separate communities that host differing agricultural industries using varying types of OPs, and a reference group of children living in a urban area.

Materials and Methods

Study design. This study was conducted as a partnership between Oregon Health and Science University (OHSU) and the Oregon Child Development Coalition (OCDC), which is the grantee for Oregon Migrant Head Start. A cross-sectional design was employed to collect serial samples of urine from preschoolers attending Head Start programs at three centers operated by OCDC in the communities of Hood River, The Dalles, and Cornelius. For comparison purposes, a reference sample of preschool-age Hispanic children who lived in an urban area, Portland, and whose parents did not work in agriculture was also assembled. Urine was collected from the agricultural communities during June–September 2001, at the beginning, mid-point, and end of each work season. The timing of the sampling varied in each community depending on the time that the farmworkers began to arrive in the community, enrolled their children in Head Start programs, and started work harvesting crops. In the reference community group, samples of urine were collected during July and November 2001.

Survey sites. The communities selected for study are geographically separate and differ in the type of agricultural industry. Hood River primarily produces pears but also produces apples. Hood River is located along the

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Columbia Gorge, approximately 100 km east of Portland, Oregon. The farmworkers in this community tend to be semipermanent residents who live in cabins, trailers, or apartments that are located in or alongside orchards. Azinphos-methyl [trade name Guthion; Chemical Abstract Service (CAS) No. 86-50-0], chlorpyrifos (CAS No. 2921-88-2), and phosmet (trade name Imidan; CAS No. 732-11-6) are used to control pests such as coddling moth and are applied May–August. Harvesting of tree fruit begins in August and extends through October. Urine samples were collected from the children at the Hood River Migrant Head Start day care centers in Parkdale and Odell in June, August, and October.

The second survey site was The Dalles, where cherries are grown. The Dalles is located along the Columbia Gorge, 30 km east of the Hood River community. Cherry harvest lasts only 1 month, usually beginning in mid-June. The cherries are hand harvested by migrant farmworkers, who live with their families in camps, cars, trailers, and tents. Chlorpyrifos can be applied to the cherry crop at prebloom in March by air blast method from the ground, whereas ultra-low-volume concentrate malathion (CAS No. 121-75-5) is applied aerially in late May and every 7–10 days throughout harvest. Urine samples were collected at the Dalles Migrant Head Start Center in June and July.

The third agricultural community, Cornelius, has both rural and suburban features and is situated in the northwest Willamette Valley. The farmworker population consists of settled farmworkers who work in the nursery, vineyard, and berry industries and a highly mobile migrant population of farmworkers who harvest the strawberry, caneberry, and blueberry crops during the summer months. The migrant population of farmworkers receive priority enrollment in Migrant Head Start and was targeted for this study. These families tend to live in camps composed of wood cabins and trailers that are adjacent to berry fields. Strawberry, caneberry, and blueberry growers apply diazinon (CAS No. 333-41-5), azinphos methyl, chlorpyrifos, and malathion to control pests. Urine samples were collected at the Cornelius Migrant Head Start Center the months of June, July, and August.

Reference subjects were recruited through Head Start and school-based programs in the Portland metropolitan area. Hispanic families were screened and invited to participate only if the parents reported that they did not work in agriculture, nursery, or landscaping businesses. Typical jobs held by families in the reference group include food service, factory work, and child care. These families lived in apartments in an urban setting.

Recruitment and data collection. Eligibility for participation was restricted to preschool-age children, 2–6 years of age, who were able to urinate while sitting on a toilet. Signed informed consent was obtained from parents as they registered their child for the Migrant Head Start Program. All participants except children from the reference population had at least one parent working in the field, orchard, or nursery while participating in the study. Demographic information was extracted from Head Start records, which were completed at time of registration. The extracted information included the child's name, sex, age, date of birth, type of residence, previous and current place of employment, and crop worked by the parents. Reference subjects were selected from summer Head Start programs at three sites in northeast and north Portland, and Gresham (a suburban community east of Portland). When reference subjects were recruited and informed consent from the mother was obtained, each parent was asked questions to confirm that their child was between the ages of 2 and 6 years, and that neither the parents nor other adult household members worked in agriculture. At the time the urine sample was collected, participants were weighed.

Human subjects review. The study protocol and procedures for informed consent were reviewed and approved by the OHSU institutional review board (protocol 4216).

Urine collection. Urine samples were collected from each participant while the child attended Migrant Head Start. Research assistants collected the single void urine samples mid-morning through early afternoon using commode inserts. The urine was transferred into a urine specimen cup, labeled, and transferred on ice to the Center for Research on Occupational and Environmental Technology analytical laboratory.

Urinalysis. Urine specimens were adjusted to pH 3.0 and stored at –20°C until extraction and analysis. Five DAPs were analyzed by gas chromatography (GC): dimethylphosphate (DMP), diethylphosphate (DEP), dimethylthiophosphate (DMTP), diethylthiophosphate (DETP), and dimethyldithiophosphate (DMDTP). Urine samples were prepared for GC analysis according to a modified method of Moate et al. (1999). Aliquots of the samples underwent azeotropic distillation with methanol and evaporation under a nitrogen stream. Sample extracts were then derivatized with 2,3,4,5,6-pentafluorobenzylbromide to convert phosphate acids to esters. Extracted samples were analyzed on a gas chromatograph (model 5890; Hewlett-Packard, Palo Alto, CA) equipped with a pulsed flame photometric detector (OI Analytical, College Station, TX). The limit of detection (LOD) for each of the metabolites was calculated from the instrument response factor corresponding to a concentration having a peak area three times the baseline noise (blank signal). The specific LODs for the five metabolites were 4.0 ng/mL for DMP, 2.0 ng/mL for DEP, 2.2 ng/mL for DMTP, 1.6 ng/mL for DMDTP, and 1.6 ng/mL for DETP. The average extraction efficiencies of the five metabolites were, respectively, 87, 84, 97, 96, and 93%. Creatinine concentrations (micrograms per deciliter) were determined by the modified Jaffe rate method (Sigma Diagnostics Creatinine Kit no. 555; Sigma-Aldrich, St. Louis, MO).

Quality control/quality assurance. Quality control data generated for each set of urine samples were used to provide an overall assessment of precision, accuracy and overall reliability of the method. Spike sample recoveries and urine blank analysis were conducted for every set of 12 samples. Urine samples known to contain low levels of DAP were used for blanks and for spike recoveries. Urine samples were spiked with DAP reference standards varying in concentration from 2 to 50 ng/mL.

LOD is defined as the lowest concentration that can be determined to be statistically different from a blank. However, in practice, the detection of an analyte by an instrument is often based on the extent to which the analyte signal exceeds peak to peak noise (U.S. Environmental Protection Agency 2000). The LOD for each set of metabolites was calculated from the instrument response factor corresponding to a concentration having a peak area three times the baseline noise (signal) corresponding to the urine blank sample.

Data analysis. To obtain stable measurements of urinary metabolites, we averaged the concentrations measured in the second and third samples, corresponding to the mid-point and end of the work season. The first sample, collected at the time the child was being registered in Head Start, was not included because it could represent exposures that had occurred before the parents began working in agriculture in the area. The second sample was obtained at least 2 weeks into the work, reflecting exposures while the parents were working in the area, and the third sample, taken at the end of the work season, also reflected exposures occurring while their parents were working in the area.

We evaluated the distribution of creatinine levels and excluded urine samples less than the 5th percentile (14 mg/dL) and greater than the 95th percentile (110 mg/dL) from the analysis because of concerns of hydration state and metabolic disorders (Loewenherz et al. 1997; Lu et al. 2001). Tampering by dilution is unlikely given the young age of the subjects and the supervision of urine collection by our field staff.

During the course of the survey, urine samples were collected from a total of 214 subjects. Nineteen subjects were excluded from the

statistical analysis because second or third samples were not obtained. Data for an additional 19 subjects were excluded because creatinine levels fell outside the range of 14–110 mg/dL. Statistical analyses were performed on a data set consisting of 176 subjects.

DMTP was the most commonly quantified DAP. The percentage of samples below the LOD was 20.5%. DMP and DMDTP were below the LOD in 33.0 and 58.8% of creatinine valid samples, respectively. For comparison, the ethyl compounds, DEP and DETP, were below LOD in 91.2 and 68.2% of samples, respectively. Because ethyl metabolites were detected with much less frequency, statistical analysis was restricted to the methyl metabolites. The infrequent detection of DEP and DETP in our survey is consistent with the experience of researchers at the University of Washington (Curl et al. 2002; Fenske et al. 2000; Lu et al. 2001). In our case, inclusion of the large proportion of unquantifiable observations would needlessly complicate analyses and not produce informative results.

Standardization of DAP metabolites to creatinine (nanomoles per gram creatinine) was performed in the statistical analysis. However, because this adjustment did not alter findings in any substantive way, we chose to present our findings in units unadjusted for creatinine (nanograms per milliliter and micromoles per liter).

We summed the molar equivalent concentrations of the methyl metabolites to create a combined methyl DAP measure. Nondetects were treated as zeroes in the summation of molar concentrations, and if the total methyl DAP value was zero, then it was assigned the value of 0.5 LOD for DMDTP (0.5×0.010 μmol/L), the methyl DAP with the lowest LOD.

Statistical methods. Demographic data were summarized using means ± SDs. Metabolite levels below the LOD were treated as 0.5 LOD for the purpose of statistical analysis and were summarized using the geometric mean, geometric SD, and percentiles. The geometric mean is a descriptive statistic suitable only for summarizing the data; however, it is an estimator for the population median. Hypothesis tests or confidence intervals based on the geometric mean make inference to the population median, and subsequent wording indicates the median is the parameter being tested/estimated. We used Kruskal-Wallis one-way rank analysis of variance to test for differences in the median concentrations of metabolites between the communities. We used separate Wilcoxon tests, with *p*-values adjusted by the method of Benjamini and Hochberg (1995), to determine which pairs of medians significantly differed at the 0.05 level. Within the three agricultural communities, potential confounding of the location effect by age, sex, and weight was controlled for using a general linear model (GLM) applied to the log-transformed data. After back-transformation, adjusted means from this model estimate the population median and additive changes become multiplicative effects (Aitkin et al. 1994; Ramsey and Schafer 2002). We used extra-sum-of-squares *F*-tests (Netter et al. 1989) to test significance of multiple effects within the GLM.

To analyze variation in metabolite levels within subjects and communities over time, DMTP concentrations were log-transformed, and paired *t*-tests were applied to assess differences between sample collection times: time 1 versus time 2 (beginning vs. middle of work season), time 2 versus time 3 (middle vs. end of work season), and time 1 versus time 3 (beginning vs. end of work season). We used Pitman's test for correlated variances (Pitman 1939; Snedecor and Cochran 1980) to determine whether the amount of dispersion changed between two points in time. All analyses were performed using R version 1.9 (R Development Core Team 2004).

Results

Table 1 presents the characteristics of the samples obtained in each community. The distribution of males and females did not vary across the four communities. Overall, 52.3% of the sample was female. There was indication that the weight of children differed significantly from one community to the next ($F_{3,210} = 6.25$, $p < 0.01$). Children from the pear community weighed significantly less than did children from either the cherry or reference community [95% confidence interval (CI), 3.6–10.4 lb less]. The mean age of children was not compared across communities because, by design, we sampled only children between 4 and 6 years of age.

In our comparisons of urinary metabolites, we averaged the concentrations measured in the second and third samples for each individual collected during the mid-point and end of their parents work season. The median concentrations of urinary DMTP and the combined methyl DAP by weight-volume concentrations units were significantly higher in the agricultural communities versus the urban reference community [Kruskal-Wallis test: χ^2 (3 df) = 27.29 and χ^2 (3 df) = 25.237, respectively; $p < 0.001$ for both) (Table 2)]. The median concentrations of DMTP and combined methyl DAPs were significantly higher in children from the pear community, relative to those observed in either the cherry or the strawberry communities (adjusted *p*-value from Wilcoxon test = 0.008 for both). Median values in samples from the cherry and strawberry communities were not significantly different (adjusted *p*-value from Wilcoxon test = 0.786).

We used a GLM applied to log-transformed data to compare urinary DMTP and the combined methyl DAP levels across the agricultural communities. The analysis adjusted for age, sex,

Table 1. Age, sex, and weight of children from three agricultural communities and reference urban community in Oregon.

	Community				
Parameter	Reference (*n* = 65)	Berries (*n* = 63)	Cherries (*n* = 38)	Pears (*n* = 48)	Significance[a]
No. of samples/child					
Mean ± SD	1.2 ± 0.4	2.9 ± 0.4	2.8 ± 0.5	2.6 ± 0.7	—
Min, max	1, 2	1, 3	1, 3	1, 3	
Age [years (mean ± SD)]	NA[b]	4.2 ± 1.0	4.3 ± 1.1	4.2 ± 1.0	—
Sex (% male)	51	44	50	46	$\chi^2(3) = 0.66$ $p = 0.88$
Weight [lb (mean ± SD)]	42.1 ± 9.6	39.2 ± 10.2	42.6 ± 11.2	35.9 ± 7.5	$F_{3,210} = 6.25$ $p < 0.01$

Abbreviations: max, maximum; min, minimum; NA, not applicable.
[a]Number of samples per child and age were controlled by design and not tested. [b]Date of birth not collected.

Table 2. Comparison of child urinary DMTP (ng/mL) and combined methyl[a] (μmol/L) DAP metabolites[b] among three agricultural communities and referent community.

	Reference (*n* = 61)		Berries (*n* = 52)		Cherries (*n* = 29)		Pears (*n* = 33)	
Summary statistics	DMTP	Combined methyl[b]	DMTP	Combined methyl	DMTP	Combined methyl	DMTP	Combined methyl
Geometric mean[c]	7.25	0.12	18.81	0.25	20.24	0.26	38.54	0.40
Geometric SD[d]	5.33	3.80	3.43	2.54	2.51	1.93	3.07	2.34
10th percentile	1.10	0.01	4.48	0.09	5.60	0.12	7.68	0.11
25th percentile	1.10	0.06	7.97	0.12	9.40	0.15	27.00	0.27
50th percentile	6.50	0.15	17.50	0.23	19.00	0.25	41.00	0.44
75th percentile	30.00	0.30	40.50	0.46	37.00	0.39	87.00	0.77
90th percentile	61.00	0.50	99.90	0.90	63.00	0.53	126.00	1.00

[a]"Combined methyl" is the summed molar equivalent concentration of DMP, DMTP, and DMDTP (μmol/L). [b]Concentrations are the average of the mid- and the end-of-season samples (ng/mL). [c]Back-transformed mean of log-transformed data (an estimate of the median). [d]Back-transformed SD of log-transformed data.

and weight. Means from this analysis (derived from β-coefficients) estimated the median effect in the population after they had been back-transformed. CIs for the effects were derived from CIs for specific β-coefficients and then back-transformed. The results indicated that the differences in the median level of DMTP in children of agricultural communities remained significant after controlling for age, sex, and weight (extra-sum-of-squares F-test, $F_{2,108} = 8.270$, $p = 0.04$). The median level in the pear community was 1.92 times higher than the level in the berry community (95% CI, 1.14–3.23). Median levels were 1.75 times higher in the pear community than in the cherry community (95% CI, 0.95–3.23), but this was only a suggestive difference ($p = 0.075$). The difference between the berry community and the cherry community was not significant when accounting for age, sex, and weight ($p = 0.719$). Controlling for age, sex, and weight reduced the statistical significance of the community effect for the combined methyl DAP data within the three agricultural communities (extra-sum-of-squares F-test, $F_{2,108} = 2.165$, $p = 0.120$).

Table 3 presents the geometric means and SDs for DMTP measurements by community and sample time. CIs for the effect were found by first calculating a confidence interval for the mean difference of the log-transformed data and then back-transforming the end points with corresponding inference made to median levels. In the berry community, concentrations tended to increase from beginning to end of the work season (Table 3). The median was estimated to be 2.18 times higher than the median at the beginning of the work season (95% CI, 1.26–4.08 times higher). In the cherry community, concentrations of DMTP sharply decreased from the beginning of the work season to the midpoint, 10 days later. At the end of the work season, three weeks after the initial sample, urinary DMTP began to increase. In the pear community, concentrations of DMTP did not change in an appreciable way from the beginning to end of the work season.

Discussion

Our analysis of urinary biomarkers adds to a growing body of literature that suggests children of agricultural workers experience more exposure to OPs than do children who live in urban areas (Lu et al. 2000). The median level of DMTP, the most commonly detected metabolite, was significantly higher in the children of migrant farmworkers relative to urban Hispanic children whose parents did not work in agriculture. More important, however, is our demonstration of substantial variation in OP metabolite levels across communities hosting differing agricultural industries, and substantial variation within a community over time. This variation was demonstrated among communities within the same geographic region, and within relatively short work seasons lasting weeks to months, conditions that could be expected to foster homogeneity.

Median levels of DMTP differed significantly among communities, despite substantial interchild variability and overlapping distributions. Although the precise reasons for the observed differences between locations cannot be inferred from our data, we attribute the variation to differences in the types of pesticides used on the fruits and berries, and the timing of application and opportunity for environmental degradation before contact with workers at the time of harvest. For example, OPs are applied to pears as needed to control infestations until the time of harvest, whereas OPs are applied very early in the development of cherry fruit and not again, or infrequently, with months of time for breakdown of residues in sunlight and moisture. It is also possible that the greater extent of contact with foliage associated with picking pears (vs. cherries) may create greater opportunity for transfer of foliar residues to the clothing and skin of adults, who may carry these residues to their children. Finally, differences in type of housing and proximity of residence to orchards and fields may also explain the observed differences. The pear community is located in a valley, and air blast spraying and drift transport occurs near the homes of migrant farmworkers, possibly increasing the opportunity for exposure. Proximity of housing to application areas has been demonstrated to influence carpet residue levels in the homes of workers in Washington (Lu et al. 2000) and Oregon (McCauley et al. 2001). Furthermore, we have observed a larger number of detectable OPs in carpet dust of homes in the pear community (Hood River) compared with homes in the berry community (Cornelius, Washington County) (McCauley et al. 2001).

Koch et al. (2002) reported a temporal pattern of pesticide exposures in children living in an agricultural community over an entire year and the impact of agricultural spraying on exposure. The children studied in the Koch et al. (2002) report were enrolled via Women, Infants, and Children clinic populations in central Washington State. There are important differences in the design of these two studies. We were unable to study children over an entire year because, by the nature of their parent's migratory work, they move on to other agricultural regions or return to their native country for a portion of the year. We were unable to assess the impact of spraying pesticides on urinary OP levels, because at the time the parents are harvesting the crops, the active spraying season is over. Instead, the goal of our study was to point to differences between the OP metabolite levels in children according to the type of harvesting work their parents were engaged in and to compare agricultural children to urban children.

The levels of urinary metabolites observed in the children of our pear community are similar to those reported for children of apple orchard workers living in central Washington State and extensively characterized by Fenske and colleagues (Loewenherz et al. 1997). The same OPs (e.g., azinphos methyl, phosmet) are applied to both crops to control coddling moth, using similar air blast spray systems, in similar settings of cultivation. In fact, apples are cultivated adjacent to pear orchards at our study site.

For reference, we collected urine samples from Hispanic children attending summer Head Start programs in the Portland, Oregon, metropolitan area. The geometric mean level of DMTP in this control group was 7.2 ng/mL. This level is slightly higher than the geometric mean of 2.7 ng/mL (95% CI, 1.85–4.01) reported for 471 children, 6–11 years of age, sampled in the 1999–2000 National Health and Nutrition Examination Survey [Centers for Disease Control and Prevention (CDC) 2003]. The difference may be attributed to age and differing hand-to-mouth behavior and floor contact in our younger children. Our combined methyl DAP median concentration was 0.15 µmol/L, very similar to the 0.11 µmol/L median reported for Seattle children 2–5 years of age (Lu et al. 2001). Presumably, the urinary metabolites observed in studies of urban

Table 3. The amount and variation of DMTP levels from children in agricultural communities across three time points in a harvest season.

	Geometric mean (geometric SD)			*p*-Values for comparison of DMTP levels[a]			*p*-Values for comparison of variation[b]		
Community	T1	T2	T3	T1 vs. T2	T1 vs. T3	T2 vs. T3	T1 vs. T2	T1 vs. T3	T2 vs. T3
Berries ($n = 50$)	7.2 (5.0)	9.6 (5.5)	15.7 (4.3)	0.34	0.02	0.10	0.67	0.54	0.30
Cherries ($n = 29$)	43.4 (2.6)	14.0 (2.5)	18.3 (4.0)	< 0.01	0.01	0.33	0.90	0.06	0.04
Pears ($n = 31$)	22.4 (3.4)	22.5 (5.3)	22.8 (5.4)	0.99	0.95	0.97	0.10	0.08	0.95

Abbreviations: T1, time 1; T2, time 2; T3, time 3. The amount and variation are summarized in terms of the geometric mean and geometric SD; *p*-values show whether the changes are significant.
[a]Based on paired *t*-test of log-transformed data. [b]Based on Pitman's test for correlated variances (performed on log-transformed data).

children derive from dietary exposures to residues, and exposures associated with residential and public (schools, parks) applications.

Our serial sampling design provided an opportunity to consider temporal variation in urinary metabolite levels. Given the short half-life of 24–48 hr for DAPs, we expected to observe variation, specifically increased excretion of metabolites, as parents began work and started to transfer residues to the home environment. Further, we expected increasing concentrations as body burdens increased and new doses were superimposed on previous doses that were being metabolized and eliminated from the children's bodies. We observed increasing levels of DMTP across the work season only in the berry community. Levels decreased at the cherry community and remained constant in the pear community. We attribute this pattern to differences in the migrant farmworker labor forces. In the berry community, migrant workers who arrived to work for the short harvest season apparently had low body burdens and low exposures to OPs in the period immediately before arriving. In the cherry community, most migrant farmworker families arrived directly from agricultural work in California and/or from the nearby pear community, and sufficient time had not yet passed for the OP metabolites to wash out of their bodies. In the pear community, the workers are settled and maintain their migrant status and Head Start benefits by returning to Mexico once per year in the winter. These workers and their children live for extended periods in the valley, in close proximity to the orchards and associated pesticides; therefore, their body burdens may reflect steady state, rather than new and accumulating doses. Although the differences that we observed could be attributed to differences in the work patterns and total exposure to OPs, it is possible that the different pharmacokinetics of specific pesticides used in these communities and their half-lives could have contributed to the observed difference.

Our findings are subject to several limitations. First, the DAP metabolites measured in this study represent a partial view of the total mix of pesticide exposures received by children. Exposures to other classes of pesticides and herbicides certainly occur, and the total exposure to all classes of agricultural chemicals is not quantified by our methods. Further, the sources and routes of exposure to OP compounds cannot be identified by measurement of urine DAPs, which provide an integrated indicator of exposure to a variety of OP compounds via ingestion, inhalation, and dermal exposure.

Despite these limitations, our survey is based on homogeneous samples of children who by virtue of their eligibility for enrollment in Migrant Head Start are of the same preschool age, share Hispanic ethnicity and come from the same socioeconomic class, and have at least one parent who works in agriculture. We collected measurements on age, sex, and weight and analyzed creatinine levels to control for physiologic variation. Appreciating the relatively short half-life of these metabolites, we collected serial samples from the children and used the average of two samples collected at the middle and end of the work season to investigate between community differences and to improve the characterization of longer-term exposure. We did not use the first urine sample, collected at the time of Migrant Head Start enrollment, because of concerns that this urine sample represented the exposure of the child before the family's move to the new work location and Head Start center. Although some methyl DAPs probably come from exposure to OP residues on foods (Curl et al. 2003), this class of biomarker has proven to be a valid and reliable measure of exposure via other pathways, including hand-to-mouth transfer, dermal absorption, and inhalation, and the observed pattern of variation between communities is consistent with differing pesticide application practices by the agricultural industries in these areas. Although it was not practically possible to measure exposure to the full suite of chemicals to which these children are exposed, OPs as a class are among the most toxic chemicals in use by the agriculture industry and, as a class, present significant health risks.

In conclusion, our findings indicate that there is substantial variation in level of exposure to OPs among the children of migrant farmworkers living in different communities. This diversity in exposure experience must be considered in exposure assessments and health risk analyses. Failure to characterize potential differences between communities may introduce exposure misclassification into epidemiologic studies. Further, our observation of substantial temporal variation within a child supports the need for multiple urine samples to accurately characterize longer term and/or cumulative exposure.

Correction

In the original manuscript published online, Michael Lasarev and William E. Lambert were listed as the first and second author, respectively. The order has been reversed here.

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Healthier Homes and Gardens
January 2008

Childhood Leukemia & Lymphoma And Pesticide Exposure

Two common childhood cancers have been linked to prenatal exposure to household pesticides according to a December 2007 study.

Using data from a large survey about childhood cancers, French scientists examined the relationship between parents' use of household pesticides and the occurrence of acute leukemia, non-Hodgkin lymphoma and Hodgkin lymphoma in their child. They found that the use of any pesticide by the mother during pregnancy was associated with an increased likelihood of acute leukemia and non-Hodgkin lymphoma in the child.

To focus on the effect of "in utero" pesticide exposure, researchers examined information about the mother's pesticide use during the pregnancy. They also looked at use of pesticides by the father during the combined period of the pregnancy and the child's early years. The pesticides were differentiated as: "weed killers" (herbicides); fungicides; or insecticides used in the home, on pets, or on garden crops.

Analysis by the type of pesticide used by mothers during pregnancy showed that acute leukemia was linked to use of home and pet insecticides, and to a lesser degree, herbicides. Using insecticides on garden crops was linked to non-Hodgkin lymphoma.

In the initial analysis, the father's use of insecticides during the pregnancy and early years was associated with acute leukemia. However, when researchers looked at the combined effects of maternal and paternal use, the risk for acute leukemia appeared to be attributable to the mother's pesticide use. Paternal use of both insecticides and herbicides was linked to non-Hodgkin lymphoma, but researchers were not able to draw conclusions about the overall strength of this effect.

Researchers also found that certain subtypes of leukemia and lymphoma were strongly associated with either maternal pesticides or paternal pesticide use.

The authors pointed out that other studies have also linked childhood leukemia and lymphoma to children's exposure to pesticides. Among theses studies, prenatal pesticide exposure appears to carry slightly more risk than childhood exposure. Given this information they concluded that perhaps pregnant women should not use pesticides.

SUMMARY: Keep the Sprays Away? Home Pesticides Linked to Childhood Cancers.
Tina Ader. *Environmental Health Perspectives* 115(12):A594
http://www.ehponline.org/docs/2007/115-12/ss.html#keep

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Biological Impacts of EMFs

Link: http://www.dirtyelectricity.net/dirty-electricity-biological-impacts/

Electromagnetic fields (EMFs) can have a variety of biological impacts. Impacts like these and others may help explain the connections between dirty electricity and a range of diseases and other ailments. EMFs (of various frequencies):

- Interfere with normal "electrical" communication between cells (This can disrupt cellular function in every part of the body and interfere with the body's natural processes, such as sleep, hormone production, neurological function, immune response, and the ability to heal.)

- Disrupt Melatonin production (Melatonin is a neuro-hormone that is vital for healthy sleep. It is also a potent, natural antioxidant that protects cells from genetic damage that can lead to cancer as well as neurological, cardiac, and reproductive damage.)

- Linked to chronic inflammation in the body (Inflammation has been linked with cellular/tissue/organ damage, cancer, heart disease, autoimmune diseases, diabetes, neurological diseases, and Alzheimer's.

- Trigger the immune system to release inflammatory substances such as histamines and cytokines as a protective mechanism (These markers of inflammation have been shown to highly correlate with asthma and other allergic reactions.)

- Overstimulate the immune system, then suppress it and decrease T-lymphocyte production. (T-lymphocytes orchestrate the immune system's response to infected or malignant cells.)

- Raise cortisol (stress hormone) in the body (This can lead to sleep disorders, depressed immunity, cardiovascular disease, autoimmune disorders, premature aging, neurological problems, and more.)

- Cause cell membranes to be locked in an inactive state (oxidative stress) that prevents toxins, or free radicals, from leaving cells. (There is evidence that this inactive state can damage DNA and prevent the body from repairing it, which is a first step to cancer.)

- Cause calcium ion efflux. (Calcium ion alteration of cells by electromagnetic radiation is linked to neurological degeneration, to cancer, to dangerous heart arhythmias, and many other health effects.)

- Can elevate blood sugar levels and blood viscosity, which can be associated with symptoms such as headaches, chest pain, higher blood pressure, blurred vision, and fatigue among others.

The Dark Side of Electronics: Are Kids suffering from Dirty Electricity?

Posted on April 6, 2012

Link: http://www.dirtyelectricity.net/380/

Kids are increasingly experiencing ADHD, asthma, diabetes, learning disabilities, headaches, sleep disturbances and mood. Why? Are these health issues being worsened by the explosion of electronics in our homes and schools?

Seminal research by Dr. Magda Havas, PhD and Dr. Sam Milham suggests that "dirty electricity" and other electrical energy may be disproportionally affecting children. Dr. Olle Johansson, PhD believes exposures to electrical energy, such as dirty electricity, aggravate children's immune systems, which may cause, or worsen illnesses and afflictions like those mentioned above.

Dirty electricity refers to unusable and potentially harmful electromagnetic energy that is generated by electronic devices as they operate. Once generated, this energy circulates throughout a building's electrical system and escapes into the immediate environment through wiring, outlets, power strips, and electronic devices themselves, contaminating the environment with electro-pollution. In turn, this invisible pollution may be silently compromising our children's health and well-being.

In a series of studies in schools in Minnesota, Wisconsin and Canada, after filtering dirty electricity, Dr. Havas demonstrated an improvement in the learning environment for children and a reduction in symptoms from ADHD, Asthma and headaches. Teachers in the same schools experienced an improvement in sleep, joint/ muscle pain and mood. Some adults with Chronic Fatigue (CFS) and Multiple Sclerosis (MS) experienced better health. In a related study, diabetics experienced lower glucose levels when dirty electricity was filtered.

Many devices generate dirty electricity, including energy efficient lighting (CFL/fluorescent bulbs), dimmer switches, computers, printers, solar power, plasma televisions, stereo equipment, video games systems, cordless (DECT) phones, Wi-Fi, microwave ovens and other kitchen appliances, washers/dryers, variable speed fans, and battery/device chargers.

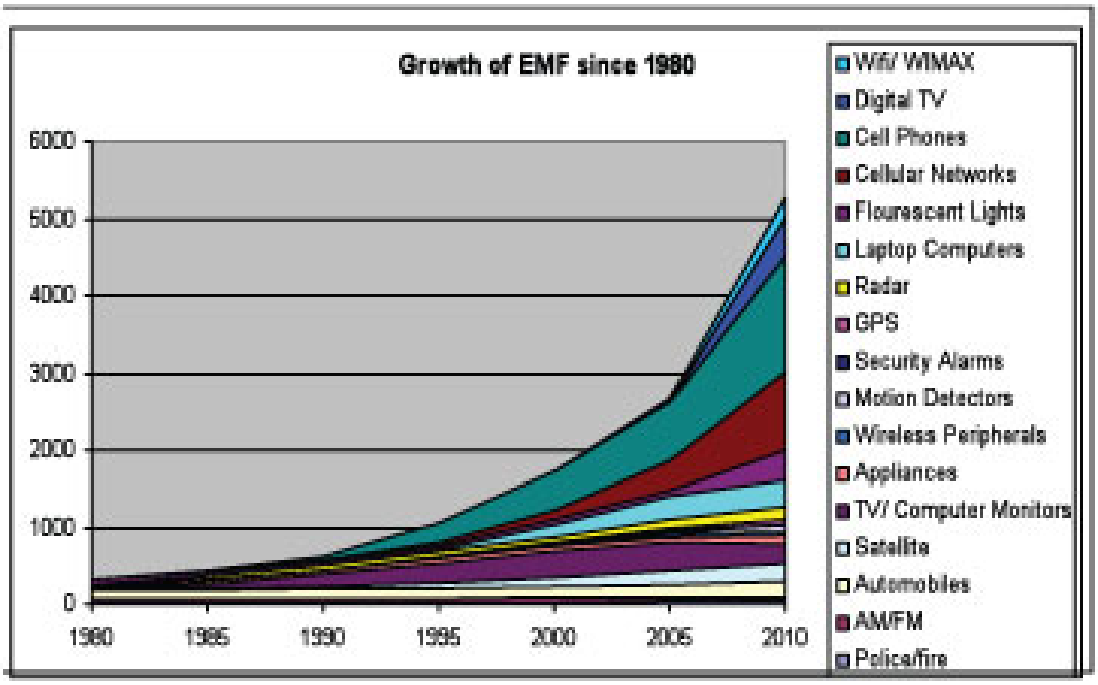
The increase in electronics, solar power systems and fluorescent lighting in schools may be putting children in harm's way. Children are becoming increasingly exposed to these devices both at home and in the school and their smaller size and developing immune systems potentially make them more vulnerable than adults.

One way to reduce the harmful effects of dirty electricity is to unplug all the devices and lights causing the potentially unhealthy electrical energy. Unfortunately, this isn't always possible. A more practical way to reduce your exposures to dirty electricity is to plug-in filters from Greenwave™ www.greenwavefilters.com or Stetzer Electric. A typical three bedroom house usually requires around 15 filters. Most classrooms can be successfully filtered with three to five filters. Both products allow you to keep your devices plugged in and still lower your exposures to this fast growing form of pollution.

Tags: add/adhd, Dirty Electricity, disabilities, electronics, learning, packages, school

The Dirty Electricity Story

Link: http://www.dirtyelectricity.net/dirty-electricity/



The sheer volume of modern electronics and appliances in homes and other settings has increased at a dizzying rate. While, this proliferation of electrical devices has made our lives more efficient and convenient, it has also contributed to a potentially harmful form of electro-pollution known as DIRTY ELECTRICITY.

Dirty electricity is unusable electromagnetic energy that is created by many electrical devices as they operate. It is caused by interruptions in the flow of normal 60-Hertz AC (alternating current) power traveling through wires and electrical systems in homes and other buildings. These interruptions result in voltage spikes, or surges, as well as frequency variations (also called high frequency voltage transients) that combine to form a complex and potentially harmful electromagnetic field.

How do electronic devices contribute to dirty electricity?

Many modern electronics and appliances include transformers that convert the AC power in a building's wiring to the DC power needed to run electronics and appliances. During this conversion process, interruptions in electrical current flow occur. In addition, many modern electronic devices (e.g., light dimmer switches, compact fluorescent light bulbs, equipment that use switching mode power supplies) utilize power in a more complicated way than more "old-fashioned" electronics and equipment. These devices are actually designed to operate with interrupted electric current flow. Rather than draw power continuously, they do so intermittently in variable amounts at a high frequency, primarily for efficiency. While this can save energy, it involves frequent interruptions in electric current flow. For example, a compact fluorescent light bulb saves energy by turning itself on and off repeatedly, thousands of times per second. Regular interruptions like these create transients (i.e., voltage spikes/surges and frequency variations) that "dirty" the normal electricity flowing along wires.

What happens to dirty electricity once it is generated? The dirty electricity created by electrical devices within a building is circulated throughout the building, and even to other buildings in the neighborhood, via wiring. It radiates into the immediate environment via outlets, power strips, electronic devices, and cords/wires, exposing the humans inside to electromagnetic pollution.

Your Mattress Could be Acting as a Cancer-Causing Radiation Antenna

Posted by: Dr. Mercola | August 18 2010 | 157,378 views

The rate of breast cancer in Western countries is 10 percent higher in the left breast than in the right. This also is true for the skin cancer melanoma.

Researchers have suggested a surprising explanation for this -- and for the dramatic increase in rates of breast cancer and melanoma over the past three decades.

In Japan, there is no correlation between the rates of melanoma and breast cancer, and there is no left-side prevalence for either disease. The rate of breast cancer in Japan is also significantly lower than in the West.

This may be due to differences in sleeping habits in Japan and Western countries. Previous research has shown that people prefer to sleep on their right sides, possibly as a way of reducing weight stress on the heart.

This is most likely the same in both the East and the West, but the futons used for sleeping in Japan are mattresses placed directly on the bedroom floor, in contrast to the elevated box springs and mattress of beds used in the West.

According to *Scientific American*:

"... [A] 2007 study in Sweden conducted between 1989 and 1993 ... revealed a strong link between the incidence of melanoma and the number of FM and TV transmission towers covering the area where the individuals lived ...

Consider, however, that even a TV set cannot respond to broadcast transmissions unless the weak electromagnetic waves are captured and amplified by an appropriately designed antenna. Antennas are simply metal objects of appropriate length sized to match the wavelength of a specific frequency of electromagnetic radiation."

In the U.S., bed frames and box springs are made of metal, and the length of a bed is exactly half the wavelength of FM and TV transmissions. The maximum strength of the field develops 75 centimeters above the mattress, so when sleeping on your right side, your left side will be exposed to the highest field strength.

Sources:

- Scientific American July 2, 2010
- Pathophysiology June 2010;17(3):157-160
- Pathophysiology June 2010; 17(3):161

Dr. Mercola's Comments:

This *Scientific American* article highlights some very interesting research from Sweden -- a country that is on the forefront when it comes to investigating the harmful health effects of electromagnetic fields (EMF), and micro wave radiation such as that from cell phones.

Although the US is quite resistant to the idea that our everyday technology might be contributing to epidemics like heart disease and cancer, other countries, particularly in Europe, are facing this problem more head on.

Sweden, for example, formed an association called FEB - The Swedish Association for the ElectroSensitive, to address the emerging problem of electromagnetic hypersensitivity. The association produces and distributes educational literature that has helped raise awareness about the phenomenon around the world. Mast Action UK is doing similar work in Great Britain, as well as the Electromagnetic Radiation Alliance in Australia.

With everything I know about the health dangers associated with electromagnetic fields (EMF) and micro waves from cell phones, WiFi routers and cell phone towers, I'm convinced electromagnetic hypersensitivity is a real and looming health disaster.

I've previously written about the health dangers of EMFs and other types of radiation, especially in your bedroom, but here the researchers are linking two very specific cancers – melanoma and breast cancer -- to commonly used beds, because coil-spring mattresses can actually act as a giant antenna!

Your Mattress – A Restorative Haven or a Disease-Promoting Zone?

In Western countries, the most common type of mattress is an elevated box spring that contains metal coils, while in Japan, people typically sleep on futon mattresses, which typically contain cotton or wool, placed directly on the floor.

According to researchers Hallberg and Johansson, a number of studies indicate that increasing rates of melanoma (the deadliest type of skin cancer) can be linked to immune-disrupting radiation from FM radio and TV transmission towers, adding that "geographical areas covered by several transmitters show higher incidences of melanoma than areas covered by one transmitter."

Studies have also linked radiation to brain tumors, and DNA damage that might precipitate a number of different diseases and health problems.

These connections are strengthened when you consider the researchers' explanation of how your box spring mattress actually acts like an antenna; *attracting and amplifying* whatever radiation might be zipping through your bedroom.

Scientific American explains this quite well:

"Antennas are simply metal objects of appropriate length sized to match the wavelength of a specific frequency of electromagnetic radiation. Just as saxophones are made in different sizes to resonate with and amplify particular wavelengths of sound, electromagnetic waves are selectively amplified by metal objects that are the same, half or one quarter of the wavelength of an electromagnetic wave of a specific frequency.

Electromagnetic waves resonate on a half-wavelength antenna to create a standing wave with a peak at the middle of the antenna and a node at each end, just as when a string stretched between two points is plucked at the center.

In the U.S. bed frames and box springs are made of metal, and the length of a bed is exactly half the wavelength of FM and TV transmissions that have been broadcasting since the late 1940s.

… Radiation envelops our bodies so that the maximum strength of the field develops 75 centimeters above the mattress in the middle of our bodies.

When sleeping on the right side, the body's left side will thereby be exposed to field strength about twice as strong as what the right side absorbs."

Could this explain why Japan has much lower rates of cancer compared to the US and Europe, and why the Japanese do not have higher rates of left- than right-sided breast cancer?

I believe it may be a part of the puzzle, yes.

Naturally, there are many other factors that come into play as well, including diet, chemical exposures, and vitamin D deficiency, just to name a few.

However, the theory that you may be promoting cancer by sleeping on a metal coil-spring mattress that amplifies ambient radiation is quite convincing.

Now, a couple of my readers have commented that the quote from Scientific American makes little sense because TV and radio broadcast on a number of different wavelengths, and beds come in many different sizes.

These concerns can also be valid, and I make no claims of having the in-depth technological expertise to either support or refute this particular Scientific American author’s explanation.

However, I believe sleeping on metal is not in your best interest health-wise, (and qualified scientist raised the question to begin with). The total effect will naturally be entirely individual, and dependent on a number of factors, such as the amount of radiation zipping through your

room; proximity to transmission towers; number and type of electronics kept in your bedroom and their proximity; your current state of health and your susceptibility to EMF; the material of the rest of your bed… I could go on, but I'm sure most of you are wise enough to get the picture.

Becoming truly health conscious is much like reaching for spiritual enlightenment. It's not about nitpicking on minor details. It's about discerning patterns and revealing the big picture. It's not about avoiding "sins" – in the case of health, avoiding everything that could possibly harm you – it's about making healthier, saner choices. You can't make those choices unless you know what's good for you, and one of the ways you discover better options is by revealing what's detrimental.

There's no question in my mind that EMFs can have a dramatic, negative effect on your health, and certain factors may, in some cases, turn up the dial on EMFs and increase it's harmful effects. Could your mattress do this? I say, "possibly, yes."

But I digress…

Sleeping in High Radiation Field May Also Trigger Development of Potent Mycotoxins

In one of my previous interviews with Dr. Dietrich Klinghardt, he discusses the research of Dr. Robert Becker, who discovered that electromagnetic fields produce more potent mycotoxins.

When you expose a bacterial culture to abnormal electromagnetic fields, the bacteria believe they are being attacked by your immune system and start producing much more virulent toxins as a protective mechanism!

In your body, these mycotoxins can wreak havoc with your health. Klinghardt believes that it's possible that some 50 percent of chronic infections are caused, and/or aggravated, by electromagnetic field exposure, leading to syndromes like chronic fatigue, fibromyalgia and other chronic pain syndromes.

When you consider that you spend one-third of your lifetime in bed, it's easy to realize the level of hazard you may be inadvertently exposed to if you're sleeping on a metal coil-spring mattress that literally bathes you in electromagnetic radiation.

Other Potential Health Hazards of Mattresses

In addition, the springs in your mattress can become magnetized, which can also pose a health hazard. In the article Is Your Mattress Making You Sick? Dr. Doris Rapp offers instructions on how to determine whether you mattress is magnetized.

The article also discusses a number of other important concerns when it comes to selecting a mattress, as metal springs is by no means the only health hazard inherent with these types of beds. Many mattresses are treated with a number of toxic flame retardant chemicals, some of which can ALSO cause cancer.

Remember that Your Cells are Driven by Electrical Impulses

I want to stress what might be one of the most fundamental issues when it comes to the potential hazard of electromagnetic radiation, which is the fact that the human body has its own internal electrical system.

All the cells in your body vibrate at different rates, and these weak electrical impulses are used for intracellular communication throughout your body.

Unfortunately, your body can literally act like a tuning fork. An external electrical influence can therefore cause your cells normal frequency or rate to be disrupted as they start vibrating to the frequency of the outside source. James Oschman explains this further in his book Energy Medicine.

Now, in the typical bedroom of a modern home, electrical exposure from external sources (live electrical wiring in ceilings, walls and floors) is thousands of times stronger than your body's own electrical system. Long-term exposure to these high-level electric fields can thus impair your body's intracellular communication, which can impact your health in a number of ways.

Biological Problems Associated with Electromagnetic Stressors

In the book Cross Currents, Dr. Robert Becker (whom I mentioned earlier) states that biological problems associated with external electromagnetic stressors fall into two major categories:

1. Brain (behavioral abnormalities, learning disabilities, altered bio-cycles and stress responses)
2. Growing tissue (embryos, genetics and cancer)

More recent studies seem to confirm his conclusions, as most of the health problems researchers now associate with EMF and microwave radiation exposure fall neatly into these two categories. One good source that demonstrates this is the 2009 special EMF issue of the Journal of Pathophysiology, which contains over a dozen different studies on the health effects of electromagnetic fields and wireless technology.

Cancer has been high on the list from the beginning, and at least as far as cell phones are concerned, the evidence is overwhelming that cell phone use significantly increases your risk of certain types of brain tumors.

Since cancer can take decades before manifesting, children are particularly at risk, as they are now growing up in an environment saturated with radiation. (Even toddlers are now talking on cell phones!) Today's children will be exposed to previously unimaginable levels of radiation over the course of their lifetimes.

The BioInitiative Report also includes studies showing evidence for exposure to electromagnetic fields and:

- Effects on Gene and Protein Expression (Transcriptomic and Proteomic Research)
- Genotoxic Effects – RFR and ELF DNA Damage
- Stress Response (Stress Proteins)
- Effects on Immune Function
- Effects on Neurology and Behavior
- Brain Tumors, Acoustic Neuromas, and childhood cancers like leukemia

It seems quite clear that sleeping on a bed that can amplify the amount of radiation you're exposed to night after night is simply not a good idea.

Shopping for a Safe Mattress

Taking into account that metal coils can act as an antenna and be potentially magnetized, and that most box spring mattresses contain toxic flame retardant chemicals, your safest bet is to look for an organic, chemical-free mattress that does not contain metal coils.

One way to find a safe mattress is to have a doctor or chiropractor write you a prescription for a chemical-free mattress, and then find a manufacturer to make one for you. You can also search for 100% wool, toxin-free mattresses. Many of these mattresses also contain latex for support in lieu of coil springs, giving you the best of both worlds.

You can also avoid the toxins by finding a mattress that uses a Kevlar, bullet-proof type of material instead of chemicals for fire-proofing. I got mine from Stearns and Foster. I'm quite happy with this mattress as it is FAR more comfortable than a number of the organic mattresses I had purchased in the past.

Also keep in mind that metal frames and headboards can amplify and distort magnetic fields, including the natural magnetic field from the earth, which can lead to non-restful sleep. Use natural materials, such as a wood frame, instead.

Five Steps to Create a Sleeping Sanctuary

Once you have addressed your mattress, you'd be well advised to address the amount of electromagnetic radiation in your bedroom – especially if you are pregnant, or planning to become pregnant.

As discussed by Dr. Klinghardt in this previous article, electromagnetic fields interfere not only with your biology, but with that of your unborn child as well. He performed a small study showing that autism can actually be **predicted** based on the EMF levels of your sleeping quarters while pregnant! He found that if you sleep in strong electromagnetic fields during pregnancy, your child will likely begin to exhibit neurological abnormalities within the first two years of life.

Here are several guidelines that can help turn your bedroom into an EMF-free zone suitable for health-promoting restorative sleep:

1. **Use only battery devices near your bed --** Many electric clocks produce high magnetic fields so use a battery powered clock instead.
2. **Turn off the fuses to your bedroom at night**
3. **Eliminate radio frequency (RF) sources --** Radio frequency signals from portable phones, cell phones, and wireless devices have been shown to interfere with your body's immune system so don't keep cell phones or charging stations in your bedroom. It's also recommended to turn off the Wi-Fi in your home at night.
4. **Shield your bed with a special metalized fabric,** to protect yourself from harmful frequencies that can disrupt cellular communication. This recommendation may be particularly important if you're pregnant.
5. If you are remodeling you can also use material called 'radiant barrier' between the dry wall and the frame of your home. This is a strengthened aluminum foil that will reflect most of the radiation from coming into your home. It should also be applied the ceiling.

   It is relatively inexpensive and will also pay for itself in reduced heating and cooling bills. However, it isn't cost effective if you have to tear out your current walls, so it's really only practical in new homes or remodeling projects. I installed this in my home when I renovated it and it virtually eliminated all external EMFs. The downside however is that it is VERY difficult to get any cell phone reception in my home.

Related Links:

- 4 Steps to Reduce Electrosmog in Your Bedroom
- Why Where You Sleep Matters If You Want a Healthy Baby
- Are You Sleeping in a Dangerous, Electrically Polluted Bedroom?

Author: Alice Elliott Brown — **Published:** Apr 03, 2011

Link: http://blogcritics.org/tastes/article/can-gmo-food-be-organic/

Can GMO Food Be Organic?

GMO Food: Genetically-modified organisms. This refers to the act of scientifically modifying the genetic structure of an organism. The resultant organism will have specifically defined characteristics. This is frequently done with seeds for crops. In the U.S., most of our soybeans, corn, cotton, and canola are genetically modified. Recently, the U.S. allowed GMO alfalfa to be planted without restriction. The genetically modified seeds can be patented. After all, the research to make them costs money. The argument for seed patents is that the investor must receive a return, or there will be no more investment money for research.

Until recently, I assumed GMO food could not be called "organic." The U.S. and Canada both prohibit 100% certified organic food from containing GMO ingredients. However, contamination of the crops may cause organic feed to contain some percentage of GMO ingredients. At the Straus Family Creamery in California, for example, Farmer Straus spent nearly $10,000 tracing back the ingredients in his organic supplies, to remove the GMO traces. Basically, the problem is that GMO crops spew pollen into the air, as all crops do. This pollen cross-breeds with organic feed, which pollutes and corrupts the organic farms.

It is a normal practice for organic farmers to save their seed for the next year. With GMO crops growing nearby, however, the organic crops become infected with the GMO seed. When the farmer saves his seed, he is then infringing on Monsanto Corporation's patent. Monsanto issued policies about patent infringement and their methods of enforcing patent law on these seeds. In these policies, Monsanto takes the position that their patented seed is desirable, and therefore the farmers who normally save their own seed are taking something away from the company.

Organic farmers point out that they are no longer organic when their seed is contaminated by genetically-engineered cross-pollination. They are the victim, not the perpetrator. The Monsanto Corporation protests that it has sued only 145 farmers for patent violation. Meanwhile, the Organic Consumers Organization has organized a Millions Against Monsanto campaign to pressure Congress to force foods to be labeled as GMO. This would allow consumers to decide whether they will risk their health with GMO foods. The FDA does not determine whether or not GMO is safe. It only determines whether evidence has been provided to declare it unsafe. No evidence, no reason not to eat it.

Of course, whether GMO food is safe will not be determined until after people have eaten it for 20 or more years. By that time, non-GMO seeds may no longer be available on the planet, as the wind and the birds cross-pollinate our fields. Although dramatic increases in the incidence of allergies and immune system diseases have occurred over the last 20 years, science have not been able to identify why this has happened.

Currently, the only way to know you are not purchasing food contaminated with GMOs is to not buy anything that contains a likely GMO food, and to buy only processed foods with the "Non-GMO Project Verified seal".

What can we do if we want the right to choose? First, take the time to learn more.
Your rights depend upon it.

Genetically modified food, also called *frankenfood* by critics, is found in most processed food in the US. If you eat food made from corn, canola, soybeans, cottonseed oil, or sugar from beets, you are most likely ingesting GMOs. Livestock animals, including dairy cows, are fed predominantly corn, canola, soy, and alfalfa, which is also genetically engineered.

Currently, a GM Atlantic salmon is up for approval for American tables, as well. Not one of these plant or animal products is required to be labeled in the U.S., even though the public has repeatedly demanded choice. Currently, the only way to know you are not purchasing food contaminated with GMOs is to not buy anything that contains a likely GMO food, and/or to buy only processed foods that have the "Non-GMO Project Verified seal".

What can we do if we want the right to choose? First, take the time to learn more.
Your rights depend upon it.

GMO Myths & Truths

Genetically modified crops are promoted on the basis of a range of far-reaching claims from the biotech industry and its supporters. A new, evidence-based report by U.K. scientists shows these claims are unsubstantiated. Here is a snapshot of their findings.

MYTH #1: Genetic engineering is just an extension of natural breeding.

TRUTH: Genetic engineering is very different from natural breeding and poses special risks. Natural breeding occurs between like life forms-a cat with a cat, not a cat with a dog or a tomato with a fish. GM transfers DNA between unrelated organisms in ways that do not occur naturally.

MYTH #2: GM foods are strictly regulated for safety.

TRUTH: GM food regulation in most countries varies from non-existent (the U.S.) to weak. In the U.S. the FDA overruled its own scientists to form a GM policy, in the 1990s. The policy required no safety testing or labeling.

MYTH #3: GM foods are safe to eat.

TRUTH: GM foods can be toxic or allergenic. Peer-reviewed studies have found serious, harmful effects on the health of livestock and lab animals fed GMOs.

MYTH #4: GM Bt insecticidal crops harm only insects and are harmless to animals and people.

TRUTH: GM Bt insecticidal crops pose hazards to people and animals that ingest them. Findings include toxic effects on the small intestine, liver, kidney, spleen, and pancreas, and disturbances in the digestive and immune systems.

MYTH #5: GM animal feed poses no risks to animal or human health.

TRUTH: GM feed affects the health of animals and may affect the humans who eat their products. Bt toxin protein has been found in the blood of pregnant women and the blood supply to their fetuses.

MYTH #6: GM crops increase yield potential.

TRUTH: GM crops do not increase yield potential-and in many cases decrease it. Dr. Doug Gurian-Sherman: "Traditional breeding ...can be solely credited with the intrinsic yield increases in the U.S. and other parts of the world that characterized the agriculture of the 20th century:'

MYTH #7: GM crops decrease pesticide use.

TRUTH: GM crops increase pesticide use. In the first 13 years since their introduction, in 1996, GM crops increased pesticide use by 383 million pounds.

MYTH #8: No-till farming with GM crops is “environmentally friendly”.

TRUTH: Claims of environmental benefits are unsound. GM herbicide-tolerant crops, such as Roundup Ready soy, have increased the use of toxic chemicals and led to glyphosate-resistant superweeds. These superweeds and other pests now require even more chemical controls.

GMO Myths & Truths

MYTH #9: Roundup (Monsanto's glyphosate) is a benign, biodegradable herbicide.

TRUTH: Roundup is not biodegradable, and was forced by law to remove that claim from its packaging. Roundup persists in the environment and has toxic effects on wildlife. Roundup (Glyphosate) is toxic, and was detected in 60%-100% of air and rain samples in the U.S. Midwest during crop growing season.

MYTH #10: GM crops can "coexist" with non-GM.

TRUTH: "Coexistence" rapidly results in widespread contamination of non-GM and organic crops. Germany passed a law making GM crop growers liable for economic damages to non-GM farmers resulting from GM contamination. The law has virtually halted the planting of GM crops in that country.

MYTH #11: GM will deliver climate-ready crops.

TRUTH: Conventional breeding outstrips GM in delivering climate-ready crops. Tolerance to extreme weather and resistance to accompanying pests and diseases are complex traits that GM cannot deliver.

MYTH #12: GM reduces energy use.

TRUTH: GM crops are energy-hungry. They depend on large amounts of herbicides which require large amounts of fossil fuels to manufacture. The U.S. food system spends 10 kilocalories of fossil energy for every 1 kilocalorie produced. Two-thirds of that energy goes to produce synthetic fertilizers and on-farm mechanization.

MYTH #13: GM crops are needed to feed the world's growing population.

TRUTH: GM crops are irrelevant to feeding the world. GM neither delivers higher yields nor produces more with fewer inputs than non-GM crops. Hunger is a problem of distribution, poverty, and loss of crop diversity – which GM crop growth in developing nations has been shown to worsen..

MYTH #14: GM crops are vital to achieving food security.

TRUTH: Agro-ecological farming is the key to food security, according to 400 scientists and experts from 80 countries, a position endorsed by 62 governments worldwide. Their report, the *International Assessment of Agricultural Knowledge, Science and Technology,* did not endorse GM crops or livestock.

Summarized from **"GMO *Myths and Truths: An evidence-based examination* of *the claims made* for *the safety and efficacy* of *genetically modified crops,"*** *by Michael Antoniou, PhD; Claire Robinson, MPhil; and John Fagan, PhD; June* 2012, *published by Earth Open Source,* a *London based not-for-profit dedicated* to *assuring the sustainability, security, and* safety *of the global food* system. *Download the free 123-page report* at ***www.earthopensource.org*** **.**

INVISIBLE GM INGREDIENTS

Processed foods often have hidden GM sources (unless they are organic or declared non-GMO). The following are ingredients that may be made from GM soy, corn, cotton, or canola.

Aspartame
baking powder
bee pollen
caramel color
cellulose
citric acid
cobalamin(Vitamin B12)
corn gluten
corn masa
corn oil
corn syrup
cornmeal
cornstarch
cyclodextrin
cystein
dextrin
dextrose
diacetyl
diglyceride
fructose
fructose (crystalline)
glucose
glutamate
glutamic acid
gluten
glycerides
glycerin
glycerol
glycerol monooleate
glycine
hemicellulose
high fructose corn syrup (HFCS)
hydrogenated starch hydrolates
hydrolyzed vegetable protein
inositol
invert sugar (colorose or inversol)
inverse syrup
isoflavones
lactic acid
lecithin
leucine
lysine
malitol
maltodextrin
maltose
mannitol
methylcellulose
milo starch
modified starch
monosodium glutamate
oleic acid
phenylalanine
phytic acid
sorbitol
soy flour
soy isolates
soy lecithin
soy protein
starch
stearic acid
tamari
tempeh
threonine
tocopherols (Vitamin E)
tofu
trehalose
triglyceride
vegetable fat
vegetable oil
Vitamin B12
Vitamin E
xanthan gum

Our understanding is that ascorbic acid (Vitamin C),although usually derived from corn, is probably not GM because it is not made in North America.

Honey and bee pollen may contain GMOs if the beehives are near GM crops.

This list is continually being updated and refined. For the most recent version, see www.responsibletechnology.org.

Info from the Non-GMO Project website:

http://www.nongmoproject.org/learn-more/

What are GMOs?
GMOs, or "genetically modified organisms," are plants or animals created through the gene splicing techniques of biotechnology (also called genetic engineering, or GE). This experimental technology merges DNA from different species, creating unstable combinations of plant, animal, bacterial and viral genes that cannot occur in nature or in traditional crossbreeding.

Virtually all commercial GMOs are engineered to withstand direct application of herbicide and/or to produce an insecticide. Despite biotech industry promises, none of the GMO traits currently on the market offer increased yield, drought tolerance, enhanced nutrition, or any other consumer benefit.

Meanwhile, a growing body of evidence connects GMOs with health problems, environmental damage and violation of farmers' and consumers' rights.

Are GMOs safe?
Most developed nations do not consider GMOs to be safe. In nearly 50 countries around the world, including Australia, Japan, and all of the countries in the European Union, there are significant restrictions or outright bans on the production and sale of GMOs. In the U.S., the government has approved GMOs based on studies conducted by the same corporations that created them and profit from their sale. Increasingly, Americans are taking matters into their own hands and choosing to opt out of the GMO experiment.

Are GMOs labeled?
Unfortunately, even though polls consistently show that a significant majority of Americans want to know if the food they're purchasing contains GMOs, the powerful biotech lobby has succeeded in keeping this information from the public. In the absence of mandatory labeling, the Non-GMO Project was created to give consumers the informed choice they deserve.

Where does the Non-GMO Project come in?
The Non-GMO Project is a non-profit organization with a mission of protecting the non-GMO food supply and giving consumers an informed choice. We offer North America's ONLY third party verification for products produced according to rigorous best practices for GMO avoidance (for more info, click here). Our strategy is to empower consumers to make change through the marketplace. If people stop buying GMOs, companies will stop using them and farmers will stop growing them.

Do Americans want non-GMO foods and supplements?
Polls consistently show that a significant majority of North Americans would like to be able to tell if the food they're purchasing contains GMOs (a 2008 CBS News Poll found that 87% of consumers wanted GMOs labeled). And, according to a recent CBS/New York Times poll, 53% of consumers said they would not buy food that has been genetically modified. The Non-GMO Project's seal for verified products will, for the first time, give the public an opportunity to make an informed choice when it comes to GMOs.

How common are GMOs?
In the U.S., GMOs are in as much as 80% of conventional processed food. Click here for a current list of GMO risk crops.

Why does the Non-GMO Project verify products that have a low risk of containing GMOs?
Some ingredients that seem low-risk may have less-visible high-risk ingredients. Take, for example, dried fruit. Raisins and similar fruit are sometimes packed with a small quantity of oil to keep them moist. This oil, when used, is sometimes high-GMO-risk. As such, it is critical that we do take the time to look carefully at ingredient spec sheets during the verification process, to ensure that risks like this are effectively mitigated, even in apparently low-risk products.

Contamination incidents have occurred with seemingly "low-risk" products (rice, starling corn, flax). Non-GMO Project Verification supports manufacturers in being able to quickly and proactively respond to unexpected contamination issues.

Verifying only high-risk products puts a heavy burden on consumers to know what products are at risk of containing GMOs. Many people, even in the world of Natural Foods, don't know what a GMO is, let alone which crops and processed ingredients are high-risk. As such, labeling only products that contain high-risk ingredients could give an unfair competitive advantage to products that contain ingredients containing corn, soy, etc. Taking the cereal aisle for our example, if we verified only high-risk products, a shopper might see the seal on a box of verified corn flakes, but not on the wheat-based cereal box next to them, produced with the same high standards by the same company. This could leave them thinking the corn flakes were non-GMO, but that they should avoid the wheat product, even though there's no GMO wheat on the market. Given the lack of understanding of the issue, this presents some serious issues.

Through verifying low-risk products, the Non-GMO Project's work builds consumer interest and industry investment in Non-GMO, even for crops that aren't genetically engineered yet. Biotech is constantly working to patent and commercialize new organisms (salmon, apples, etc.), and the more companies that have committed to Non-GMO production, the more resistance these new developments will see prior to release.

What are the impacts of GMOs on the environment?
Over 80% of all GMOs grown worldwide are engineered for herbicide tolerance. As a result, use of toxic herbicides like Roundup has increased 15 times since GMOs were introduced. GMO crops are also responsible for the emergence of "super weeds" and "super bugs:' which can only be killed with ever more toxic poisons like 2,4-D (a major ingredient in Agent Orange). GMOs are a direct extension of chemical agriculture, and are developed and sold by the world's biggest chemical companies. The long-term impacts of GMOs are unknown, and once released into the environment these novel organisms cannot be recalled.

How do GMOs affect farmers?
Because GMOs are novel life forms, biotechnology companies have been able to obtain patents with which to restrict their use. As a result, the companies that make GMOs now have the power to sue farmers whose fields are contaminated with GMOs, even when it is the result of inevitable drift from neighboring fields. GMOs therefore pose a serious threat to farmer sovereignty and to the national food security of any country where they are grown, including the United States.

How can I avoid GMOs?
Choose food and products that are Non-GMO Project Verified! Click here to see a complete list.

Learn more about GMOs:

To learn how to make Non-GMO food choices:

The Non-GMO Project: www.nongmoproject.org/

The Non-GMO Shopping Guide: www.nongmoshoppingguide.com/

To Learn More In-Depth Information About GMOs:

Watch the informative documentary, "Genetic Roulette": http://geneticroulettemovie.com/

Visit the Institute for Responsible Technology: http://responsibletechnology.org/

Visit Earth Open Source and download their paper "GMO Myths & Truths: http://earthopensource.org/

Read the GM Watch research paper "GM Crops – Just the Science": http://www.gmwatch.eu/gm-crops-research-documenting-the-limitations-risks-and-alternatives

Watch the award-winning documentary "The Future of Food": http://www.thefutureoffood.com/

Watch "One Mom's Story": http://justlabelit.org/right-to-know/why-labels-matter-to-moms/

To learn more about the economic and social damage caused by GMOs:

Impacts of Genetically Engineered Crops on Pesticide Use in the United States: The First Thirteen Years: http://www.organic-center.org/reportfiles/13Years20091126_FullReport.pdf

The GMO Emperor has No Clothes: http://www.navdanya.org/attachments/Latest_Publications1.pdf

The Union of Concerned Scientists 2009 GMO research paper "Failure to Yield":

http://www.ucsusa.org/food_and_agriculture/science_and_impacts/science/failure-to-yield.html

Watch the award-winning documentary, "Bitter Seeds":

http://teddybearfilms.com/2011/10/01/bitter-seeds-2/

To contact food companies for more information about their products, and to ask them to become a part of the Non-GMO Project:

Food Manufacturer List: http://nutritionwonderland.com/2009/02/organic-corporate-hierarchy/

This flyer is brought to you by Oregonians for Farm & Food Rights.
To learn more, visit: www.FarmAndFoodRights.org

Prop 37: Your right to know.

GMOs: Corporate Charlatans Versus Organic Heroes

Corporate agribusiness fighting Prop 37
Donated: $23,500,000

VS.

Organic leaders supporting Prop 37
Donated: $3,537,450

MONSANTO
$4,208,000

PEPSICO
$1,716,300

COCA COLA
$1,164,400

CONAGRA
$1,076,300

KELLOGG
$632,500

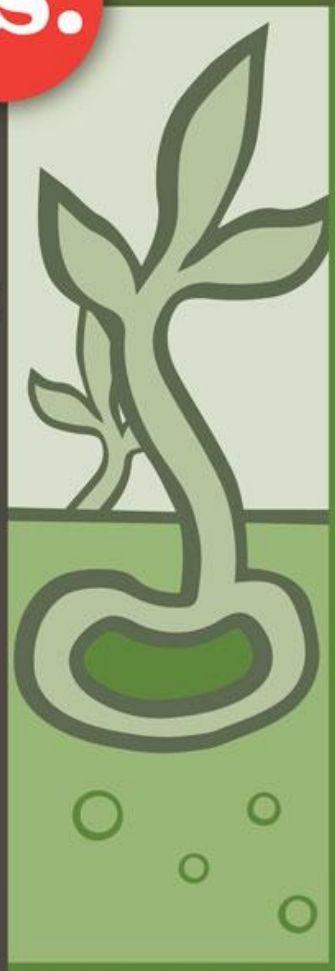
GENERAL MILLS
$520,000

SMUCKER
$387,000

DEAN FOODS
$253,000

RICH PRODUCTS
$225,000

COUNSEL FOR BIOTECHNOLOGY INFORMATION (DONATED $375,000), **GROCERY MANUFACTURERS ASSOCIATION** ($375,000), **BIOTECHNOLOGY INDUSTRY ORGANIZATION**: ($250,000)



ALEX BOGUSKY [FEARLESS REVOLUTION] ($100,000), **MICHAEL FUNK** ($50,000), **PRESENCE MARKETING** ($20,000), **BURROUGHS FAMILY FARM** ($5,000), **BAKER CREEK HEIRLOOM SEEDS** ($4,000), **MINTWOOD MEDIA COLLECTIVE** ($1,250)

DR. JOSEPH MERCOLA
$1,100,000

$600,000

$100,000

$52,500

$50,000

$20,000

$10,000

$10,000

$10,000

$5,000

$2,500

$40,000 (ASSOC)/
$730,000 (FUND)

$290,000

$200,000

$50,000

$25,000

$12,500

$10,000

$10,000

$7,500

$7,200

$3,000

$2,500

$2,500

As Goes California, So Goes the Nation.

Democratic and Republican administrations, and Congress, have repeatedly ignored the overwhelming majority of Americans who favor labeling genetically engineered (GE) food in the marketplace. Our politicians seem to be listening to the corporate executives (donors) instead of the citizenry. But in California, the people have a right to craft laws of their choosing. Proposition 37, on the ballot in California on November 6, would mandate labeling of foods containing GE ingredients. If we win this fight in California, manufacturers will likely begin to label food nationally for GE ingredients.

Please make your voice heard by signing the petition at cornucopia.org

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WWW.CORNUCOPIA.ORG

United States Dept. of Agriculture | Agricultural Marketing Service

National Organic Program

Genetically Modified Organism (GMO)

Miles McEvoy, Deputy Administrator

GMO OVERVIEW

The Organic Food Production Act (OFPA) of 1990 does not mention biotechnology, genetic engineering or genetically modified organisms. OFPA prohibits synthetics unless they are allowed, allows natural substances unless they are prohibited.

The first National Organic Program proposed rule (1997) did not prohibit GE substances or GMOs. There was a huge public outcry against GMOs being considered in organic production and handling. Proposed rule withdrawn.

The second National Organic Program proposed rule (2000) excluded the use of GMOs in organic production and handling.

GMOs are prohibited as excluded methods: 7 CFR § 205.105

Allowed and prohibited substances, methods, and ingredients in organic production and handling.

To be sold or labeled as "100 percent organic," "organic," or "made with organic (specified ingredients or food group(s))," the product must be produced and handled **without the use of**:

(e) Excluded methods, except for vaccines: Provided, That, the vaccines are approved in accordance with §205.600(a);

Excluded Methods

A variety of methods used to genetically modify organisms or influence their growth and development by means that are not possible under natural conditions or processes and are not considered compatible with organic production. Such methods include cell fusion, microencapsulation and macroencapsulation, and recombinant DNA technology (including gene deletion, gene doubling, introducing a foreign gene, and changing the positions of genes when achieved by recombinant DNA technology).

Such methods do not include the use of traditional breeding, conjugation, fermentation, hybridization, in vitro fertilization, or tissue culture.

NOP Policy

- In 2004 the NOP responded to a letter from the National Association of State Departments of Agriculture (NASDA) regarding the use of GMOs in organic production and handling.
- In September 2010 the NOP published the Program Handbook: Guidance and Instructions for Accredited Certifying Agents and Certified Operations.
- In April 2011 the NOP issued NOP Policy Memo 11-13 to address GMOs in organic production and handling. The policy reiterates the policy outlined in the 2004 letter to NASDA and clarifies questions concerning GMOs and organic production and handling.

NOP Policy Memo 11-3

Issue:

If a producer adheres to all aspects of the NOP regulations, Including never utilizing genetically modified seeds, but a Certifying agent tests and detects the presence of genetically modified material in the crop, is that crop's status determined to be no longer certified organic?

Continued on next page-

NOP Policy Memo 11-3

Reply:

Organic certification is process based. That is, certifying agents attest to the ability of organic operations to follow a set of production standards and practices which meet the requirements of the Organic Foods Production Act of 1990 and the NOP regulations. The NOP regulations prohibit the use of excluded methods (i.e., "GMOs") in organic operations. If all aspects of the organic production or handling process were followed correctly, then the presence of a detectable residue from a genetically modified organism alone does not constitute a violation of this regulation. This policy was established at the promulgation of the NOP Regulation in the Preamble to the Final Rule (FR Vol. 65, No. 246, p. 80556), December 21, 2000. The Preamble stated that:

Continued on next page-

NOP Policy Memo 11-3

Continued from previous page-

Reply-

As long as an organic operation has not used excluded methods and takes reasonable steps to avoid contact with the products of excluded methods as detailed in their approved organic system plan, the unintentional presence of the products of excluded methods should not affect the status of the organic operation or its organic products

NOP Policy

Issue:

Is the inadvertent presence of GMOs in organic seeds a violation of the NOP regulations? Can organic producers use seeds that contain the inadvertent presence of GMOs?

Continued on next page-

NOP Policy

Reply:

7 CFR § 205.105 of the NOP regulations prohibits the use of GMOs as excluded methods in organic production and handling. The use of excluded methods, such as planting genetically modified seeds, would require a specific intent, and would render any product ineligible for organic certification. However, the inadvertent presence of GMOs in organic seeds does not constitute a use because there was no intent on the part of the certified operation to use excluded methods. The presence of detectable GMO residues alone in an organic seed does not constitute a violation of the NOP regulations

NOP Policy

Issue: How do organic producers avoid contact with GMOs?

Reply:

Organic producers utilize a variety of methods to avoid contact or the unintentional presence of GMOs including testing seed sources for GMO presence, delayed or early planting to get different flowering times for organic and GMO crops, cooperative agreements with neighbors to avoid planting GMO crops adjacent to organic crops, cutting or mowing alfalfa prior to flowering, posting signs to notify neighboring farmers of the location of organic fields, and thorough cleaning of farm equipment that has been used in non-organic crop production.

NOP Policy

Issue:

What are organic producers required to do in order to avoid the presence of GMOs in their products?

Reply:

In order to become a certified organic operation, a producer must submit an organic system plan to a NOP accredited certifying agent for approval. The producer's organic system plan must include a description of management practices and physical barriers established to prevent contact of organic crops with prohibited substances. Certifying agents evaluate the preventative practices and buffer zones to determine if the producer has taken reasonable steps to avoid contact with GMOs.

NOP Policy

Issue:

Could a farm's organic certification status be threatened if sufficient buffers and barriers are not established and inadvertent contact with GMO material occurs?

Reply:

Organic producers that implement preventive measures to avoid contact with GMOs will not have their certification threatened from the inadvertent presence of the products of excluded methods (GMOs). Crops grown on certified organic operation may be sold, labeled and represented as organic, even with the inadvertent presence of GMOs, provided that all organic requirements under 7 CFR Part 205 have been followed.

NOP Policy

Issue:

Is there a working definition of the word "contamination" within the NOP?

Reply:

There is no definition in the NOP regulations for the word "contamination," even though it is mentioned frequently in the standards. The use of excluded methods in organic production is prohibited, as cited in 7 CFR § 205.105.

NOP Policy

Issue:

What actions are authorized or required when organic crops or products are found to contain unintended or inadvertent genetically modified substances?

Reply:

The inadvertent presence of genetically modified material does not affect the status of the certified operation and does not result in loss of organic status for the organic product, provided it was produced in accordance with all of the organic requirements under 7 CFR Part 205. Certifying agents are responsible for working with organic producers to identify the source of the inadvertent GMOs and to implement reasonable steps to avoid contact with GMOs in the future.

NOP Policy

Issue:

Are organic products tested for genetically modified substances?

Reply:

Under 7 CFR § 205.670(b) certifying agents may test organic products when there is reason to believe that excluded methods were used in the production or handling of an organic agricultural product. Certifying agents may also collect and test organic products from organic handlers to ensure that practices are in place to prevent commingling or contamination during handling and processing.

NOP Policy

Issue:

Are organic products free of GMO contaminants?

Reply:

Organic standards are process based. The NOP regulations prohibit the use of genetically modified organisms, prohibit commingling or contamination during processing and handling, and require preventative practices to avoid contact with GMOs. Organic agricultural products should have minimal if any GMO contaminants; however, organic food products do not have a zero tolerance for the presence of GMO material.

NOP Policy

Issue:

Has a tolerance level (e.g. 5%) been established for the presence of GMOs in organic agricultural products?

Reply:

The NOP regulations do not establish GMO tolerance levels. The NOP regulations establish a tolerance for the presence of pesticides registered by the U.S. Environmental Protection Agency (EPA) that is set at 5% of the EPA tolerance level for the specific residue detected. No federal agency, including EPA or USDA has established tolerance levels for the inadvertent presence of the products of excluded methods (GMOs).

NOP Policy

Issue:

Processed foods sold as “organic” must contain at least 95% organic ingredients. Are GMOs allowed in the remaining 5% of ingredients? Likewise, processed foods sold as “made with organic (specified ingredients or food group(s))” must contain at least 70% organic ingredients. Are GMOs allowed in the remaining 30% of ingredients for these products?

Reply:

The use of GMOs is prohibited in all ingredients in “organic” and “made with organic (specified ingredients or food groups(s)).” There is no provision within the NOP regulations that allows the use of excluded methods (GMOs) in ingredients or processing aids under the “organic” or “made with organic (specified ingredients or food group(s))” label categories.


USDA
ORGANIC

When Organic Isn't Really Organic

By Jyoti Thottam Wednesday, Mar. 14, 2007 TIME MAGAZINE

Link: http://www.time.com/time/health/article/0,8599,1599110,00.html

Learn More

At the Straus Family Creamery. Straus Family Creamery

When you buy a gallon of organic milk, you expect to get tasty milk from happy cows who haven't been subjected to antibiotics, hormones or pesticides. But you might also unknowingly be getting genetically modified cattle feed.

Albert Straus, owner of the Straus Family Creamery in the small northern California town of Marshall, decided to test the feed that he gives his 1,600 cows last year and was alarmed to find that nearly 6% of the organic corn feed he received from suppliers was "contaminated" by genetically modified (GM) organisms. Organic food is, by definition, supposed to be free of genetically modified material, and organic crops are required to be isolated from other crops. But as GM crops become more prevalent, there is little that an organic farmer can do to prevent a speck of GM pollen or a stray GM seed from being blown by the wind onto his land or farm equipment and, eventually, into his products. In 2006, GM crops accounted for 61% of all the corn planted in the U.S. and 89% of all the soybeans. "I feared that there weren't enough safeguards," Straus says.

So Straus and five other natural food producers, including industry leader Whole Foods, announced last week that they would seek a new certification for their products, "non-GMO verified," in the hopes that it will become a voluntary industry standard for GM-free goods. A non-profit group called the Non-GMO Project runs the program, and the testing is conducted by an outside lab called Genetic ID. In a few weeks, Straus expects to become the first food manufacturer in the country to carry the label in addition to his "organic" one. With Whole Foods in the ring, the rest of the industry will soon be under competitive pressure to follow.

Earning the non-GMO label, at least initially, requires nearly as much effort as getting certified organic. To root out the genetically modified corn, Straus spent several months and about $10,000 testing, re-testing and tracing back his products: from his own dairy's milk, to other dairies that supply some of his milk, to the brokers who sell them feed, to their mills that grind the corn, to farmers who grow it. To put the GM-free label on his ice cream, Straus will have to trace the chickens that provided the egg yolks, the grain used in the alcohol that carries his vanilla extract and the soy lecithin used as an emulsifier for his chocolate chips.

So why bother? The organic and natural foods industry sees a huge opportunity in telling consumers even more about what's in their food. Few consumers would think about the pesticides and hormones in conventional foods without the organic alternative to remind them. Similarly, genetically modified crops have become so prevalent in the U.S. that chances are you've been buying and eating them for years. You just wouldn't know it from the label: the U.S. Department of Agriculture, unlike agencies in Europe and Japan, do not require GM foods to be labeled. While scientists have not identified any specific health risks from eating GM foods, anti-GM activists say there is not enough research yet into their long-term risks or impact on biodiversity. By telling consumers loud and clear which products are GM-free, organic-food producers will give them one more reason to choose organic. Says Jeffrey Smith, a longtime activist against genetically modified food: "The people served by the organic industry are very sensitive to GMO." And, the industry hopes, willing to pay to avoid it.

2012 Shopper's Guide
to Pesticides in Produce

News Release - EWG Releases 2012 Shopper's Guide to Pesticides in Produce

Researchers Highlight Pesticides in Produce, Baby Food, Tap Water

Published June 19, 2012

Washington, D.C. – Environmental Working Group has released the eighth edition of its Shopper's Guide to Pesticides in Produce [1] with updated information on 45 popular fruits and vegetables and their total pesticide loads. EWG highlights the worst offenders with its new Dirty Dozen Plus™ list and the cleanest conventional produce with its list of the Clean Fifteen™.

"The explosive growth in market share for organic produce in recent years testifies to a simple fact that pesticide companies and the farmers who use their products just can't seem to grasp: people don't like to eat food contaminated by pesticides," said EWG president Ken Cook. "Our shopper's guide to pesticides in produce gives consumers easy, affordable ways to eat a diet rich in fruits and vegetables while avoiding most of the bug killers, fungicides and other chemicals in produce and other foods."

"This year's guide will also give new parents pause," Cook added. "Government scientists have found disturbing concentrations of pesticides in some baby foods. And the U.S. Department of Agriculture has found weed killers widespread in finished tap water. Environmentalists have had important successes in forcing pesticides that presented unacceptably high dietary risks off the market. The latest USDA tests show we have much more work to do."

EWG researchers analyzed annual pesticide residue tests conducted by the USDA and federal Food and Drug Administration between 2000 and 2010. The samples were first washed or peeled prior to being tested so the rankings reflect the amounts of the crop chemicals likely present on the food when is it eaten.

The USDA and FDA tests have produced hard evidence of widespread presence of pesticide residues on conventional crops. The most recent round of tests show that as late as 2010, 68 percent of food samples had detectable pesticide residues. EWG found striking differences between the number of pesticides and amount of pesticides detected on the Dirty Dozen Plus™and Clean Fifteen™ foods.

Notable findings:

Some 98 percent of conventional apples have detectable levels of pesticides.

Domestic blueberries tested positive for 42 different pesticide residues.

Seventy-eight different pesticides were found on lettuce samples.

Every single nectarine USDA tested had measurable pesticide residues.

As a category, grapes have more types of pesticides than any other fruit, with 64 different chemicals.

Thirteen different pesticides were measured on a single sample each of celery and strawberries.

New to the Shopper's Guide: The Dirty Dozen Plus

2012 Shopper's Guide
to Pesticides in Produce

For the past eight years, EWG has scrutinized pesticide testing data generated by scientists at USDA and FDA and has created its signature Dirty Dozen™ list of foods most commonly contaminated with pesticides. As well, we publish our Clean Fifteen™ list of the foods least likely to be pesticide-tainted.

This year we have expanded the Dirty Dozen™ with a Plus category to highlight two crops -- green beans and leafy greens, meaning, kale and collard greens – that did not meet traditional Dirty Dozen™ criteria but were commonly contaminated with highly toxic organophosphate insecticides. These insecticides are toxic to the nervous system and have been largely removed from agriculture over the past decade. But they are not banned and still show up on some food crops. For this reason, EWG lists these on the new Dirty Dozen Plus™ as foods to avoid or to buy organic.

"Organophosphate pesticides are of special concern since they are associated with neurodevelopmental effects in children," said EWG toxicologist Johanna Congleton. "Infants in particular should avoid exposure to these pesticides since they are more susceptible to the effects of chemical insult than adults."

The American Academy of Pediatrics advises parents [2] to "minimize using foods in which chemical pesticides or herbicides were used by farmers."

Pesticides in Baby Food

For the first time since the inception of its pesticide testing program in 1991, USDA looked at pesticide residues on baby food. Department scientists analyzed about 190 samples each of prepared baby food consisting of green beans, pears and sweet potatoes.

Green beans prepared as baby food tested positive for five pesticides, among them, the organophosphate methamidiphos, which was found on 9.4 percent of samples, and the organophosphate acephate, on 7.8 percent of samples. EWG analyzed baby food samples in 1995 and found the two organophosphates in surprisingly similar concentrations.

Pears prepared as baby food showed significant and widespread contamination. Fully 92 percent of the pear samples tested positive for at least one pesticide residue, with 26 percent of samples containing 5 or more pesticides and 15 different pesticides on all samples. Disturbingly, the pesticide iprodione, which EPA has categorized as a probable human carcinogen, was detected on three baby food pear samples. Iprodione is not registered with the EPA for use on pears. Its presence on this popular baby food constitutes a violation of FDA regulations and the federal Food, Drug, and Cosmetic Act.

"Federal testing of pesticide residue in baby food was long overdue, as infants are especially vulnerable to toxic compounds," said Andrew Weil, MD, Founder and Director, Arizona Center for Integrative Medicine and a renowned medical expert on natural health and wellness. "Now that it has begun, the results are highly disturbing. It is bad enough that baby food contains pesticides at all; the fact that pears contain a likely human carcinogen is an outrage. Parents should purchase organic baby foods, or better yet, prepare their own by putting organic foods through a simple hand-turned food mill (search the internet for "baby food mill"). It is vital that an infant's developing brain and nervous system receive only uncontaminated, nutrient-dense foods."

Sweet potatoes sold as baby food, a Clean Fifteen™ crop, had virtually no detectable pesticide residues.

The extent of pesticide contamination document by USDA's baby food tests highlight the need for the department to accelerate testing of baby foods and for EPA to reduce further the organophosphate pesticide exposures allowed for Americans, especially infants.

Pesticides in Drinking water

In 2010 USDA analyzed samples from 12 community drinking water systems that use surface water such as reservoirs, lakes and rivers as their water sources. Tests of 284 samples taken after treatment detected 65 pesticides or their metabolites. The toxic herbicide atrazine or its metabolites were found in every single sample. The herbicides 2,4-D and metolachlor were detected in more than 70 percent of the samples. Six other pesticides were found in at least half the samples.

Clean Fifteen™

The footprint of pesticide residues for those items placed on the Clean Fifteen™ looked much better.

The produce least likely to test positive for pesticides were asparagus, avocado, cabbage, grapefruit, watermelon, eggplants, pineapples, mushrooms, onions, frozen peas and sweet potatoes.

More than 90 percent of cabbage, asparagus, sweet peas, eggplant and sweet potato samples had one or fewer pesticides detected. Of the Clean Fifteen™ vegetables, no single sample had more than 5 different chemicals, and no single fruit sample from the Clean Fifteen™ had more than 5 types of pesticides detected.

The EWG's Shopper's Guide is not built on a complex assessment of pesticide risks but instead reflects the overall pesticide loads of common fruits and vegetables. This approach best captures the uncertainties of the risks of pesticide exposure. Since researchers are constantly developing new insights into how pesticides act on living organisms, no one can say that concentrations of pesticides assumed today to be safe are, in fact, harmless.

The Shopper's Guide aims to give consumers confidence that by following EWG's advice, they can buy foods with consistently lower overall levels of pesticide contamination.

EWG is a nonprofit research organization based in Washington, DC that uses the power of information to protect human health and the environment. http://www.ewg.org [3]
Source URL:
http://www.ewg.org/release/ewg-releases-2012-shopper-s-guide-pesticides-produce

Links:
[1] http://www.ewg.org/foodnews/
[2] http://www.healthychildren.org/English/safety-prevention/all-around/Pages/Pesticides-Herbicides-and-Children.aspx?nfstatus=401&nftoken=00000000-0000-0000-0000-000000000000&nfstatusdescription=ERROR%253a+No+local+token&nfstatus=401&nftoken=00000000-0000-0000-0000-000000000000&nfstatusdescription=ERROR%3a+No+local+token
[3] http://www.ewg.org

EWG's 2012 Shoppers Guide to Pesticides in Produce™

Cut along line

Instructions:

1. Cut along outside line.
2. Fold along middle line.

Fold together

EWG's 2012 Dirty Dozen™ Plus

Shoppers Guide to Pesticides in Produce™

Buy these organic

Domestic
Imported

- Apples
- Bell Peppers
- Blueberries
- Celery
- Cucumbers
- Grapes
- Lettuce
- Nectarines
- Peaches
- Potatoes
- Spinach
- Strawberries

Plus

- Green Beans
- Kale/Greens

Pesticides residues of special concern.
Questions? foodnews.org

EWG's 2012 Clean Fifteen™

foodnews.org

Shoppers Guide to Pesticides in Produce™

Lowest in pesticides

- Asparagus
- Avocado
- Cabbage
- Cantaloupe
- Corn
- Eggplant
- Grapefruit
- Kiwi
- Mangoes
- Mushrooms
- Onions
- Pineapples
- Sweet Peas
- Sweet Potatoes
- Watermelon

Blood levels of organochlorine pesticide residues and risk of reproductive tract cancer among women from Jaipur, India.

Mathur V, **John PJ**, **Soni I**, **Bhatnagar P**.

Department of Zoology, University of Rajasthan, Jaipur, India.

Residues of organochlorine pesticides are integral part of our environment. Because of their strong lipophilic and non-biodegradable nature, organisms at higher trophic levels in the food chain tend to accumulate them. The aim of the present study was to assess the influence of organochlorine pesticides upon the occurrence of reproductive tract cancers in women from Jaipur, India. Blood samples were collected from 150 females. In that group, 100 females suffered from reproductive tract cancers like cervical, uterine, vaginal and ovarian cancers, while the rest did not suffer from cancers or any other major disease and were treated as control group. The collected blood samples were subjected to pesticide extraction and analyzed with the help of gas chromatography. The pesticides detected were benzene hexa chlororide and its isomers, dieldrin, heptachlor, dichloro diphenyl trichloro ethane and its metabolites. The data obtained indicate that the organochlorine pesticide residue levels were significantly higher in all the cancer patients as compared with the control group.

PMID: 18497062 [PubMed - in process]

Biomonitoring of organochlorines in women with benign and malignant breast disease.

Siddiqui MK, **Anand M**, **Mehrotra PK**, **Sarangi R**, **Mathur N**.

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Established risk factors for breast cancer explain breast cancer risk only partially. Organochlorines are considered to be a possible cause for hormone-dependent cancers. A hospital-based case-control study, the first from India, was conducted among 50 women undergoing surgery for breast disease to examine the association between organochlorine exposure and breast cancer risk. Blood, tumor, and surrounding adipose tissue of the breast were collected from the subjects with benign (control) and malignant breast (study) lesions and analyzed to determine organochlorine insecticides using a gas-liquid chromatograph equipped with an electron capture detector. The alpha, beta, gamma, and delta isomers of hexachlorocyclohexane (HCH), p,p'-dichlorodiphenyltrichloroethane (DDT), o,p'-DDT, p,p-dichlorodiphenyldichloroethylene, and p,p'-dichlorodiphenyldichloroethane were frequently detected in three specimens. Total HCH and total DDT levels were higher in the blood of the study group (25 cases) than in those of the controls (25 cases) with only gamma-HCH being significantly different ($P<0.05$). However, both total HCH and total DDT were higher in the tumor tissues of the controls than in those of the study group; gamma-HCH was significantly different ($P<0.05$). The level of total HCH (alpha-HCH was significantly different, $P<0.05$) was higher in the

breast adipose tissue of the study group, whereas total DDT was higher in the breast adipose tissue of the control group. The distribution of known confounders of breast cancer including age, body mass index, age at menarche and menopause, duration of breast feeding, and family history related to breast disease did not differ significantly between benign and malignant groups. This pilot study with limited statistical power does not support a positive association between exposure to organochlorines and risk of breast cancer but paves the way for a larger Indian study with greater statistical power encompassing different regions of the country to enable statistically sound conclusions.

PMID: 15820732 [PubMed - indexed for MEDLINE]

Relative abundance of organochlorine pesticides and polychlorinated biphenyls in adipose tissue and serum of women in Long Island, New York.

Stellman SD, **Djordjevic MV**, **Muscat JE**, **Gong L**, **Bernstein D**, **Citron ML**, **White A**, **Kemeny M**, **Busch E**, **Nafziger AN**.

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Some organochlorine pesticides (OCPs) and PCBs are under investigation as possible risk factors for breast cancer because of their estrogenic properties and widespread presence in the environment. It is important to know whether adipose tissue used by some investigators and serum assays used by others can provide comparable information on body burden. Concentrations of seven OCPs or their breakdown products as well as 14 PCB congeners were measured in the adipose tissue and serum of 293 women enrolled as controls in a case-control study of environmental factors for breast cancer in Long Island, New York, a high-risk region. Adipose OCP/PCB levels were measured using a supercritical fluid extraction method developed by the authors. 1,1-Dichloro-2,2-di(4-chlorophenyl)ethylene (p,p'-DDE) was detected in all adipose and serum samples; two chlordane derivatives, beta-hexachlorocyclohexane (a lindane isomer) and hexachlorobenzene, were detected in at least 92% of adipose samples. The di-ortho hexachlorinated PCB congeners 2,4,5,2',4',5'-hexachlorobiphenyl and 2,3,4,2',4',5'-hexachlorobiphenyl were detected in all adipose and over 98% of serum samples. 1,1-Dichloro-2,2-di(4-chlorophenyl)ethylene comprised 77% of total pesticide residues in adipose and 71% in serum. 2,4,5,2',4',5'-Hexachlorobiphenyl comprised 24% of adipose and 21% of serum PCBs. The relative concentration patterns of the 14 PCB congeners were similar to those reported in other human studies and were also typical of patterns reported in environmental samples from various biota, including mammals and birds, but differed substantially from patterns reported in occupationally exposed workers. All adipose-serum correlations for pesticides and most PCBs were statistically significant. Either serum or adipose OCP/PCB levels of a variety of environmental organochlorine compounds may serve as useful biomarkers of body burden.

PMID: 9641493 [PubMed - indexed for MEDLINE]

Chlororganic pesticides and polychlorinated biphenyls in breast tissue of women with benign and malignant breast disease.

Güttes S, **Failing K**, **Neumann K**, **Kleinstein J**, **Georgii S**, **Brunn H**.

Staatl. Medizinal-, Lebensmittel- und Veterinäruntersuchungsamt Mittelhessen, Marburger Str. 54, D-35396 Giessen, Germany.

Persistent chlorinated hydrocarbons assimilated through the diet may, as a result of their carcinogenic, immunotoxic, and, at least in regard to certain of these substances, estrogenic properties, play a role in the etiology of human breast cancer. As a consequence, increased concentrations of these ubiquitous environmental contaminants may be found in breast tissue of women suffering from malignant breast disease. To examine this possibility, surgically removed breast tissue samples from 65 women in Hesse, Germany were examined by capillary gas chromatography for p, p'-dichloro(diphenyl)trichloroethane (p,p'-DDT), p, p'-dichloro(diphenyl)-dichloroethane (p,p'-DDD), p, p'-dichloro(diphenyl)dichloroethene (p,p'-DDE), hexachlorobenzine (HCB), alpha-, beta-, and gamma-hexachlorocyclohexane (HCH) as well as the polychlorinated biphenyls (PCB) no. 28, 31, 49, 52, 101, 105, 118, 138, 153, 156, 170, and 180. Of the 65 patients, 45 were diagnosed with breast cancer. The control group of 20 women suffered from benign breast disease such as mastopathy. After statistical adjustment for age differences, higher concentrations of p,p'-DDT, p, p'-DDE, HCB as well as PCB-congeners no. 118, 138, 153, and 180 were detected in tissue from women with breast cancer than in tissue from control persons. These differences were weakly significant for p, p'-DDE ($p = 0.017$), for PCB 118 ($p = 0.042$) and for PCB no. 153 barely not significant ($p = 0.083$). On an average, a 62% higher concentration of p,p'-DDE was found in cancer tissue (cancer patients: 805 microg/kg fat; controls: 496 microg/kg fat) and 25% higher concentration of PCB no. 118 (81 microg/kg fat; 65 microg/kg fat). The concentrations of beta-HCH, PCB no. 156 and 170 were lower (not significant) in cancer tissue than in tissue from women with benign disease. PCB-congeners no. 105 and 149 as well as gamma-HCH could only be detected in individual tissue samples; congeners no. 28, 31, 49, 52, and 101 as well as alpha-HCH and p,p'-DDD were not detected in any of the samples. To rule out the possibility that the concentrations of chlorinated hydrocarbons measured were influenced by the surgical procedure, 20 samples of tissue that were at a distance (minimum 1 cm and maximum 3 cm) from the tumor, tissue that was in direct proximity to the tumor (no more than 5 mm from the tumor), and tumor tissue itself (center of tumor) were separately prepared and analyzed. The average concentrations of chlorinated hydrocarbons varied to differing degrees and only minimally in tumor and surrounding breast tissue, indicating that the surgical procedure did not influence the results.

PMID: 9601932 [PubMed - indexed for MEDLINE]

Are chemicals making us fat? - *Pesticide Action Network*

Source: **http://www.panna.org/blog/are-chemicals-making-us-fat**

The rate of obesity in very young children — even infants — continues to climb. Evidence is building that obesity-promoting chemicals called obesogens are contributing to this alarming trend.

Some of these obesogens are pesticides that — as the ongoing study of endocrine disruption clarifies — can act at very low doses to interfere with all kinds of physiological processes. This includes, it turns out, triggering increased fat cell production.

With children affected so early in life, it's become increasingly clear that genetic and behavioral factors alone cannot explain the rising rates of obesity. Scientists are now examining the contribution of environmental factors, researching whether chemical exposures may be promoting obesity, and if so, how.

A fungicide that promotes fat

A recent study of triflumizole (TFZ) suggests that this fungicide is a likely obesogen. In 2010, over 40,000 pounds of TFZ's active ingredient was used in over 6,000 agricultural applications in the state of California alone. Widely used on food and ornamental crops, TFZ is not particularly toxic nor carcinogenic — but it does promote fat cells.

Researchers found that TFZ treatment of human stem cell cultures activated the pathway leading to fat cell production. Stem cells have the potential to become different kinds of cells (i.e., a stem cell can become a bone cell or fat cell).

The study also examined pre-birth exposure to TFZ in the drinking water of female mice. The lowest dose produced a significant increase in stored fat, while higher doses had no effect. This type of dose response — when different levels of exposure lead to different effects — is showing up in more and more studies, leading EPA to rethink how it evaluates pesticide risk.

In addition, stem cells prepared from the fat of female mice exposed to TFZ had significant genetic changes. Expression of a genetic marker that inhibits fat cell production was reduced, while genes known to promote fat cell production had increased expression.

Evidence growing stronger

Findings from this study suggest that exposure to a widely used fungicide may promote the rate of fat cell production from stem cells, and that female offspring exposed to TFZ *in utero* may have genetic changes that lead to increased fat cell production.

And the TFZ paper is just one of many recent studies on pesticides and obesogens. While this is a fairly new field of study, evidence is accumulating that chemicals are likely contributors to the obesity epidemic.

In our recent report, *A Generation in Jeopardy*, we discussed recent epidemiological studies focusing on children's health and putative obesogens. At this point, most of the studies focus on adult and teenage obesity. The widespread public health consequences of the obesity epidemic suggest that we should pay very close attention to this rapidly emerging science.

Environ Res. 2010 Apr;110(3):251-257. Epub 2010 Feb 2.

Body burdens of brominated flame retardants and other persistent organo-halogenated compounds and their descriptors in US girls.

Windham GC, Pinney SM, Sjodin A, Lum R, Jones RS, Needham LL, Biro FM, Hiatt RA, Kushi LH.

CA Department of Public Health, DEODC, 850 Marina Bay Pkwy, Bldg. P, Richmond, CA 94804, USA.

BACKGROUND: Levels of brominated flame retardants are increasing in US populations, yet little data are available on body burdens of these and other persistent hormonally active agents (HAAs) in school-aged children. Exposures to such chemicals may affect a number of health outcomes related to development and reproductive function. OBJECTIVE: Determine the distribution of biomarkers of polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), and organo-chlorinated pesticides (OCPs), such as DDT/DDE, in children, and their variation by key descriptor variables. METHODS: Ethnically diverse cohorts of girls 6-8y old at baseline are being followed for growth and pubertal development in a multi-site, longitudinal study. Nearly 600 serum samples from the California and Ohio sites were analyzed for lipids, 35 PCB congeners, 11 PBDE congeners, and 9 OCPs. The biomarker distributions were examined and geometric means compared for selected analytes across categories of age, race, site, body mass index (BMI), parental education, maternal age at delivery, and breast feeding in adjusted models. RESULTS: Six PBDE congeners were detected among greater than 70% of samples, with BDE-47 having the highest concentration (median 42.2, range 4.9-855ng/g lipid). Girls in California had adjusted geometric mean (GM) PBDE levels significantly higher than girls in Ohio. Furthermore, Blacks had significantly higher adjusted GMs of all six PBDE congeners than Whites, and Hispanics had intermediate values. GMs tended to be lower among more obese girls, while other variables were not strongly associated. In contrast, GMs of the six PCB congeners most frequently detected were significantly lower among Blacks and Hispanics than Whites. PCBs and the three pesticides most frequently detected were also consistently lower among girls with high BMI, who were not breast-fed, whose mothers were younger, or whose care-givers (usually parents) were less educated. Girls in California had higher GMs than in Ohio for the pesticides and most PCB congeners, but the opposite for CB-99 and -118. CONCLUSIONS: Several of these potential HAAs were detected in nearly all of these young girls, some at relatively high levels, with variation by geographic location and other demographic factors that may reflect exposure pathways. The higher PBDE levels in California likely reflect differences in fire regulation and safety codes, with potential policy implications.

PMID: 20129604 [PubMed - as supplied by publisher]

science café

Flame Retardants, PCBs & Pesticides Found in Blood of Young Girls

By Jeffrey NorrisMarch 17, 2010

Banned chemicals — present in amounts higher than levels found in recent years in US adults — are turning up in the blood of young girls being studied in California and Ohio.

Robert Hiatt

Researchers from Kaiser Permanente Northern California, UCSF and the California Department of Public Health (CDPH) led the study to measure blood levels of the chemicals. They assessed levels of PCBs, DDT and related pesticides, and flame retardants in nearly 600 girls, ages six to eight. These chemicals can act like hormones. Scientists and public health experts want to know how they might affect children as they develop through adolescence and into adulthood.

The main difference found between girls in Ohio and California was that the California girls had higher levels of all three classes of chemicals in their blood, on average.

The potentially hormonally active fire retardants measured in the study are called polybrominated diphenyl ethers (PBDEs). They have been banned in Europe, but they are still used in products sold in the United States to help meet fire safety standards. Products containing PBDEs include foam upholstery, textiles and home furnishings.

In the United States, PCBs were used in transformers and industrial applications until 1979, when their use was banned. DDT and related pesticides also are banned but persistent. In addition, these pesticides are still used on crops in Mexico.

The study identified additional differences in average blood levels of chemicals among the girls. Black girls had significantly higher levels of PBDEs in their blood in comparison to white girls. In general, levels of PCBs and pesticides were significantly lower among girls born to less educated mothers. Mexican-American girls had the highest levels of pesticides in their blood. Chemical levels also tended to be lower in girls who were obese.

The research report is in press and already available online in the scientific journal *Environmental Research*. The findings are among the first to be reported from a long-term, multi-site collaboration called the Breast Cancer and the Environment Research Centers (BCERCs). The project is funded primarily by the National Cancer Institute and the National Institute of Environmental Health Sciences.

The main exposures to these chemicals occur in utero or through breast feeding, according to the study authors, but additional exposures occur during childhood.

”This study demonstrates that measurable levels of chemicals that might interfere with normal hormonal pathways are present in the blood of girls during the time of breast development,” says Robert Hiatt, MD, PhD, director for population sciences for the UCSF Helen Diller Family Comprehensive Cancer Center and lead scientist for the Bay Area BCERC.

“The next step is to determine if we can detect any evidence that these chemicals have effects on pubertal development at the levels we have detected them.”

‘Cause for Concern’

According to study leader Lawrence Kushi, ScD, associate director for epidemiology at Kaiser Permanente Northern California, these findings indicate that exposures to these chemicals are widespread. “Although we don’t know what the health implications of these exposures are, the fact that they are found in almost all of our study population suggests some cause for concern,” Kushi says.

Childhood and especially adolescence are thought to represent a window of time when girls might be especially vulnerable to environmental exposures that can influence breast cancer

risk in later years. This idea stems from lab studies of animals and population studies of women who had received radiation treatment when they were girls. Reproductive factors — including early age of first menstruation, late age of menopause and delayed childbearing — already are known to increase a woman's risk for breast cancer.

That's why cancer researchers are interested in identifying influences on reproductive development of young girls. When they entered the study, the girls had not yet begun to experience the hormone-driven changes that eventually lead to puberty and adolescence.

The procedure used in the study to prepare and analyze blood samples is the same one used by the Centers for Disease Control and Prevention (CDC), and the samples were analyzed by the same CDC laboratories. Every two years for the past decade, the CDC has conducted "biomonitoring" surveys to measure levels of chemicals in the blood and urine of people across the nation.

According to the study authors, "Our study provides data that was previously lacking on body burdens of hormonally active agents in young children from a large, racially diverse population using the most sensitive assays now available."

The study was led by Gayle Windham, PhD, chief of the Epidemiology Surveillance Unit at the Environmental Health Investigations Branch of the CDPH, along with Kushi, and Hiatt. These three were joined in the research by additional collaborators from the University of Cincinnati, Cincinnati Children's Hospital Medical Center, the US Centers for Disease Control and Prevention, and the CDPH.

Body burdens of brominated flame retardants and other persistent organo-halogenated compounds and their descriptors in US girls

Gayle C. Windham, Susan M. Pinney, Andreas Sjodin, Raymond Lum, Richard S. Jones, Larry L. Needham, Frank M. Biro, Robert A. Hiatt and Lawrence H. Kushi

Environmental Research (April 3, 2010)

Summary

Carefree Outdoor Living Without the Mosquitoes -- or the Poisons! – by Dr. Mercola

Those nasty mosquitoes can drive even the sanest person 'crazy'. And it's really not a good idea to spray a bunch of chemicals on your skin to solve the problem.

But at the same time, I realize that gazillions of mosquitoes can become so annoying that you'll find yourself willing to spray ***anything*** on!

So when daylight transitions to evening -- or anytime the bugs are bugging you -- **Dr. Mercola's BUG OFF: The Natural Anti-Insect Spray** offers just the solution you need in an all-natural formulation free of DEET.

DEET -- More Dangerous Than You Think!

DEET (N,N-diethyl-3-methylbenzamide) is a chemical that was patented by the US Army in 1946 and is still widely recognized as an effective mosquito repellent. In fact, most commercial insect repellents are made of varying concentrations of DEET.

Every year approximately one-third of the American population uses insect repellents to deter mosquitoes and other pests. Currently, DEET is used in up to 230 different products in concentrations of up to an astounding 100%.

However, all is not well with DEET...

DEET is a pesticide intended to ***kill*** insects! In case that idea by itself doesn't scare you, read on to discover the health issues it is known to cause.

If It Melts Plastics...

DEET sprays can melt plastic bags and fishing lines. Does that make you wonder what it can do to you?

Duke University Medical Center pharmacologist Mohamed Abou-Donia spent 30 years researching the effects of pesticides.

He discovered that prolonged exposure to DEET can impair cell function in parts of your brain -- demonstrated in the lab by death and behavioral changes in rats with frequent or prolonged use.[i]

When these rats had their skin treated with the average human dosage equivalent (40 mg/kg body weight) of DEET, they performed far worse than control rats on physical tests requiring muscle control, strength, and coordination.[ii]

This is consistent with reports of symptoms after military use of DEET in the Persian Gulf War.

Exposure causes neurons to die in several parts of your brainiii -- including areas that control muscle movement, memory, concentration and learning. Abou-Donia says rats given small doses of DEET for 60 days had a harder time accomplishing even the easiest tasks, things as simple as walking.

Heavy exposure to DEET and other insecticides can cause eye and skin irritation, memory loss, headaches, weakness, fatigue, muscle/joint pain, nausea, tremors and shortness of breath. Symptoms can appear months or even years after use.

Abou-Donia believes that although short-term exposure to DEET *might* not be harmful, he warns against ***ever*** using any product with more than a 30 percent concentration. To me, even that seems a dangerous and risky amount to use.

But wait! It gets worse...

Worse Than DEET

"We found that the combined exposure to DEET and other chemicals is more dangerous than just DEET alone," says Abou-Donia.

Exposure causes neurons to die in several parts of your brain[iv] -- including areas that control muscle movement, memory, concentration and learning. Abou-Donia says rats given small doses of DEET for 60 days had a harder time accomplishing even the easiest tasks, things as simple as walking.

Insecticides aren't the only problem though. **Skin care products** containing various chemicals can put you at increased risk of chemical contamination with DEET. Products you use on a daily basis, like deodorants, soaps, make-up and *sunscreens* (except **Natural Sunscreen**), when combined with DEET, create greater exposure than DEET alone.

Medications, both prescription and over the counter, can also react with DEET and increase your risk of problems.

Long-term and regular use of DEET -- especially combined with these other chemicals or medications -- can cause brain deficiencies in vulnerable groups, particularly children.[v]

Children are more susceptible than adults to subtle brain changes caused by chemicals in their environment, because their skin more readily absorbs them. Their still-developing nervous systems are potentially more affected.[vi]

Never, ever, ever use any DEET-containing product on infants! And be very hesitant to use it on anyone who you care about -- including yourself.

Many Potential Hazards Lurk in Commercial Bug Sprays

Other potential hazards can lurk in commercial bug sprays, such as the chemical permethrin. It's part the synthetic pyrethroid family, all of which are neurotoxins.

At relatively high doses, its effects are known to include tremors, loss of coordination, elevated body temperature, aggressive behavior, and learning disruption.[vii] Even at sub-lethal doses it can cause aggressive behavior, disruption of eating habits, and agitation.[viii] Lab results suggest that it is more dangerous for children than adults[ix]

The Environmental Protection Agency labeled it as a carcinogen because it causes lung tumors in female mice and liver tumors in mice of both sexes. It's also implicated in chromosome abnormalities in human and hamster cells, and hinders immune function.

But that's not all. It causes environmental damage too.

Permethrin is toxic to honeybees and other beneficial insects, fish, crayfish, and shrimp. It causes deformities in tadpoles and reduces the number of oxygen-carrying cells in the blood of birds. Unfortunately it's found in streams and rivers throughout the United States.

I'm sure you can agree that using sprays containing permethrin is not only bad for you, but bad for the environment!

Then there's S.D. alcohol, used as an anti-bacterial agent, denatured by toxic solvents such as acetone, turpentine and benzene which make it poisonous in moderate to large amounts. Ingestion may cause nausea, vomiting, impaired perception, stupor, coma and death.

And that's just for starters...

Menaces Without Names

Oh ... and have you heard about 'inert ingredients'?

Product containers tell you that it contains a certain percentage of inert ingredients. Two popular commercial insect repellent brands have ***unspecified*** inert ingredient levels of 68% and 77%.

Unfortunately, it's impossible to know for sure what 'inert' includes, since companies choose not disclose it on their labels or to the public. Care to speculate?

Turns out there's a much better alternative for you and your loved ones ... Because it really doesn't have to be a choice of either mosquitoes or poisons. You can be rid of both!

New! All-Natural Anti-Insect Alternative for Carefree Outdoor Living

Knowing that you'd be appalled to use insect sprays containing DEET and other suspect ingredients (revealed or not) -- yet that you might just get annoyed enough to consider it anyway -- I knew I had to find a better option for you.

That's why I had **Dr. Mercola's BUG OFF** formulated for your summer comfort, fun, and wellbeing.

Dr. Mercola's BUG OFF is chemical-free, has an appealing scent, and is effective against the harassment of biting insects! Plus, it's not harmful to the environment.

It's specially formulated for effectiveness against mosquitoes, fleas, chiggers, ticks, and other biting insects -- giving you back your freedom to enjoy the great outdoors.

Mosquito Defeater Special Ingredients

Each active ingredient in this special formulation of **Dr. Mercola's BUG OFF** is a known natural deterrent to bug bites, so you can feel totally confident in using it freely. You'll love how natural and gentle it is to your skin.

Citronella, lemongrass oil, and peppermint oil have been known to be effective bug deterrents for many years. Many people also consider pure vanillin to be effective against mosquitoes. But vanilla combined with citronella, lemongrass oil, and peppermint oil -- now there's a **smart combination**.

Citronella:
Oil of citronella has been used for over 50 years as an insect repellent. It protects you from insects without harming or killing them. Citronella's distinctive odor may make it difficult for pests to locate a host. Oil of citronella has been used extensively since 1948 without reports of adverse side effects or concern.

Lemongrass Oil:
Lemongrass oil is an herb widely cultivated in the tropics and subtropics, long appreciated for its ability to repel insects. It has an amazingly fresh, earthy and lemony scent. The amount of citral, the aldehyde responsible for its lemon scent, determines its quality.

If you have sensitivity to lemongrass, please do not use this product.

Peppermint Oil:
Researchers in India -- a nation with a large mosquito problem -- discovered that peppermint oil is effective as more than a flavoring or digestive aid. It also repels adult mosquitoes and kills the larvae. It has been particularly effective against the Anopheles culicifacies mosquito, which is a tyrannical menace in the northern plains of India.

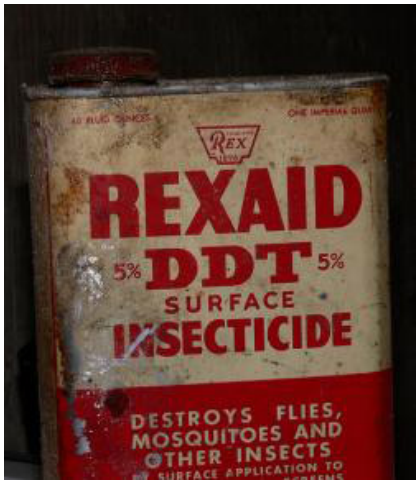
Vanillin:
Vanillin (the real deal, not imitation vanilla!) is also considered a substance that keeps bugs at bay, and is included in the **BUG OFF** formulation.

Naturally-occurring sodium benzoate and **potassium sorbate** are included as natural preservatives to prevent bacterial growth. Sodium benzoate occurs naturally in cranberries, prunes, plums, cinnamon, ripe cloves, and apples. It is this food-form of sodium benzoate that's in **BUG OFF**.

Because **Dr. Mercola's BUG OFF** doesn't contain neurotoxins or other dangerous or suspicious ingredients, you can apply generously without fear of reactions or long-term issues. *Caveat: Do not use this product if you are sensitive to lemongrass..*

DDT & Obesity: New science on harms of old pesticide

Wed, 2010-10-13 18:27
Kristin Schafer



Plenty of calcium, fruits, vegetables & exercise. No drinking, no smoking, cut down on caffeine. Oh, and avoid DDT breakdown products — they may put your soon-to-be-born baby on the road to obesity.

Researchers in Spain say they were surprised to find this link between DDT and overweight infants. Turns out when women of normal weight have higher levels of DDE (DDT's breakdown product) in their blood during pregnancy, their babies are twice as likely to grow quickly during the first 6 months of life, and 4 times as likely to be overweight when they reach the 14-month mark.

No one quite understands why — are the babies less active? eating more? — but there are plenty of animal studies suggesting an answer: some pollutants (at very low levels) interfere with normal hormone functions to slow the metabolism and cause obesity.

Janet Raloff of *Science News* describes it this way:

"… a growing body of data has been indicating that some pollutants — known colloquially as obesogens — can trigger the body to put on the pounds. In animals, these pollutants will sometimes lead a mouse to become rotund despite eating no more and exercising no less than its lean cousins."

It should be easy to avoid contact with DDT though, right? After all, it's been banned in the U.S. for nearly 40 years, and use has been pretty much phased out around the world. (In some countries, small quantities of DDT are still used for malaria control — though the World Health Organization is working hard to help put safer tools in place.)

So how is it that 99%* of us carry DDT breakdown products in our blood? The short answer: DDT and DDE hang around for decades.

Women who were exposed as children back when DDT was used may have passed the chemical on to their children during pregnancy and breastfeeding. Root crops such as carrots and potatoes can pull DDE from the soil for years after the parent chemical was applied. And these long lasting pesticides can travel the globe for years after use, stopping along the way as they travel to the polar regions, where contamination levels can be astonishing.

On top of all that, Canadian scientists are now saying that climate change may increase our exposure to legacy chemicals like DDT. As global temperatures rise, lingering pollutants are likely to be released from frozen water and soil where they've been trapped, putting them back into circulation for another round of contamination.

Since obesity is just one more in a long list of known ways DDT (and other persistent chemicals) can damage human health, this is disturbing news for us all — whether we're expecting mothers or no.

*DDT breakdown products were found in the blood of 99% of the people tested by CDC.

US Drinking Water and Watersheds Widely Contaminated by Hormone Disrupting Pesticide, Atrazine

Natural Resources Defense Council (NRDC) August 24, 2009

Analysis of Water Data Reveals Broad Contamination Ignored by EPA Monitoring

CHICAGO - August 24 - A widely used pesticide known to impact wildlife development and, potentially, human health has contaminated watersheds and drinking water throughout much of the United States, according to a new report released today by the Natural Resources Defense Council (NRDC). Banned by the European Union, atrazine is the most commonly detected pesticide in U.S. waters and is a known endocrine disruptor, which means that it affects human and animal hormones. It has been tied to poor sperm quality in humans and hermaphroditic amphibians.

"Evidence shows Atrazine contamination to be a widespread and dangerous problem that has not been communicated to the people most at risk," said Jennifer Sass, PhD, NRDC Senior Scientist and an author of the report. "U.S. EPA is ignoring some very high concentrations of this pesticide in water that people are drinking and using every day. This exposure could have a considerable impact on reproductive health. Scientific research has tied this chemical to some ghastly impacts on wildlife and raises red flags for possible human impacts."

"People living in contaminated areas need to be made aware -- and the regulators need to get this product off the market," said Sass.

The report, **"*Poisoning the Well: How the EPA is Ignoring Atrazine Contamination in Surface and Drinking Water in the Central United States*"** creates a ground breaking analysis of atrazine pollution by bringing together data from watershed monitoring and drinking water compliance programs for the first time.

The report reveals that all of the watersheds monitored by EPA and 90% of the drinking water sampled tested positive for atrazine. Contamination was most severe in Illinois, Iowa, Indiana, Missouri, and Nebraska. An extensive U.S. Geological Survey study found that approximately 75 percent of stream water and about 40 percent of all groundwater samples from agricultural areas contained atrazine, and according to the New York Times, an estimated 33 million Americans have been exposed to atrazine through their drinking water systems.

"The extent of contamination we found in the data was breathtaking and alarming," said Andrew Wetzler, Director of NRDC's Wildlife Conservation Program and Deputy Director of NRDC's Midwest Program, as well as one of the report's authors. "The EPA found atrazine almost everywhere they looked. I think that the public will find this hard to swallow and I hope it will help force the EPA to address the situation more aggressively."

Click here for the full report, including detailed maps of affected areas and Google Earth applications.

The contamination data in the report was obtained as the result of a legal settlement and Freedom of Information Act requests. "*Poisoning the Well"* highlights watersheds and municipal water treatment systems most affected by the chemical contamination, offers policy solutions,

and describes actions that people can take to protect themselves from exposure to this dangerous chemical in their water.

Atrazine is regulated by the U.S. Environmental Protection Agency (EPA). Under the Safe Drinking Water Act (SDWA), EPA has determined that an annual average of no more than 3 parts per billion (ppb) of atrazine may be present in drinking water. One of the chief findings of the report was that this reliance on a "running annual average" allows levels of atrazine in drinking water to peak at extremely high concentrations.

Given the pesticide's limited economic value and the fact that safer agricultural methods can be substituted to achieve similar results, NRDC recommends phasing out the use of atrazine, more effective atrazine monitoring, the adoption of farming techniques that can help minimize the use of atrazine to prevent it from running into waterways. The report also underscores the importance of using home filtration systems.

The effects associated with atrazine have been documented extensively. Reproductive effects have been seen in amphibians even at low levels of exposure. Concentrations as low as 0.1 ppb, for example, have been shown to alter the development of sex characteristics in male frogs, resulting in male frogs with female sex characteristics and the presence of eggs in male frog testes. Some scientists are concerned about exposure for children and pregnant women, as small doses could impact development of the brain and reproductive organs. Research has also raised concerns about atrazine's "synergistic" affects, showing potential for the chemical having a multiplier affect to increase toxic affects of other chemical co-contaminants in the environment.

The report includes information on actions people can take to protect themselves from Atrazine and other dangerous contaminants. NRDC recommends that consumers concerned about atrazine contamination in their water use a simple and economical household water filter, such as one that fits on the tap. Consumers should make sure that the filter they choose is certified by NSF International to meet American National Standards Institute (ANSI) Standard 53 for VOC (volatile organic compounds) reduction and therefore capable of significantly reducing many health-related contaminants, including atrazine and other pesticides.

Additionally, NRDC's SimpleSteps Web site includes an online form to allow people to take on a watchdog role by collecting information on how their public water systems are treating these issues. Visit **www.simplesteps.org/atrazine** for more information.

The Natural Resources Defense Council is a national, nonprofit organization of scientists, lawyers and environmental specialists dedicated to protecting public health and the environment. Founded in 1970, NRDC has 1.2 million members and online activists, served from offices in New York, Washington, Chicago, Los Angeles, San Francisco and Beijing. Natural Resources Defense Council (NRDC) Links: **Homepage**

Josh Mogerman, 312-651-7909 (office) or 773-531-5359 (mobile) or jmogerman@nrdc.org

Herbicide Poisons

Commonly used by timber companies
Source: ODF notifications

Chemical/Brand Names	Action(s)
Atrazine	
Clopyralid (Transline, Stinger, Reclaim)	Synthetic auxin—mimics natural plant hormones. Mixed with other herbicides: Confront, Curtail, Scorpion, Hornet & Accent Gold
Dicamba (Banvel, Vanquish, Trimec)	Synthetic auxin—mimics natural plant hormones
Duron (Valpar, Karmex, Durex)	
Glyphosate (RoundUp, Rodeo, Mirage)	Inhibits three amino acids and protein synthesis
Hexazinone	
Imazapyr (Arsenal, Chopper, Assult, Stalker, Habitat)	Inhibits the plant enzyme acetolactate, which prevents protein synthesis
Metsulfuron methyl (Ally, Allie, Gropper, Escort)	Sulfonylurea—inhibits acetolactate synthesis, protein synthesis inhibitor, blocks formation of amino acids.
Picloram (Grazon, Tordon)	Restricted Use Herbicide. Synthetic auxin--mimics Natural plant hormones
Sulfometuron methyl (Oust)	Sulfonylurea—inhibits actolactase synthase, a key step in Branch chain amino acid synthesis
Triclopyr (Garlon, Pathfinder, Remedy, Turflon, Release)	Synthetic auxin—mimics natural plant hormones
2,4-D (weedone, Weedar, many more)	Synthetic auxin—mimics natural plant hormones

For information about a map of Lane County that depicts the amounts and types of herbicides used, call:

Oregon Toxic Alliance
541-465-8860
1192 Lawrence St.
Eugene, OR 97401

Forestland Dwellers
P.O. Box 5954
Eugene, OR 97405
541 342-8332

Signs of Herbicide Poisoning

Source: interviews with poison survivors

Immediate

Chemical smell
Chemical taste
Red face
Itchy eyes
"Fuzzy" feel on front of tongue
Nausea/vomiting
Uncontrollable dry cough

Soon

Loss of appetite (humans and animals)
Skin rash
Burning throat
Extreme headaches
Mental fogginess: "chemo brain"
Depression
Aggression
Vaginal hemorrhaging
Vaginal bleeding in pre-pubescent and post-menopausal women
Involuntary abortion
Sudden disappearance of birds
Vegetation kill
Crop damage
Hair loss in humans and animals

Eventual

Skin plaques
Chloracne --(cysts & pustules on face)
Chemical sensitivity
Unexplained tiredness— --Chronic fatigue syndrome
GERD: gastro-esophageal reflux
Porphyria (sunlight allergy)
Gait disturbance --humans & animals-- --hind end drag
Childhood leukemia
Low sperm count, infertility
Birth defects
Attention Deficit Disorder (ADD)
Autism
Parkinson's disease
Esophageal cancer
Breast cysts
Breast cancer
Lymphoma
Brain tumors
Death

Forestland Dwellers
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Reagentless Bidirectional Lateral Flow Bioactive Paper Sensors for Detection of Pesticides in Beverage and Food Samples

S. M. Zakir Hossain, Roger E. Luckham, Meghan J. McFadden and John D. Brennan*
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Anal. Chem., 2009, 81 (21), pp 9055–9064

DOI: 10.1021/ac901714h

Publication Date (Web): September 29, 2009

A reagentless bioactive paper-based solid-phase biosensor was developed for detection of acetylcholinesterase (AChE) inhibitors, including organophosphate pesticides. The assay strip is composed of a paper support (1 × 10 cm), onto which AChE and a chromogenic substrate, indophenyl acetate (IPA), were entrapped using biocompatible sol−gel derived silica inks in two different zones (e.g., sensing and substrate zones). The assay protocol involves first introducing the sample to the sensing zone via lateral flow of a pesticide-containing solution. Following an incubation period, the opposite end of the paper support is placed into distilled deionized water (ddH_2O) to allow lateral flow in the opposite direction to move paper-bound IPA to the sensing area to initiate enzyme catalyzed hydrolysis of the substrate, causing a yellow-to-blue color change. The modified sensor is able to detect pesticides without the use of any external reagents with excellent detection limits (bendiocarb ~1 nM; carbaryl ~10 nM; paraoxon ~1 nM; malathion ~10 nM) and rapid response times (~5 min). The sensor strip showed negligible matrix effects in detection of pesticides in spiked milk and apple juice samples. Bioactive paper-based assays on pesticide residues collected from food samples showed good agreement with a conventional mass spectrometric assay method. The bioactive paper assay should, therefore, be suitable for rapid screening of trace levels of organophosphate and carbamate pesticides in environmental and food samples.

Month Of Conception Linked To Birth Defects In United States

Co-authors of this study, which was funded by the Division of Neonatalogy of the Department of Pediatrics of the IU School of Medicine, were Jordan Huskins, B.A., a fourth year I.U. School of Medicine student, and Jun Ying, Ph.D. of the University of Cincinnati.

New research suggests that birth defect rates in the United States may be highest for women conceiving in the spring and summer.

(Mar. 30, 2009) — A study published in the April 2009 issue of the medical journal Acta Pædiatrica is the first to report that birth defect rates in the United States were highest for women conceiving in the spring and summer.

The researchers also found that this period of increase risk correlated with increased levels of pesticides in surface water across the United States.

Studying all 30.1 million births which occurred in the U.S. between 1996 and 2002, the researchers found a strong association between the increased number of birth defects in children of women whose last menstrual period occurred in April, May, June or July and elevated levels of nitrates, atrazine and other pesticides in surface water during the same months. While many of these chemicals, including the herbicide atrazine which is banned in European countries but permitted in the U.S., are suspected to be harmful to the developing embryo, this is the first study to link their increased seasonal concentration in surface water with the peak in birth defects in infants conceived in the same months.

The correlation between the month of last menstrual period and higher rates of birth defects was statistically significant for half of the 22 categories of birth defects reported in a Centers for Disease Control database from 1996 to 2002 including spina bifida, cleft lip, clubfoot and Down's syndrome.

"Elevated concentrations of pesticides and other agrochemicals in surface water during April through July coincided with significantly higher risk of birth defects in live births conceived by women whose last menstrual period began in the same months. While our study didn't prove a cause and effect link, the fact that birth defects and pesticides in surface water peak during the same four months makes us suspect that the two are related," said Paul Winchester, M.D., Indiana University School of Medicine professor of clinical pediatrics, the first author of the study.

"Birth defects, which affect about 3 out of 100 newborns in the U.S., are one of the leading causes of infant death. What we are most excited about is that if our suspicions are right and pesticides are contributing to birth defect risk, we can reverse or modify the factors that are causing these lifelong and often very serious medical problems," said Dr. Winchester, a Riley Hospital for Children neonatalogist.
Birth defects are known to be associated with risk factors such as alcohol, smoking, diabetes or advanced age. However, the researchers found that even mothers who didn't report these risk factors had higher overall birth defect rates for babies conceived from April to July.

The study relies on findings by U.S. Geological Survey, the U.S. Environmental Protection Agency and other agencies on the seasonal variations in nitrates, atrazine and other pesticides in the surface water.

"These observations by Dr. Winchester are extremely important, as they raise the question for the first time regarding the potential adverse effect of these commonly used chemicals on pregnancy outcome – the health and well-being of our children," said James Lemons, M.D., Hugh McK. Landon Professor of Pediatrics at the IU School of Medicine. Dr. Lemons is director of the section of neonatal-perinatal medicine at Riley Hospital.

Co-authors of this study, which was funded by the Division of Neonatalogy of the Department of Pediatrics of the IU School of Medicine, were Jordan Huskins, B.A., a fourth year I.U. School of Medicine student, and Jun Ying, Ph.D. of the University of Cincinnati.

Journal reference:

Paul D Winchester, Jordan Huskins, Jun Ying. Agrichemicals in surface water and birth defects in the United States. Acta Paediatrica, 2009; 98 (4): 664 DOI: 10.1111/j.1651-2227.2008.01207.x

Non-Hodgkin Lymphoma Linked to Herbicides in Two New Studies

November 2008

Non-Hodgkin lymphoma (NHL) is a group of different types of malignant lymphatic diseases that share some features but not others. In two recent health surveys, herbicide exposure was highlighted as a significant NHL risk factor. A Swedish study focused on risks of exposure to various types of pesticides while a German study concentrated on occupational risk factors for a mostly rural population.

Both studies compared two groups of people who were similar with respect to age, sex, and regional residence. Using information from responses to survey questions as well as subjects' medical records, researchers compared people who had been diagnosed with non-Hodgkin lymphoma over a set period of years and others who did not have non-Hodgkin lymphoma.

Pesticide Exposure

Swedish scientists, following up on earlier studies, investigated the role that pesticides might play in the development of non-Hodgkin lymphoma. In their new study, they found that a significant risk factor was exposure to both herbicides and wood preservatives. Researchers did not find any overall increased risk from exposure to insecticides, fungicides or rodenticides.

Confirming earlier studies, phenoxy herbicides were found to be a link to increased risk for NHL. Researchers grouped data relating to 2,4-D and 2,4,5-T together to mirror pesticide use in earlier studies.

Examined separately, MCPA was found to have the highest risk factor for NHL. In the United States, MCPA is used on lawns and in agriculture.

Exposure to all other herbicides (i.e. non-phenoxy herbicides) also increased cancer risk. Of this group, glyphosate — the chemical in Roundup products — posed the greatest risk.

Researchers noted that the use of herbicides in Sweden has changed over the years. Sweden banned 2,4,5-T in the 1970s and later also banned of 2,4-D because of concerns about contamination by various dioxins. The use of MCPA and newer herbicides such as glyphosate became more prominent.

In Sweden, the incidence of non-Hodgkin lymphoma has le veled off in recent years and the researchers suggest this trend may result from the move away from 2,4,5-T and 2,4-D and other pesticides like DDT.
New protective measures may also have reduced risks.

Occupational Risk

To assess occupational factors associated with NHL, German researchers surveyed residents of mostly rural counties in northern Germany, asking about work history and exposure to 50 "agents," such as herbicides, textile dust, electromagnetic fields, and paints.

The study looked at long-term employment trends. The results showed that those who worked in agricultural occupations (including forestry and fishermen) had an elevated risk for both high malignancy and low malignancy NHL when compared to their counterparts in the study.

Researchers also found elevated risk of high malignancy NHL for people with estimated exposure to herbicides. Farmers and gardeners who worked in horticulture or tree nurseries were the main occupations categorized as likely to be exposed to herbicides.

Other occupations were also linked. Technical salesmen, manufacturers' agents and construction workers had elevated risk for both the high and low malignancy groups. Low malignancy NHL was more common among blacksmiths, toolmakers, and machine tool operators. Other occupational agents associated with both high and low malignancy NHL included diesel fuel, nitrates, organic dusts, chlorophenols and arsenic compounds. Potential exposure to arsenic compounds was largely linked to horticultural and tree nursery workers. The published study did not explain how these workers would have been exposed to arsenic, but arsenic has been used as a wood preservative and pesticide in Sweden.*

SOURCES

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Pesticide exposure as risk factor for non-Hodgkin lymphoma including histopathological subgroup analysis.
International Journal of Cancer 123:1657-1663
Abstract

Richardson, D.B., Terschuren, C., and W. Hoffmann. 2008.
Occupational risk factors for non=Hodgkin's lymphoma: a population-based case-control study in Northern Germany.
American Journal of Industrial Medicine 51:258-268.
Abstract

* Navas-Acién, A et al. 2002. Occupation, exposure to chemicals and risk of gliomas and meningiomas in Sweden. American Journal of Industrial Medicine 42(3):214-227.

Comments to the FIFRA Scientific Advisory Panel
on
FIELD VOLATILIZATION OF
CONVENTIONAL PESTICIDES

Meeting of December 1-4, 2009

EPA-HQ-OPP-2009-0687; 74 FR 47578(Sept 16, 2009)

Comments supported by:

Alaska Community Action on Toxics (Pam Miller)
Center for Environmental Health (Caroline Cox)
Earthjustice (Patti Goldman, Janette Brimmer)
Farmworker Justice (Virginia Ruiz)
Hoosier Environmental Council (Rae Schnapp)
Institute for Agriculture and Trade Policy (David Wallinga, MD)
Oregon Toxics Alliance (Dona Hippert)
Pesticide Watch Education Fund (Paul Towers)

BACKGROUND

EPA Office of Pesticide Programs (OPP) describes the goal of this meeting in its November 4th memorandum as follows:[1]

> Recently, the Agency has been exploring the development of an approach for assessing inhalation exposure resulting from one specific chemical trespass scenario, field volatilization of conventional pesticides. The following issues have been identified as key elements for this exposure scenario: (1) the use of the available air monitoring data; (2) development of a tiered approach for estimating exposure when air monitoring data are

[1] Memorandum from T. Levine to S. Matten, EPA. Transmission of background reference materials and charge to the panel for the December 1-4, 2009 session of the FIFRA SAP. November 4, 2009. EPA-HQ-OPP-2009-0687-0004

not available; (3) use of models to predict emissions of conventional pesticides; (4) the use of oral toxicity studies when inhalation toxicity studies are not available; and (5) use of inhalation studies conducted with aerosolized pesticides to assess toxicity caused by exposure to vapors.

GENERAL COMMENTS

Recommend an eventual goal to phase out or drastically reduce pesticides of high toxicity and high volatility

The SAP should recommend that OPP set a goal of developing policies and practices that will lead to an eventual phase out or drastic reduction in the amount of high toxic, volatile pesticides in use. Pesticides of particular concern include those pesticides that are highly toxic, are highly volatile, and pose specific risks to children.

The long-range transport of volatile pesticides is a particularly outrageous form of chemical trespass. The US National Park Service reported on the atmospheric deposition of semi-volatile pesticides in the seasonal snowpack of seven national parks in the Arctic and sub-Arctic northern regions. The most frequently detected pesticides still in current-use were dacthal, chlorpyrifos, endosulfan, and lindane (γ-hexachlorocyclohexane).[2]

All the soil fumigants are quite toxic and are also used at high application rates. The fumigants either are volatile chemicals that become gases at relatively low temperatures, or are chemicals that react to produce a gas (e.g., dazomet and metam sodium converting to methyl isothiocyanate or MITC). As a gas, these chemicals move from soil into air.

Recommend that Fumigant Management Plans should be public, and monitoring should be expanded

In May, 2009 OPP released amended REDs for the soil fumigants cluster.[3] In those REDs, OPP announced a requirement that all fumigant applicators prepare a written, site-specific "fumigant management plan" (FMP) that includes site information, application procedures, how buffers were determined, air monitoring procedures, posting, hazard communication, record keeping, and an emergency plan.[4] The applicator must verify in writing that the FMP is accurate before fumigating, and must provide a post-fumigation summary report if any deviations from the FMP occurred. However, OPP is not requiring that they be submitted to any enforcement agency, or made public. Instead, they only have to be kept on file for two years by the applicator and the owner/operator of the property.

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[2] Hageman KJ, SL Simonich, DH Campbell, GR Wilson, DH Landers. 2006. Atmospheric deposition of current-use and historic-use pesticides in snow at national parks in the Western United States. Environ Sci & Technol, 40(10):3174-3180

[3] Implementation of risk mitigation measures for soil fumigant pesticides. EPA website. Updated June 3, 2009. http://www.epa.gov/oppsrrd1/reregistration/soil_fumigants/

[4] Fumigant management plan and postapplication summary reports fact sheet. EPA website. http://www.epa.gov/oppsrrd1/reregistration/soil_fumigants/fmp-fs.htm

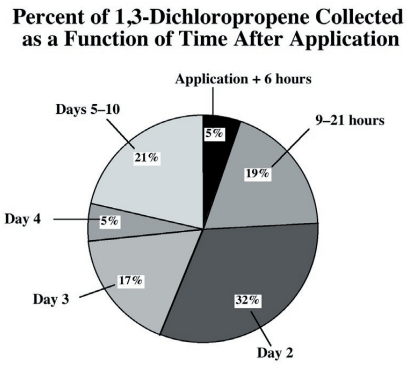
The SAP should recommend that all FMPs be filed with local and federal enforcement agencies and deposited into a publicly accessible database. This way, the public and regulatory enforcement staff can access the information by searching for the pesticide name, active ingredient, location of the application, and any post-application incidents. A published report of these data by USGS and USEPA scientists found that the percentage of total pesticide due to transport was highest for pesticides with low vapor pressures and shorter half-lives in air.

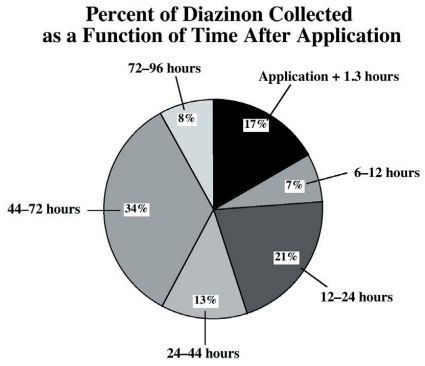
As a new label requirement, applicators of fumigants are now required to conduct some air monitoring in cases where there is an indication that concentrations are high enough for respirators to be used or to clear the area. This air monitoring is meant to protect handlers and others in the area, and may stimulate an emergency action plan. The results of this monitoring are required to be reported on the FMP, but are not required to be publicly accessible under current OPP requirements. This information should be made public in a searchable database, and the monitoring requirements should be expanded significantly. The FMPs should be public, so that this information would be available to citizens, impacted communities, regulators, and others.

SAP can recommend that upon a condition of continued registration, all registrants of the soil fumigants should provide funding for government/independent air monitoring at the periphery of application sites. The FMPs could be used to identify in advance where and when an application will take place.

Recommend that the regulatory definition of drift be expanded

The definition that OPP uses for spray drift only accounts for the drift that occurs during and shortly following a chemical application. It fails to account for the pesticide that moves off-site 8 hours or more after an application (see air monitoring Figures below, from PANNA report).[5] OPP's limited definition ignores as much as 80-90% of total drift for the volatile pesticides, and as much as 45% of most pesticides, according to a 2003 report by PANNA. [6]





Industry argues that technical improvements to application methods provide a solution to the drift problem. It is important to note that technical improvements such as those that increase droplet size or improve the angle of spray may reduce the amount of spray drift that occurs during application, but will not improve the post-application drift.

[5] Laws governing drift. PANNA website. http://www.panna.org/drift/laws

[6] Secondhand Pesticides: Airborne pesticide drift in California. Report by Pesticide Action Network North America (PANNA), 2003. http://www.panna.org/files/secondhandDriftAvail.dv.html

If spray drift is to be addressed adequately, the definition of drift used by the regulatory agencies must include post-application drift over several days after the chemical application. For example, an appropriate and scientifically-supported definition of spray drift would be "The physical movement of a pesticide through air at the time of application or within [X] days, to any site other than that intended for application (often referred to as off target)."

Recommend incorporating monitoring data into models and to truth-test regulatory assumptions

The SAP should recommend that the OPP implement or require water quality monitoring for pesticides in urban watersheds throughout the nation. Volatile pesticides may end up in waterways from off-site transport through air, rain, fog, and even snow.[7] In California, water quality monitoring conducted by local agencies has documented widespread pesticide contamination, triggering pollution prevention efforts. Unfortunately, in most regions of the country neither the EPA nor state agencies have required or provided water quality monitoring for pesticides. Hazardous pesticides are used throughout the nation by homeowners and commercial exterminators, leaving urban waterways everywhere at risk.

The SAP should recommend that OPP check their regulatory decisions against robust reliable monitoring data to determine compliance rates and the effectiveness of mitigation measures. The Agency has determined that the risks from continued use of hazardous fumigants posed to workers, consumers, and bystanders for the fumigants are acceptable; i.e. there is a reasonable certainty that no harm will result from aggregate exposure to the pesticide chemical residue provided that the risk mitigation measures are adopted and labels are amended to reflect these measures. However, OPP does not collect any information on the compliance rate with the mitigation measures, or conduct any follow up on the effectiveness of the mitigation. Without enhanced monitoring, including environmental monitoring and biomonitoring, OPP cannot know if its required mitigation measures are being complied with and are effective.

The SAP should recommend that OPP consider the methods to estimate cumulative pesticide exposure recently described by EPA experts. The Centers for Disease Control and Prevention (CDC) provides extremely useful and reliable blood and urine biomonitoring data relevant to pesticide exposures. Of most relevance to this SAP, EPA experts recently reported on an elegant approach to estimate cumulative doses, using a deterministic steady-state model and incorporating robust government data.[8] These authors reviewed the CDC biomonitoring data and reported that approximately 40% of children in the US have body burdens of organophosphate pesticides that are within 1000-fold or less of the dose that causes neurological harm in animal studies (MOE less than 1000).[9] Moreover, children had about twice as high a risk as adults, at the high end of the estimate cumulative exposure curve, with Mexican American children having a

[7] Hageman KJ, SL Simonich, DH Campbell, GR Wilson, DH Landers. 2006. Atmospheric deposition of current-use and historic-use pesticides in snow at national parks in the Western United States. Environ Sci & Technol, 40(10):3174-3180

[8] Sturges-Payne D, J Cohen, R Castorina, DA Axelrad, TJ Woodruff. Evaluating cumulative organophosphorus pesticide body burden of children: A national case study. *Environ. Sci. Technol.*, 2009, 43 (20), pp 7924–7930

[9] Sturges-Payne D, J Cohen, R Castorina, DA Axelrad, TJ Woodruff. Evaluating cumulative organophosphorus pesticide body burden of children: A national case study. *Environ. Sci. Technol.*, 2009, 43 (20), pp 7924–7930

higher risk than white children. These data provide evidence that current regulations are not protecting children as much as intended.

The Sentinel Event Notification System for Occupational Risks (SENSOR) for Pesticides is a state-based pesticide incident surveillance program administered by the National Institute for Occupational Safety and Health (NIOSH) and twelve US state health agencies (six states receive NIOSH funding, and six states are unfunded). This program provides valuable real-world monitoring data that could be used by OPP to validate and truth-test its model assumptions and model output. EPA co-funds this program.[10] The website for this program estimates that "10,000-20,000 physician-diagnosed pesticide poisonings occur each year among the approximately 2 million U.S. agricultural workers. Agricultural workers, groundskeepers, pet groomers, fumigators, and a variety of other occupations are at risk for exposure to pesticides including fungicides, herbicides, insecticides, rodenticides, and sanitizers."[11] These poisoning events are either a result of label violations (off-label uses), or a result of legal pesticide use (label compliance). In either case, it suggests that OPP needs to adjust its assumptions to better protect workers.

The USGS National Water Quality Assessment Program (NAWQA) tests for pesticides, volatile organic compounds, metals, and other environmental contaminants in US waterways. This program provides valuable real-world monitoring data that could be used by OPP to validate and truth-test its model assumptions and model output. Findings from their most recent report show that "at least one pesticide was detected in water from all streams studied and… pesticide compounds were detected throughout most of the year in water from streams with agricultural (97 percent of the time), urban (97 percent), or mixed-land-use watersheds (94 percent)." [12] These contaminations are either a result of label violations (off-label uses), or a result of legal pesticide use (label compliance). In either case, it suggests that OPP needs to adjust its assumptions to better protect our nation's waterways and the wildlife that depends on those waterways.

Thank you for the opportunity to provide comments.

Respectfully,

Jennifer Sass, Ph.D.
Senior Scientist, Natural Resources Defense Council

[10] SENSOR-pesticides program. http://www.cdc.gov/niosh/topics/pesticides/overview.html

[11] NIOSH Pesticide Illness and Injury Surveillance. http://www.cdc.gov/niosh/topics/pesticides/

[12] The Quality of Our Nation's Waters--Pesticides in the Nation's Streams and Ground Water, 1992-2001 (USGS Circular 1291) (Released March 2006). http://water.usgs.gov/nawqa/pnsp/

Organic Pesticides Not Always Best Choice

Posted By Dr. Mercola | July 17 2010 | 22,392 views

Consumers cannot assume that a product is environmentally friendly just because it is organic. A new study shows that some organic pesticides can have a higher environmental impact than conventional pesticides.

Researchers compared the effectiveness and environmental impact of organic pesticides to those of conventional and reduced-risk synthetic products on soybean crops.

An organic mineral oil-based product was among the less effective ones, as it killed ladybugs and flower bugs, which are important regulators of aphid population and growth.

According to *Science Daily*:

"These predator insects reduce environmental impact because they naturally protect the crop, reducing the amount of pesticides that are needed."

Sources:

- Science Daily June 23, 2010
- PLoS ONE June 22, 2010; 5(6): e11250.

Dr. Mercola's Comments:

Coming up with safe, effective alternatives to the toxic chemical pesticides sprayed on the majority of U.S. food crops is extremely important, but simply swapping out synthetic pesticides with organic versions may not be the best solution.

The problem that Canadian researchers stumbled upon in their new study was that the natural mineral-oil based pesticide, which works by smothering pests like aphids, also smothered beneficial insects like ladybugs and flower bugs.

Because these latter two bugs are naturally useful in controlling aphid populations, the researchers ruled the organic mineral oil pesticide to be even more harmful, environmentally speaking, than the synthetic varieties.

I certainly don't agree with this conclusion, as synthetic pesticides are notoriously hazardous not only for human health but also to the environment, but it does highlight the increasing need for integrated pest management programs that use a variety of natural, non-toxic methods to keep pests at bay.

Synthetic Pesticides are a Disaster for Your Health and the Environment

The Environmental Protection Agency (EPA) considers 60 percent of herbicides, 90 percent of fungicides, and 30 percent of insecticides to be carcinogenic, and most are also damaging to your nervous system as well. In fact, these powerful, dangerous chemicals have been linked to numerous health problems such as:

- Neurotoxicity
- Disruption of your endocrine system
- Carcinogenicity
- Immune system suppression
- Male infertility and reduced reproductive function
- Miscarriages
- Parkinson's disease

Ironically, studies have shown that often less than 0.1 percent of an applied pesticide reaches the target pest, leaving 99.9 percent as an unintended pollutant in the environment.

Thousands of pounds of these poisons then find their way, intentionally or unintentionally, into your food and water supplies on an annual basis. It's well known that conventionally grown fruits and vegetables are often tainted with unacceptable levels of pesticide residues, but you're also exposed when you eat animal products.

Factory-farmed animals eat feed full of pesticides, and these toxins accumulate in their flesh and fat over the course of their lifetimes. When you eat factory-farmed meat, you then ingest these accumulated pesticides.

Pesticides also linger in the environment, where they can cause even more damage over time. For some "high-persistence" pesticides, the half-life is greater than 100 days in soil. The more difficult a pesticide is to break down, the more damage it can cause to the environment and living beings, because it is more susceptible to soil runoff and evaporation into the air.

In addition, measurable amounts can move through the atmosphere and accumulate in more distant locations, including in waterways, fish and vegetation.

Because you and your family are at the top of the food chain, you are all exposed to these high levels of toxins whenever you consume fish or other animals that have bioaccumulated pesticides or other organic chemicals in their bodies.

You're also exposed if you live near agricultural fields or drink water that's contaminated with pesticide run-off. Your children may even be exposed at school, as many routinely use pesticides in the building and on the grounds.

Even "Inert" Pesticide Ingredients May be Toxic

Most pesticides contain unregistered and untested "inert ingredients." These so-called inert substances can be more dangerous (or can contaminate an area longer) than the active or "registered" poisons in the pesticide formula itself, but they're not required to be listed on the label.

According to BeyondPesticides.org:

"In general, inert ingredients are minimally tested, however, many are known to state, federal and international agencies to be hazardous to human health. For example, the U.S. government lists creosols as a "Hazardous Waste" under Superfund regulations, yet allows these chemicals to be listed as inert ingredients in pesticide products.

Creosols are known to produce skin and eye irritations, burns, inflammation, blindness, pneumonia, pancreatitis, central nervous system depression and kidney failure.

Some inert ingredients are even more toxic than the active ingredients. One of the most hazardous ingredients in the commonly used herbicide RoundUp® is a surfactant, which is classified as an inert, and therefore not listed on the label. The pesticide naphthalene is an inert ingredient in some products and listed as an active ingredient in others."

It should be noted, too, that even with their harsh, synthetic chemicals, pesticides are not 100 percent effective. Over time, pesticides can become ineffective because pests develop resistance to them. Most farmers and other growers became familiar with pesticide resistance in the 1950s, as a result of widespread insect resistance to DDT.

Since then, growers have come to expect the eventual loss of pesticide effectiveness because of resistance. By the mid-1980s, there were records of about 450 resistant species of insects and mites.

When pests do become resistant, more virulent and dangerous pesticides are rolled out to address the resistance, causing greater human and environmental damage. It is estimated that the cost of catering to pest resistance costs the government at least $1.5 billion annually.

Safer, Integrated Pest Management Practices are Available

The risks of dousing virtually all U.S. food crops in toxic pesticides are steep … especially when there are far less toxic options available.

At the forefront of this area is Integrated Pest Management (IPM), a program of prevention, monitoring and control that can eliminate or drastically reduce the need for synthetic pesticides.

What makes IPM different is that there is no one program. Rather, it works by utilizing a variety of methods, including sanitation, structural repairs, mechanical and living biological controls and other non-chemical methods to control pests. Prevention is also key, and preventive measures are used as a primary means of pest control in IPM.

If there's still a problem after the non-chemical options have been exhausted, then only the least toxic pesticides, such as boric acid, diatomaceous earth, or those made with essential oils, are used.

Better still, pesticides that meet any of the below criteria are *banned* from use in natural IPM programs, according to BeyondPesticides.org:

- Determined by EPA to be a possible, probable, or known carcinogen, mutagen, teratogen, reproductive toxin, developmental neurotoxin, endocrine disruptor, or immune system toxin
- A pesticide in EPA's toxicity category I or II
- Any application of the pesticide using a broadcast spray, dust, tenting, fogging, or baseboard spray application

There are some chemical-based programs out there that are IPM "imposters," but at its roots, this is a system that prides itself on finding non-toxic methods of pest control that are safe and effective for you, your family and the environment, for the long run.

Simple Options for Avoiding Pesticides at Home and in Your Food

Despite all the influx of pesticides into your environment, there are ways you can protect yourself from exposure, and minimize future exposure. One of the best strategies if you live in a geographical area that uses a great deal of pesticides, is to move to a more protected area.

Next, seek out organically grown food as much as possible. If you have to choose, pick animal products first, as these will have a greater pesticide load if purchased non-organic, and then try to buy organic versions of these most highly contaminated fruits and veggies.

Also, unless you have an Artisan well or well water that has been tested so you know it is safe and clean, then the water you use for showering, bathing, washing dishes, cooking, and drinking is likely to be contaminated with pesticides, herbicides, and other toxins.

I recommend you use a Reverse Osmosis water system, or at very least, a good charcoal filter, to keep pesticides out of your water supply.

Finally, do not use synthetic pesticides in your home or garden, or in the form of insect repellant, lice shampoo, pet sprays or otherwise. There are safe and effective natural alternatives for virtually every pest problem you come across.

For instance, boric acid powder is a very effective deterrent to roaches and ants. Sprinkle some in the inner corners of your cabinets and in the corners under your cabinets. Pests will carry it back to their nests on their feet and kill the remainder of the infestation. Boric acid is non-toxic for animals and only kills the insects.

Or, for a homemade garden spray that will discourage most pests, use some mashed garlic paste combined with a little cayenne pepper or horseradish. Add a small amount to a gallon jug of water and let it sit for a day or two, shaking it occasionally. Just spray a small amount onto a few leaves first to make sure it's not so strong that it will burn them.

For more details on these types of natural solutions to pests of all kinds, I recommend the book Dead Snails Leave No Trails by Nancarrow and Taylor, or visit the website BeyondPesticides.org. They have a section on do-it-yourself natural solutions to a wide range of pest problems along with a resource to find pest management companies that use non-toxic products.

Out for the count: Why levels of sperm in men are falling

Submitted by Drew Kaplan on May 18, 2010

Source: http://www.independent.co.uk/news/science/out-for-the-count-why-levels-of-sperm-in-men-are-falling-1954149.html

If scientists from Mars were to study the human male's reproductive system they would probably conclude that he is destined for rapid extinction. Compared to other mammals, humans produce relatively low numbers of viable sperm – sperm capable of making that long competitive swim to penetrate an unfertilised egg. As many as one in five healthy young men between the ages of 18 and 25 produce abnormal sperm counts. Even the sperm they do produce is often of poor quality. In fact only between 5 and 15 per cent of their sperm is, on average, good enough to be classed as "normal" under strict World Health Organisation rules – and these are young, healthy men. By contrast, more than 90 per cent of the sperm of a domestic bull or ram, or even laboratory rat, are normal.

Human males also suffer a disproportionately high incidence of reproductive problems, from congenital defects and undescended testes to cancer and impotency. As these also affect fertility, it's a minor miracle men are able to sire any children at all. In fact, an increasing number of men are finding themselves childless. Among the one in seven couples now classed as infertile, the "male factor" has been found to be the most commonly identified cause.
Next year marks the 20th anniversary of the WHO conference where a Danish scientist first alerted the world to the fact that Western men are suffering an infertility crisis. Professor Niels Skakkebaek of the University of Copenhagen presented data indicating sperm counts had fallen by about a half over the past 50 years. Sperm counts in the 1940s were typically well above 100m sperm cells per millilitre, but Professor Skakkebaek found they have dropped to an average of about 60m per ml. Other studies found that between 15 and 20 per cent of young men now find themselves with sperm counts of less than 20m per ml, which is technically defined as abnormal. In contrast, a dairy bull has a viable sperm count in the billions.

Experts in human reproductive biology were astonished by the Danish study. The declining trend seemed to indicate that men were on a path to becoming completely infertile within a few generations (although recent studies suggest the fall in sperm counts may have bottomed out). Professor Skakkebaek could offer no explanation for the trend other than to suggest that the fall may have something to do with the equally alarming rise in other reproductive disorders, such as cancer of the testes and cryptorchidism, the incomplete descent of the testes into the scrotum.

Experts began to talk of a new phenomenon affecting the human male, a collection of disorders known as testicular dysgenesis syndrome. They wanted to know what was causing it, because the changes were occurring too quickly to be a result of genetics. It must have something to with changing lifestyles or the environment of men, and almost everything was suggested, from exposure to chemical pollutants to the modern fashion for tight underpants. There is now an emerging consensus among some experts that whatever it is that is exacerbating the problems of

male infertility, it probably starts in the womb. It is not the lifestyle of men that is problem, but that of their mothers.

The process of sperm production, called spermatogenesis, starts in adolescence, but the groundwork is laid down in the few months before and immediately after birth. An increasing number of studies point to a crucial "window" of testicular development that begins in the growing foetus and ends in the first six months of life. Interfere with this critical developmental period, and a baby boy will suffer the lifetime consequences of being a suboptimally fertile man.

So are we anywhere nearer to finding an explanation for why are so many more men today are suffering from reproductive problems?

"It's most likely a reflection of the fact that many environmental and lifestyle changes over the past 50 years are inherently detrimental to sperm production," says Professor Richard Sharpe, fertility research expert at the Medical Research Council. "It may be that different factors come together to have a combined effect." A number of studies point to a connection between early development in the womb and male reproductive problems in later life, especially low sperm counts. For example, men whose pregnant mothers were exposed to high levels of toxic dioxins as a result of the 1976 industrial accident in Seveso, Italy have been found to have lower-than-average sperm counts. But men exposed to dioxins in adulthood showed no such effect. Another study found women who ate large amounts of beef during pregnancy, a diet rich in potentially damaging chemicals called polycyclic aromatic hydrocarbons (PAHs), had sons with relatively low sperm counts. But eating beef as an adult man shows no similar impact.

Meanwhile, studies of migrants between Sweden and Finland, showed that a man's lifetime risk of testicular cancer tends to follow the country he was born in rather than the country where he was brought up. It was his mother's environment when she was pregnant with him, rather than his own as a boy or as an adolescent, that seems to have largely determined a man's risk of testicular cancer.

One of the strongest pieces of evidence in support of this idea comes from studies of people who smoke. A man who smokes typically reduces his sperm count by a modest 15 per cent or so, which is probably reversible if he quits. However, a man whose mother smoked during pregnancy has a fairly dramatic decrease in sperm counts of up to 40 per cent – which also tends to be irreversible.

Professor Sharpe said such findings can be explained by understanding how the first cells of the testes form. Sertoli cells, which in the adult act as guardians for the development of sperm cells, are the very first cells to form from a "genital ridge" of the human male foetus. The number of sperm that can be produced in an adult man is critically dependent on the number of Sertoli cells that develop in his foetus, so anything that interferes with the formation of Sertoli cells in a mother's womb will affect sperm production many years later. "Maternal-lifestyle factors in pregnancy can have quite substantial effects on sperm counts in sons in adulthood, and the most logical mechanism by which this could occur is via reducing the number of Sertoli cells," Professor Sharpe says.

But the key question now is to identify the relevant lifestyle and environmental factors.

This is proving tricky. Obesity, for instance, is a growing problem and it has been linked with reproductive problems in both men and women. One study has also indicated that overweight pregnant women tend to produce sons with poor semen quality. But is it being fat that is the cause, or the environmental chemicals stored in fat?

There has been a lot of interest in chemicals in the environment, especially those that can either mimic female sex hormones – oestrogenic chemicals – or block male sex hormones, specifically testosterone which plays a critical role in stimulating the development of Sertoli cells in the womb. So far, the Seveso study provides the clearest link between human foetal development, low sperm counts and prenatal exposure to an environmental chemical. But the dioxin concentrations from this industrial accident were exceptionally high.

It is more difficult trying to establish a similar, significant link between male reproductive problems and exposure to low concentrations of the many other environmental chemicals that may have weak oestrogenic or androgen-blocking properties, including substances as wide-ranging as pesticides, traffic fumes, plastics and even soya beans. Professor Sharpe says that much of the evidence to date is weak or non-existent.

“Public concern about the adverse effects of environmental chemicals on spermatogenesis in adult men are, in general, not supported by the available data for humans. Where adverse effects of environmental chemicals have been shown, they are usually in an occupational setting rather than applying to the general population,” he says.

So although scientists are closing in on the critical window of foetal development in the womb that determines a man’s fertility status in later life, they are still not sure about what it is that could be affecting this change in his reproductive status. But one thing is clear, it is his mother who almost certainly holds the key.

Pesticides Linked to Allergic Asthma in Farm Women

By Northwest Coalition for Alternatives to Pesticides (NCAP) January 2009

Link: http://www.pesticide.org/the-buzz/pesticides-linked-to-allergic-asthma-in-farm-women/?searchterm=Pesticides%20Linked%20to%20Allergic%20Asthma%20in%20Farm%20Women

A recent study of adult-onset asthma in farm women shows that allergic asthma may be linked to using pesticides. Many studies have looked at respiratory hazards of farming activities for men, but there is little research on respiratory risks to farm women.

Using information from a survey of more than 25,000 farm women, researchers focused on the 702 who were diagnosed with asthma as adults. Of these, 60 percent had non-allergic asthma and 40 percent had allergic asthma. (Allergic asthma is triggered by inhalation of allergens such as pet dander, pollen, mold or an occupational exposure such as latex.)

The farm women in this study who grew up on a farm were less likely to develop allergic asthma as adults. In fact, growing up on a farm had a strong protective effect against allergic asthma and a smaller protective effect against non-allergic asthma.

Sixty-one percent of all farm women in the study grew up on farms while other women experienced farm life only after marrying farmers as adults. The study showed that the women least likely to develop allergic asthma were women raised on farms who had never used pesticides; they had a lower risk than women who had never used pesticides and did not grow up on a farm. But this study showed that despite the protective effect of growing up on a farm, using pesticides, particularly insecticides, increased the risk of allergic asthma.

Among all the farm women in the study, 57 percent reported using pesticides at some point in their lives . While women who used pesticides three or more times were more likely to have allergic asthma than women who never applied pesticides, evidence did not show an increased risk in asthma for those who used pesticides for longer periods of time or more frequently than other women.

Information on the number and type of pesticides applied, in addition to the frequency of use, allowed for a more detailed analysis of risks. Among the pesticides that women reported using, 10 were linked

to allergic asthma, including two herbicides, one fungicide, and seven insecticides. Among specific classes of insecticides, organophosphates posed the highest risk. Some of these chemicals are used only in agriculture, but others are also commonly used around homes. For example, the two herbicides linked to allergic asthma were glyphosate, which is found in commonly used Roundup, and 2,4-D, a common weed and feed ingredient.

Two of the insecticides associated with asthma risk, malathion and carbaryl (Sevin), are found in garden bug killers. Another insecticide, permethrin, has residential uses both indoors and outdoors. In this study, using permethrin on animals was associated with the risk of allergic asthma, while using it on crops was linked to non-allergic asthma.

References

Hoppin, JA et al. 2008.
Pesticides and atopic and nonatopic asthma among farm women in the Agricultural Health Study.
American Journal of Respiratory and Critical Care Medicine 177:11-18
NOTE: atopic asthma = allergic asthma and nonatopic asthma = nonallergic asthma

American Academy of Allergy Asthma & Immunology:
* Topic of the Month - March - Is your asthma allergic? (2008)
* Tips to Remember: Occupational asthma (2007)

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Pesticides Related to Vitamin D Levels

A recently published study indicates that low-dose organochlorine pesticide exposure is associated with low serum concentration of Vitamin D in humans. Previous animal and field studies suggest that chemicals such as organochlorine pesticides that are lipophilic (lipid-loving) and deposit in the fat tissue may influence levels of Vitamin D, which is also fat-soluble.

The subjects included 1,275 adults age 20 years or older. The researchers evaluated serum concentrations of 25-hydroxyvitamin D and seven organochlorine pesticides detectable in 80 or more of the participants.

Three of the seven organochlorine pesticides showed an inverse association with serum concentrations of vitamin D, meaning that as the levels of these pesticides increased, the level of serum vitamin decreased. Adjusting the data for age, race and various health conditions, p,p'-DDT (dichlorodiphenyltrichloroethane) showed a consistent inverse association in all subgroups, but showed a stronger association among subjects with old age, white race or chronic health conditions.

The researchers concluded that the background exposure to some organochlorine pesticides may lead to vitamin D deficiency in humans.

Reference:

Yang JH, Lee YM, Bae SG, Jacobs Dr. Jr, Lee DH. Assocoations between Organochlorine pesticides and Vitamin D Deficiency in the U.S. Population. PloS One. 2012;7(1):e30093. Published Online Ahead of Print.

What's on my food?

Link: http://www.whatsonmyfood.org/

Pesticides :: A Public Problem

Pesticides
…on our food, even after washing;
…in our bodies, for years;
…& in our environment, traveling many miles on wind, water and dust.

What's On My Food? is a searchable database designed to make the public problem of pesticide exposure visible and more understandable.

How does this tool work? We link pesticide food residue data with the toxicology for each chemical, making this information easily searchable for the first time.pesticides are a public health problem requiring public engagement to solve.

Use the tool, share it with others: we built it to help move the public conversation about pesticides into an arena where you don't have to be an expert to participate.

At Pesticide Action Network (PAN), we believe that pesticides are a public health problem requiring public engagement to solve. We want you to have the information you need to take action based on a solid understanding of the issues. *What's On My Food?* builds on PAN's 28-year tradition of making pesticide science accessible.

OTHER NAMES FOR PARABENS

Look at how the chemical industry confuses the public by calling parabens all these different names.

Don't you think is about time you became chemical free and used 100% Synthetic Chemical FREE products.

You can do this by choosing to use CERTIFIED ORGANIC skincare made to FOOD GRADE standards.

For complete safety use organic cosmetics free of ALL toxic synthetic chemicals

COMMON NAME	THE SYNONYMS FOR PARABEN
Benzylparaben	Benzoic acid, 4-hydroxy-, phenylmethyl ester 4-Hydroxybenzoate de benzyle benzyl 4-hydroxybenzoate Benzyl-4-hydroxybenzoat 4-hidroxibenzoato de bencilo Benzyl p-hydroxybenzoate benzoate, 4-hydroxy-, benzyl 4-(Benzyloxycarbonyl)phenol 4-Hydroxybenzoic acid benzyl ester Benzoic acid, p-hydroxy-, benzyl ester p-Hydroxybenzoic acid benzyl ester
isobutylparaben	Benzoic acid, 4-hydroxy-, 2-methylpropyl ester 4-Hydroxybenzoate d'isobutyle isobutyl 4-hydroxybenzoate Isobutyl-4-hydroxybenzoat 2-Methylpropyl p-hydroxybenzoate Benzoic acid, p-hydroxy-, isobutyl ester Iso-Butyl p-hydroxybenzoate Isobutyl p-hydroxybenzoate p-Hydroxybenzoic acid isobutyl ester
Butylparaben	Benzoic acid, 4-hydroxy-, butyl ester 4-Hydroxybenzoate de butyle butyl 4-hydroxybenzoate Butyl-4-hydroxybenzoat 4-hidroxibenzoato de butilo 4-Hydroxybenzoic acid butyl ester 4-hydroxybenzoesaeure-butylester benzoate, 4-hydroxy-, butyl p-oxybutylbenzoate 4-(Butoxycarbonyl)phenol Aseptoform Butyl Benzoic acid, p-hydroxy-, butyl ester Butyl p-hydroxybenzoate n-Butyl 4-hydroxybenzoate n-Butyl p-hydroxybenzoate n-Butylparabenp-Hydroxybenzoic acid butyl ester

n-Propylparaben Benzoic acid, 4-hydroxy-, propyl ester
4-Hydroxybenzoate de propyle propyl 4-hydroxybenzoate Propyl-4-hydroxybenzoat
4-hidroxibenzoato de propilo
4-Hydroxybenzoic acid propyl ester
4-hydroxybenzoesaeure-propylester
4-hydroxybenzoic acid propylester
propyl p-hydroxybenzoate
propyl paraben
benzoate, 4-hydroxy-, propyl

Benzoic acid, p-hydroxy-, propyl ester
n-Propyl 4-hydroxybenzoate
p-Hydroxybenzoic acid propyl ester
p-Hydroxybenzoic acid, propyl ester
p-Hydroxybenzoic propyl ester

Ethylparaben Benzoic acid, 4-hydroxy-, ethyl ester
4-Hydroxybenzoate d'ethyle ethyl 4-hydroxybenzoate
Ethyl-4-hydroxybenzoat
4-hidroxibenzoato de etilo
4-hydroxybenzoesaeure-aethylester
benzoate, 4-hydroxy-, ethyl
ethylparaben
ethyl parasept
4-(Ethoxycarbonyl)phenol
4-Carbethoxyphenol
4-Hydroxybenzoic acid ethyl ester
Benzoic acid, p-hydroxy-, ethyl ester
Ethyl p-hydroxybenzoate
p-(Ethoxycarbonyl)phenol
p-Carbethoxyphenol
p-Hydroxybenzoate ethyl ester
p-Hydroxybenzoic acid ethyl ester

Methylparaben Benzoic acid, 4-hydroxy-, methyl ester
4-Hydroxybenzoate de methyle
methyl 4-hydroxybenzoate
Methyl-4-hydroxybenzoat
4-Hidroxibenzoato de metilo
4-Hydroxybenzoic acid methyl ester
4-hydroxybenzoesaeure-methylester benzoate
4-hydroxy-, methyl methyl p-hydroxybenzoate
p-hydroxybenzoic acid
methyl ester
methyl paraben
methyl ester of p-hydroxy benzoic acid
4-(Carbomethoxy)phenol
4-(Methoxycarbonyl)phenol
Benzoic acid, p-hydroxy-, methyl ester
Methylben
Methylparaben
p-Carbomethoxyphenol
p-Methoxycarbonylphenol

Parabens: Dangers and Uses

by Ingrid Schuetz, MA

From the Townsend Letter
November 2007

Parabens have been used as preservatives since the 1920s. Chemically, parabens have a simple structure. They consist of a six-member carbon ring with a hydroxyl group on one side (-OH) of the ring and a side chain called an alkyl ester on the opposite side of the ring. The side chains can be of varying lengths. One of the most widely quoted sources of information on the use of, exposure to, and safety of parabens was published in 1984 in a report authored by Elder.1 This report estimated that parabens were used in over 13,200 different cosmetic products. Parabens are colorless and odorless. They also have activity against a wide range of bacteria. They are less active against fungi and, therefore, are usually combined with other biocides such as formaldehyde releasers, isothiazolinones, or phenoxyethanol to provide a broader antiseptic action.

Products Commonly Containing Paraben Preservatives

Cosmetics

Foundations, powders, concealers, eye makeup (liners, shadows, mascara), facial makeup (blushes), bronzes, makeup removers, lipstick, quick-dry nail products

Pharmaceutical Products

Topical dermatological medications, eye, ear and nose drops, rectal and vaginal medications, bandages, parenteral products, including antibiotics, corticosteroids, local anesthetics, radiopharmaceuticals, vitamins, antihypertensives, diuretics, insulin, heparin, and chemotherapeutic agents

Personal Care Products

Moisturizing lotions and creams, dentifrices, sunscreens, cleansers and other skin care products, antiperspirants and deodorants, soaps, including liquid hand soap and toothpastes, shampoos and conditioners, colognes, and perfumes

Food Products (E210-219)

Marinated fish products, salad dressings, mayonnaise, mustard, spiced sauces, processed vegetables, frozen dairy products, jams and jellies, soft drinks and fruit juices, baked goods, and candies

Industrial Products

Parabens are used industrially in oils, fats, shoe polishes, textiles, and glues.

The Dangers of Parabens

Studies demonstrate the health risks of parabens. Some scientists have raised concerns

that further assessment of parabens may be needed. This is based on recent evidence from scientific studies indicating that several types of parabens can bind to the estrogen receptor and can cause estrogen-like responses when tested in laboratory animals or in a variety of tissue culture assays.2 Parabens produced a positive uterotrophic response in vivo and also damaged the late stages of spermatogenesis, altered proportion of pups born alive, and affected body weight of offspring. They reduced the number of sperm in the epididymis and reduced the sperm motile activity in male offspring. Parabens could compete with [3H] 17beta-estradiol for binding to the estrogen receptor. The proliferation of two estrogen-dependent cell lines MCF-7 and ZR-75-1 could be increased by parabens. They also increased expression of both transfected and endogenous estrogen-regulated genes in MCF-7 cells. The studies showed parabens were weakly estrogenic.3

The following studies of estrogenic activity/antispermatogenic potential of parabens indicated a possible relationship between paraben exposure and breast cancer and/or male reproductive function: Dr. S. Oishi of the Department of Toxicology, Tokyo Metropolitan Research Laboratory of Public Health, Japan reported that exposure of postweaning rats and mice to butylparaben or propylparaben (but not methylparaben or ethylparaben) adversely affected the secretion of testosterone and the function of the male reproductive system.4 British researcher Dr. Philippa Darbre and colleagues at the University of Reading5 proposed that parabens may contribute to the increasing incidence of breast cancer. Darbre et al. carried out tests on 20 samples of human breast tissue taken from patients undergoing surgery at the Edinburgh Breast Unit in Scotland, UK. The study by P. Darbre and colleagues was conducted to assess whether any of the six parabens commonly used in consumer products in Europe could be detected in human breast tumors. The parabens studied were methylparaben, ethylparaben, propylparaben, isobutylparaben, butylparaben, and benzylparaben.

The Scottish study is the first report of the detection of parabens in human breast tumors. It enabled identification and measurement of mean concentrations of individual parabens in samples of 20 human breast tumors. Comparison of individual parabens showed that methylparaben was present at the highest level (with a mean value of 12.8 +/- 2.2 ng x g [-1] tissue) and represents 62% of the total parabens recovered in the extractions. This investigation did demonstrate that five of the six parabens widely used in consumer products could be detected intact (not changed or metabolized) in human tissues. The study did not, however, make any attempt to find out the source of the parabens. It is not known if the major exposure was due to the parabens from food or via topical application of a certain type or a variety of personal care products. Since parabens can be measured intact in the human breast and possess oestrogenic properties, it has been suggested that they could contribute to an aberrant burden of oestrogen signalling in the human breast and so play a role in the rising incidence of breast cancer.

In the sixteenth century, Paracelsus said, "It is the dose that makes the poison." However, over a lifetime, with daily use of products containing parabens, we don't actually know what the cumulative dose really is. Future work will need to address the extent to which parabens can accumulate in hormonally sensitive tissues and also the extent to which their weak oestrogenic activity can add to the more general environmental oestrogen

problem.

Allergenicity of Parabens

Type IV Delayed-Type Hypersensitivity Reactions to Topical and Ingested Parabens in Orally Administered Products

Numerous individual reports describing cases of contact dermatitis as a result of contact with parabens in topical products – such as reactions to facial cosmetic products and formulations, gel-like toy products, ultrasound gel, topical creams, etc. – have been published. Occupational cases of paraben contact dermatitis (among cooks and food handlers with hand dermatitis caused by paraben-containing foods) have also been reported. Allergic contact dermatitis has most commonly been described when paraben-containing products are used on damaged skin. In a phenomenon known as the "paraben paradox" by Fisher,6 inflamed skin reacts to parabens whereas intact skin does not. This concept is important, because patch tests may produce false-negative findings in patients who are truly sensitive to parabens. There are a few reports of a systemic allergic contact dermatitis presenting as a generalized eczematous eruption after ingestion of paraben-containing medications or foods.

Type I Immediate Hypersensitivity Reactions to Topical and Parenteral Parabens

The same paraben compounds that cause delayed-type reactions can also cause type I immediate reactions such as contact urticaria.7 Several cases of immediate hypersensitivity reactions (including bronchospasm, pruritus, localized angioedema, and generalized dermatitis) to parenterally administered compounds containing parabens have been reported.1

Type IV Hypersensitivity Reactions to Parenteral Parabens

Fine and Dingman reported one case of generalized eczematous dermatitis following suction-assisted lipectomy when a local anesthetic containing methylparaben was used.8

Patch Testing with Paraben Mix

Two or more paraben esters are often found in a single product, so it is useful to test paraben sensitivity with paraben mix, as there is a high incidence of cross-reactions between the esters. Paraben mix is a mixture of five different paraben esters: methyl-, ethyl-, propyl-, butyl-, and benzyl-parahydroxybenzoic acids. Paraben-mix sensitivity produces classic allergic contact dermatitis reactions. Sometimes, the reactions may be seen as a flare or spread of an existing treated rash. It appears that repeated applications of relatively low concentrations of parabens in medications and cosmetics may lead to sensitivity. Paraben-mix allergy is diagnosed from the clinical history and by performing special allergy tests, i.e., patch tests. Patch testing with 15% paraben mix in petrolatum (three percent each of methyl-, ethyl-, propyl-, butyl-, and benzyl-parahydroxybenzoic acids) is used. This mix caused ACD in one percent of patients patch-tested by the North American Contact Dermatitis Group (NACDG).9

Cross Reactions

The "para" group of antigens (para-aminobenzoic acid [PABA] esters,

paraphenylenediamine) are frequent sensitizers and consist of chemicals with a free amino group in the para position of a benzene ring. A debate exists about the cross-reactivity of parabens and the "para" group, because parabens have a hydroxyl group instead of an amino group in the para position. Although PABA itself does not cross-react with parabens, the esters of PABA may show cross-reactivity.7

Parabens as Urinary Biomarkers of Exposure in Humans

A team from the Division of Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Georgia, has now provided the field with new biomarkers that could help researchers document exposures to parabens.10 Until now, the only biomarker used for human paraben exposure was p-hydroxybenzoic acid in urine. However, that metabolite is produced by the hydrolysis of all the various paraben compounds, so it is nonspecific to individual parabens, which vary widely in estrogenic bioactivity.

Ye et al. measured the presence of free and conjugated parent parabens in urine to determine their suitability to be biomarkers of human exposures. They analyzed the urinary concentrations of methyl, ethyl, n-propyl, butyl (n- and iso-), and benzyl parabens in 100 human adults with no known industrial exposure to the compounds. The results appear to support the viability of those measures as biomarkers of exposure. Methyl and n-propyl parabens, the parabens most commonly used in cosmetics and foods, were found at the highest median concentrations in almost all the samples—99% contained the former and 96% the latter. The authors say this could result from the widespread use of these compounds; from differences in the absorption, distribution, metabolism, and excretion of the various parabens; or from a combination of both factors. Other parent compounds, such as ethyl and butyl paraben, appeared in more than half of the samples. Regardless of the reason for such high frequencies of detection, the researchers say their results suggest that urinary parabens and their conjugates could be valid biomarkers of exposure to these chemicals. The detection and measurement methodologies used by Ye et al. could help investigators as they seek to characterize the potential health risks associated with exposure to the individual paraben compounds.10

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40 Women With Breast Cancer Had This "Cosmetic Ingredient" in Their Tissues by Dr. Mercola

Source: http://articles.mercola.com/sites/articles/archive/2012/04/02/toxic-parabens-on-breast-cancer-patients.aspx?e_cid=20120402_DNL_art_1

New research has detected the presence of paraben esters in 99 percent of breast cancer tissues sampled.[1]

The study examined 40 women who were being treated for primary breast cancer.

In 60 percent of cases, five of the different esters were present.

Parabens are chemicals with estrogen-like properties, and estrogen is one of the hormones involved in the development of breast cancer.

The study notes that:[2]

"Variation was notable with respect to individual paraben esters, location within one breast and similar locations in different breasts.

Overall median values in nanograms per gram tissue for the 160 tissue samples were highest for n-propylparaben and methylparaben; levels were lower for n-butylparaben, ethylparaben and isobutylparaben...

The source of the paraben cannot be identified, but paraben was measured in the 7/40 patients who reported never having used underarm cosmetics in their lifetime."

Sources and Dangers of Parabens

Deodorants and antiperspirants are some of the primary sources of parabens, but the fact that even those who reportedly never used them still had parabens in their breast tissue clearly demonstrates that these chemicals, regardless of what products they're added to, can, and apparently will, accumulate in breast tissue.

It's important to recognize that whatever you spread on your skin can be absorbed into your body and potentially cause serious damage over time, as this research demonstrates.

(To learn more about the potential toxicity of your cosmetics, I urge you to review the EWG's extensive *Skin Deep Report*.)[3] Parabens inhibit the growth of bacteria, yeast, and molds, and are used as preservatives.

On the label they may be listed as:

Methyl paraben	Propyl paraben	Isobutyl paraben
Ethyl paraben	Butyl paraben	E216

These chemicals are commonly used in:

Deodorants and antiperspirants	Shampoos and conditioners	Shaving gel	Toothpaste
Lotions and sunscreens	Make-up / cosmetics	Pharmaceutical drugs	Food additives

Studies have shown that parabens can affect your body much like the estrogens, which can lead to diminished muscle mass, extra fat storage, and male gynecomastia (breast growth). Other studies besides the one featured here have also linked parabens to breast cancer. The US Environmental Protection Agency (EPA) has linked methyl parabens in particular to metabolic, developmental, hormonal, and neurological disorders, as well as various cancers.

How to Avoid Some of the Most Common Culprits

Avoiding parabens and other harmful chemicals requires becoming an avid label reader. Beware that products boasting "all-natural" labels can still contain harmful chemicals, including parabens, so make sure to check the list of ingredients.

Another alternative is to make your own personal care products. In many cases it's much easier than you might think. Michael DeJong, environmentalist and author of books on green living has a book called *Clean Cures*,[4] which is chockfull of affordable, easy, natural remedies you can prepare at home to treat ordinary ailments with items you have in your own refrigerator and pantry.

When it comes to deodorants, one option is to skip it altogether. Simple soap and water has served me quite well. For some additional odor-protection, try a pinch of baking soda mixed with a small amount of water.

Beware: There's a Brand NEW Class of Cancer-Causing "Estrogens..."

Recent research has also confirmed the existence of a previously unknown class of cancer-causing materials that can be found in thousands of consumer products. Some of them are even added to supplements and foods as "nutrients". These estrogen-mimicking compounds are: *metals*.

Yes, a broad range of metals have been shown to act as "metalloestrogens" with the potential to add to the estrogenic burden of the human breast, thereby increasing the risk of breast cancer. The following metals have been identified as being capable of binding to cellular estrogen receptors and then mimicking the actions of physiological estrogens:[5]

Aluminum	Antimony	Arsenite	Barium	Cadmium	Chromium	Cobalt
Copper	Lead	Mercury	Nickel	Selenite	Tin	Vanadate

According to GreenMedInfo:[6]

"...[E]xposure to sodium selenite (and sodium selenate) is difficult to avoid, as it is the primary source of supplemental selenium in mass market vitamins, foods, beverages, etc. The same is true for inorganic forms of chromium, copper, nickel, tin and and vanadium, which you will find on the labels of many mass market multivitamins. Another daily source of metalloestrogen exposure for millions of consumers is aluminum-based antiperspirants."

Cadmium Linked to Higher Breast Cancer Risk

A recent study published in the journal *Cancer Research* indicates that women whose diets contain higher levels of cadmium are at a greater risk of developing breast cancer. Cadmium is a heavy metal long known to be carcinogenic, and, as you can see by its inclusion on the list above, it's also been identified as a metal that can bind to estrogen receptors, effectively mimicking the female hormone estrogen. The study found that among close to 56,000 women, those with the highest intakes of cadmium were 21 percent more likely to develop breast cancer.[7]

Cadmium leaches into crops from fertilizers, or when rainfall or sewage sludge deposit it onto farmland. Potatoes and whole grains are a couple of the primary sources cadmium, but it's also present in air pollution from the burning of fossil fuel, and can therefore also be inhaled. According to the *Los Angeles Times*:[8]

"The study offers new evidence in a large human population that environmental chemicals that mimic the effects of the female hormone estrogen may contribute to women's risk of certain cancers, including endometrial and breast cancers... The finding comes just three months after the Institute of Medicine, a prestigious body of independent biomedical researchers, concluded that a host of other factors — most within a woman's power to control, such as obesity and hormone-replacement medication — were the most important sources of breast cancer risk.

The report they're referring to is *Breast Cancer and the Environment: A Life Course Approach* by the Institute of Medicine (IOM)[9] issued in December of last year, which discusses environmental impacts on breast cancer risk.

The report is a step in the right direction, as it recognizes the need to further investigate the role environmental toxins play in the development of breast cancer. This is important, because while individuals can do their best to avoid harmful chemicals, if we really want to quell the rise in cancers of all kinds, we must remove chemicals linked with cancer from consumer products, manufacturing, and other sources of exposure. Furthermore, the IOM report also identifies ionizing radiation as one of the primary contributors to breast cancer, which of course includes mammograms...

Could More Women Be Harmed than Helped with Mammography?

Mammograms expose a woman's body to radiation that can be 1,000 times greater than that from a chest x-ray, which increases the risk of cancer. Mammography also compresses breasts tightly (and often painfully), which could lead to a lethal spread of cancerous cells, should they exist.

Earlier this year, the Nordic Cochrane Collaboration issued a report stating that mammography screening may cause more harm than good. Their informative leaflet, *Screening for Breast Cancer with Mammography*,[10] is an important read for every woman. Even more provocative is the new book, *Mammography Screening: Truth, Lies and Controversy* by *Peter C. Gøtzsche, Professor of* Clinical Research Design and Analysis Director at The Nordic Cochrane Centre, and Chief Physician. The very first paragraph of the book's ad reads:[11]

"The most effective way to decrease women's risk of becoming a breast cancer patient is to avoid attending screening."

While this may sound too shocking to be true for some, the available data fully supports that conclusion. According to the Cochrane Collaboration, for every 2,000 women invited for screening over the course of 10 years, just ONE woman will have her life prolonged. Meanwhile, 10 healthy women, who would not have been diagnosed with cancer had it not been for the mammography screening, will be misdiagnosed as having breast cancer, and will be treated unnecessarily. Additionally, more than 200 women will experience significant psychological distress for many months due to false positives.

The Cancer Industry is Fraught with Corruption...

There's plenty of damning information out there that can, and ultimately will, be used to call for a congressional hearing on the mammography cover-up. As far back as 1974, professor Malcolm C. Pike at the University of Southern California School of Medicine warned the National Cancer Institute (NCI) that a number of specialists had concluded that "giving a women under age 50 a mammogram on a routine basis is close to unethical." In the 1990's, Dr. Samuel Epstein warned about the dangers of mammography, stating:

"The premenopausal breast is highly sensitive to radiation, each 1 rad exposure increasing breast cancer risk by about 1 percent, with a cumulative 10 percent increased risk for each breast over a decade's screening... The high sensitivity of the breast, especially in young women, to radiation-induced cancer was known by 1970. Nevertheless, the establishment then screened some 300,000 women with X-ray dosages so high as to increase breast cancer risk by up to 20 percent in women aged 40 to 50 who were mammogramed annually."

Yet despite all the evidence against the routine use of it, mammography has remained the number one recommended "prevention" strategy for all women over the age of 40. Alarmingly, there's evidence indicating the U.S. Food and Drug Administration (FDA) has been negligent (to put it mildly) in their approval of a number of cancer-detecting devices. It recently became known that whistleblowers within the agency had been secretly monitored for two years; all of whom worked in the office responsible for reviewing medical devices, including cancer screening devices. The monitored employees had warned Congress that the agency was approving medical devices that posed unacceptable risks to patients.

Jeffrey Shuren, director of the FDA's Center for Devices and Radiological Health has repeatedly tried to take action against the employees, claiming they had disclosed information that undermined the integrity and mission of the FDA. Shuren is also the official who oversees mercury dental fillings, which they have been fraudulently referring to as 'silver fillings.' Shuren promised to make an announcement about dental amalgam by the end of 2011, but just before the end of the work year, the agency conceded that no announcement was forthcoming – not in 2011, and maybe not at all.

Again and again, Mr. Shuren demonstrates loyalty to industry interests rather than public health and safety... But he's not the only one. In a 2009 letter from an unknown number of FDA employees to President Obama's transition team, the authors clearly spell out the need for a complete overhaul of the agency due to deep-rooted systemic corruption at the highest levels.[12] They write:

"Currently there is an atmosphere at FDA in which the honest employee fears the dishonest employee, and not the other way around. Disturbingly, the atmosphere does not yet exist at FDA where honest employees committed to integrity and the FDA mission can act without fear of reprisal. ...America urgently needs change at FDA because FDA is fundamentally broken, failing to fulfill its mission, and because re-establishing a proper and effectively functioning FDA is vital to the physical and economic health of the nation."

Mammograms No Longer Recommended for Women in Their 40's

The US Preventative Task Force revised its recommendations on mammograms in October 2009,[13] stating that women in their 40's should no longer get routine mammograms for early detection of breast cancer. Instead, the panel recommended waiting until the age of 50, and only doing one mammogram every other year rather than annually. The Canadian task force followed suit in November last year.

While many cancer organizations were outraged and have shunned the task forces' new directive, it's important to realize that the main reasons for this change in guidelines were the documented

dangers and short-comings of mammographic screening. All in all, there's convincing evidence that mammography is not all it's cracked up to be, and the FDA is not doing its stated job to protect your health. Instead, they're busy catering to industry and skirting the boundaries of the law to protect a lucrative business model. This is a tragedy, considering how many alternatives there are that could help stem the tide of cancer...

There's a wide variety of prevention and treatment strategies that appear to be both safer and more effective than conventional strategies like mammograms and the "cut-poison-burn" model of cancer treatment... To learn more, please review the related articles listed below.

Baby Bottles Leach Toxic Chemical According to New U.S. and Canadian Study - Feb. 7, 2008

Oregon Environmental Council joins with other environmental health groups to call for immediate moratorium on Bisphenol A in baby bottles, food, and beverage containers

PORTLAND, Ore.—Feb. 7, 2008—Dozens of state and national environmental health organizations in the United States and Canada are calling for an immediate moratorium on the use of bisphenol A (BPA) in baby bottles and other food and beverage containers, based on the results of a new study that demonstrates the toxic chemical BPA leaches from plastic baby bottles when heated.

Results of the study, "Baby's Toxic Bottle: Bisphenol A Leaching from Popular Baby Bottles," commissioned by Environmental Defence of Canada and researched by the laboratory of Frederick vom Saal, PhD, at the University of Missouri, show that, when new bottles are heated, those manufactured by Avent, Evenflo, Dr. Brown's and Disney/First Years leached between 4.7 – 8.3 parts per billion of BPA.

Recent research on animals shows that at doses below these levels BPA can harm health by disrupting development. BPA is a synthetic sex hormone that mimics estrogen, and is used to make hard polycarbonate plastic. Ninety-five percent of all baby bottles on the market are made with BPA.

Studies conducted on laboratory animals and cell cultures have also linked low doses of BPA to obesity, diabetes, thyroid disease, breast cancer, prostate cancer, and other illnesses. BPA exposure is widespread and has been found in 95 percent of Americans tested. Scientists, physicians, and public health professionals suspect that existing scientific evidence on BPA indicates a real risk to human health.

"Once again we see the unfortunate effects of the loopholes in our chemical policy," said Andrea Durbin, Executive Director of the Oregon Environmental Council. "Sadly, there are no existing safety standards for BPA under U.S. laws. We and our legislators need to become proactive in defending the health of our youngest, and most vulnerable citizens."

Nine states have introduced legislation that would restrict the use of BPA in children's products, including baby bottles; California, Connecticut, Hawaii, Maine, Maryland, Massachusetts, Minnesota, Minnesota, New York, Pennsylvania. Oregon has not as yet introduced legislation.

BPA is also used to make hard plastic used in some toddler sippy cups, polycarbonate water bottles such as some Nalgene bottles, dental sealants, and the linings of many food and beverage cans, including all infant formulas.

On the Web

The full study, “Baby’s Toxic Bottle: Bisphenol A Leaching from Popular Baby Bottles,” is available to download for free on the website www.chej.org

The U.S. version of “Baby’s Toxic Bottle” was written by the Center for Health, Environment and Justice, and Clean Water Action, in collaboration with Environment Defence, and released in the U.S. by a broad coalition of public health and environmental non-governmental organizations including: The Oregon Environmental Council, Alliance for a Healthy Tomorrow, Boston Common Asset Management, Breast Cancer Fund, Center for Health, Environment and Justice, Clean New York, Clean Water Action, Environment America, Environmental Health Fund, Environmental Health Strategy Center, Healthy Legacy, Learning Disabilities Association of America, MOMS (Making Our Milk Safe), and US PIRG.

About the Oregon Environmental Council

The Oregon Environmental Council safeguards what Oregonians love about Oregon – clean air and water, an unpolluted landscape and healthy food produced by local farmers. For 40 years we’ve been a champion for solutions to protect the health of every Oregonian and the health of the place we call home. Our vision for Oregon includes solving global warming, protecting kids from toxins, cleaning up our rivers, building sustainable economies, and ensuring healthy food and local farms. Find out more at www.oeconline.org.

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BPA Revealed: the nasty secret in your kitchen cupboard

18 of 20 most popular tins made with controversial bisphenol A in lining

By Martin Hickman , Consumer Affairs Correspondent Thursday 01 April 2010

Some of Britain's best-known foods contain the controversial chemical bisphenol A, The Independent can reveal.

Tins of Heinz baked beans, soup and beans, John West and Princes fish, and Napolina tomatoes are lined with a membrane containing bisphenol A, or BPA, a molecule of which is pictured top left. Other companies using it in their tins include the biggest retailers in the UK, Tesco, Sainsbury's and Asda, who use it for tins of tuna and sardines.

Britain's Food Standards Agency (FSA) has given the chemical the all-clear, in contrast to the US Food and Drug Administration, which in January expressed concern over its impact on the brains and development of young children and said it was "taking reasonable steps to reduce human exposure" to it in the food supply. After the American U-turn, the EU-funded European Food Safety Authority (EFSA) launched and is still carrying out a review of BPA.

Some scientists fear that exposure to minute doses of the chemical in food and other products may be damaging to the health of individuals.

BPA is an endocrine disruptor that interrupts hormones and, in laboratory experiments on animals, has been linked with breast cancer, prostate cancer, hyperactivity and other metabolic and behavioural problems, diseases which are all on the rise in the West. But the plastics and chemicals industries insist its use is safe and accuse campaigners of misleading the public, pointing to industry-funded studies involving large numbers of rodents that have shown no harm.

At stake is the future of one of the highest production volume chemicals in the world. BPA is widely used to harden the plastic casings of mobile phones and computers and makes baby bottles shatterproof. In food products, it commonly lines the inside of cans and tins to protect their contents from being contaminated by the metal.

To establish its prevalence in food, The Independent surveyed manufacturers of the UK's 20 best-selling tinned foods. Although it is not stated on tins, BPA is used in the linings of 18 out of the 20 products, which have combined annual sales of £921m, or 43 per cent of UK tinned food sales. All the companies said their products were safe because the levels of BPA leaching out into food were so low that they were safe.

However Heinz said it was looking to phase out BPA once alternatives could be found. In a statement, the US tinned food giant said: "Although UK and European food authorities have stated that minute levels of BPA in can coatings are safe, Heinz remains committed to moving to alternatives. For beans, pasta and many soups a protective coating is only applied to the can ends

which would not provide any trace of BPA or would be at the limit of detection of a few parts per billion. This compares with the safe legal limit of 600 parts per billion. Heinz continues to advance research into alternative coatings in response to consumer opinion but safety remains our first priority before making any changes."

Princes, the tinned fish company which also owns the Napolina brand, said: "The inside of most food cans requires a protective coating. Bisphenol A (BPA) is used industry wide as a component part of this coating. It is an approved food contact material and there is guidance from both the FSA and the EFSA regarding its use."

John West said: "Some of John West's tinned products are lined with a lacquer that contains a derivate of Bisphenol. By contact tiny amounts of Bisphenol-A are able to migrate within the EU regulation limits." Baxters, the Scottish soup-maker, said its cans contained "minute" amounts of BPA at levels "substantially lower" than that approved by the EFSA.

Tesco, Sainsbury's and Asda, and other producers such as Premier Foods, General Mills and Hormel Foods, the US company which makes Spam, insisted their tins were safe and produced in accordance with current safety regulations.

Tinned drinks also include a membrane with BPA. A spokeswoman for Coca-Cola UK confirmed: "We use BPA in the linings of our cans. Our top priority is to ensure the safety and quality of our products and packaging through rigorous standards that meet or exceed government requirements... All available scientific evidence and testing shows that drinks in aluminium and steel cans are safe."

According to the FSA, studies have shown that BPA is not harmful to laboratory animals when fed in amounts equivalent to more than exposure levels in humans. However the last review of the safety of BPA in tinned foods in Britain was eight years ago by the Committee on Toxicity. Since then several peer-reviewed scientific studies have detected low-dose effects on animals. These low-dose effects are not currently recognised by British or European regulators.

Breast Cancer UK is among several campaigning organisations which wants to see reductions in BPA used in food and other products. Claire Dimmer, chair of trustees, said: "We welcome the research that the food packaging industry is undertaking to find potential BPA alternatives. But these efforts need to be stepped up significantly. " She called on manufacturers to introduce clear BPA labelling – "otherwise it's impossible for us to make a decision on ways of limiting our and our families exposure to this chemical."

BPA makes canned food risky for pregnant women

By Liz Szabo, USA TODAY

Most food cans use lining with the chemical bisphenol A. "Fresh fruits and vegetables may be more expensive, but I believe that the risk is too high not to spend the extra. The entire life of that individual may be altered by a few months of BPA exposure in pregnancy," says obstetrician Hugh Taylor who wasn't involved in the report but advises pregnant patients to avoid canned foods.

Pregnant women should limit their intake of canned foods and drinks, according to a report that finds 92% of food from metal cans is contaminated with an estrogen-like chemical called BPA, or bisphenol A.

The chemical is used in countless products, from plastic bottles and paper receipts to the linings of metal cans. The National Toxicology Program has said it has "some concern" that BPA alters development of the brain, behavior and the prostate gland in children, before and after birth.

BISPHENOL A: What to know about 'everywhere chemical'

Researchers found that BPA levels vary dramatically even between cans of the same product, according to the study, released Tuesday by the National Workgroup for Safe Markets, a coalition of 19 environmental groups. For example, one can of Del Monte French Style Green Beans had 36 micrograms of BPA per serving, while another can of the same product had 138 micrograms per serving — a level that has been linked to changes in prostate cells and increased aggression in animals.

The report calls on Congress to ban BPA in food and drink containers, noting that companies such as Eden Foods already sell vegetables in BPA-free cans; Muir Glenn also plans to begin packaging tomatoes in BPA-free cans this year. Canada and Denmark restrict the use of BPA in certain children's products, as do five U.S. states, three counties in New York and the city of Chicago, the report says.

The Grocery Manufacturers Association says the report ignores evidence showing BPA is safe.

And there is "no replacement for BPA that will work across the board for all foods," the association's Robert Brackett said in a statement. "The performance of any technology that could impact the safety of food or beverages must be proven over the entire shelf life of the product before it can be used."

Obstetrician Hugh Taylor of Yale University School of Medicine, who wasn't involved in the new report, says he now advises pregnant patients to avoid canned foods.

"Fresh fruits and vegetables may be more expensive, but I believe that the risk is too high not to spend the extra," says Taylor, who studies the effect of BPA on prenatal development. "The entire life of that individual may be altered by a few months of BPA exposure in pregnancy. This is where the greatest risk lies. We are programming the hormonal response of the next generation. The worst effects may not become apparent for years."

New study confirms bisphenol A found in plastic is linked to heart disease

by S. L. Baker, features writer. Originally published January 19 2010

(NaturalNews) According to the American Heart Association, cardiovascular disease is the number one killer in the U.S. Various forms of the disease take the lives of over 80 million Americans a year. And while we've all heard about the risk factors for cardiovascular disease -- including smoking, being overweight, high cholesterol and lack of exercise -- it appears it's time to add bisphenol A, better known as BPA, to that list.

This chemical has been used for decades in polycarbonate plastic products including refillable drink containers, plastic eating utensils and baby bottles as well as the epoxy resins that line most food and soft-drink cans. Now a new study just published in the journal PLoS ONE provides the most compelling evidence so far that BPA exposure is dangerous to the cardiovascular system.

Using 2006 data from the US government's National Health and Nutrition Examination Survey (NHANES), researchers from the Peninsula Medical School at the University of Exeter in the UK studied urinary BPA concentrations and found a significantly strong link between BPA exposure and heart disease. In 2008, these same scientists discovered that higher urinary BPA concentrations were associated with a long list of medical problems in adults, including liver dysfunction, diabetes and obesity. This research team was also the first to report evidence that BPA was linked to cardiovascular disease -- and their new research offers further confirmation of a strong connection between BPA and heart ailments.

Despite the fact the new study found that urinary BPA concentrations were one third lower than those measured from 2003 to 2004, higher concentrations of BPA were still associated with heart disease. "This is only the second analysis of BPA in a large human population sample. It has allowed us to largely confirm our original analysis and exclude the possibility that our original findings were a statistical 'blip'," David Melzer, Professor of Epidemiology and Public Health at the Peninsula Medical School and the research team leader, said in a statement to the media.

"We now need to investigate what causes these health risk associations in more detail and to clarify whether they are caused by BPA itself or by some other factor linked to BPA exposure. The risks associated with exposure to BPA may be small, but they are relevant to very large numbers of people. This information is important since it provides a great opportunity for intervention to reduce the risks," added scientist Tamara Galloway, Professor of Ecotoxicology at the University of Exeter and senior author of the paper.

As NaturalNews has previously reported, BPA exposure has been shown in other studies to be associated with neurological problems (http://www.naturalnews.com/025801_B...), diabetes and aggressive behavior in little girls (http://www.naturalnews.com/027382_B...). Unfortunately, the FDA has demonstrated little ability or interest in taking decisive measures to protect consumers from this chemical (http://www.naturalnews.com/024593_t...).Your best strategy to avoid BPA? Eat natural, fresh foods and stay away from cans, bottles and other plastic containing products that are not certified BPA-free.

For more information:
http://www.pms.ac.uk/news.php?id=85

http://www.naturalnews.com/BPA.html

Does Breast Cancer Start in the Womb?

Alarming Findings in Bisphenol A Studies

By Eric Seaborg*

What has caused the steep rise in breast cancer, now the second most common cancer in women? In the 1950s, it struck 1 in 22 women. Just 5 years ago the figure was 1 in 8, and has now reached 1 in 7. Ana Soto, M.D., of Tufts University School of Medicine has devoted much research to this question, and she believes she is finding some answers.

"This is a 3-fold increase of risk in a little more than one generation. So we cannot say that it is because of a change in the genes. It has to be a change in the environment, and by environment, I mean diet, lifestyle, and exposure to chemicals," Dr. Soto said in an interview with ***Endocrine News.***

Dr. Soto, professor of anatomy and cellular biology at the Sackler School of Graduate Biomedical Sciences, has been investigating the contribution of chemicals. One group that seems particularly suspicious is environmental estrogens, or xenoestrogens, because their introduction into the environment preceded the rise in breast cancer incidence. Dr. Soto presented fresh data about their effects at ENDO 07, The Endocrine Society's annual meeting, in a plenary lecture entitled "Does Breast Cancer Start in the Womb?" She cited strong evidence that the effects of certain ubiquitous estrogenic compounds in the modern environment begin prenatally and seem to be contributing to the higher risk of breast cancer.

Tracks Back to DES

The first clues about prenatal estrogen exposure and breast cancer risk came from the potent synthetic estrogen diethylstilbestrol (DES), which was used as an anti-abortive therapy from 1948 to 1971 in the United States, Europe, and Australia. DES use was stopped when young women exposed to it in the womb began exhibiting an extremely rare cancer—clear cell adenocarcinoma of the vagina—as well as other abnormalities. An interesting clue was that only those girls exposed to DES before the 13th week of pregnancy developed the clear cell carcinoma; those exposed later developed malformations of the genital tract, but not cancer, which pointed to an interplay between DES and the stage of fetal development. Women exposed to DES prenatally are now reaching the age at which breast cancer is commonly diagnosed, and the first epidemiological data are revealing a higher incidence of breast cancer in this population.

DES is a powerful estrogen, but retrospective studies using health registries from Scandinavian countries have given evidence that even small increases in estrogen exposure can raise breast cancer risk. In twin births involving a boy and a girl, meaning two placentas, each twin is believed to be exposed to more estrogen than in a single birth. The studies found that in these twins, the girls were more likely to have breast cancer and the boys prostate cancer in adulthood, compared with single births.

Of course, the actual estrogen levels were not tested in these studies, so the hormone's role can only be inferred; but similar studies have looked at the children of mothers who experienced preeclampsia and eclampsia during pregnancy, implying a lower exposure to estrogen in utero. These offspring experienced a lower incidence of breast cancer in adulthood.

"DES is a powerful estrogen, but retrospective studies using health registries from Scandinavian countries have given evidence that even small increases in estrogen exposure can raise breast cancer risk."

Looking at Bisphenol A

To follow up on such effects, Dr. Soto, Carlos Sonnenschein, M.D., and their team exposed mice and rats in utero to the estrogenic chemical bisphenol A (BPA) because of its ubiquity in the human environment. BPA is widely used to manufacture polycarbonate plastics and epoxy resins. It is present in a multitude of products, including the interior coating of food cans, water bottles, food storage containers, baby formula bottles, water pipes, dental materials, wine storage vats, optical lenses, protective coatings, adhesives, protective window glazing, compact discs, and thermal paper. In 2003, BPA production exceeded 6 million pounds worldwide. The chemical is known to leach from polycarbonate plastics and has been detected at measurable levels in maternal and fetal plasma and in 95% of urine samples in a Centers for Disease Control and Prevention study involving a reference human population. Dr. Soto reported that in mice, prenatal exposure to environmentally relevant BPA levels from gestational day 9 to postnatal day 2 accelerated development of the fetal mammary gland and induced altered mammary gland architecture during puberty and adulthood, long after the exposure had ended.

Her team turned to a surrogate rat model to test whether BPA induces mammary gland neoplasia, because in its hormone dependence and histopathology the rat more closely mimics human disease. BPA induced the development of ductal hyperplasias at all four doses tested and carcinoma in situ at the two highest doses.

"Exposure to these so-called weak estrogens at environmentally relevant levels increases the propensity to mammary cancer in rats, and alters the development of the mammary glands in a way that can be noticed from fetal life to adulthood," Dr. Soto said. "The mammary gland develops in a different way in animals that have been exposed in utero to this chemical. So the end point is when the animals are adults and they show intraductal hyperplasia (a precancerous lesion) and carcinoma in situ, but in fetal life, when we look at the fetal mammary gland of animals that were not exposed and animals that were exposed, we already see these changes."

Challenging Traditional Cancer Concepts

Dr. Soto noted that BPA is not a mutagen, so its role in causing cancer cannot be explained by the conventional wisdom of the somatic mutation theory, which posits that cancer is caused by accumulated mutations in the DNA of individual cells that then go on to divide endlessly. "We think that cancer is development gone wrong," said Dr. Soto. The phrase, "cancer as development gone wrong," summarizes a bold theory that challenges medical professionals to re-examine the way they look at cancer. Dr. Soto notes that one genotype can result in several phenotypes, so genetics is not destiny, as demonstrated by studies in the United Kingdom on the effect of malnutrition during pregnancy. Children born to mothers subjected to food restriction during World War II were prone in adulthood to becoming diabetic and having heart disease.

And more specifically on the cancer front, Dr. Soto cited reports that cancer cells can be induced to revert to normal. Mary J. Hendrix, Ph.D., of the Northwestern University Feinberg School of Medicine in Chicago, injected aggressive melanoma cancer cells in zebra fish embryos and the cells normalized. Another group of researchers tested hepatocyte

carcinoma cells in rats. If injected subcutaneously, the cancer cells killed the animal, but if injected into the liver of healthy animals, they normalized and integrated into the liver.

"If you think cancer is caused by mutations that alter the control of cell proliferation, how can you normalize something that is hardwired … if those mutations were dictating the cancer phenotype?" she asked.

Testing Cancer in Developmental Stages

Dr. Soto's team has done similar experiments on the interplay of developmental stages and cancer. They removed the ducts from mammary glands and injected cancer cells into the stroma. "If we injected them into young animals, those cancer cells would form tumors," Dr. Soto said. "We found that if we injected the cells into animals that had been pregnant twice, they formed normal ducts and no tumors at all. So there are many examples of the reversal of the cancer phenotype that contradict the notion that it is due to a rigid change."

If cancer can be reverted to normal, it calls into question the whole concept that the disease is caused by a "single cell that went mad," Dr. Soto said. In contrast, she suggested that cancer is a problem of tissue organization—a belief that Dr. Sonnenschein and she proposed in a book entitled *The Society of Cells*. The authors termed their concept the tissue organization "field theory," which sees cancer as the result of a breakdown in communication among the various cell types of a tissue. "We know that for a mammary gland to form, there is a dialogue between the epithelial cells and the stroma cells that goes in both directions. Cancer is an alteration of that dialogue. Cancer is a problem of misunderstanding among different cell types," she said.

> **That phrase, "cancer as development gone wrong," summarizes a bold theory that challenges medical professionals to re-examine the way they look at cancer.**

Tying Back to BPA

One may not accept this overall interpretation about how to characterize cancer, but it is hard to deny the data showing the effects of the prenatal exposure to chemicals such as BPA. Rather than causing mutations, BPA and other xenoestrogens are causing alterations in development, and Dr. Soto's team has studied the way that estrogens affect mammary gland development in minute detail.

For example, as early as embryonic day 18 in mice, fetuses of mothers exposed to a high dose of BPA exhibited changes in the appearance of the mammary epithelium, such as decreased cell size and delayed lumen formation, as well as increased ductal area. In the stroma, BPA exposure promoted advanced maturation of the fat pad and altered localization of fibrous collagen. By 6 months of age, the mammary glands of perinatally exposed virgin mice resembled those of pregnant mice, with an increased percentage of ducts, terminal ends, terminal ducts, and alveolar buds.

"There is a lot of plasticity in the fetus, and part of that plasticity is the propensity to develop cancer," Dr. Soto said. "If these chemicals are possibly producing these effects, then it is a problem of public health, and should be addressed at that level, and people should be protected. The environmental estrogens are acting in addition to the estrogens that are indigenous, so maybe if you exceed by a little bit the amount that is indigenous, you are really in a critical zone."

A BPA Effect on Humans

Even seemingly insignificant amounts seem to be having an impact, and BPA is just one of many xenoestrogens and other endocrine disrupters in our environment. The relationship between the xenoestrogens and breast cancer is likely to be just one of many effects to be elucidated in coming years. In addition to its estrogenic properties, BPA is also a thyroid hormone antagonist. Exposed animals have shown many other effects, including an increased tendency toward obesity and alterations in behavior, indicating that the chemicals are affecting the formation of tissues in the nervous system.

"We have to know about these things because probably we are going to see them in clinical practice—as obesity, as behavioral problems, problems of reproduction, of early menopause, and so forth," Dr. Soto said. "And all these outcomes touch endocrinology, they touch us in every aspect of our profession, from the very beginning of development." ■

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Issue: September 2006

Living Better with Plastics

Plastic is hailed as one of the world's greatest inventions. While its use in products such as helmets and computers makes lives safer and tasks easier, I'm wary of many kitchen plastics used to protect and serve meals. Recent studies show that when some plastics come in contact with food, certain chemicals migrate to the food and may cause an array of health problems. Scientists fear that even low levels of these chemicals may reduce immunity and alter behavior in adults and cause cancers and irreversible organ damage in fetuses and children. The good news is that you don't have to give up the convenience of your favorite plastic, because in almost all cases there are similar products made from a safer plastic or other material.

A Closer Look at Four Problem Plastics

In June, San Francisco banned the manufacturing, sale, and distribution of child-care products and toys containing certain phthalates and bisphenol A, both hormone-disrupting chemicals found in some plastics that are thought to interfere with childhood development. The European Union has similar bans on phthalates.

Bisphenol A (BPA). BPA is found in many polycarbonate plastic products (often labeled as #7), such as plastic labeled as microwavable, eating utensils, linings for metal food and beverage containers, baby bottles, and other products. I believe San Francisco's measure to ban BPA in children's products is justified. BPA is a hormone disrupter, a chemical that alters the body's normal hormonal activity and mimics the effects of estrogen. In mice and rats, exposure to BPA has led to miscarriages, birth defects, and mental retardation, as well as early puberty, breast and prostate cancers, and reduced sperm counts. In laboratory studies, this chemical has been shown to inhibit the formation of connections in a part of the brain called the hippocampus, or memory center.

The Centers for Disease Control and Prevention detected BPA in the urine of a majority of the thousands of people it tested in the United States. Frederick vom Saal, PhD, a professor of reproductive biology at the University of Missouri-Columbia, has been researching the effects of these chemicals for more than 30 years and is convinced they're a danger to health, especially for children and fetuses. He and his colleagues looked at human cells and BPA and were astonished at the small amount of chemicals needed to alter the cells. Since then, more than 40

studies have confirmed the negative effect of low doses of BPA on human cells and mouse cells, where they produced an almost identical response. More studies show BPA's cell-altering effects at higher levels.

I'm concerned about these findings. Normal wear and tear of plastics causes chemicals to leach. "In adults, high levels affect behaviors and the immune system, but babies are ruined for life," says vom Saal. Of particular concern is the abundance of baby bottles on the market containing BPA. *Consumer Reports* tested hard plastic baby bottles and found that even after multiple washings, they continued to leach BPA. "When you boil them in water, you have an estrogen cocktail," says vom Saal.

"Many countries, in fact, have banned polycarbonate dishes and cutlery," says vom Saal. "In Japan, consumer use of polycarbonate is crashing." Recent state bills in California, Maryland, and Minnesota that proposed a ban on children's products containing high levels of BPA haven't fared as well as legislation did in San Francisco.
My advice:
• Microwave food in glass or ceramic, never in plastic.

• Unfortunately, cans are not labeled that they contain BPA. Once opened, notice the canned products that are lined with plastic and try to avoid them.
• Switch to baby bottles made of glass or opaque pastel-colored #4 or #5 plastic. (Evenflo and Gerber offer nontoxic bottles.) I'm not concerned about toddlers' sippy cups, which are made from safe #5 and polyethylene #2 plastic.

Polystyrene. Polystyrene, labeled as #6, is often found
in foam containers and cups and sometimes in clear disposable takeout containers, plastic cutlery, and cups. Polystyrene may leach styrene, a possible human carcinogen, into food it comes in contact with. Studies involving mice show that when it's ingested over several weeks, this chemical can damage the liver, kidneys, lungs, and brain.
My advice:
• Avoid consuming hot liquids, fatty foods, or alcoholic drinks from Styrofoam containers, since heat and alcohol may increase leaching of the chemical.
• Transfer foods from takeout containers made of Styrofoam to glass or ceramic.

Adipates and Phthalates. Many plastic-wrapped foods in grocery stores, such as meats and cheeses, are wrapped in polyvinyl chloride (PVC), often labeled as #3 plastic. To make PVC plastic flexible, manufacturers add chemicals called plasticizers. These chemicals, known as adipates and phthalates, can leach out of the PVC into food, especially hot, fatty foods. In mice, these chemicals have been shown to cause birth defects, fetal death, and damage to the liver, kidneys, lungs, and reproductive systems. Di-(2-ethylhexyl) adipate, or DEHA, is one plasticizer used in many plastic wraps. In one study

by the Consumers Union, 19 pieces of cheese wrapped in plastic were analyzed and the seven that were wrapped in PVC plastic contained consistently high levels of DEHA. Levels averaged 153 parts per million; the European Union's limit is 18 parts per million for DEHA migration from plastic to food.
My advice:
• Since many plastics used to wrap meat and cheese are made from PVC, I take these items out of the plastic and wrap them in one of my favorite kitchen tools—wax paper. You can also store them in a glass or ceramic dish covered with a lid or a piece of PVC-free plastic wrap (not touching the food), such as Glad Cling Wrap or Saran Cling Plus.
• Never let plastic wrap touch food in the microwave.
• Some cooking oils are sold in plastic bottles made from PVC. Only store oils in glass bottles.

Selecting the Right Water Bottle

All soft plastics have the potential to leach chemicals into food and beverages, including disposable and reusable bottles. Here are a few practical tips:
• Avoid reusing single-use plastic bottles. Frequent washing of disposable plastic bottles may accelerate its breakdown and cause chemicals called adipates to leach.
• Toss the bottle of water that's been sitting in your car. Solar heat can do as much damage as the microwave.
• Some plastic bottles are made from BPA, so use stainless steel water bottles (*visit kleankanteen.com*).

Good and Bad Plastics by the Numbers

Plastics to Avoid

#3 polyvinyl chloride (V or PVC)
Where you'll find it: plastic wrap, cooking oil bottles

#6 polystyrene (PS)
Where you'll find it: Styrofoam

#7 other (usually polycarbonate made with bisphenol A)
Where you'll find it: microwavable plastics, eating utensils, linings for metal food and beverage containers, baby bottles

Safer Plastics

#1 polyethylene terephthalate (PET or PETE)
Where you'll find it: soft drink and water bottles

#2 high-density polyethylene (HDPE)
Where you'll find it: milk and water bottles

#4 low-density polyethylene (LDPE)
Where you'll find it: wrapping films, grocery bags

#5 polypropylene (PP)
Where you'll find it: yogurt containers, syrup bottles

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Get Plastic Out Of Your Diet

PAUL GOETTLICH 16nov03

A similar version of this was published in
Living Nutrition magazine v.15, Spring (April) 2004

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By popular demand . . . **Alternatives to Plastic** Paul Goettlich 3aug2005

You Are What You Eat

When you eat or drink things that are stored in plastic, taste it, smell it, wear it, sit on it, and so on, plastic is incorporated into you. In fact, the plastic gets into the food and food gets into the plastic and you. So, quite literally, you are what you eat[1]. . . drink. . . and breathe — plastic! These plastics are called "Food Contact Substances" by the US Food and Drug Administration (FDA), but until April 2002, they were called "Indirect Food Additives."[2] The new name is cleansed of the implication that plastic gets into your food. In spite of this semantic deception, migration is a key assumption of the FDA.

According to Dr. George Pauli, Associate Director of Science Policy, FDA Office of Food Additive Safety, the regulations mandated in 1958 assume that all plastics migrate toxins into the food they contact. Migration is the movement of free toxins from plastic into the substances they contact — in this case it's your food. The manufacturer must "prove" that the migrations fall within an acceptable range.[3] I agree with the assumption of migration from all plastics, but I find a critical disparity between the level of science employed by the regulations and the current scientific knowledge regarding the levels at which they migrate and the effects they can have. In particular, I am more concerned with extremely low concentrations. There is also a conflict of interest in allowing the manufacturer to submit its own testing to the FDA as proof of anything. We invite the fox into the henhouse and are surprised when there's nothing left but eggshells and feathers.

The amount of migration and corresponding toxicological effects are highly disputed topics, even within the FDA, which has commonly acquiesced to industry in its regulation of

technologies that are used in the production of our foods — plastics, pesticides, growth hormones, irradiation, and microwave. This is clear from the mass of expert and citizen testimony against such technologies that regulatory agencies bend over backwards and jump through flaming hoops to please their corporate clients, as they are called.

There is a worst plastic for any purpose — polyvinylchloride (vinyl or PVC). However, there is no best plastic to contain food or drink. It is my hope that this article will clarify this viewpoint. By the time you've finished reading, you should be closer to forming your own evaluation of plastics.

Its Uses

Plastic is used in contact with nearly all packaged foods. Most cardboard milk containers are now coated with plastic[4] rather than wax. It is sprayed on both commercial and organic produce to preserve its freshness. Plastic is even used to irrigate, mulch, wrap, and transport organic food. Organic bananas now come from wholesalers with a sticky plastic wrapping the cut stem to protect the bananas from a black mold.[5] The mold is controlled on non-organic bananas by dipping the cut ends in a fungicide. Chiquita would only reveal that it's a "food grade plastic," which means that it meets minimum regulatory standards. But since it has a sticky feel to it, I suspect it either carries a fungicide or its physical characteristics act as a fungicide. Either way, if it is or acts as a fungicide, the EPA regulates it as a pesticide, which fungicides are considered a subset of. [6] In a way, this is similar to the regulation of corn that is genetically engineered to carry the toxic bacterium bacillus thuringiensis (Bt) in every cell. Rather than the FDA regulating it as a food, the EPA regulates it as a pesticide. Incredible as it may seem, they see our food as a pesticide.

According to the FDA scientist I spoke with, it's a proprietary formula that he doesn't know about and would offer nothing beyond that. Disclosure of proprietary information is a criminal offense.[7] All plastic manufacturers hide behind trade secrets. This is true with nearly all consumer products. It is quite impossible to know the chemical makeup of any plastic without paying a substantial amount of money for an independent lab analysis.

How is it made?

In a nutshell, plastic is made by combining monomers into polymers under great heat and pressure in a process called polymerization. Each manufacturer has its own proprietary formula for each plastic. And each uses a variety of additives such as plasticizers for flexibility, UV filters for protection from sunlight, antistatic agents, flame-retardants, colorants, antioxidants, and more. Heavy metals such as cadmium, mercury, and lead are common additives. There are also chemicals used to facilitate production such as mold releases, and countless other toxic chemicals regularly added to plastic consumer goods without our knowledge or approval. Many of the products and byproducts of the intermediary steps of plastics production are used in other plastics or industrial processes and products such as pesticides or fertilizer. For holistic thinkers, the mention of plastics and pesticides in the same sentence should begin an informative thought process, while keeping in mind that they all have complete regulatory approval.

The True Cost of Plastic

Plastic is ubiquitous in our lives because it is convenient and relatively inexpensive. It is advertised as safe and that it saves lives.[8] Its safety is based on outdated science and regulations. And while it saves lives in the short run, the record against plastic is looking quite different.

Its convenience comes from being lightweight and its ability to absorb impact shock without breaking, which on its own merit, is hard to argue with. It comes in an endless range of colors and finishes, is pliable, and is easily formed and molded. Most would say it's a perfect material, right? Here's where the bad news begins.

Its inexpensiveness is the result of a large portion of the costs associated with its life — production, use and disposal — being put onto society as a whole. This unsolicited financial burden on society manifests itself as increased taxes to finance municipal curbside recycling programs, landfill space, and incineration. It also increases health care and insurance costs as a result of its incineration polluting the air, water, and food. I'll give much more detail on the negative health effects later, but for now, suffice to say that a full and truthful lifecycle analysis would reveal that the long-term negative health and socioeconomic effects at the local and global scales far outweigh the benefits realized by the use of plastics.

What's so bad about plastic?

For decades, the plastics industry has deceived us with assurances that the polymerization process binds the constituent chemicals together so perfectly that the resulting plastic is completely nontoxic and passes through us without a hitch. In spite of this industry disinformation,[9] the polymerization process is never 100% perfect. Logically then, there are always toxicants available for migration into the many things they contact — your food, air, water, skin, and so on. Both the FDA and the industry know this. However, because of many millions of dollars worth of advertising and public relations work, consumers are educated to think that plastics are safe.

The additives utilized are not bound to the already imperfect plastic, leaving them quite free to migrate. One quick example: without a plasticizer additive, PVC would be rigid. The plasticizer resides between the molecules of the PVC, acting as a lubricant that allows those molecules to slide by each other, and thus flex. Many containers used for food or water are made of it. Even Barbie dolls are made of it. The plasticizer migrates out from day one. And as it ages, the migration can visibly weep out of it.[10]

Plastics, their additives and other processing chemicals can be toxic at extremely low concentrations. In fact, some are significantly more toxic at extremely low concentrations than at much higher concentrations, which is contrary to the FDA scientist's paradigm that, "The dose makes the poison," meaning that the higher the concentration, the more toxic something is. It is an interpretation of the writings of Paracelsus, an alchemist who wrote in the 16th century that, "*Alle Ding sind Gift und nichts ohne Gift; alein die Dosis macht das ein Ding kein Gift ist*" [All things are poison and nothing without poison; alone it is the dose that makes a thing no poison].[11] It's now 500 years later and that assumption of Paracelsus is still the basis for the many regulations. Except on chemical-by-chemical investigations by various independent, institutional, and academic labs, plastics are not explored for harmful effects or regulated in any meaningful way.

Extremely Low Doses and Synergy

Since it is known that all plastics migrate into food, it behooves us to look for the evidence at meaningful levels of detection, at and below single-digit parts-per-trillion (ppt) or ng/kg. Extremely low doses are especially relevant because they can upset the natural balance of the endocrine system. To paraphrase the report of an EPA workshop in 1996, endocrine disruptors (EDs) are external agents that interfere with the production, release, transport, metabolism, binding, action or elimination of natural hormones in the body responsible for maintaining internal balances and the regulation of developmental processes.[12]

Current knowledge of EDs turns the work of Paracelsus — that guy born in the 15th century — upside down. Some chemicals can be more toxic at extremely low doses than extremely high doses. The timing of the exposure can be much more relevant than its dose. Most vulnerable times are in periods of rapid growth, such as those in embryo and children right up to puberty. They can be exposed in the womb and before conception, if sperm and/or ovum are contaminated. The maladies of the children of Gulf War veterans are a prime example of this type of exposure.[13]

Synergy is an important issue that is mostly disregarded by the FDA. Many will even debunk the idea that low dose synergy is real. In combination with other commonly used products, the toxicity of the migratory chemicals from plastics can be potentiated by synergy. A synergy can occur between two or more chemicals that elevate the combination's toxicity to hundreds of times greater than that of the individual chemicals. Besides plastics, other household chemicals can be part of a synergy with plastics.

Nuclear radiation can also severely damage the endocrine system. According to Dr. Ernest Sternglass, Professor Emeritus of Radiological Physics at the University of Pittsburgh Medical School, the synergy between nuclear radiation and chemical toxicants is well documented.[14] Gulf War vets (I and II) were and still are being exposed to depleted uranium (DU) from the tons of armour-busting shells they fired being distributed across the Gulf Region as an aerosol smaller than the size of a virus.[15] The hazardous materials (MOPP) suit that soldiers are given do not protect them from the infinitesimally small particles of DU because the high efficiency particulate air (HEPA) filters do not work below 1/10 of a micron (0.1µ). Each one of us is exposed to extremely low levels of radiation from the nuclear power plants scattered about the US.[16]

On the home front, even the products in our day-in and day-out humdrum lives are coated with, contain, or are made of synthetic chemicals that can interact synergistically with each other. The list is endless but includes beauty products such as nail polish, eyeliner, deodorant and aftershave; household cleaning products such as tile and carpet cleaners, air fresheners that are solid, plug-in, or spray. Even gas and diesel engine exhaust are included. Quite frankly, the FDA doesn't even consider all sources of a chemical in its review of industry product applications.

Consider that there between 87,000 to 100,000 chemicals in commercial production. At the time I wrote this, there were 22,241,247 organic and inorganic substances registered with Chemical Abstracts Service (CAS) registry.[17] Only eight months before that, there were 1,112,474 fewer chemicals.[18] They are regulated and tested in what I would call a "don't look — don't see" style of science that boggles the minds of those who look just a little below the surface of the

happy little corporate-science myths. The focus is on the wonders of plastic with a purposeful avoidance of the painfully evident negative human and environmental health effects. Using the more conservative 87,000 chemicals, there are approximately $1.063725377 \times 10^{86,991}$ different combinations possible that could have a synergistic effect on toxicity.[19] For the purposes of this article, that number is roughly 1 with 87,000 zeros after it. Even if researchers had the time and money to test them all, they still wouldn't know what to look for, because there is no precedent. In addition, one must account for the uniqueness of each living organism and its unique environment, which further expand the possible synergies and possibilities.

Water Stored in Plastic

Water bottles are be made from various types of plastic — polycarbonate (PC), polyethylene terephthalate (PET), Polypropylene (PP), high-density polyethylene (HDPE), low-density polyethylene (LDPE), polyvinyl chloride (PVC or vinyl), and others. To reiterate, they all migrate to some degree. I will focus on just one chemical that migrates out of one plastic that is used to make products with high use and sales profiles.

Bisphenol-A (BPA) is a monomer used in the synthesis of PC plastics, epoxy resins, and composites, as well as a heat stabilizer in PVC. The list of products containing BPA is long. Some rigid containers such as water and baby bottles are made of PC. The popular Nalgene® water bottles are made of Lexan® brand PC. In the medical industry, it is used for syringes, containers, lenses, and dental products. Keep in mind that the FDA regulates only plastics in contact with foods and not any of the other exposures a person might commonly experience every day at home, school, or the office. Because the FDA approves plastics for specific uses rather than for individual chemicals, BPA is not explicitly regulated.[20] It is important to note that all exposures, no matter what origin, are relevant and cumulative. Even other chemicals that act in the body in similar ways can be part of the total effect. The body's natural defenses try to breakdown toxins as they enter. These are called metabolites and can be significantly more toxic than the original chemical.

Today it is common that dentists coat children's teeth with dental sealants [21] that harden (polymerize) within the mouth. This exposure to BPA is large enough to have biologic effects. [22] Just as with other plastics, dental sealants polymerize imperfectly, leaving free monomers to be ingested or absorbed through the skin within the mouth. When it comes to dental solutions without plastic, the choices are limited. And I must say that I am extremely frustrated by the situation. One orthodontist I spoke with creates retainers from metal wire that can replace the standard polycarbonate ones. In tooth replacement, even some materials that dentists call ceramic have a polymer matrix. Gold caps or crowns are an excellent choice, but they too are glued into place with a volatile polymer. By far, the best alternative is to keep your teeth healthy by brushing and flossing regularly, and by eating a healthy diet.

Food and beverages cans are coated with a BPA-containing plastic. During the processing of canned food, it is sterilized in the can at 250°F for 1 hour. Because heat increases its migration, this is an especially large exposure for people who eat canned foods. As PC plastics grow old, BPA and other chemicals are released. But even when they are new BPA migrates out of PC plastic.

The Code of Federal Regulations section on PC plastics allows for migratory chemicals in the hundreds of parts-per-million (ppm) range as well as a percentage of the plastic's total weight. While concentrations of ppm and higher are relevant, there is vast area of exposure that falls well below the FDA's radar in the parts-per-trillion (ppt) range and lower. Testing methods are available, but the cost would be far greater. Because the industry is responsible for testing, it protests madly about the idea that these concentrations are relevant. If the table was turned and the burden of proof was on the consumer, the FDA would demand the most up to date testing methods. A graphic example of 1 ppt is one drop of liquid in 660 rail tank cars. That's a train 6 miles long!

In the year 2000, Consumers Union (CU) tested water from five-gallon PC plastic bottles for BPA, They found from 0.5 ppb to 11 ppb in water samples from eight of the ten 5-gallon jugs.[23] After industry spin-meisters discredited the study as being flawed, not many regulatory red flares were sent up within the FDA. This type of industry disinformation is standard operating procedure. Most times, the statements made could be compared it to one child calling another derogatory names, hoping that the recipient will become *persona non grata* with the other children. However, the CU study was indeed valid and the concentrations of BPA that were found are extremely relevant.

CU also found BPA in samples from baby bottles at worrisome levels.[24] CU advised its readers to avoid exposure to BPA by "dispos[ing] of polycarbonate baby bottles and replac[ing] them with bottles made of glass or polyethylene, an opaque, less-shiny plastic that does not leach bisphenol-A."[25] That advice attracted the wrath of the plastics industry. But I will go further and advise readers not to serve or store any food — liquid or solid, water-based or fatty, hot or cold — in any plastic.

In April 2003, a study was published about BPA accidentally killing mice that had been held in polycarbonate cages at a lab.[26] It was found accidentally when it ruined a lab experiment that heated yeast in PC flasks to find out if the yeast produced estrogens. It was discovered that BPA from the PC flasks was the material that was estrogenic, and that it competed with the natural estrogen in a rat's body. [27] I asked one noted researcher why labs still use plastics considering what it has been known since 1993 that BPA migrates and is hormonally active. The response was, "What are we supposed to do, go back to glass?" The tone of voice made it seem as if I had advised going back in time to live in the Stone Age. This is the state of what is still amazingly called science. There is a lack of reason and logic that goes well beyond what I knew possible before I began looking at the many aspects of this technology. Truth is sought, but the obvious is knocked to the ground and trampled over in the stampede to secure funding.

BPA's Rap Sheet

The list of negative health effects associated in some way with exposure to BPA is remarkably long. The most visible effect may be aneuploidy, a chromosome abnormality found in more than 5% of pregnancies. Most aneuploid fetuses die in utero. About one-third of all miscarriages are aneuploid, making it the leading known cause of pregnancy loss. Among conceptions that survive to term, aneuploidy is the leading genetic cause of developmental disabilities and mental retardation. About 1 in 300 liveborn infants and 1 in 3 miscarriages are aneuploid. It is associated with Down syndrome,[28] Patau syndrome, [29] Edwards syndrome,[30] Klinefelter syndrome, [31] Turner syndrome, [32] Cri du chat syndrome, [33] and Alzheimer's disease.[34] And each of

these bears its own extensive list of maladies covering all parts and functions of the human body — both physical and mental. The condition at birth is directly related to the type of chromosome abnormality present in the embryo at the time of conception.[35] It is well documented that aneuploidy contributes to the increased risk of spontaneous abortion when the female partner is older, but it is also thought that males more than 30 years old may increase the risk of spontaneous abortion when the female partner is less than 30 years of age.[36]

Being one of many known endocrine disruptors, BPA affects development, intelligence, memory, learning, and behavior, skeleton, body size and shape, significant increase in prostate size, decreased epididymal weight and a longer anogenital distance,[37] prostate cancer, [38] reduced sperm count,[39] both physical and mental aspects of sexuality. It may have something to do with obesity,[40] and so many more that a separate article is required to list them all. In other words, if the fetus lives, any one or many parts of its body can be permanently affected. The problems may become evident at any age.

Alzheimer's disease generally occurs after the age of 50. In those afflicted with it, areas of brain become smaller with cell death and the cavities left become enlarged. The areas most affected are control memory, logical thinking, and personality. Only 5-10% of the cases are inherited. 14 million people with Alzheimer’s disease are predicted by 2050.

BPA is about 10,000-fold less potent than 17ß-estradiol, a potent estrogen that is synthesized primarily in the ovary, but also in the placenta, testis and possibly adrenal cortex. Because of the disparity, industry representatives claim it causes no harm at the levels that the majority of people are exposed to. However, a study in 2001 showed that even at such low potency, when combined with other xenoestrogens (estrogens from outside the body), they act together additively, effectively raising the body load of estrogen to dangerous levels.[41] Another study showed that there is an increased sensitivity to BPA during the perinatal period, which begins with completion of the twentieth to twenty-eighth week of gestation and ends 7 to 28 days after birth.[42] Exposure to BPA increases risk of mammary tumors.[43] To reiterate, there is no shortage of research published on the negative health effects of BPA.

Avoiding Plastic

While it’s impossible to avoid all plastics, we must rid our diets and lives of this toxic material as much as possible. There is a huge amount of data confirming the migration of plastic monomers and additives in all steps of food processing.[44] And in my opinion and that of many top research scientists, it is only a matter of time and money spent on new studies before the harm is found. Because of corporate political campaign financing, meaningful regulations resulting from studies will take even longer to become law. We must protect our families while the obvious results trickle in.

I strongly advise individuals and governments to ban plastics wherever possible by utilizing the precautionary principal. The Wingspread Statement on the Precautionary Principle is the consensus statement of a conference in 1998. Simply put it states that if you have reasonable suspicion of harm coming from (plastic in this case) then you must stop it from happening; the burden of proof must be on industry, not consumers; alternatives must be fully explored before using a new material or technology; and any decisions regarding such activities must be "open, informed, and democratic" and "must include affected parties."[45]

Evidence of the negative health effects of plastics already exists in sufficient quantity to halt the use of it in contact with food. More importantly, I feel that the manufacture of plastic itself must be halted for a multitude of reasons. Besides causing an endless number of human deaths, disabilities, and diseases, plastic is clogging all habitats of the world and destroying the ecosystem. There is now 6 times more plastic than plankton floating around in the middle of the Pacific Ocean. Plankton is a major food source for sea animals.[46] A large portion of it is preconsumer plastic that has not been made into a product yet. Called nurdels, they look very much like plankton in size and color. According to a paper by Arrigo et al in *Geophysical Research Letters* in October 2003, plankton production has been declining for the last 20 years with rising ocean surface temperatures. Along with increasing plastic quantities, the ratio of plastic to plankton is increasing, making it more of a target for hungry animals.

The researcher who found this, Captain Charles Moore, Director of the Algalita Marine Research Foundation, told me that new data indicate that the ratio of plastic to zooplankton is even higher in two so-called floating plastic "Garbage Patches" that are each bigger than the State of Texas.[47], [48]

Nurdles are incorporated into all strata of the oceans with no known method of removal. DDE, a metabolite of DDT, and other dioxin-like chemicals concentrate on the surface of the plastic nurdles at a rate up to a million times that found in the ocean.[49] Captain Moore's presentation includes images of sea animals that have suffocated and starved as a result. Even more startling is seeing plastic bits incorporated into the flesh of the sea animals.

Conclusion

I spent about two years answering telephone inquiries at an environmental organization in Berkeley. A great number of the callers asked what the safest plastic to use in contact with food or water is. They also wanted to know what the safest plastic is to microwave food in. My answer was that plastic should never contact food. And that one should never microwave food — it's probably as bad or worse than putting it in plastic because it creates free radicals in the food that damage cells in your body. It also heats the plastic, thus increasing the rate of migration into the food. That answer wasn't popular with either the caller or the organization, which likes to point out positive alternatives. However, plastic *is* the alternative! And glass, wood, metal, and ceramics are the real things. Plastic is merely a foul imitation thereof. By using the least offensive plastic, one only prolongs and increases the toxic load on the Earth and in our bodies. If saving trees is your aim, stop using so much stuff. But in the mean time, don't further degrade the environment with more plastic.

As consumers, we always look for ways to maintain the status quo of our modern lives. However, the only logic I can see in the regulation of food contact plastics is profit at the expense of our health, the economy, society, and environment. You needn't be a polymer scientist to know that plastic shouldn't contact food. What is essential though is a firm standing in reality and a good grip on logic. It also requires being free of ties to the industry before that logic becomes evident.

First set aside your assumptions and look at the known long- and short-term negative effects of plastic on health, economy, environment, and society, as well as the long-term viability of the

human race. Next contrast that with what you find as benefits. I guarantee that the stack of chips will be far larger in the negative pile.

If one were to listen only to nonprofits and the industry, it would be natural to think that only the additives are toxic and migrate. But everything about plastics is toxic — both the additives and the base plastics. And both migrate in quantities that are problematic at extremely low concentrations. Some chemicals are obviously more so than others. But it is undeniable that they all migrate all the time into everything that they touch.

Consider that:

- **Ubiquitous** — plastics are everywhere. . . our bodies, the air, water, oceans and so on
- **Toxic** — plastics are toxic. Both additives and base plastics
- **World** — almost everything we touch is made of or coated with plastics
- **People** — we are all exposed during every every minute of every day
- **Unknown** — almost nothing is known about individual chemicals that make up plastics
- **Synergies** — even less than nothing is known about the effects of combinations of plastics and other things including ionizing radiation
- **Wishful thinking** — the process by which plastics are considered safe by the FDA, the industry, nonprofits and users

Further Reading

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- Be sure to browse through the Plastics index of Mindfully.org
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[17] CAS Registry Numbers for new compounds and assistance with nomenclature can be obtained by writing to Chemical Abstracts Service (CAS) Client Services, 2540 Olentangy River Road, P.O. Box 3343, Columbus, OH 43210, or by visiting their website at http://www.cas.org
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Prenatal exposure to BPA might explain aggressive behavior in some 2-year-old girls

Tuesday, October 06, 2009

Source: The University of North Carolina at Chapel Hill

http://uncnews.unc.edu/content/view/2944/1/

Daughters of women exposed to a common chemical found in some plastics while they were pregnant are more likely to have unusually aggressive and hyperactive behaviors as 2-year-olds, according to a new study by researchers at Simon Fraser University, the University of North Carolina at Chapel Hill and Cincinnati Children's Hospital.

The study, published Oct. 6, 2009 in the journal Environmental Health Perspectives, is the first to examine if there is a link between prenatal bisphenol A (BPA) exposure and behavior problems in children. Results suggest that if a woman is exposed to BPA early in her pregnancy, development of the baby's nervous system might be adversely affected.

BPA is commonly used in the production of polycarbonate plastics and epoxy resins that can be found, for example, in some types of plastic bottles, canned food linings, water supply pipes and medical tubing. About 93 percent of people in the United States have detectible levels of BPA in their urine, according to the Centers for Disease Control and Prevention.

Researchers found that daughters of women who had higher concentrations of BPA in their urine samples during pregnancy were more likely to have aggressive and hyperactive behaviors than children of women with lower BPA levels, especially if higher exposure was seen earlier in pregnancy.

"In other words, girls whose mothers had higher BPA exposure were more likely to act like boys than girls whose mothers had lower BPA levels, especially if the exposure was seen earlier in pregnancy," said the study's lead author Joe Braun, a doctoral student in epidemiology at the UNC Gillings School of Global Public Health. "Boys' behavior did not seem to be affected, although there was some evidence of increased internalizing scores among BPA-exposed boys."

Researchers do not know why girls seem to be affected by the exposure more or differently than boys.

BPA has been used in products for decades, and concerns about its safety have been growing in recent years, Braun said. Previous studies in mice have shown that the offspring of mothers with high BPA exposure during pregnancy were more aggressive than offspring not exposed to high prenatal levels of BPA.

"We wanted to know if there was a risk in humans for neurodevelopment problems," he said. "Study results indicate that exposure to BPA early in the pregnancy seems to be the most critical issue. The most damaging exposure might happen before a woman even knows she's pregnant."

Braun worked with researchers at the Cincinnati Children's Hospital Medical Center, the University of Cincinnati, the Centers for Disease Control and Prevention, and Simon Fraser University in Vancouver, British Columbia.

For the study, urine samples were taken from 249 pregnant women in Cincinnati, Ohio, at 16 weeks and 26 weeks of pregnancy, and again at birth. BPA concentrations in the samples were measured. Then, when the children were 2 years old, behavior problems were assessed, using the Behavioral Assessment System for Children-2 (BASC-2).

"Many government agencies and consumers in the U.S., Canada and around the world have expressed concerns about BPA exposure, especially in children," said Dr. Bruce Lanphear, professor of children's environmental health in the Faculty of Health Sciences at Simon Fraser University and the study's senior author. "Canada has banned BPA in baby bottles and other baby products, but that might not be sufficient to protect children. Although this is the first study of its kind, it suggests that we may also need to reduce exposures during pregnancy."

The study was funded in part by the National Institute of Environmental Health Sciences and the U.S. Environmental Protection Agency.

For more information on the study, visit: www.ehponline.org.

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Teens carry 30 per cent more BPA than rest of population

Submitted by Drew Kaplan on August 30, 2010 – 12:48 pm

Teenagers may carry the highest levels of bisphenol A – about 30 per cent more than the rest of the population, according to the first national survey about the compound conducted by Statistics Canada, but exposure to the estrogen-mimicking chemical is widespread, with detectible levels in 91 per cent of Canadians.
The survey, released Monday, found that the average level of BPA, as the substance is known, was just over one part per billion, an exceedingly small amount, but still a thousand times higher than natural levels of estrogen found in the body.

Statistics Canada said its data, based on urine samples collected from more than 5,400 people aged six to 79, suggest there is "continual widespread exposure in the Canadian population" to BPA. The Statscan sampling is the largest such effort done to date in the world.
"Although BPA may constitute a health risk, no guidance values are currently available in Canada for urinary BPA," the federal agency said of its findings.

The everyday chemical is used to produce everything from CDs to the liners of nearly all tin cans, and has emerged as one of the most debated substances in use because of concerns that exposures amount to receiving an extra dose of estrogen.

Two years ago, Canada was the first country in the world to propose declaring it a toxic substance, although it has yet to do so. Both the U.S. Environmental Protection Agency and the Food and Drug Administration have announced that they are conducting safety of the chemical.

Some scientists and public-health advocates are worried about even these trace amounts, saying they could contribute to increased risk of breast cancer and precocious puberty in girls, among other hormonally caused health impacts that have been observed in animal experiments using low-level exposures to the compound.

Health Canada said it wasn't surprised by the findings because the results are in line with research from other advanced countries showing nearly everyone has some BPA.
As for possible health concerns, it said its scientists are conducting research "related to the potential effects of low concentrations of BPA on human health."

Food is considered the major source of BPA. Teenagers had the highest average amounts, possibly because they eat more food relative to their body size or have metabolic differences.

Monday's survey has led to calls to further regulate BPA.

"The No. 1 priority at the moment has got to be getting it out of the lining of tin cans," said Rick Smith, executive director of Environmental Defence, an advocacy group.

“When nine out of 10 Canadians have a hormonally active chemical in their body, for which easy alternatives are available … why not make some further changes with respect to BPA?”

The trade association representing companies making BPA said the Statscan findings show that Canadians shouldn’t be worried about any possible health hazards.
Martin Mittelstaedt

From Tuesday’s Globe and Mail
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BPA linked to male sex problems
More health risks

View

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The trade association representing companies making BPA said the Statscan findings show that Canadians shouldn't be worried about any possible health hazards.

The new data on consumer exposure to BPA in Canada "is very reassuring and confirms that people are exposed to only minute levels that are eliminated from the body," said Steven Hentges, a spokesman for the American Chemistry Council.

When people ingest BPA through food, about half is broken down in the digestive track into a harmless compound that doesn't have estrogenic activity every six hours or so.

The fact that most people have the chemical in their urine suggests they were having regular exposures in the 24 hours to 36 hours before their tests, says Frederick vom Saal, a biologist at the University of Missouri and a leading U.S. researcher on BPA.

Dr. vom Saal said he found it "really concerning" that younger people had higher levels than those who are older because exposure to hormones during key points in childhood development can cause permanent, lifetime changes in the way cells are organized and operate.

But the view that BPA represents a threat is disputed.

"The presence of a substance doesn't mean anything other than that it's there. It doesn't mean that it causes any harm," said Joe Schwarcz director of McGill University's Office for Science and Society.

Dr. Schwarcz says the bio-monitoring data would need to be follow up by decades of surveillance to find out whether health outcomes varied by exposure to the chemical.

http://www.theglobeandmail.com/life/health/teens-carry-30-per-cent-more-bpa-than-rest-of-population/article1674153/

Web only | posted February 8, 2005

What's in your water bottle besides water?

by Vincent Standley

A reader writes *The Green Guide*:

I just read your article on plastic bottles. The article says to avoid #7 plastic because the polycarbonate contains bisphenol-A. I had been under the impression that the Nalgene bottles made of #7 plastic were the safest plastic bottle. I had been told that the bottles were made of "medical-grade" plastic and did not leach any chemicals. Are all #7 plastics polycarbonate? I have been using these water bottles during my entire pregnancy, because they were supposed to be "safe". I'm alarmed by your report and would appreciate any info you can provide. Thanks.

Sheri Sheibani

***The Green Guide* responds:**

Not all #7 plastic is polycarbonate, nor are all Nalgene bottles made from polycarbonate. Unlike #1-#6,#7 is the official "other" plastics category. Nalgene makes several varieties of water bottle, made from different kinds of plastic, including polyethylene and polypropylene. Nalgene's Lexan bottles are made of polycarbonate plastic (PC), a plastic known to leach the hormone-disrupting chemical bisphenol-A (BPA). This puts it in the company of two other plastics which studies have determined are prone to leaching and pose environmental and/or health concerns: 1) Polyvinyl Chloride (PVC) which can leaches phthalates, a hormone disruptor, and dioxin, a carcinogen; and 2) polystyrene, which can leach styrene, a possible human carcinogen.

Does that mean Nalgene Lexan water bottles are unsafe? We cannot say categorically that Nalgene Lexan bottles are unsafe or even that they leach BPA, until the product has been properly tested. According to Our Stolen Future: "No tests on bisphenol-A leaching have been carried out specifically on Nalgene water bottles, to the knowledge of www.OurstolenFuture.org, nor were Nalgene bottles the brand used in the experiments demonstrating a link between polycarbonate and chromosomal aberrations. There may be some reason why Nalgene bottles do not leach bisphenol-A. This would be highly unexpected, however, given their chemical composition."

What We Know

PC is a durable and heat resistant plastic, making it a popular material for food storage and laboratory equipment, including baby bottles, water bottles, petri dishes and animal cages. In the late 1990s, studies testing PC baby bottles found they leached low levels of BPA. After several tests of the same bottles the presence of BPA decreased dramatically. The studies suggested that while residual amounts of BPA may be present on some baby bottles, it would disappear after a short period of use. The studies concluded that the PC baby bottles currently on the market are not a health risk to children.

In 2003, a study, published in *Environmental Health Perspectives* (EHP), reproduced the same results as the earlier studies when new bottles were tested. However, after repeated washings and scrubbings, the levels of BPA leaching from the bottles increased significantly. The study concludes, "The increased migration levels may be due to polymer degradation."

During the same year, two more studies were published in EHP, which came about after researchers traced BPA in lab mice to the PC cages in which the mice were housed. These studies share several of the same conclusions: 1) Polycarbonate exposed to harsh detergent is prone to leaching; 2) The older the polycarbonate, the more it leaches; 3) High temperatures cause higher rates of leaching. One study found that polycarbonate will leach into water at room temperature. Of even greater concern, the laboratory plastics studies were initiated by sudden abnormalities in mice egg cells after polycarbonate animal cages were mistakenly washed with the wrong detergent.

Erring on the Side of Caution

Until all of the facts are in, we recommend stainless steel water bottles, such as Klean Kanteen and polypropylene bottles such as Rubbermaid's Chuggables listed in The Green Guide Plastics for Kitchen Use report. Stainless steel bottles should have a metal cap as well. Aluminum bottles with an enamel inner coating are also a healthy alternative, though aluminum requires more energy to produce and has greater environmental impact than stainless steel. The Swiss Sigg bottle is made from extruded aluminum and coated on the inside with a taste-inert, food-compatible stove enamel.

If you're still attached to your Nalgene water bottle, you can lower any potential health risk by following a few common sense guidelines. Based on what we know from the

current research, PC is most likely to leach BPA during it initial use and after prolonged use. Heat increases the likelihood of leaching as well. It makes sense, then, to replace your bottle every six months or whenever it appears worn. When you buy a new bottle wash it out with warm water and a mild detergent and let it dry at room temperature. Do not wash your bottle in the dish machine or leave it for prolonged periods in direct sunlight. Only use the bottle to carry cold water; don't fill it with hot water or other liquids. While it is important to keep the bottle clean and bacteria-free, avoid strong detergents and bleach as they can facilitate leaching.

This is an unfolding story, and *The Green Guide* will be following all related studies as they become available to us. We will continue to keep our readers updated on this important issue.

Resources
Stainless steel water bottles with stainless steel caps:
Klean Kanteen w/ cap 27 fluid ounces $21.00
emagazine.greenhome.com

Stainless steel thermos:
MLS Stainless Steel Thermos Bottle
1 liter $23.95
mls-group.com

Nissan Thermos
34 ounces $34.99
coffee-makers-espresso-machines.com

Enamel coated aluminum bottles:
Sigg, 1 liter $15.95
www.sigg.ch/

Polypropylene bottles:
Platypus Play Sport collapsible water bottle
.5 liter $4.50
www.rei.com

Nalgene Silo
48 ounces $9.25
www.rei.com

For more, see The Green Guide's Plastics for Kitchen Use product report

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MEDICAL DISPATCH

THE PLASTIC PANIC

How worried should we be about everyday chemicals?

BY JEROME GROOPMAN

Bisphenol A, commonly known as BPA, may be among the world's most vilified chemicals. The compound, used in manufacturing polycarbonate plastic and epoxy resins, is found in plastic goggles, face shields, and helmets; baby bottles; protective coatings inside metal food containers; and composites and sealants used in dentistry. As animal studies began to show links between the chemical and breast and prostate cancer, early-onset puberty, and polycystic ovary syndrome, consumer groups pressured manufacturers of reusable plastic containers, like Nalgene, to remove BPA from their products. Warnings went out to avoid microwaving plasticware or putting it in the dishwasher. On May 6th, the President's Cancer Panel issued a report deploring the rising number of carcinogens released into the environment—including BPA—and calling for much more stringent regulation and wider awareness of their dangers. The panel advised President Obama "to use the power of your office to remove the carcinogens and other toxins from our food, water, and air that needlessly increase health care costs, cripple our Nation's productivity, and devastate American lives." Dr. LaSalle Leffall, Jr., the chairman of the panel, said in a statement, "The increasing number of known or suspected environmental carcinogens compels us to action, even though we may currently lack irrefutable proof of harm."

The narrative seems to follow a familiar path. In the nineteen-sixties, several animal studies suggested that cyclamates, a class of artificial sweetener, caused chromosomal abnormalities and cancer. Some three-quarters of Americans were estimated to consume the sweeteners. In 1969, cyclamates were banned. Later research found that there was little evidence that these substances caused cancer in humans. In the nineteen-eighties, studies suggesting a cancer risk from Alar, a chemical used to regulate the color and ripening of apples, caused a minor panic among parents and a media uproar. In that case, the cancer risk was shown to have been overstated, but still present, and the substance remains classified a "probable human carcinogen." Lead, too, was for years thought to be safe in small doses, until further study demonstrated that, particularly for children, even slight exposure could result in intellectual delays, hearing loss, and hyperactivity.

There is an inherent uncertainty in determining which substances are safe and which are not, and when their risks outweigh their benefits. Toxicity studies are difficult, because BPA and other, similar chemicals can have multiple effects on the body. Moreover, we are exposed to scores of them in a lifetime, and their effects in combination or in sequence might be very different from what they would be in isolation. In traditional toxicology, a single chemical is tested in one cell or animal to assess its harmful effects. In studying environmental hazards, one needs to test mixtures of many chemicals, across ranges of doses, at different points in time, and at different ages, from conception to childhood to old age. Given so many variables, it is difficult to determine how harmful these chemicals might be, or if they are harmful at all, or what anyone can do to avoid their effects. In the case of BPA and other chemicals of its sort, though, their increasing prevalence and a number of human studies that associate them with developmental issues have become too worrisome to ignore. The challenge now is to decide a course of action before there is any certainty about what is truly dangerous and what is not.

In 1980, Frederica Perera, a professor at Columbia's Mailman School of Public Health and a highly regarded investigator of the effects of environmental hazards, was studying how certain chemicals

in cigarette smoke might cause cancer. Dissatisfied with the research at the time, which measured toxic substances outside the body and then made inferences about their effects, she began using sophisticated molecular techniques to measure compounds called polycyclic aromatic hydrocarbons, or PAH—which are plentiful in tobacco smoke—in the body. Perera found that after entering the lungs the compounds pass into the bloodstream and damage blood cells, binding to their DNA. She hoped to compare the damaged blood cells from smokers with healthy cells, and decided to seek out those she imagined would be uncontaminated by foreign substances. "I thought that the most perfect pristine blood would come from the umbilical cord of a newborn," Perera said.

But when she analyzed her samples Perera discovered PAH attached to some of the DNA in blood taken from umbilical cords, too. "I was pretty shocked," she said. "I realized that we did not know very much about what was happening during this early stage of development."

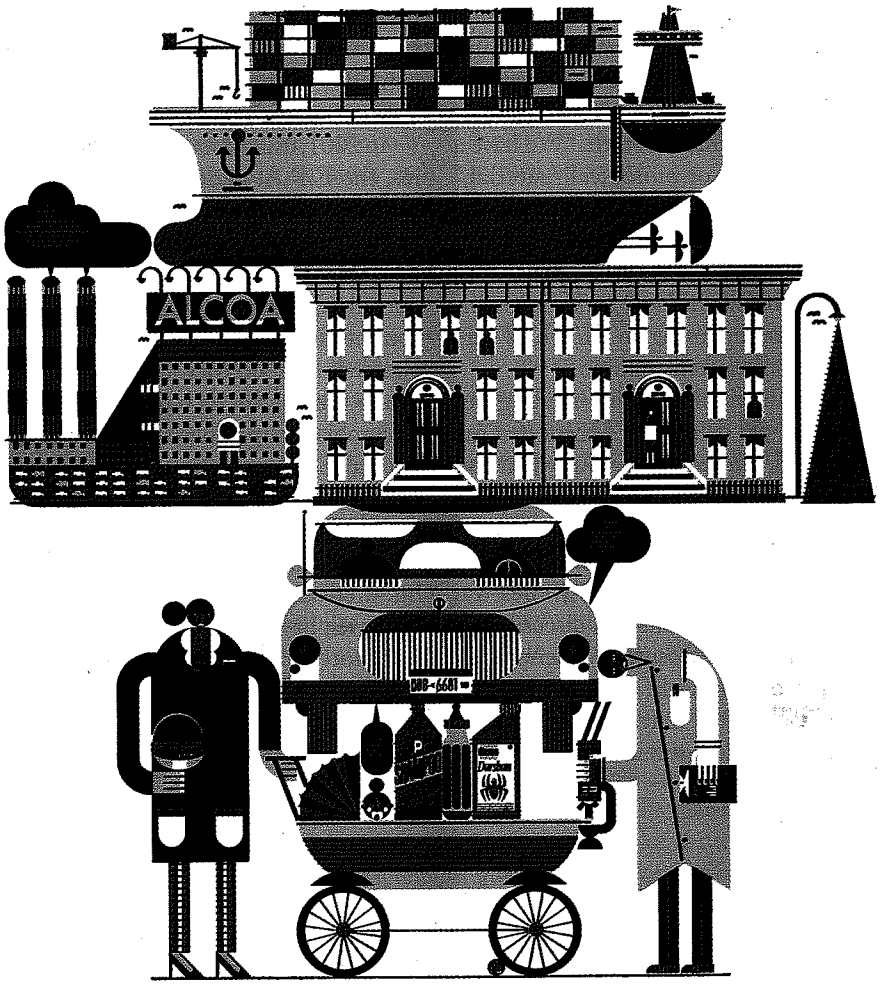
Perera's finding that chemicals like PAH, which can also be a component of air pollution, are passed from mother to child during pregnancy has now been replicated for more than two hundred compounds. These include PCBs, chemical coolants that were banned in the United States in 1979 but have persisted in the food chain; BPA and phthalates, used to make plastics more pliable, which leach out of containers and mix with their contents; pesticides used on crops and on insects in the home; and some flame retardants, which are often applied to upholstery, curtains, and other household items.

Fetuses and newborns lack functional enzymes in the liver and other organs that break down such chemicals, and animal studies in the past several decades have shown that these chemicals can disrupt hormones and brain development. Some scientists believe that they may promote chronic diseases seen in adulthood such as diabetes, atherosclerosis, and cancer. There is some evidence that they may have what are called epigenetic effects as well, altering gene expression in cells, including those which give rise to eggs and sperm, and allowing toxic effects to be passed on to future generations.

In 1998, Perera initiated a program at Columbia to investigate short- and long-term effects of environmental chemicals on children, and she now oversees one of the largest and longest-standing studies of a cohort of mothers and newborns in the United States. More than seven hundred mother-child pairs have been recruited from Washington Heights, Harlem, and the South Bronx; Perera is also studying pregnant women in Kraków, Poland, and two cities in China, and, since September 11, 2001, a group of three hundred and twenty-nine mothers and newborns from the downtown hospitals near the World Trade Center. In all, some two thousand mother-child pairs have been studied, many for at least a decade.

This March, I visited Columbia's Center for Children's Environmental Health, where Perera is the director, and met with a woman I'll call Renee Martin in an office overlooking the George Washington Bridge. Martin was born in Harlem, attended a community college in Queens, and then moved to 155th Street and Broadway, where she is raising her five children. She entered the study eleven years ago, when she was pregnant with her first child. "I was asthmatic growing up," Martin said. "And I was concerned about triggers of asthma in the environment. So when they asked me to be in the study I thought it would be a good way to get information that might tell me something about my own health and the health of my child." She showed me a small black backpack containing a metal box with a long plastic tube. During her pregnancy, Martin would drape the tube over her shoulder, close to her chin, and a vacuum inside the device would suck in a sample of air. A filter trapped particles and vapors of ambient chemicals, like pesticides, phthalates, and PAH. "I walked around pregnant with this hose next to my mouth, but, living in New York, people hardly notice," she said with a laugh.

The Columbia team also developed a



Environmental hazards may cause lasting harm to children.

RAYMOND BIESINGER

comprehensive profile of Martin's potential proximity to chemicals, including an environmental map that charted her apartment's distance from gas stations, dry cleaners, fast-food restaurants, supermarkets, and major roadways. They took urine samples and, at delivery, blood samples from her and from the umbilical cord, along with samples from the placenta. Nearly a hundred per cent of the mothers in the study were found to have BPA and phthalates in their urine. Urine and blood samples are taken as the babies grow older, as well as samples of their exhaled breath. "We have a treasure trove of biological material," Perera said. The researchers track the children's weight and sexual development, and assess I.Q., visual spatial ability, attention, memory, and behavior. Brain imaging, using an M.R.I., is performed on selected children.

Martin was still breast-feeding her two-year-old daughter. "I bottle-fed my first child," she told me. "But when you learn what can come out of plastic bottles and all the benefits of breast-feeding—my other children were nursed." The Columbia group regularly convenes the families to hear results and discuss ways to reduce their exposure to potential environmental hazards. At one meeting, Martin found out that some widely used pesticides could result in impaired learning and behavior. "I told the landlord to stop spraying in the apartment" to combat a roach infestation, she said. On the advice of the Columbia researchers, Martin asked him to seal the cracks in the walls that were allowing cockroaches to enter, and Martin's family meticulously swept up crumbs. This approach has now become the New York City Department of Health's official recommendation for pest control. "You don't need to be out in the country and have compost," Martin said. "This has made me into an urban environmentalist."

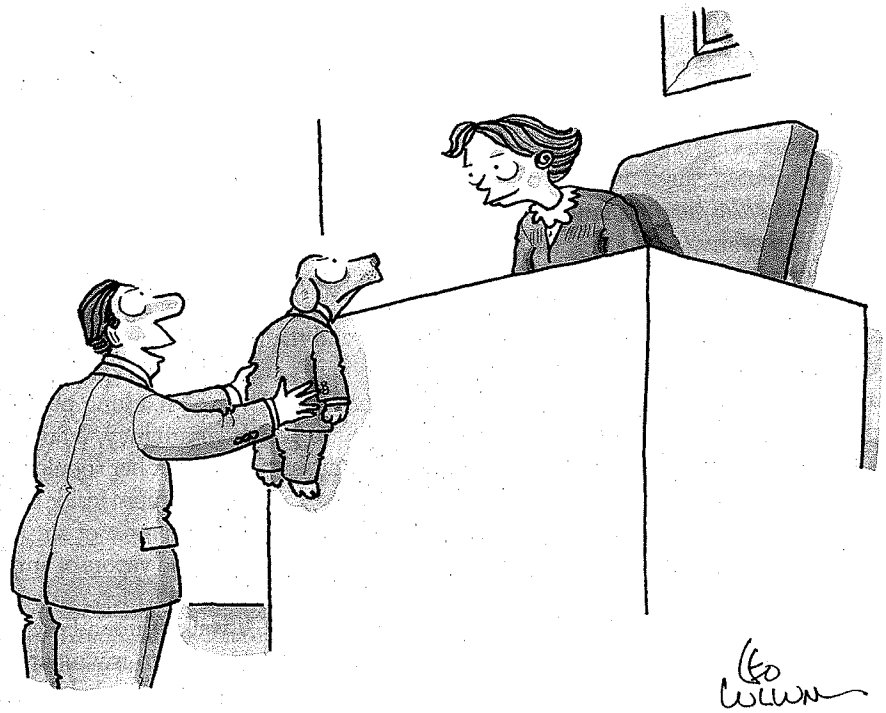
In 2001, using data from animal studies, the E.P.A. banned the sale of the pesticide chlorpyrifos (sold under the name Dursban) for residential and indoor use. Many agricultural uses are still permitted, and farming communities continue to be exposed to the insecticide. Residues on food may affect those who live in urban areas as well. In 2004, the Columbia group published results in the journal *Environmental Health Perspectives* showing that significant exposure during the prenatal period to chlorpyrifos was associated with an average hundred-and-fifty-gram reduction in birth weight—about the same effect as if the mother had smoked all through pregnancy. Those most highly exposed to the insecticide were twice as likely to be born below the tenth percentile in size for gestational age. The researchers found that children born after 2001 had much lower exposure levels—indicating that the ban was largely effective.

For those children who were exposed to the pesticide in the womb, the effects have seemed to persist. The children with the greatest exposure were starting to fall off the developmental curve and displayed signs of attention-deficit problems by the time they were three. By seven, they showed significant deficits in working memory, which is strongly tied to problem-solving, I.Q., and reading comprehension. Another study, published this month in *Pediatrics,* using a random cross-section of American children, showed that an elevated level of a particular pesticide residue nearly doubled the likelihood that a child would have A.D.H.D.

"The size of this deficit is educationally meaningful in the early preschool years," Virginia Rauh, the leader of Columbia's research, said. "Such a decline can push whole groups of children into the developmentally delayed category."

First used in Germany, in the nineteen-thirties, bisphenol A has a chemical structure similar to that of estrogen, but was considered too weak to be developed into a contraceptive pill. Recent animal studies have shown that, even at very low levels, BPA can cause changes that may lead to cancer in the prostate gland and in breast tissue. It is also linked to disruption in brain chemistry and, in female rodents, accelerated puberty. Japanese scientists found that high levels of BPA were associated with polycystic ovary syndrome, a leading cause of impaired fertility.

Phthalates are also ubiquitous in cosmetics, shampoos, and other personal-care products. They may have effects on older children and adults as well as on neonates. A study at Massachusetts General Hospital found an association of high levels of certain phthalates with lower sperm concentrations and impaired sperm motility; young girls in Puerto Rico who had developed breasts prematurely were more likely to have high levels of phthalates in their blood. Immigrant children in Belgium who exhibited precocious puberty also showed greater exposure to the pesticide DDT, which has estrogenlike effects and has been banned in the U.S., but is still



"He's actually my co-counsel, but you may scratch his head."

used in Africa to help control malaria.

Long-term studies have provided the most compelling evidence that chemicals once considered safe may cause health problems in communities with consistent exposure over many years. Researchers from SUNY Albany, including Lawrence Schell, a biomedical anthropologist, have worked over the past two decades with Native Americans on the Mohawk reservation that borders the St. Lawrence River, once a major shipping thoroughfare, just east of Massena, New York. General Motors built a foundry nearby that made automobile parts, Alcoa had two manufacturing plants for aluminum, and the area was contaminated with PCBs, which were used in the three plants. Several Mohawk girls experienced signs of early puberty, which coincided with higher levels of PCBs in their blood.

The Albany researchers also observed that increased levels of PCBs correlated with altered levels of thyroid hormone and lower long-term memory functioning. Similar results have been found in an area of Slovakia near heavy industry. "Folks have complained about reproductive problems," Schell said, of the residents of the Mohawk reservation. "They talked a lot about rheumatoid arthritis, about lupus, about polycystic ovary syndrome. And, you know, you hear these things and you wonder how much of it is just a heightened sensitivity, but, when you see elevated antibodies that are often a sign of autoimmune disease of one kind or another, it could be the beginning of discovering a biological basis for their complaints about these diseases."

Beginning in 2003, Antonia Calafat, a chemist at the Centers for Disease Control and Prevention, and Russ Hauser, of the Harvard School of Public Health, set out to evaluate the exposure of premature infants to certain environmental contaminants. The researchers hypothesized that infants treated in the most intensive ways—intravenous feedings and delivery of oxygen by respirators—would receive the most exposure, since chemicals like phthalates and BPA can leach from plastic tubing. They studied forty-one infants from two Boston-area intensive-care units for BPA. Calafat told me, "We saw ten times the amounts of BPA in the neonates that we are seeing in the general population." In several children, the levels of BPA were more than a hundred times as high as in healthy Americans.

Calafat, who came to the United States from Spain on a Fulbright scholarship, developed highly accurate tests to detect BPA, phthalates, and other compounds in body fluids like blood and urine. This advance, she explained, "means that you are not simply doing an exposure assessment based on the concentration of the chemicals in the food or in the air or in the soil. You are actually measuring the concentrations in the body." With this technology, she can study each individual as if he or she were a single ecosystem. Her studies at the Centers for Disease Control show that 92.6 per cent of Americans aged six and older have detectable levels of BPA in their bodies; the levels in children between six and eleven years of age are twice as high as those in older Americans.

Critics such as Elizabeth Whelan, of the American Council on Science and Health, a consumer-education group in New York (Whelan says that about a third of its two-million-dollar annual budget comes from industry), think that the case against BPA and phthalates has more in common with those against cyclamates and Alar than with the one against lead. "The fears are irrational," she said. "People fear what they can't see and don't understand. Some environmental activists emotionally manipulate parents, making them feel that the ones they love the most, their children, are in danger." Whelan argues that the public should focus on proven health issues, such as the dangers of cigarettes and obesity and the need for bicycle helmets and other protective equipment. As for chemicals in plastics, Whelan says, "What the country needs is a national psychiatrist."

To illustrate what Whelan says is a misguided focus on manufactured chemicals, her organization has constructed a dinner menu "filled with natural foods, and you can find a carcinogen or an endocrine-disrupting chemical in every course"—for instance, tofu and soy products are filled with plant-based estrogens that could affect hormonal balance. "Just because you find something in the urine doesn't mean that it's a hazard," Whelan says. "Our understanding of risks and benefits is distorted. BPA helps protect food products from spoiling and causing botulism. Flame retardants save lives, so we don't burn up on our couch."

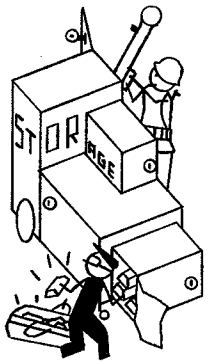
Several studies also contradict the conclusion that these chemicals have deleterious effects. The journal *Toxicological Sciences* recently featured a study from the E.P.A. scientist Earl Gray, a widely respected researcher, which indicated that BPA had no effect on puberty in rats. A study of military conscripts in Sweden found no connection between phthalates and depressed sperm counts, and a recent survey of newborns in New York failed to turn up an increase in a male genital malformation which might be expected if the effects from BPA seen in rodents were comparable to effects in humans. Richard Sharpe, a professor at the University of Edinburgh, and an internationally recognized pioneer on the effects of chemicals in the environment on endocrine disruption, recently wrote in *Toxicological Sciences*, "Fundamental, repetitive work on bisphenol A has sucked in tens, probably hundreds of millions of dollars from government bodies and industry, which, at a time when research money is thin on the ground, looks increasingly like an investment with a nil return."

With epidemiological studies, like those at Columbia, in which scientists observe people as they live, without a control group, the real-life nature of the project can make it difficult to distinguish between correlation and causation. Unknown factors in the environment or unreported habits might escape the notice of the researchers. Moreover, even sophisticated statistical analysis can sometimes yield specious results.

Dr. John Ioannides, an epidemiologist at the University of Ioannina, in Greece, has noted that four of the six most frequently cited epidemiological studies published in leading medical journals between 1990 and 2003 were later refuted. Demonstrating the malleability of data, Peter Austin, a medical statistician at the Institute for Clinical Evaluative Sciences, in Toronto, has retrospectively analyzed medical records of the more than ten million residents of Ontario. He showed that Sagittarians are thirty-eight per cent more likely to fracture an arm than people of other astrological signs, and Leos are fifteen per

cent more likely to suffer a gastrointestinal hemorrhage. (Pisces were more prone to heart failure.)

To help strengthen epidemiological analysis, Sir Austin Bradford Hill, a British medical statistician, set out certain criteria in 1965 that indicate cause and effect. Researchers must be sure that exposure to the suspected cause precedes the development of a disease; that there is a high degree of correlation between the two; that findings are replicated in different studies in various settings; that a biological explanation exists that makes the association plausible; and that increased exposure makes development of the disease more likely.



When epidemiological studies fulfill most of these criteria, they can be convincing, as when studies demonstrated a link between cigarettes and lung cancer. But, in an evolving field, dealing with chemicals that are part of daily life, the lack of long-term clinical data has made firm conclusions elusive. John Vandenbergh, a biologist who found that exposure to certain chemicals like BPA could accelerate the onset of puberty in mice, served on an expert panel that advised the National Toxicology Program, a part of the National Institute of Environmental Health Sciences, on the risks of exposure to BPA. In 2007, the panel reviewed more than three hundred scientific publications and concluded that "there is some concern" about exposure of fetuses and young children to BPA, given the research from Vandenbergh's laboratory and others.

Vandenbergh is cognizant of the difficulty of extrapolating data from rodents and lower animals to humans. "Why can't we just figure this out?" he said. "Well, one of the problems is that we would have to take half of the kids in the kindergarten and give them BPA and the other half not. Or expose half of the pregnant women to BPA in the doctor's office and the other half not. And then we have to wait thirty to fifty years to see what effects this has on their development, and whether they get more prostate cancer or breast cancer. You have to wait at least until puberty to see if there is an effect on sexual maturation. Ethically, you are not going to go and feed people something if you think it harmful, and, second, you have this incredible time span to deal with."

The inadequacy of the current regulatory system contributes greatly to the atmosphere of uncertainty. The Toxic Substances Control Act, passed in 1976, does not require manufacturers to show that chemicals used in their products are safe before they go on the market; rather, the responsibility is placed on federal agencies, as well as on researchers in universities outside the government. The burden of proof is so onerous that bans on toxic chemicals can take years to achieve, and the government is often constrained from sharing information on specific products with the public, because manufacturers claim that such information is confidential. Several agencies split responsibility for oversight, with little coördination: the Food and Drug Administration supervises cosmetics, food, and medications, the Environmental Protection Agency regulates pesticides, and the Consumer Product Safety Commission oversees children's toys and other merchandise. The European Union, in contrast, now requires manufacturers to prove that their compounds are safe before they are sold.

According to the E.P.A., some eighty-two thousand chemicals are registered for use in commerce in the United States, with about seven hundred new chemicals introduced each year. In 1998, the E.P.A. found that, among chemicals produced in quantities of more than a million pounds per year, only seven per cent had undergone the full slate of basic toxicity studies. There is no requirement to label most consumer products for their chemical contents, and no consistent regulation throughout the country. Although the F.D.A. initially concluded that BPA was safe, some states, including Massachusetts and Connecticut, either have banned it or are considering a ban. (In January, the F.D.A. announced that it would conduct further testing.)

There has been some movement toward stricter controls: in July, 2008, Congress passed the Product Safety Improvement Act, which banned six phthalates from children's toys. But so far removal from other products has been voluntary. The President's Cancer Panel report advised people to reduce exposure with strategies that echo some of what the mothers in Frederica Perera's study have learned: choose products made with minimal toxic substances, avoid using plastic containers to store liquids, and choose produce grown without pesticides or chemical fertilizers and meat free of antibiotics and hormones.

Mike Walls, the vice-president of regulatory affairs at the American Chemistry Council, a trade association that represents manufacturers of industrial chemicals, agrees that new laws are needed to regulate such chemicals. "Science has advanced since 1976, when the last legislation was enacted," he said. But Walls notes that some eight hundred thousand people are employed in the companies that the A.C.C. represents, and that their products are found in ninety-six per cent of all American manufactured goods. "The United States is the clear leader in chemistry," Walls said. "We have three times as many new applications for novel compounds as any other country in the world. We want to make good societal decisions but avoid regulations that will increase the burden on industry and stifle innovation."

Academic researchers have found that the enormous financial stakes—the production of BPA is a six-billion-dollar-a-year industry—have prompted extra scrutiny of their results. In 2007, according to a recent article in *Nature*, a majority of non-industry-supported studies initially deemed sound by the National Toxicology Program on the safety of BPA were dismissed as unsuitable after a representative of the A.C.C. drafted a memo critiquing their methods; experimental protocols often differ from one university lab to another. Researchers are now attempting to create a single standard protocol, and a bill introduced by Representative Louise Slaughter, of New York, would fund a centralized research facility at the National Institute of Environmental Health Sciences.

Other legislation aims to completely overhaul the 1976 law. "It's clear that the current system doesn't work at all," Ben Dunham, a staffer in

the office of Senator Frank Lautenberg, of New Jersey, who crafted the bill now before the Senate, told me. Henry Waxman, of California, and Bobby Rush, of Illinois, have released a companion discussion draft in the House. Lautenberg's bill seeks to allow the E.P.A. to act quickly on chemicals that it considers dangerous; to give new power to the E.P.A. to establish safety criteria in chemical compounds; to create a database identifying chemicals in industrial products; and to set specific deadlines for approving or banning compounds. The bill also seeks to limit the number of animals used for research. (Millions of animals are estimated to be required to perform the testing mandated under the E.U. law.) How much data would be needed to either restrict use of a chemical or mandate an outright ban is still unclear. Lautenberg's bill resisted the call of environmental groups to ban certain compounds like BPA immediately.

Dr. Gina Solomon, of the Natural Resources Defense Council, said that the Lautenberg bill is "an excellent first step," but noted several "gaps" in the bill: "There is what people call lack of a hammer, meaning no meaningful penalty for missing a deadline in evaluating a chemical if E.P.A. gets bogged down, and we know from history that it can be easily bogged down." The language setting a standard for safety is too vague, she added. "You could imagine industry driving a truck through this loophole."

Linda Birnbaum, the director of the N.I.E.H.S. and its National Toxicology Program, helps assess chemicals for the federal government and, if Slaughter's bill passes, could become responsible for much of the research surrounding these safety issues. Birnbaum's branch of the National Institutes of Health is working with the National Human Genome Research Institute and the E.P.A. to test thousands of compounds, singly and in combination, to assess their potential toxicity. Part of the difficulty, she points out, is that "what is normal for me may not be normal for you. We all have our own balance of different hormones in our different systems." When it comes to development and achievement, incremental differences—such as the drop of five to ten I.Q. points, or a lower birth weight—are significant. "We're all past the point of looking for missing arms and legs," Birnbaum said.

"I know of very little science where you will ever get hundred-per-cent certainty," Birnbaum says. "Science is constantly evolving, constantly learning new things, and at times decisions have to be made in the presence of a lot of information, but maybe not certainty. The problem is we don't always want to wait ten or twelve or twenty years to identify something that may be a problem."

Perera, who is keenly aware of the potential pitfalls of epidemiological research, told me that her team employs rigorous statistical methods to avoid falsely suggesting that one chemical or another is responsible for any given result. And she objects to the characterization of her research as fear-mongering. "Our findings in children increasingly show real deleterious effects that can occur short-term and potentially for the rest of the child's life," Perera said. In January, the Columbia group published data from the mothers and infants it studied following September 11th. Cord-blood samples saved at the time of birth had been analyzed for the presence of flame retardants. Each year, the children were assessed for mental and motor development. As a point of reference, low-level lead poisoning results in an average loss of four to five I.Q. points. Those children in Columbia's group with the highest levels of flame retardant in their blood at birth had, by the age of two, I.Q. scores nearly seven points lower than normal.

How do we go forward? Flame retardants surely serve a purpose, just as BPA and phthalates have made for better and stronger plastics. Still, while the evidence of these chemicals' health consequences may be far from conclusive, safer alternatives need to be sought. More important, policymakers must create a better system for making decisions about when to ban these types of substances, and must invest in the research that will inform those decisions. There's no guarantee that we'll always be right, but protecting those at the greatest risk shouldn't be deferred. ♦

NEWYORKER.COM/GO/ASK
Jerome Groopman takes readers' questions.

Labour Environmental Alliance Society
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On the Trail of Water Bottle Toxins

Are Hikers and Others Quenching Outdoor Thirsts with H2-UH-OH?

For years, hikers, bikers, campers, and other outdoor recreationalists have favored wide-mouthed water bottles made from Lexan® polycarbonate plastic, like those sold under the brand name Nalgene®. Lexan,s advantages have been as clear as the water that flows from containers made from it. It,s tough, lightweight, absorbs no flavors, and imparts no unpleasant tastes to liquids stored inside. According to new research, it may, however, be imparting unhealthy doses of a chemical called bisphenol-A.

According to several recent studies, polycarbonate plastic readily leaches a chemical called bisphenol-A (BPA) into foods and liquids that are stored in containers made from it. BPA has been identified as an endocrine disrupting chemical, or a chemical that easily mimics hormones when absorbed by the human body. In the case of BPA, the hormone being mimicked is estrogen. Exposure to this compound at the wrong time can cause a cell division problem called aneuploidy in which chromosomes do not evenly split as a cell divides, leaving the two resulting cells with more or fewer chromosomes than normal. This uneven distribution of genetic material can in turn lead to cancer, miscarriage, and birth defects that include Down,s Syndrome.

Low levels of BPA, including those well below the current regulatory safety threshold, have also been shown to affect prostate development, promote prostate tumors, affect breast tissue development and sperm counts, and even possibly create and enlarge fat cells.

The problem with BPA contamination from polycarbonate water containers first came to light in 1998 during research at Case Western Reserve University. Scientists conducting a study on aneuploidy in mice noticed a sudden inexplicable 8-fold increase in the condition in their test subjects. An investigation found that a lab worker had washed out their water bottles with a particularly harsh detergent that had caused large amounts of BPA to be released from the plastic into the animals, drinking water. The researchers found that the BPA levels the mice were exposed were similar to the levels experienced by people under normal conditions and that chromosomes in cells affected by BPA appeared to have been "shot with a shotgun.‰ To verify their unexpected conclusions, the scientists duplicated the detergent accident and achieved the same results. To verify that BPA was, in fact, the culprit of this genetic damage, they then gave mice a daily dose of pure BPA and found that results of this intentional exposure were the same.

As a result, for several years now, experts have warned against washing polycarbonate bottles with harsh detergents and/or using older bottles for food or beverage storage, especially those with scratches, discolorations, or other readily apparent signs of age. The belief was that BPA generally only leached from well-used bottles or those that had come into contact with the strong chemicals found in many commercial dishwashing detergent formulas.

Additionally, a University of Missouri study, as reported in the July 2003 issue of Environmental Health Perspectives, found that the BPA leaching problem isn,t restricted to older polycarbonate bottles. Instead, researchers found detectable levels of BPA leaching out of brand new bottles at room temperature. This new finding calls into question the safety of any kind of polycarbonate plastic container, new or used, for food or beverage use.

Unfortunately, polycarbonate plastic bottles and containers are identified by the plastic recycling symbol #7, which is used for a wide variety of plastics and plastic mixtures that fall into the "Other category. Unless this #7 symbol is accompanied by the letters "PC, there,s no sure way to tell if the container in question is made from polycarbonate or some other kind. To be safe, environmental advocates suggest simply avoiding #7 plastics altogether and opting for safer choices for food and beverage storage. These better options include polypropylene (#5 PP), high density polyethylene (#2 HDPE), and low density polyethylene (#4 LDPE). No evidence has been found to suggest that these plastics leach toxic materials. Scientists advise against the repeated use of plastic water bottles made from plastic type #1 PETE as there is evidence to suggest that such bottles leach a compound known as DEHA, which is classified by the EPA as a possible human carcinogen, as well as acetaldehyde, which has received the same designation from the International Agency for Research on Cancer.

In addition to outdoor water bottles, it should be noted that polycarbonate plastic has also been used in the past for many baby bottles. Although environmental experts note that manufacturers have quietly substituted other, safer plastics for their baby bottle products since the BPA leaching issue first emerged in 1998, these products have not been banned and may still be found in some stores. Parents and other child care professionals are advised to check new and existing bottles for the #7 code and dispose of them. Safer substitutes (see above) are easy to find.

Babies aside, consumers unwilling to part with their polycarbonate water containers should wash any they own only with very mild soap and water and throw away any that have interior scratches, discolored areas, or other signs of aging. Stainless steel and glass containers, though heavier, are healthier. Nalgene also manufactures a model of their classic water bottle made from safe HDPE plastic.

From **Non-Toxic Times**, the newsletter produced by **Seventh Generation**

http://leas.ca/On-the-Trail-of-Water-Bottle-Toxins.htm
Updated: October 13, 2005

Book Review: *Hormone Deception*

By Dr. Lindsey Berkson

I have known Dr. Lindsey Berkson for many years, dating from the time that she lived in northern California In her excellent book ***Hormone Deception*** (Contemporary Books), Lindsey educates woman about the myriad of substances found in our food and environment that masquerade as hormones and can disrupt a woman's delicate hormonal system.

Lindsey's personal story of exposure to dangerous synthetic estrogen call DES(or diethylstilbestrol) is one of the most poignant and heart-wrenching tales of American women between the years 1938 and 1971, was given DES by her obstetrician in order to prevent a miscarriage while pregnant. This ghastly mistake on the part of conventional medicine has caused incredible suffering to millions of women and their families.

In Lindsey's case, exposure to DES threw her into a lifelong struggle with female-related health problems. The DES predisposed her to a state of hormone imbalance in which she produced unopposed estrogen without the balancing effect of sufficient progesterone. As a result of her hormone imbalance, she suffered from heavy menstrual bleeding, a 10-day menstrual period, and severe cramps. At 19 years of age she was diagnoses with severe cervical dysplasia—a pre-cancerous condition.

Over the next few decades she also developed other hormone-related health problems. In 1976, she underwent a lumpectomy for a benign tumor in her right breast; in the 80s she was diagnosed with endometrial polyps; and finally, in 1991 she had to undergo a hysterectomy for large fibroid tumors, numerous cysts of the fallopian tubes, and severely painful intrauterine endometriosis.

You would think that her hysterectomy marked the end o fall her DES-related female problems, but this was not the case. In the 90s she was also diagnosed with estrogen-dependent breast cancer—all before the age of 50. In ***Hormone Deception***, Lindsey unravels this complex world of hormone disruptors, how they adversely affect your health, and what you can do to protect yourself.

What Message Are You Sending?

According to Dr. Berkson, more than 87,000 different chemicals have been introduced into the world in the last 50+ years, and very few of these have been adequately tested to determine if they pose a health risk.

In the meantime, these chemicals have been entering our bodies via inhalation, ingestion, or through skin contact and having a profound impact on our hormonal signal/response system. Normally, hormonal signals come from inside our body, but thanks to the new, alien chemicals, our systems are being bombarded with outside signals—and we aren't equipped to properly, and safely, translate these messages.

Dr. Berkson states, "An incorrect message sent by chemical that mimics a hormone can tell the genes to turn on when they should be turned off, or vice versa. Creating a disturbance at any one point can throw things off balance anywhere else in the body."

She goes on to explain, "In wildlife, when hormone disruptors enter an animal's body, these 'wrong messages' have been show to mimic, amplify, or block the working of the animal's own hormones. What happens? Male fish start making female egg proteins. Panthers are born with undescended testicles. Male Alligators have such small penises they cannot reproduce.

Clearly, we have a critical problem on our hands.

Environmental Pollutants and Breast Cancer

Julia Green Brody and Ruthann A. Rudel

Silent Spring Institute, Newton, Massachusetts, USA

Breast cancer is the most common cancer in women and the leading cause of cancer death among women 35–54 years of age. Rising incidence, increased risk among migrants to higher risk regions, and poor prediction of individual risk have prompted a search for additional modifiable factors. Risk factors for breast cancer include reproductive characteristics associated with estrogen and other hormones, pharmaceutical hormones, and activities such as alcohol use and lack of exercise that affect hormone levels. As a result, investigation of hormonally active compounds in commercial products and pollution is a priority. Compounds that cause mammary tumors in animals are additional priorities. Animal models provide insight into possible mechanisms for effects of environmental pollutants on breast cancer and identify chemical exposures to target in epidemiologic studies. Although few epidemiologic studies have been conducted for chemical exposures, occupational studies show associations between breast cancer and exposure to certain organic solvents and polycyclic aromatic hydrocarbons (PAHs). Population-based studies have been limited to a few organochlorine compounds and PAHs and have been mostly negative. A variety of challenges in studies of breast cancer and the environment may have contributed to negative findings. Lack of exposure assessment tools and few hypothesis-generating toxicologic studies limit the scope of epidemiologic studies. Issues of timing with respect to latency and periods of breast vulnerability, and individual differences in susceptibility pose other challenges. Substantial work is needed in exposure assessment, toxicology, and susceptibility before we can expect a pay-off from large epidemiologic studies of breast cancer and environment. *Key words:* **benzene, breast cancer, carcinogens, endocrine-disrupting compounds, estrogen, hormonally active agents, organic solvents, PAHs, pesticides.** *Environ Health Perspect* **111:1007–1019 (2003). doi:10.1289/ehp.6310 available via** *http://dx.doi.org/* **[Online 19 May 2003]**

Breast cancer is the most common cancer in women (Parkin et al. 2001). Incidence is highest in North America, Northern Europe, and Australia, where age-adjusted rates are 75–92 per 100,000 women (standardized to year 2000 world population), and lowest in Asia and Africa, where incidence is less than 22 per 100,000 (Parkin et al. 2001). Mortality has increased steadily from the 1960s until the late 1980s, when rates declined in many countries, including the United States (Parkin et al. 2001). Mortality continued to climb, however, for African Americans, whose mortality rates have exceeded the U.S. average since the 1980s (SEER 2002). Worldwide, breast cancer incidence continues to rise in all age groups, with an increase in U.S. age-adjusted incidence of more than 40% from the early 1970s to the late 1990s (Clegg et al. 2002; SEER 2002). An estimated 203,500 new invasive breast cancer diagnoses are expected in the United States this year, 54,300 *in situ* cases, and 45,000 deaths (ACS 2002). About 40% of new invasive cases are diagnosed in women younger than 60 years of age (ACS 1996), and breast cancer is the leading cause of cancer death among women 35–54 years of age (National Center for Health Statistics 1997).

The threat to women in mid life coupled with observations of substantial temporal and geographic variation and poor prediction of individual risk has prompted a search for modifiable risk factors. Because breast cancer risk changes over time and varies across geographic locations, factors associated with these variations may provide clues that can lead to prevention. Thus far, many correlates of risk have been identified, including a constellation of hormone-related reproductive factors. These factors account for a substantial portion of the variation in incidence, while also providing evidence that additional factors, probably modest in magnitude, remain to be discovered.

Taken together, epidemiologic studies of hormonal factors in breast cancer and animal studies of the hormonal activity and carcinogenic potential of certain synthetic chemicals suggest environmental pollutants as possible sources of risk. Compounds identified in laboratory studies as mammary carcinogens or hormonally active are in common commercial products and are ubiquitous pollutants to which women in industrial societies are widely exposed, so identifying effects on breast cancer has the potential for substantial public health impact, even if the relative risk associated with exposure is low.

In this article we identify promising leads in the study of environmental pollutants and breast cancer and the challenges in pursuing them. As background, we provide an overview of incidence trends and well-established and suggested breast cancer risk factors that inform environmental research. We review animal studies of chemicals that may be breast carcinogens, promote growth of breast cells and hormonally sensitive tumors, or affect mammary gland development and susceptibility. We assess current knowledge from the few epidemiologic studies of environmental pollutants, discuss the barriers to further progress, and identify research needs.

Background

Trends in incidence and mortality. The association between breast cancer risk and industrial development, historically and worldwide, is one indicator of modifiable risk. Increased access to mammography and other forms of screening is generally believed to play a role in rising incidence, particularly during the early to mid-1980s, but does not explain increases in risk before 1980 or increasing risk for younger and older women who are less likely to be screened or in developing countries with low screening rates (Ursin et al. 1994).

Currently, incidence is rising most rapidly in low-risk populations both internationally (Parkin et al. 2001) and in the United States (SEER 2002), suggesting that ongoing cultural change is a primary contributor. For example, incidence for Asian-American women at the beginning of the 1990s was 40% lower than for U.S. non-Hispanic white women but increased 19% by 1998 compared with 7% increase for non-Hispanic whites (SEER 2002).

In Los Angeles County, California, where ethnic diversity allows for more detailed analysis of trends in ethnic populations, incidence among non-Hispanic whites is 20% higher than for African Americans and roughly double the rate for Hispanics and Asian Americans; in contrast, the rates of change are highest among Asian Americans. Los Angeles County breast cancer incidence rose by 1.1% per year in 1993–1997 among non-Hispanic whites, 2.1% in Hispanics, and 4.6% in Asians, while declining by 0.3% for

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African Americans (Deapen et al. 2002). By the late 1990s, rates for women of Japanese and Filipino heritage were approaching rates for non-Hispanic whites.

Surveillance data for Asian-American women are consistent with studies of migrant populations showing that when women migrate from low- to high-risk countries and vice versa, their risk and the risk in successive generations change to approximate the levels in the destination country (Kliewer and Smith 1995). Further, a population-based case–control study of Asian migrants to California and Hawaii showed higher risk associated with longer residence in the United States (Ziegler et al. 1993); and for U.S.-born Asian women, the study showed higher risk for those with more U.S.-born grandparents, an indicator of acculturation. The relative risk associated with migration changed only slightly after controlling for menstrual and reproductive factors, providing evidence that other factors contribute to migration effects (Wu et al. 1996).

Although migration studies provide insight into the contribution of sociocultural factors and support the idea that heritable factors are not predominant determinants of breast cancer risk, studies of heritable genes add a complementary perspective. Mutations in the breast cancer genes *BRCA1* and *BRCA2* are estimated to account for fewer than 10% of cases (Claus et al. 1996), although additional genes that affect hormone synthesis and metabolism and DNA repair likely add to heritable risk (Martin and Weber 2001). The effect of the broader range of heritable genes is seen in studies of identical (monozygotic) and fraternal (dizygotic) twins. In a study of 45,000 twin pairs, 14% of monozygotic twins and 9% of dizygotic twins were concordant for breast cancer diagnosis (Lichtenstein et al. 2000), and Mack et al. (2002) reported slightly higher concordance.

Reproductive and other previously studied risk factors. The fact that reproductive characteristics affect breast cancer risk has been known since 1700, when Ramazzini reported higher incidence among nuns (Spratt et al. 1995). Factors now known to confer higher risk include older age and being female, younger at menarche, older at menopause, nulliparous, and older at a first live birth or stillbirth; whereas higher parity, longer lactation, and bilateral ovariectomy are protective (Davis et al. 1997; Kreiger et al. 1999; Parazzini et al. 1997).

Reproductive risk factors are associated with exposure to estradiol, progesterone, and other hormones; and reproductive hormones are also believed to underlie increased risk associated with alcohol consumption, lack of physical activity, higher body mass index and weight gain after menopause, and low premenopausal body mass index (Bernstein et al. 2002). In addition, recent studies provide some evidence that *in utero* hormonal exposures characteristic of certain pregnancies affect breast cancer risk in the offspring. Daughters exposed to lower hormone levels in pregnancies with toxemia or pre-eclampsia are at lower breast cancer risk, whereas higher hormone levels in pregnancies with twins result in higher risk (Bernstein et al. 2002). This is a new area of research with some inconsistencies within the limited number of studies completed.

Pharmaceutical hormones similarly affect risk. Both estrogen-only and estrogen-progesterone hormone replacement therapy (HRT) for postmenopausal women increase breast cancer risk. In a pooled analysis of 51 studies involving about 54,000 postmenopausal women, the relative risk of breast cancer for women with at least 5 years of recent use was 1.35 [95% confidence interval (95% CI), 1.21–1.49] (Collaborative Group on Hormonal Factors in Breast Cancer 1997). Women who stopped using HRT more than 5 years before were not at higher risk. Additional large-scale population-based epidemiologic studies show 10% increased risk after 5 years of use for estrogen alone and 40% after 15 years, and 30% increased risk for less than 5 years of use for combination HRT (Bernstein et al. 2002). In a clinical trial of combination HRT versus placebo, the Women's Health Initiative reported a hazard ratio of 1.26 (95% CI, 1.00–1.59) about 5 years after enrollment and higher risk for women with prior HRT use up to a hazard ratio of 1.81 (95% CI, 0.6–5.43) (Women's Health Initiative Investigators 2002). For oral contraceptives, recent, but not long-term, use is associated with higher risk (Bernstein 2002), with about 26% increased risk for current users (Collaborative Group on Hormonal Factors in Breast Cancer 1996). Additional information will become available as more women with long-term oral contraceptive use reach the ages of higher breast cancer risk. Diethystilbestrol (DES), a potent synthetic estrogen, has been linked to increased breast cancer risk in women who took DES during pregnancy (Colton et al. 1993; Titus-Ernstoff et al. 2001).

Diet seems very likely to affect breast cancer risk, as it does in animals, but epidemiologic studies have failed to identify specific dietary constituents that increase or decrease risk. Effects of fat and fruits and vegetables have been extensively studied, so far providing no consistent evidence of dietary risk factors (Gandini et al. 2000; Holmes et al. 1999; Hunter and Willett 1996; Michels 2002; Smith-Warner et al. 2001; Willett 1999). High soy intake in Asia has been proposed as a factor in reduced breast cancer rates there, although epidemiologic studies so far provide limited evidence of a protective effect (Adlercreutz 2002; Hilakivi-Clarke et al. 2001; Trock et al. 2000). One recent study of Asian Americans reported a protective effect for soy that was most pronounced for high soy intake beginning in adolescence (Wu et al. 2002), and this study illustrates newer approaches to diet that explore possible effects of the timing of exposure. Other new approaches focus on possible interactions of multiple aspects of diet, for example, alcohol and folate (Feigelson et al. 2003; Zhang et al. 2003), or between diet and genetic polymorphisms (Zheng W et al. 2002).

Ionizing radiation is a clearly established environmental cause of breast cancer (NRC 1990). Studies of atomic bomb survivors and women exposed to X-ray medical treatments in childhood indicate that exposures early in life impart greater risk than adult exposures. In studies of exposed Japanese women 35 years after the atomic bomb, risk of breast cancer was 4-fold greater in women younger than 4 years of age and 2-fold greater in women 10–14 years of age compared with women 20–30 years of age at the time of the bombing. Women younger than 40 years of age had a greater risk than those older than 40 at the time of bombing (Land 1995; Tokunaga et al. 1987).

Higher socioeconomic status (SES), usually measured by education level and income, is consistently associated with higher breast cancer risk, although education and income clearly are not themselves causal. This relationship is often seen even after controlling for breast cancer risk factors such as parity and age at childbearing, which are themselves associated with SES. The possibility that some part of this relationship is due to chemical exposures, for example, from use of consumer products and pesticides, warrants further study. In a small exploratory survey of breast cancer risk factors in high- and low-incidence neighborhoods, higher SES women reported significantly higher use of several different pesticides (home and lawn chemicals, repellents, and lice control) and of dry cleaning (Maxwell et al. 1999).

Role of previously studied risk factors in incidence patterns. Women diagnosed with breast cancer, as with other diseases, often ask themselves, Why me? In recent years, communities with high incidence have struggled with that question as well. A few studies have tried to address these questions at both the individual and population levels, and these studies are interesting because unexplained variation can motivate and inform studies of new hypotheses.

At the individual level, Gail et al. (1989) developed a model that predicts risk from a woman's age, age at menarche, age at first live birth, number of previous biopsies, and

number of first-degree relatives with breast cancer; and this model has been used, among other things, as a basis for identifying women considered high risk as candidates for chemoprevention trials of treatments such as tamoxifen and raloxifene. Using data on breast cancer incidence and risk factors in two large national surveys, Madigan et al. (1995) estimated that 41% of breast cancer risk in the United States is explained by later childbearing, nulliparity, higher income, and family history of breast cancer.

Regarding geographic patterns within the United States, mortality is highest in the Northeast and West and intermediate in the Midwest compared with the South (National Cancer Institute et al. 1999). Sturgeon et al. (1995) reported in an ecologic analysis that recognized breast cancer risk factors accounted for nearly all regional variation in mortality among women younger than 50 years of age; however, among older women, adjustment reduced excess incidence by 50% for the Northeast and Midwest and 10% for the West compared with the South. A similar analysis of the Nurses' Health Study improved on the Sturgeon et al. method by adjusting at the individual level rather than regional level for established risk factors (Laden et al. 1997). However, little variation in breast cancer risk across regions was observed either before or after adjustment, perhaps due to the relative homogeneity in the risk-factor profile of nurses nationwide, so results are not informative.

The extent to which known breast cancer risk factors account for geographic variation is a subject of particular interest in areas such as Cape Cod, Massachusetts, and Marin County, California, where incidence is higher than in a comparison population such as the entire state. Surveillance data show about 20% higher risk on Cape Cod in 1982–1994 (Silent Spring Institute 2000), and case–control data from a statewide study (the Collaborative Breast Cancer Study) show about 20% excess risk for Cape Cod women older than 50 years of age compared with others in Massachusetts, after controlling at the individual level for many recognized and hypothesized breast cancer risk factors (Silent Spring Institute 1998).

In Marin County, where elevated rates of breast cancer were first reported in the 1990s, incidence increased 6 times faster than statewide during the 1990s, rising 3.6% per year (Clarke et al. 2002). A comparison of Marin County with California census block groups that were comparable for census characteristics associated with breast cancer risk showed similar incidence rates in block groups with similar percentage white population, urban status, average parity, median household income, percentage with a college degree, percentage with a working class occupation, and percentage below the poverty line (Prehn and West 1998). Another study reached similar conclusions but relied on risk factor data for women 20–55 years of age, an age group unlikely to be representative of most women with breast cancer, who tend to be older (Robbins et al. 1997). Analysis of demographic factors is not a stopping point for analysis of rate variations, however, because the SES variables are not explanatory for disease.

Aside from the role of established breast cancer risk factors, higher rates of screening mammography could contribute to higher reported incidence in a region. For both Cape Cod and Marin County, available evidence from patterns of stage at diagnosis (based on the expectation of more early-stage diagnoses with mammography) and surveys of mammography use, although not conclusive, is on the whole not consistent with screening as an explanation for higher incidence (Clarke et al. 2002; Silent Spring Institute 1998).

An earlier experience in Marin County illustrates the public health value of drawing etiologic clues from geographic variation. Rapidly increasing incidence of endometrial cancer in Marin County and other affluent neighborhoods in the San Francisco Bay Area led to the identification in the 1970s of estrogen HRT as a causal factor (Austin and Roe 1979).

Insights from Animal Studies

Epidemiologic studies that consistently show increased risk associated with multiple sources of exposure to endogenous and pharmaceutical estrogen and other hormones strongly point to the hypothesis that hormonally active agents in commercial products and pollution also increase risk. Studies in laboratory animals, *in vitro* assays, and wildlife provide further evidence of mechanisms for effects of environmental pollutants on breast cancer risk through exposure to compounds that mimic or disrupt hormones that promote or inhibit tumor growth, act as breast carcinogens, or affect the development and vulnerability of the breast. Although the processes by which breast cancers develop are poorly understood, a review of the primary features of mammary gland development and the effects of hormones and chemicals on mammary gland carcinogenesis in animal models shows that the mechanisms that underlie the recognized risk factors for breast cancer in humans are also seen in animal studies. This section outlines current research related to biological mechanisms for breast cancer, including chemical and hormonal factors and the hypothesis that hormonally active chemicals—also known as endocrine disruptors—affect breast cancer. This information provides the essential scientific foundation for evaluating existing hypotheses about environmental factors in breast cancer and generating new hypotheses and directions for future research.

Mechanistic models for cancer. Historically, carcinogenesis has been characterized by three separate stages: initiation, promotion, and progression. Although the process of carcinogenesis is now recognized as more complex than this simple model suggests, the three-stage model still provides a useful paradigm by which chemicals can be described based on a potential mechanism of action (Barrett 1993; Pitot et al. 2000). Initiation is characterized as an irreversible change in a cell, very probably a genetic change or mutation, resulting in a latent neoplastic cell (Appel et al. 1990; Pitot 1993; Pitot and Dragan 1991). Promotion is the process by which an initiated cell expands clonally into a visible, benign tumor (Barrett 1993). Experimental evidence demonstrates that chemically modulated promotion of a cell requires repeated exposure; endogenous estrogen is thought to affect the process of mammary carcinogenesis primarily by this mechanism. Progression is the term used to describe the irreversible transition from a benign to malignant tumor, which involves additional genetic events, although not necessarily point mutations in DNA (Barrett 1993; Pitot 1993; Pitot and Dragan 1991).

Agents that are carcinogens are often genotoxic, or able to damage DNA. Both initiation and progression steps involve some level of genotoxicity, whereas tumor promotion more typically involves stimulation of cell proliferation. Many agents stimulate cell proliferation, and there is controversy over whether these should be considered carcinogens unless they can also induce some level of genetic damage (Alden 2000; Klaunig et al. 2000). Of course, increasing cell proliferation also increases the opportunity for spontaneous mutations, so even promoters can have some impact on DNA integrity.

Another model for carcinogenesis focuses on cell–cell interactions that maintain tissue organization in normal tissue and break down in carcinogenesis (Sonnenschein and Soto 1999). The role of stromal cells in inhibiting or promoting carcinogenic progression in breast epithelia is an ongoing area of research (Barcellos-Hoff 2001; Barcellos-Hoff and Ravini 2000; Mueller et al. 2002), and this work suggests that the study of chemical carcinogenesis must consider effects on cell signaling as well as traditional genotoxic effects.

Mammary gland development and susceptibility. The breast is one of the few organs that is not fully developed at birth. It reaches its fully differentiated state only through the hormonal stimuli induced by pregnancy and lactation, resulting in portions of the life cycle with increased susceptibility to carcinogens. Aspects of development that

are known to affect gland susceptibility include rates of cell proliferation, stages of cell differentiation, and prenatal imprinting of hormonally sensitive tissues.

Greater susceptibility to genotoxic agents is expected during periods of rapid breast cell proliferation, such as prenatal, perinatal, and pubertal time periods and during pregnancy (Russo and Russo 1996; Wolff et al. 1996). Rodent studies of dimethylbenzanthracene (DMBA)-induced mammary tumors have shown a greater number of tumors and shorter latency when the carcinogen is administered to immature animals (Dunnick et al. 1995). Similar findings of increased risk for earlier age at exposure are observed in human studies of atomic bomb survivors (Tokunaga et al. 1987).

In addition to susceptibility during periods of cell proliferation, the susceptibility of the mammary gland to carcinogen exposure decreases after the first full-term pregnancy, when formerly undifferentiated cells have developed into fully differentiated cells, which are less susceptible to genetic damage and subsequent propagation of the damaged cell (Neumann et al. 1996; Russo and Russo 1996; Wolff et al. 1996). Epidemiologic studies have consistently shown that early age of first full-term pregnancy is a protective factor for breast cancer, and studies in animal models demonstrate that virgin rats are significantly more susceptible to chemically induced mammary gland cancers than are age-matched parous rats, which are relatively resistant to tumors (Brisken 2002; Russo and Russo 1998). Indeed, ductal and lobular carcinomas tend to originate from undifferentiated cells, whereas benign breast tumors tend to originate from the more differentiated cells (Russo and Russo 1996). Characterizing the specific hormonal factors that are responsible for the refractoriness of mammary glands postpregnancy is a topic of ongoing research (Brisken 2002; Sivaraman and Medina 2002).

Because the breast is particularly susceptible to carcinogen exposure up until the first full-term pregnancy, there may be an interaction between risk associated with age at first pregnancy, an established breast cancer risk factor, and risk associated with chemical exposure. In other words, in a hypothetical group of women with similar lifetime exposures to a mammary carcinogen beginning in childhood, those who were youngest at their first full-term pregnancy would experience the lowest increase in risk, and those who were oldest would experience the greatest increase in risk.

In addition, a number of studies in humans and animal models suggest that the *in utero* environment affects subsequent breast cancer risk in offspring (see preceding discussion of human studies). Animal studies have shown that administration of estradiol or DES during pregnancy increases breast cancer rates in female offspring (reviewed in Hilakivi-Clarke et al. 2001). One mechanism that has been proposed involves imprinting of mammary gland tissues *in utero*, resulting in an effect on the responsiveness of the tissues to estrogen later in life.

Hormonal factors in mammary carcinogenesis. Throughout the life cycle, the hormonal environment plays a critical role in the development of breast cancer. Removal of both ovaries reduces risk, and increased risk has been observed for women with higher levels of endogenous and pharmaceutical estrogen exposure (Henderson and Feigelson 2000). In animal studies, treatment with chemical carcinogens does not produce mammary tumors in the absence of endogenous hormones (Russo and Russo 1996, 1998). In other words, animals that have had their ovaries removed do not develop mammary tumors even after exposure to carcinogens. Supplementing animals with extra estrogens produces tumors even in the absence of specific chemical exposures (Russo and Russo 1996, 1998). These findings are consistent with the idea that estrogens are promoters of mammary tumors, which act over a long period of time by causing cell proliferation and clonal expansion of initiated cells. In addition, estrogens appear to be required for mammary carcinogenesis to occur.

Studies of normal mammary gland development and chemically induced mammary carcinogenesis in animal models have provided useful information for clarifying how the interplay of ovarian, pituitary, and placental hormones, while influencing the structure, organization, and function of the mammary gland, modulate its response to chemical carcinogens. Many hormones and growth factors have been demonstrated to affect the tumorigenic response of rats to genotoxic mammary carcinogens, including ovarian, placental, pituitary, and thyroid hormones, as well as androgens, insulin, and many growth factors (Brisken 2002; Neumann et al. 1996; Russo and Russo 1998; Sivaraman and Medina 2002; Swanson and Unterman 2002). In human studies, androgens and insulin-like growth factor 1 have been shown to be associated with risk of breast cancer (Toniolo et al. 2000; Wang et al. 2000).

Some researchers characterize certain estrogens, including the primary active endogenous estrogen 17β-estradiol, common pharmaceutical estrogens, and the synthetic estrogen DES, as carcinogens on the basis of their significant role in hormonally mediated cancers in humans and animals (Tsutsui and Barrett 1997). Others do not consider endogenous hormones to be carcinogenic themselves but acknowledge their role as promoters of carcinogenesis because they allow neoplastically transformed cells initiated by other carcinogens to establish and grow by modifying the target tissue (Russo and Russo 1996, 1998). In addition to acting as promoters, DES, 17β-estradiol, and certain metabolites of 17β-estradiol, including 16β-hydroxyestrone, have been shown to exhibit specific types of genotoxic activity under certain conditions (Liehr et al. 1990; Telang et al. 1992; Tsutsui and Barrett 1997). Steroidal estrogens are listed as known human carcinogens in the *Report on Carcinogens, Tenth edition* by the U.S. National Toxicology Program (NTP 2002).

Chemical factors in mammary carcinogenesis. Experimental studies in animals offer an alternative means for identifying potential carcinogens in the environment, given that epidemiologic studies require a large number of women, a long duration, and adequate exposure information. The NTP has studied the carcinogenic potential of about 500 chemicals in animal carcinogenicity bioassays. Of these chemicals, 42 caused mammary tumors in the tests (Bennett and Davis 2002; Dunnick et al. 1995). These are listed in Table 1, along with information about their common uses. These chemicals include halogenated chemicals and solvents, including components of gasoline; aromatic amino/nitro compounds; dyes; and epoxides. Other research organizations that have conducted animal carcinogenicity bioassays on specific chemicals have identified about 160 additional chemicals as mammary carcinogens (Wolff et al. 1996). These include, for example, products of combustion [polycyclic aromatic hydrocarbons (PAHs), nitro-PAHs], ionizing radiation, common industrial solvents and other industrial chemicals (vinyl chloride, vinyl fluoride, vinylidene chloride, styrene, acrylamide), pesticides (atrazine, dichlorvos), and other substances (IARC 1999; Pinter et al. 1990). Many of the chemicals identified as mammary carcinogens in these bioassays also show evidence of genotoxicity. For example, in their review of 34 chemicals identified as mammary carcinogens by the NTP, Dunnick et al. (1995) report that 26 showed evidence of mutagenicity in the *Salmonella* assay.

Chemicals identified as mammary carcinogens in animal studies are priorities for follow-up study in humans. Only four of the 42 chemicals tested by the NTP (benzene, 1,3-butadiene, ethylene oxide, C.I. acid red 114) have adequate human evidence of carcinogenicity to be classified as carcinogenic in humans (NTP 2000). Although the breast is not the primary tumor site for any of these four chemicals, many of the human cohorts studied were all or predominantly male, and

some limited epidemiologic evidence supports the breast as a tumor site for ethylene oxide (the sterilant) and benzene (in gasoline) (see additional discussion further below) (Hansen 2000; Petralia et al. 1998; Tompa et al. 1999). In addition, some animal mammary carcinogens identified in other testing programs also have epidemiologic evidence of breast cancers from occupational studies, including, for example, methylene chloride, PAHs, and chlorinated solvents (Hansen 1999, 2000; IARC 1999; Petralia et al. 1999).

Potential role of hormonally active chemicals. Recent research sheds light on a class of hormonally active chemicals, referred to as endocrine disruptors, that may affect breast cancer primarily by promotional mechanisms, as well as by affecting mammary gland development and responsiveness to other carcinogens. The hypothesis has been put forward that exposure to endocrine disruptors, including chemicals that mimic estrogens, might play a role in breast cancer risk (Davis et al. 1993). To date, more than 500 chemicals have been found to be weakly estrogenic in various assays, including many chemicals in common use, such as constituents of detergents, pesticides, and plastics (Jobling et al. 1995; Nishihara et al. 2000; Soto et al. 1995). Table 2 lists selected classes of these chemicals, specific examples, and common uses. Many of these chemicals have been shown to mimic estrogen in a variety of short term *in vitro* assays; they bind the estrogen receptor, initiate transcription of estrogen-regulated genes, and can stimulate breast cancer cells *in vitro* to proliferate (Korach and McLachlan 1995; Shelby et al. 1996; Soto et al. 1995). Short-term *in vivo* assays, such as increase in uterine weight in rodents, are also used to demonstrate estrogenic activity (O'Connor et al. 1996). In addition, effects of these compounds have been frequently observed in wildlife; for example, widespread sexual disruption of wild fish has been reported in rivers receiving wastewater effluent, which contains a mixture of endogenous and pharmaceutical estrogens and industrial chemical endocrine disruptors (Jobling et al. 1998).

Table 1. Chemicals associated with increased incidence of mammary gland tumors in rats and/or mice in testing by the NTP.[a]

Chemical	Use
Acronycine	Pharmaceuticals
Benzene[b]	Gasoline, solvent
2,2-bis(Bromomethyl)-1,3-propanediol	Flame retardant
1,3-Butadiene[c]	Auto exhaust, rubber manufacture, gasoline
C.I. acid red 114[c]	Dye for silk, jute, wool, leather
C.I. basic red 9 monohydrochloride[d]	Dye for textiles, leather, paper, biological stain
2-Chloroacetophenone	Flame retardant
Chloroprene[d]	Used in neoprene manufacture
Clonitralid	Molluskicide
Cytembena	Pharmaceuticals
2,4-Diaminotoluene[d]	Intermediate in dye synthesis
1,2-Dibromo-3-chloropropane[d]	Soil fumigant, pesticide
1,2-Dibromoethane[d]	Soil fumigate, lead scavenger in gasoline
2,3-Dibromo-1-propanol	Flame retardant
1,1-Dichloroethane	Solvent
1,2-Dichloroethane	Solvent, chemical intermediate in insecticide formulations, gasoline
1,2-Dichloropropane (propylene dichloride)	Chemical intermediate, solvent in dry cleaning fluids, fumigant
Dichlorvos	Pesticide
1,2-Dimethoxybenzidine dihydrochloride[d]	Dye intermediate
3,3′-Dimethylbenzidine dihydrochloride	Dye intermediate
2,4-Dinitrotoluene	Dye intermediate, explosives, propellants
Ethylene oxide[b]	Sterilizing gas for medical equipment
Furosemide	Pharmaceutical
Glycidol[d]	Stabilizer in vinyl polymers, intermediate in pesticides and fragrances
Hydrazobenzene[d]	Dye intermediate, tobacco pesticides, motor oil
Isophosphamide	Pharmaceuticals
Indium phosphide	Microelectronics, semiconductors, injection lasers, diodes
Isoprene	By-product of ethylene production
Methylene chloride	Solvent, furniture stripper, adhesives
Methyleugenol	Food additive, flavoring, also naturally occurring
Nithiazide	Antiprotozoal compound
5-Nitroacenaphthene	Research chemical
Nitrofurazone	Antibiotic
Nitromethane	Rocket and engine fuel, solvent, mining explosive
Ochratoxin A[d]	Mycotoxin
Phenesterin	Pharmaceuticals
Procarbazine hydrochloride[d]	Pharmaceuticals
Reserpine[d]	Pharmaceuticals
Sulfallate[d]	Herbicide
2,4- and 2,6-Toluene diisocyanate[d]	Used in manufacture of flexible polyurethane foams
o-Toluidine hydrochloride[d]	Dye intermediate
1,2,3-Trichloropropane[d]	Chemical intermediate, former solvent and paint remover

Data from Bennett and Davis (2002), Dunnick et al. (1995), IARC (1999), and NTP (2000).
[a]Listed chemicals caused cancer in mammary glands in one or more of the four typical gender–species experiments conducted on each chemical (i.e., male rats, female rats, male mice, female mice); for example, benzene caused mammary gland tumors in female mice, whereas glycidol induced tumors of the mammary gland in male and female rats and in female mice. Overall number of chemicals evaluated in NTP long-term carcinogenesis experiments, 500. Animal mammary carcinogens that were not studied by the NTP are not listed (e.g., PAHs, nitro-PAHs, ionizing radiation, vinyl chloride, vinyl fluoride, vinylidene chloride, atrazine, styrene, acrylamide; and others). [b]Listed as "known human carcinogen" in *Report on Carcinogens, Ninth edition* (NTP 2000); some epidemiologic evidence of breast cancer. [c]Listed as "known human carcinogen" in *Report on Carcinogens, Ninth edition* (NTP 2000). [d]Listed as "reasonably anticipated to be human carcinogen" in *Report on Carcinogens, Ninth edition* (NTP 2000).

As research in this area continues to identify estrogenic compounds, significant questions are raised about how to evaluate the potential adverse health effects (Rudel 1997). These questions are far from being resolved. On the one hand, the potency of many of these endocrine-disrupting pollutants is typically much lower than the potency of endogenous estrogens, and so it has been proposed that their effects will be insignificant (Safe 1995). On the other hand, there is particular concern about the effects of endocrine-disrupting chemicals for exposures that take place when levels of endogenous hormones are very low, such as *in utero* or during prepubertal, or postmenopausal time periods. Also, a number of studies have demonstrated that multiple estrogenic chemicals can act together to produce an effect even when each individual component of the mixture is present below a threshold for effect, so these pollutants can act in combination (Silva et al. 2002). Finally, comparison of the *in vivo* estrogenic effects of a range of compounds demonstrates that estrogenic compounds exhibit diversity in both mechanism and effects (Gould et al. 1998; Rudel 1997). This diversity is attributed, at least in part, to the fact that the shape of the estrogen receptor ligand (either estradiol or an endocrine disruptor) affects the binding of the receptor–ligand complex to DNA sequences and subsequent gene expression. Current research into pharmaceutical selective estrogen response modifiers (SERMs) for menopause and breast cancer prevention is an outgrowth of this phenomenon (Emmen and Korach 2001). Recent discovery of a second estrogen receptor, ER-β, complicates matters further because many hormonally active compounds have differential binding affinities for the two receptors, and cellular responses to such stimuli are difficult to predict (Pennie

et al. 1998). Thus, just because two estrogenic chemicals cause a similar effect on one outcome (e.g., uterine weight) does not mean they will cause a similar effect on all estrogen receptor–mediated outcomes.

It is of particular interest that certain dietary constituents that have been hypothesized to be preventive of breast cancer, such as genistein in soy, are also estrogenic in many endocrine disruptor screening bioassays (Adlercreutz et al. 1995). As discussed above, the relationship between soy food intake and breast cancer risk in humans is controversial. In animal studies, genistein treatment often, but not always, reduced the rate of breast cancer, with the effect being strongest with treatment before puberty (Hilakivi-Clarke et al. 2001). It is hypothesized that the genistein treatment before puberty mimics the effect of an early pregnancy (this effect has been demonstrated with estradiol also), thus reducing the susceptibility of the mammary gland to carcinogenesis (Hilakivi-Clarke et al. 2001). Additional data from animal and *in vitro* studies suggest that phytoestrogens such as genistein have mixed estrogen agonist/antagonist activity and can inhibit the biological response to endogenous estrogens, although this apparent antagonist action may not take place directly via the estrogen receptor or may be due to the differential binding of genistein to ER-α and ER-β (An et al. 2001; Ford 2002; Fotsis et al. 1993; Lamartiniere et al. 1995; Markaverich et al. 1995; Po et al. 2002). This remains an active area of research.

Table 2. Selected endocrine-disrupting chemicals.

Compound	Exposures/uses
Pesticides	
Atrazine	Selective herbicide
Chlordane	Insecticide, acaricide, veterinary pharmaceutical
Chlorpyrifos	Insecticide, acaricide
Cypermethrin	Insecticide
2,4-Dichlorophenoxyacetic acid	Herbicide
DDT (and associated compounds)	Contact insecticide
Dieldrin, aldrin, endrin	Formerly as insecticide
Lindane	Insecticide
Malathion	Insecticide
Methoxychlor	Insecticide, veterinary pharmaceutical
Pentachlorophenol	Insecticide for termite control, wood preservative
Permethrin, sumithrin	Insecticide
Toxaphene	Insecticide
Tributyl tin (chloride)	Biocide, rodent repellent
Vinclozolin	Agricultural fungicide
Persistent nonpesticide OCs and PAHs	
PAHs	Compounds present in industrial air pollutants, smoke from coal or coke-burners, tobacco tar, some foods
Polybrominated biphenyls	Formerly as flame retardant
Polybrominated diphenyl ethers	Flame retardants
PCBs (Aroclor 1254)	No longer produced commercially—since 1974, in closed electrical capacitors and transformers; before 1972, in transformers and other electrical equipment, carbonless copy paper
Dioxins and furans	Produced during incineration, paper manufacturing, and production of chlorine aromatics; impurity in some herbicides
Phenols and alkylphenols	
Bisphenol A	Polycarbonate and polyester–styrene resins
4-*tert*-Butylphenol	Intermediate in the manufacturing of varnish and lacquer resins, soap antioxidant
Nonylphenol polyethoxylate, 4-nonylphenol, 4-octylphenol	Surfactant, detergent, defoaming agent, some pesticide formulations, degradation product of alkylphenol ethoxylated antioxidant in some plastics
o-Phenylphenol	Disinfectant fungicide, in the rubber industry
Phthalates	
bis(2-Ethylhexyl) phthalate, butyl benzyl phthalate	Commonly used plasticizer for polyvinyl chloride polymers
Di-*n*-butyl phthalate, diethyl phthalate	Personal care products such as nail polish, perfume, hair spray, plasticizers, inks, adhesives, other uses
Parabens	
Butyl, ethyl, methyl, propyl paraben	Pharmaceutical aid (antifungal), preservative in foods; in creams, lotions, ointments, other cosmetics
Other organics	
Amsonic acid	In manufacturing of dyes, bleaching agents, optical brighteners or fluorescent whitening agents
Styrene	Manufacturing plastics, synthetic rubber, resins; insulator
Vinyl acetate	Used in the production of a wide range of polymers, including polyvinyl acetate, polyvinyl alcohol; widely used in production of adhesives, paints, food packaging
Metals	
Cadmium, lead	Batteries, plastic stabilizers, pigments
Mercury	Thermometers, dentistry, pharmaceuticals, agricultural chemicals, antifouling paints, many other uses
Phytoestrogens	
Genistein, coumestrol, zearalenone	Soy, grains, grain molds

Data from Budavari (1996), Harris et al. (1997), IARC (1998), Illinois Environmental Protection Agency (1997), Routledge et al. (1998), Smith and Quinn (1992), Soto et al. (1995), and SRI International (1995).

Another new and important area of research related to hormonally active chemicals concerns imprinting of the mammary gland from *in utero* exposures to hormones or hormonally active chemicals. As discussed above, animal studies and limited human studies have shown that *in utero* exposure to estradiol or DES increases mammary tumor formation in the offspring (reviewed in Hilakivi-Clarke et al. 2001). In experiments related to dietary constituents, maternal intake of fatty acids and genistein, but not soy, increased DMBA-induced mammary carcinogenesis in the offspring (even though the soy diet increased pregnancy estrogen levels) (Hilakivi-Clarke et al. 2001). Limited research has been conducted on the effects of *in utero* exposures to environmental chemicals on mammary gland development and carcinogenesis (reviewed in Birnbaum and Fenton 2003). However, two studies of *in utero* exposure of rats to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD) show effects on mammary gland development, and one shows increased susceptibility to chemically induced mammary tumors (Brown et al. 1998; Fenton et al. 2002). In addition, increased susceptibility to chemically induced mammary tumors was observed in one study of a mixture of organochlorines [OCs; e.g., dichlorodiphenyltrichloroethane (DDT), dichlorodiphenyldichloroethylene (DDE), polychlorinated biphenyls (PCBs)] given neonatally to rats (Desaulniers et al. 2001), and gestational exposure to atrazine and bisphenol A have also been shown to affect mammary gland development in rodents (reviewed in Birnbaum and Fenton 2003). It is interesting to note that all of the compounds that have been shown to affect mammary gland development after gestational exposure possess some type of direct endocrine-modulating activity (e.g., estrogen agonist, androgen antagonist, etc.).

Endocrine disruptors can also act indirectly, for example, by up- or down-regulating the enzymes that metabolize endogenous estrogens or by affecting synthesis

of endogenous hormones (NRC 1999). For example, effects of alcohol on breast cancer are hypothesized to be due to a variety of impacts on cellular signaling pathways, including increased circulating estrogen and androgen levels (Ginsburg et al. 1995; Singletary and Gapstur 2001). Although the focus of research in this area has been on measuring circulating serum or urinary levels of endogenous hormones, it is important to note that human breast tissue can metabolize hormones and create its own local hormonal environment independent of circulating levels (Adams 1991; Adams et al. 1992). Thus, effects of chemicals on the local hormone environment in the breast may be more relevant than effects on circulating hormone levels.

Overall, studies in lab animals, *in vitro* assays, and wildlife help characterize factors that influence breast development and carcinogenesis. These insights in turn inform hypothesis generation for human studies and help interpret findings in these studies. Toxicological research is a critical avenue for achieving breast cancer risk reduction because occupational epidemiology provides little information on women's cancers (see next section). Priorities for toxicologic research are outlined in the final section of this article.

Human Epidemiologic Evidence

Occupational studies. Despite the strength of toxicologic evidence for effects of certain pollutants on breast cancer risk, very little human evidence has accrued. In other areas of cancer research, leads from the laboratory often are first translated into human research in occupational studies where exposures are higher and better characterized compared with community settings, but few occupational studies have included women, so this resource is limited for evaluating breast cancer risk.

Elevated incidence has been observed repeatedly among women in white-collar jobs, due partly to reproductive risk factors, such as later childbearing, that are associated with the higher educational attainment required in these jobs and with higher SES more broadly. In some studies, associations are seen for white-collar jobs after controlling for SES and other possible confounders. For example, Band et al. (2000) observed elevated risk for teachers and medical workers. Calle et al. (1998) reported elevated risk for executives and secretaries but not teachers, librarians, or nurses, in a study that included a crude measure of physical activity, a potentially important source of confounding in studies of occupation and breast cancer. White-collar jobs do involve chemical exposures that may be related to breast cancer, including exposures to indoor pesticides, solvents, second-hand tobacco smoke, and flame retardants (Spengler et al. 2000), but these exposures are so poorly understood that most white-collar job categories are not informative with respect to questions about environmental pollutants.

Few studies have investigated breast cancer risk for women in occupations with more obvious chemical exposures, even among nurses, many of whom have substantial chemical exposures and for whom a large prospective cohort study is already in place (Nurses' Health Study 2002). Nurses are likely to have been exposed to the mammary carcinogen ethylene oxide (NTP 1998), which is used to sterilize medical equipment, and to hormonally active compounds, including nonylphenol (used in detergents and plastics) and bisphenol A (used in polycarbonate plastics) (Aschengrau et al. 1998). Two studies (Norman et al. 1995; Tompa et al. 1999) provide weak evidence of an association between ethylene oxide and breast cancer among nurses.

A few studies provide evidence of breast cancer risk associated with exposures to the mammary carcinogens benzene, PAHs, and certain organic solvents. Hansen (2000) reported higher risk of breast cancer for men exposed to gasoline and vehicular combustion products, benzene, 1,2-butadiene, 1,2-dibromoethane, 1,2-dichloroethane, and PAHs. With a lag time of at least 10 years, the odds ratio, adjusted for SES, was 2.5 (95% CI, 1.3–4.5) for exposed men, and the relative risk was more than 5-fold for men younger than 40 years of age at diagnosis (odds ratio = 5.4, 95% CI, 2.4–11.9).

Petralia et al. (1999) used interview-based lifetime job histories and a job-exposure matrix to assess women's exposure to benzene and PAHs, adjusted for breast cancer risk factors. Exposed jobs involved bus and truck operators and engine mechanics, molding and casting machine operators, and garage and service-station occupations. PAH exposures independent of benzene are also found in traffic and shipping jobs, and benzene exposures without PAHs are found among clinical laboratory technologists, painters, and sculptors. The highest risk was seen for women exposed to both benzene and PAH, with about 2-fold increased risk for women ever exposed and higher risk for women exposed for 4 or more years. Increased risk of premenopausal breast cancer was seen among women exposed to benzene. The risk of PAH exposure could not be evaluated independent of benzene because of small numbers. Results provide some evidence of higher risk with longer duration of exposure and a latency period of 20 or more years.

Organic solvents, many of which are animal mammary carcinogens, have also been associated with breast cancer in an occupational study of 7,802 Danish women diagnosed at 20–55 years of age. Breast cancer risk was increased 20–66%, adjusted for childbearing and SES, for women employed longer than a year in jobs with extensive organic solvent use (Hansen 1999). Exposed women were employed in nonadministrative jobs in industries that involved metal products, wood and furniture, printing, chemicals, and textiles. Risks were more elevated for women who worked more than 10 years in these industries and for analyses with 15 or more years lag time. A 2-fold increased risk was seen for those with more than 10 years of employment.

In a case–control study of 995 incident breast cancers in British Columbia, Band et al. (2000) reported elevated risk among women in job titles associated with exposure to solvents and pesticides. In a study of Shanghai Cancer Registry data, Petralia et al. (1998) found breast cancer standardized incidence ratios (SIRs) were most elevated for women in professional jobs, but SIRs were also 40% higher for women with high probability of exposure to organic solvents and elevated for exposure to benzene and medium and high probability of pesticide exposure, based on a small number of cases. On the basis of "usual occupation" in mortality records for 33,509 cases and 117,794 controls in 24 states in the United States, Cantor et al. (1995) reported higher risk associated with higher probability and level of exposure to styrene; the widely used organic solvents methylene chloride, carbon tetrachloride, and formaldehyde; acid mists; and several metals.

Among 115 earlier studies of occupation and breast cancer reviewed by Goldberg and Labreche (1996), a few notable associations were seen. Two cohort studies reported evidence of higher risk for women in pharmaceutical manufacturing, and higher risk was also reported for women employed as cosmetologists or beauticians. Pollan and Gustavsson (1999) similarly reported elevated incidence for pharmacists, hairdressers, and beauticians with SES controlled in a cohort of women employed in 1970. Both historical and current risk among hairdressers is of interest because the mammary carcinogen vinyl chloride was used in hairspray until the early 1970s. Knowledge of workplace practices, more generally, may lead to better understanding of potentially informative inconsistencies among occupational studies.

Elevated risk was observed in other chemical-exposed jobs among metal platers and coaters (Pollan and Gustavsson 1999), whereas Goldberg and Labreche (1996) found little support for higher breast cancer risk for women in textile production (with exposure to dyes), dry cleaning (with exposure to organic solvents), or the nuclear industry. The negative finding in the nuclear industry

despite clear evidence that ionizing radiation increases risk could mean that most workers were not actually exposed, or it could be due to protective characteristics of the workforce in that setting. For example, some jobs may attract or require women with high levels of physical activity, or sensitive workers may develop acute effects such as dermatitis and central nervous system symptoms that cause them to leave the workplace. This well-known phenomenon, referred to as the "healthy worker effect," complicates interpretation of negative occupational studies.

Similarly, breast cancer risk among farm women is of interest because of possible exposure to pesticides, but in general, observed breast cancer risk is lower among U.S. farm women, perhaps due to greater levels of physical activity or patterns in other established risk factors. Consistent with other studies, the Carolina Breast Cancer Study found that women who lived or worked on a farm had lower risk, but among those who did not wear protective clothing when applying pesticides, a 2-fold higher risk of breast cancer was observed (Duell et al. 2000). Research under way in the Agricultural Health Study will provide much better information about farm-related risk (Alavanja et al. 1994).

Overall, occupational studies provide fairly consistent evidence that elevated risk independent of SES is associated with a few specific exposures—benzene, organic solvents, and PAHs—especially for younger workers, and it is interesting to note that the chemicals with the most consistent human evidence have also been identified as animal mammary carcinogens (Table 1). Leads from previous occupational findings and new directions based on animal studies are priorities for further research, although follow-up studies will be challenging. Some of the challenges are typical of occupational studies; for example, workers are typically exposed to mixtures of chemicals, so specific exposures and exposure histories are difficult to reconstruct. In addition, using surveillance methods that are common in occupational studies makes it hard to separate out the effects of chemical exposures in populations that have protective characteristics, such as higher physical activity or lower-risk reproductive patterns. Other challenges arise from women's typical work histories, with exposed women likely to move into and out of the workforce and to be employed in dispersed, small-scale settings such as beauty shops. Goldberg and Labreche (1996) identify a number of weaknesses common in the studies they reviewed: reliance on administrative data and broad job categories as an indicator of exposure; lack of information on confounders, including childbearing and SES; use of mortality as an outcome rather than incidence, which limits the relevance to etiology; and low statistical power. Concerted efforts to overcome these limitations are important because occupational studies are the primary means by which chemicals become identified as human carcinogens (IARC 1998).

In future studies, possible confounding by work-related physical activity could be assessed using job matrix methods that parallel the assessment of chemical exposures. However, studies that contact workers to assess a broader range of established breast cancer risk factors concurrently with workplace exposures are needed to deal with other potential confounders. These studies will be most useful in evaluating chemical exposures that result in cancers diagnosed during women's working years, and longitudinal follow-up will be required to pick up effects among older women. Studies of health outcomes that are known or suspected to be related to breast cancer risk, including breast density, fertility outcomes, and age at menopause, also provide avenues to learn about breast cancer through occupational studies without waiting for workers to reach the older years when breast cancers are typically diagnosed. The likelihood, based on effect sizes for established breast cancer risk factors, that effects of occupational exposures may be modest in size means that large sample sizes or meta-analysis of multiple studies will be needed to discern effects. As more women move into jobs with substantial chemical exposure, assessment of occupational risks will become even more important.

Population-based studies. Population-based studies have investigated a narrow range of the compounds identified in the toxicologic literature as plausibly relevant to breast cancer. Certain OC compounds (DDT, PCBs) have been most studied; because they are persistent and lipophilic, residues can be measured in adipose tissue and blood years after exposure. Most studies to date have measured residues at the time of diagnosis or interview and assumed that these recent measures can be used as proxies for historical exposures. A few studies have assessed PAHs, some of which are potent mammary carcinogens in animals, and tobacco smoke, mixtures with complex toxicologic properties. Accidental exposures have led to studies of dioxin (TCDD) and perchloroethylene (PCE, also called tetrachloroethylene).

The largest recent report is from the Long Island Breast Cancer Study Project case–control study that assessed PAHs and certain OCs, based on blood samples drawn near the time of diagnosis (cases) or interview (controls) (Gammon et al. 2002a, 2002b). PAH exposure was assessed by measuring PAH–DNA adducts, a measure of DNA damage from exposure over the previous months to a few years. Results showed 49% higher risk, adjusted for breast cancer risk factors, for the highest compared with the lowest quintile of adducts (95% CI, 1.00–2.21), with no evidence of a dose–response relationship (Gammon et al. 2002a). Although the authors expected grilled food and tobacco smoke to be the primary sources of PAH, the lack of relationship between these exposures and PAH–DNA adducts suggests that other sources, for example, air pollution, may be more important. PAH–DNA adducts represent combined effects of intake and individual response, so the lack of dose response could mean that this measure is a better indicator of individual response than exposure (within the range of exposures in this study).

The Long Island study showed no significantly elevated risk associated with lipid-adjusted blood levels of the OC compounds DDE (the primary metabolite of DDT), chlordane, dieldrin, or the sum of the four most common PCB congeners, although small increases in risk were observed for the highest compared with the lowest exposure groups, with no dose–response trend, for DDE, DDT, and dieldrin (Gammon et al. 2002b). No consistent associations were seen for subgroups defined by reproductive risk factors, body size, years of residence on Long Island, or tumor estrogen- or progesterone-receptor status.

The results for DDE are consistent with scientific evidence that accumulated over the years during which the Long Island study took place. Although a few early studies reported an association with breast cancer, only 6 of 27 studies reviewed by Snedeker (2001) reported statistically significant positive associations. In her review, Snedeker offers a potential explanation for the many negative studies. She points out that most studies rely on DDE as an indicator of previous exposure to DDT because DDT is not currently detectable in blood in countries where DDT was banned years ago. However, diet (especially meat, fish, and dairy) is a major ongoing route of exposure to DDE, so DDE levels in blood represent exposure from diet as well as DDE metabolized from previous DDT exposure. DDE is much less hormonally active, so it may be that DDT, but not DDE, contributes to breast cancer, and if exposure to DDT is poorly measured by current blood levels of DDE, studies that rely on DDE are not informative. In fact, a recent study by Hoyer et al. (2000a) showed a significant relationship, with dose response, for breast cancer risk and *p,p′*-DDT measured prospectively in the late 1970s and early 1980s but no association for DDE. In addition, preliminary results from a California study using blood drawn during active DDT use showed increased risk of breast cancer

diagnosed before age 50. Serum levels were measured prospectively in 131 case–control pairs. The odds ratio was 3.9 (95% CI, 1.4–10.9) for the second versus first tertile of DDT and 10.4 (95% CI, 2.5–43.2) for the third versus first tertile, with a highly statistically significant *p*-value for trend (Cohn et al. 2002). Additional studies of DDT levels in women currently exposed around the world or in blood drawn during years when DDT was in use in the United States may be informative.

A series of analyses of the association between breast cancer and blood levels of the pesticide dieldrin in Danish women have shown significant associations and dose–response trends for 1970s blood levels and breast cancer incidence (Hoyer et al. 1998) and mortality (Hoyer et al. 2000b). Mortality was increased more than 5-fold for women with the highest dieldrin levels averaged across two measurements from the 1970s and early 1980s (relative risk = 5.76; 95% CI, 1.86–17.92) (Hoyer et al. 2000b). Subgroup analyses showed the strongest associations with breast cancer risk for estrogen-receptor–negative tumors (Hoyer et al. 2001) and for tumors with *p53* mutations (Hoyer et al. 2002). One potential explanation for these positive findings compared with other OC results is that blood measures were taken closer to the time of dieldrin use, which ended in the late 1970s, so they are better indicators of exposure.

Given the many difficulties of measuring historical exposures and characterizing variation among individuals in community settings, studies of unusual accidental exposures are a valuable resource. In a study of dioxin in women who were infants to 40 years of age at the time of a 1976 industrial accident in Seveso, Italy, Warner et al. (2002) reported a 2-fold increase in breast cancer risk among women with a 10-fold increase in serum level of dioxin (hazard ratio = 2.1; 95% CI, 1.0–4.6). Aschengrau et al. (2002) reported small to moderate increases in risk for women on Cape Cod, Massachusetts, exposed to PCE that leached from vinyl-lined water distribution pipes (adjusted odds ratios = 1.5–1.9 for > 75th percentile with 0–15 years of latency). Both of these studies have significance beyond the accidental exposure scenarios because dioxin and PCE are common exposures in everyday settings that could be reduced through changes in public policy. Dioxin is a widespread environmental contaminant, for example, from waste incineration. PCE is a solvent commonly used in industry and in dry cleaning, leading to both worker and consumer exposures.

Studies of breast cancer and tobacco smoke, including active smoking or passive exposure to environmental smoke from spouses or co-workers or in commercial and leisure settings, are more numerous than for other environmental pollutants, in part because exposure can be easily and inexpensively measured in interviews. Many early studies found no increased risk among smokers, and a recent meta-analysis of 53 studies comparing "ever" to "never" smokers found no association with breast cancer risk (Collaborative Group on Hormonal Factors in Breast Cancer et al. 2002). However, recent studies that separate active from passive exposure, consider a woman's age at exposure, and take into account genetic polymorphisms that affect the mechanism for ridding the body of smoke provide some evidence for an association, although the data are still inconsistent (Band et al. 2002; Bartsch et al. 2000; Dunning et al. 1999; Kropp and Chang-Claude 2002; Perera 2000).

In general, studies of genetic polymorphisms and breast cancer have focused on genes related to PAH and steroid metabolism (e.g., *CYP*, *GST*, *NAT2*), and studies of interaction between genetic polymorphisms and environmental pollutants have focused on tobacco smoke, with two studies of PCBs. Overall, results of these studies have been inconsistent (Bartsch et al. 2000; Basham et al. 2001; Dunning et al. 1999), with some evidence of effects of *CYP*, *GST*, and *NAT2* polymorphisms and smoking on breast cancer risk, particularly in subgroup analyses (Ambrosone et al. 1996; Bartsch et al. 2000; Chang-Claude et al. 2002; Firozi et al. 2002; Hunter et al. 1997; Morabia et al. 2000; Zheng W et al. 2002; Zheng T et al. 2002, 2003), and two positive reports for PCBs and *CYP* polymorphisms in postmenopausal women (Laden et al. 2002; Moysich et al. 1999).

Overall, the population-based studies of breast cancer and environment represent a very sparse literature. Particularly notable is the focus on smoking and a small number of persistent OCs. Even for the most-studied chemicals, the number of studies is relatively small. In comparison, the recent meta-analyses of pharmaceutical estrogens and breast cancer are based on nearly twice as many studies as have been reported for DDT/DDE.

Challenges and Priorities

A variety of challenges in conducting studies about breast cancer and the environment may have discouraged work in this area, and these challenges define areas where future study will likely have the greatest impact. In particular, lack of exposure assessment tools and lack of toxicologic studies to develop hypotheses limit the scope of epidemiologic studies. In addition, issues of timing with respect to latency and periods of breast vulnerability, and individual differences in genetic susceptibility are challenges in research design that require attention. A substantial investment is needed in basic areas that are the foundation of successful human research—exposure assessment, toxicology, and susceptibility—before we can expect a pay-off from large epidemiologic studies of breast cancer and environment.

Exposure assessment. Multiple aspects of exposure assessment present methodological challenges. As in other cancer studies, latency means that exposures must be assessed for a time period long before diagnosis. For breast cancer specifically, evidence from both animal and epidemiologic studies suggests that there may be vulnerable periods, perhaps during gestation or adolescence or between menarche and birth of a first child, when exposure is most important. In addition, effects of environmental exposures may differ before and after menopause, as seen with some previously studied risk factors (e.g., body mass index and a recent report on smoking; Band et al. 2002). These multiple timing considerations are a particular challenge in studying exposures, such as air and water pollutants, that women cannot report retrospectively, in contrast with exposures, for example, childbearing history, that comprise the recognized risk factors. As yet, none of the available biomarkers can assess exposure dating back many years, let alone decades, and it is a particular challenge to characterize exposures for specific periods of the life span (e.g., during puberty). The complexity of mixtures in both occupational and community settings is another difficulty, along with simultaneous exposure to poorly understood degradation products and metabolites of pollutants.

Recent studies include efforts to improve exposure assessment in light of these challenges. Thus, the Long Island study and new research on tobacco smoke have included a relatively novel measure of PAH–DNA adducts. The Cape Cod Breast Cancer and Environment Study, now under way, defined development of new exposure assessment methods as a core goal (Brody et al. 1996). The study developed a geographic information system (GIS), a computer-mapping database, designed first to generate hypotheses and conduct ecologic analyses and later to assess exposures to wide-area pesticide use and drinking water contamination at individual addresses of 2,100 women in a case–control study (Brody et al. 2002). GIS is also being used in exposure reconstruction in several other epidemiologic studies (Beyea and Hatch 1999; Lynberg et al. 2001; Stellman et al. 2003; Ward et al. 2000). Capitalizing on geographically based research makes sense in studies of pollutants because many exposures vary geographically in relation to sources. Examples of nationally available data include the Toxics Release

Inventory (http://www.epa.gov/tri), which documents point sources of pollutants, and records generated under the Safe Drinking Water Act (1974) for every public drinking water supply (Caldwell et al. 1998). Although some exposure data are available nationally, developing additional GIS exposure data is often more practical in a geographically limited area.

Because of enormous gaps in previous research about breast cancer and environmental pollutants, beginning with a lack of basic knowledge about the frequency and level of exposure to compounds identified as hormonally active or as animal mammary carcinogens, exposure studies that investigate these questions without yet tackling the link to breast cancer are an efficient way to proceed. For example, the Cape Cod Study developed an environmental sampling program for hormonally active compounds and mammary carcinogens in groundwater and drinking water, household air and dust, and women's urine. Results documented a potential pathway of exposure to endocrine disruptors that travel from septic systems to groundwater and drinking water, and identified 72 different hormonally active target compounds in homes, showing substantial opportunity for exposure (Rudel et al. 1998, 2001, 2002). Compounds for which frequent or high exposures have been identified and methods for measuring exposures developed might then be targeted in toxicologic and epidemiologic studies.

Considering that the ideal exposure assessment would provide information about the agent, dose, exposure pathway, timing in relation to latency, and timing in relation to lifecycle development, no one measurement technique is likely to provide a "gold standard." Self-report is vulnerable to response bias and cannot assess pollutant exposures unknown to the study participant. GIS offers a new approach to historical exposures and is independent of knowledge or bias among study participants, but it is vulnerable to missing data and faulty models of relationships between indicators and individual exposures. Environmental and biological sampling methods also may not accurately reconstruct individual historical exposure. Further, measurement methods have been developed for only a limited range of compounds, and measurements are expensive and sometimes intrusive to collect, resulting in small sample sizes with low statistical power and possible bias from nonparticipation. Analyses of relationships among environmental, biological, self-report, and GIS measures can help inform interpretation of studies using each of these exposure assessment methods and help identify sources of exposure. Studies to characterize environmental and biological exposures can also help identify populations or settings with high exposures that may provide unique opportunities for study.

Toxicology and mammary gland biology. Among 70,000 chemicals in commerce, fewer than 1,000 have been tested in cancer bioassays, and there has been no systematic testing for hormonal activity (U.S. EPA 1999). The challenge of analyzing mixtures and the idiosyncratic dose–response relationships (e.g., U-shaped) for hormones and hormonally active pollutants adds another layer of complexity. In addition, the biological and hormonal regulation of mammary gland development and carcinogenesis is poorly understood, so forming hypotheses about how chemicals will affect these processes is difficult.

Although standard animal bioassays for identifying carcinogens provide important direction for study in humans, improvements are needed in the development and application of animal models for mammary tumors specifically. For example, current protocols may not adequately address increased susceptibility to carcinogens for early-life exposures because dosing typically begins in pubertal animals (Bennett and Davis 2002). In addition, the rodent strains typically used for carcinogenesis bioassays may not be optimal for identifying mammary carcinogens, either because of a reduced susceptibility to such tumors ($B6C3F_1$ mice), because a high background rate of mammary tumors makes results difficult to interpret (Fischer 344 rats), or because hormonal regulation of the rodent mammary gland differs from that in humans (Bennett and Davis 2002; Dunnick et al. 1995; Snedeker 2001).

Another important issue for animal models is that, although it is important to identify chemical carcinogens that are genotoxic, which the current protocols are designed to do, it may also be important to identify chemicals that effectively promote the growth of cells after they have been initiated by some other carcinogen. The powerful role of endogenous hormones in promoting breast tumor development suggests that environmental chemicals that act as promoters could play an important role in breast cancer. Assays to look for tumor-promoting activity involve treating with a single dose of an initiator and then following with the promoter. In an assay like this, DDT was found to accelerate the rate of mammary tumor formation in male rats (females were not tested), suggesting that it could be active as a tumor promoter (Scribner and Mottet 1981), and wheat bran was shown to decrease the incidence of DMBA-initiated mammary tumors (Zile et al. 1998). Finally, it is also a priority to develop animal models that characterize the effects of *in utero* chemical exposures on development and susceptibility of the mammary gland in the offspring because *in utero* hormonal environments have been shown to affect later susceptibility to carcinogens (Hilakivi-Clarke et al. 2001).

Individual susceptibility and intermediate outcomes. Consideration of individual susceptibility is another area where limitations in previous research have led to recent innovation. Although high-risk breast cancer genes account for a small fraction of cases, lower risk, more common genetic polymorphisms that affect metabolism of endogenous estrogen and other chemicals are promising directions for study, as discussed above. However, studies to date have yielded conflicting results, in part because of the need for large sample sizes to achieve adequate statistical power and because of limited information on specific functional outcomes of the polymorphisms in relation to mechanisms of breast carcinogenesis (Dunning et al. 1999; Friedberg 2001; Perera 2000; Pharoah et al. 2002). This is another aspect of basic biology that could advance our ability to study breast cancer.

The difficulties of linking exposures with disease may also be remedied by studies of intermediate outcomes and of interactions or effect modification associated with recognized breast cancer risk factors. Studies of effects of chemical exposures on puberty, breast density, and *in situ* disease—all recognized risk factors for breast cancer—reduce the time lag between exposure and outcome measurement. Research to identify new intermediate outcomes, such as hallmarks of mammary gland development, will add to tools available for addressing breast cancer etiology.

Conclusion

Although journalistic reports have recently implied that scientific evidence shows that environmental pollutants are unrelated to breast cancer (Associated Press 2002; Kolata 2002), a review of research in this area reveals a much different picture of major knowledge gaps, difficult challenges in research design, and contrasting bodies of evidence from toxicologic and epidemiologic studies. Strong toxicologic evidence points to a large number of ubiquitous pollutants that are plausibly linked to breast cancer because they mimic or disrupt hormones known to affect breast cancer risk, initiate mammary tumors in animals, or permanently alter breast development, affecting susceptibility. Epidemiologic research is far more limited because very few of the compounds identified as endocrine disruptors or animal mammary carcinogens have ever been targeted in a human breast cancer study. A small but interesting body of occupational studies that link higher risk with jobs involving likely exposures to organic solvents and PAHs is generally consistent with animal studies. The relatively few population-based

epidemiologic studies have been mostly negative overall, with positive results often limited to subgroups. Many plausible reasons for null epidemiologic results have been advanced in this article and elsewhere, including poor historical exposure measurement, restriction to a small number of pollutants, failure to study compounds in current use, low statistical power to detect modest effects, and failure to take into account genetic susceptibility or life-cycle effects. Limited study of women in occupational settings where exposures are relatively high and well defined is another barrier to understanding chemical risks. Given the modest relative risks associated with the recognized breast cancer risk factors, an integrated research agenda for study of environmental pollutants in both laboratory and human settings has great potential. Even if the relative risks of environmental factors are modest, discovery of a risk that can be modified would save many thousands of lives.

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Action must now be taken

About one in 10 women in Europe will be diagnosed with breast cancer at some time in their lives.

Individuals can try to minimise their own exposure to oestrogen mimics (see box below) but these actions are not enough. Only EU and governmental action to ensure effective regulation of chemicals will provide the major health and environmental benefits that can be achieved for society as a whole.

HEAL and CHEM Trust are working to have man-made chemicals known to disrupt hormones removed from the market, and replaced with safer alternatives.

What you can do to minimise exposure

1. Eat plenty of fruit and vegetables.
2. Buy organic food whenever possible.
3. Avoid unnecessary exposure to chemicals, particularly garden and indoor pesticides, homecare products, such as paints and detergents, and personal care products including cosmetics.
4. When possible, instead of using sunscreen to avoid sunburn, keep in the shade or cover up with loose fitting but tightly woven clothes and a hat.
5. Do not microwave food in plastic containers or wrapping.
6. Visit or write to your government representative or Member of the European Parliament (MEP) to express your concerns about hormone disrupting chemicals and their links to breast cancer. Ask for tighter controls over synthetic chemicals that disrupt our hormone systems.

This leaflet has been prepared by the Chemicals Health Monitor Project (CHM), which aims to improve public health by ensuring that key scientific evidence on the links between chemicals and ill-health are translated into policy as quickly as possible.
Please see: **http://www.chemicalshealthmonitor.org**

The Chemicals Health Monitor project was launched by the Health and Environment Alliance (HEAL) with other partner organisations.

Health and Environment Alliance (HEAL) is an international non-governmental organisation that aims to improve health through public policy that promotes a cleaner and safer environment.

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CHEM Trust is a UK charity which aims to protect humans and wildlife from harmful chemicals so that they play no part in causing impaired reproduction deformities, disease or deficits in neurological function.

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Breast cancer: Preventing the preventable

Exposure to certain man-made chemicals may be contributing to the breast cancer epidemic.

Most women diagnosed with breast cancer have acquired the disease over their lifetime, rather than it being set in their genes.

Preventing breast cancer – the way forward

Expert scientists consider that enough is now known about hormone disrupting chemicals to call for action.

"Given the known role of oestrogens in breast cancer, it would be prudent to reduce exposures to chemicals that can mimic oestrogen."

A review paper, *"Breast cancer and exposure to hormonally active chemicals: An appraisal of the scientific evidence"*, by Professor Andreas Kortenkamp, published in April 2008, is the latest in a series of documents highlighting the need to reduce health risks associated with hormone disrupting chemicals.

"Despite the uncertainty, it is prudent to minimise exposure of humans, especially pregnant women to hormone disrupting chemicals."

Recommendations from the **Royal Society in the UK** in 2000.

"In view of the magnitude of the potential risks, we strongly believe that scientific uncertainty should not delay precautionary action for risk reduction."

In 2005, **the Prague Declaration on Endocrine Disruption** called for measures to reduce the risks associated with endocrine disrupting chemicals. More than 200 scientific experts from across Europe and the USA have signed the Prague Declaration.

Stopping breast cancer before it starts

The number of women developing breast cancer has increased dramatically throughout the European Union over the past 20 years.

Although women welcome the advances in screening and treatment that are helping to improve survival rates, they are also starting to ask questions: *"Could more be done to prevent so many women from developing breast cancer, and how much evidence is enough before taking action?"*

Each day, we are all exposed to dozens of synthetic chemicals found in food, cosmetics, and household cleaning products. Some of these chemicals are coming under increasing suspicion. Scientific evidence is growing that synthetic chemicals which mimic oestrogen and disrupt the so-called sex hormones may be playing an important role in the rise in breast cancer.

Currently, the different established risk factors for breast cancer account for only 50% of the cases diagnosed. They include age, genetics, alcohol consumption, exercise, use of hormone-replacement therapy (HRT) and the oral contraceptive pill. They also include a woman's total lifetime exposure to her own natural oestrogen production. This means that breastfeeding, pregnancy, or early menopause, which lower a woman's exposure to oestrogen by reducing her lifetime exposure to monthly periods, reduces the risk.

In recent years, scientists have investigated whether the man-made chemicals that mimic natural oestrogens might be contributing to the rapidly increasing number of cases of breast cancer. Although synthetic oestrogens, also known as hormone disruptors, are less potent than natural ones, their combined effects may be adding to the risks.

Laboratory studies clearly demonstrate that a mixture of hormone disrupting chemicals can cause adverse effects even when each chemical is individually at a level that should cause no problem. Other studies suggest that exposures to these chemicals in the womb and around the time of puberty may be especially important. Recently, a study in Spain showed that some women newly diagnosed with breast cancer had higher levels of these synthetic oestrogenic mimics in their body fat.

More and more scientists are concluding that the combined findings of the various studies are strong enough to require precautionary action to reduce exposure to certain chemicals, particularly hormone disruptors.

For more information, see the following reports:

- ***"Breast cancer and exposure to hormonally active chemicals: An appraisal of the scientific evidence"***, by Professor Andreas Kortenkamp, Head of the Centre of Toxicology, School of Pharmacy, London University.
- ***"Factors influencing the risks of breast cancer – established and emerging"*** by CHEM Trust, UK. A briefing for the public and breast cancer sufferers available in English and several other European languages.

Both publications were produced in the context of **the Chemicals Health Monitor** project and are available on the project website (http://www.chemicalshealthmonitor.org/) and the CHEM Trust website (http://www.chemtrust.org.uk/).

breast cancer

Breast cancer and exposure to hormonally active chemicals: An appraisal of the scientific evidence

A background briefing paper
by Professor Andreas Kortenkamp,
Head of the Centre for Toxicology,
The School of Pharmacy,
University of London

April 2008

The Health & Environment Alliance (HEAL) is an international non-governmental organisation that aims to improve health through public policy that promotes a cleaner and safer environment. Our work draws on the findings of the environmental health science revolution, which is revealing the impact of environmental degradation on health in an ever widening range of diseases and conditions. We represent a diverse network of more than 50 citizens', patients', women's, health professionals' and environmental organisations across Europe and we have a strong track record in bringing environmental health science and policy to an increasing number of fora. Our vision is that of a healthy planet for healthy people.

http://www.env-health.org/

CHEM Trust is a UK charity whose aim is to protect humans and wildlife from harmful chemicals. CHEM Trust's particular concerns are related to hormone disruptors, the cocktail effect of chemicals and the role of chemical exposures in early life. Exposure to undesirable chemicals may arise from contamination of the food chain and from the use and disposal of many everyday products such as TVs, computers, cars, construction materials, toys, toiletries and cosmetics. CHEM Trust is working towards a goal where chemicals play no part in causing impaired reproduction, deformities, disease or deficits in neurological function. CHEM Trust is committed to engaging with medical, scientific and patient communities to raise the level of dialogue on the role of chemicals in chronic disease, and the wider implications this may have for disease prevention strategies.

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Chemicals Health Monitor *aims to improve public health by ensuring that key scientific evidence on the links between chemicals and ill-health are translated into policy as quickly as possible. The strategy involves fostering dialogue, sharing perspectives and promoting greater collaboration between policy makers and governments on the one hand and scientific researchers, medical and health professionals, patient groups, environmental organisations and the public on the other. We work to highlight the compelling scientific basis for added controls over certain chemicals; and encourage EU policies that are precautionary and participatory, especially with regard to the implementation of REACH, and the substitution of hazardous chemicals.*

The project was launched by the Health and Environment Alliance (http://www.env-health.org/) in collaboration with other partner organisations across Europe in March 2007.

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Breast cancer and exposure to hormonally active chemicals:
An appraisal of the scientific evidence

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Summary

The number of new breast cancer cases among women is increasing in almost all Western countries. Although late age at first child birth and genetics are shown to contribute to the increase in breast cancer, the sheer number of newly diagnosed cases cannot solely be explained by these factors. Evidence is emerging that environmental influences, including chemical exposure, also play a role.

Studies among identical twins have shown that the most important contributors to the causation of breast cancer are environmental and lifestyle factors that differ between the pair, even under circumstances where the genetic predisposition is very similar. In families with a heritable predisposition to breast cancer, time of birth, physical activity and obesity can profoundly influence risk.

There is overwhelming evidence that oestrogens are strong determinants of breast cancer risks. This is not limited to natural oestrogens formed in a woman's body, but extends to synthetic hormones used as pharmaceuticals, including those employed for the alleviation of menopausal symptoms. The demonstration of breast cancer risks from oestrogen-only and, more pronounced, from combined oestrogen-progesterone regimens is another case in point. Very recent, rapid decreases in breast cancer incidence in the USA, Canada and in parts of Germany have followed a reduction in hormone therapy use.

Given that natural oestrogens and man-made oestrogens used as pharmaceuticals have a role in breast cancer, concerns arise about the potential contribution of industrial chemicals and pesticides with hormonal activity. Such chemicals include several that have been banned already, but can still be found in human tissues, such as polychlorinated biphenyls (PCBs) and compounds related to 1,1,1-trichloro-2,2-bis(4-chloro-phenyl)ethane (DDT). A large number of chemicals currently used in consumer products also fall in this category (phthalates, bisphenol A, UV-filter substances and many more).

To date, the few studies carried out to examine whether certain environmental chemicals are implicated in breast cancer leave much uncertainty about a possible link. But to avoid wrongly dismissing a role for chemicals in breast cancer, two issues must be addressed:

First, the available studies have largely focused on single chemicals and have ignored the possibility that large numbers of agents may act in concert. Recent evidence from Spain suggests that *cumulative* exposure to oestrogenic chemicals is associated with breast cancer risks.

Second, instead of looking at exposures later in a woman's life, when the breast tissue is perhaps less vulnerable, critical periods of vulnerability during puberty and development in the womb must be considered. Very recent studies demonstrating breast cancer risks from exposure to the pesticide DDT before or during puberty, and from in-utero exposure to the oestrogenic anti-miscarriage drug diethylstilboestrol (DES) further underline the importance of early life chemical exposure in breast cancer.

Taken together, there is a case for relinquishing the dominant view of breast cancer as a life-style and genetic disease and for reappraising the role of environmental factors, including chemical exposures. With UK breast cancer incidence at an all time high, risk reduction will not be achievable without considering preventable causes, particularly exposure to chemicals.

Breast cancer incidence rates

With a few exceptions, the number of new breast cancer cases among women is increasing in almost all Western countries. Thanks to improvements in early detection methods and the introduction of large-scale screening, the chances of surviving the disease have changed for the better, but the continuing rise in new cases places a heavy burden on health services and causes immense private suffering. The risk of contracting breast cancer is highest in Northern and Western Europe where incidence rates are rising slowly or are levelling off at high values[1]. Eastern European countries are currently experiencing the fastest rises in breast cancer. In some countries, including the USA and parts of Germany, a down-turn in the number of newly diagnosed women has been noted recently.

The UK has one of the highest breast cancer rates in the world. The number of women who received a diagnosis of breast cancer has risen steadily from 24,174 in 1980 to 43,711 in 2003[1]. As demonstrated by the latest available statistic (44,335 new cases in 2004)[2], there is a continued upwards trend in breast cancer incidence in the UK. Now in the UK, one in nine women will be diagnosed with breast cancer during their lifetime.

The rise in breast cancer incidence in the UK is often attributed to improved diagnosis by screening. There is no doubt that the introduction in 1988 of large-scale screening mammography in the National Health Service has led to a rising number of diagnosed cases, particularly in women aged 50–64 years. Typically however, the additional effect on incidence is only transient and disappears as screening measures reach saturation. In the UK this effect has lasted for 4–7 years, until 1992–1996[3]. Although the upwards trend in incidence has become more pronounced with the introduction of mammography, this has not masked a general increase. The underlying increase in breast cancer incidence in the UK predated screening and continues today. Thus, current rises in incidence are not solely due to screening.

Changes in childbearing contribute to the increase in breast cancer in the UK and in most other countries. For example, it is well established that breast cancer risks are higher among women who have their first baby late in life, or who do not have children at all. Very likely, this plays a role in the current rapid rise of breast cancer in Eastern Europe. Other factors that contribute to increased risks are lack of physical activity, weight gain and obesity after the menopause. Genetics explains a small fraction of breast cancers. Around 1 in 20 cases are believed to be due to an inherited predisposition, but for the overwhelming majority of women the disease is not passed on through genes but acquired during their lifetime[4]. Alcohol consumption[5], but not high fat diets[6] contributes to breast cancer risk.

But the sheer number of newly diagnosed cases cannot be explained solely by childbearing, genetics, lack of physical exercise or alcohol. Experts estimate that more than half of all breast cancers are due to as yet unidentified causes[7]. So what are these unexplained factors? This briefing document will appraise the evidence for a role of environmental factors, particularly chemicals, in breast cancer.

What are "Environmental factors" in breast cancer causation?

The term "environmental factors" is used ambiguously and with different connotations in the medical literature. In its broadest sense, it describes all non-genetic factors in cancer causation, such as life style, diet and infectious agents. Used in this way, the term is not very discriminating and consequently "the environment" can be implicated in the causation of most cancers. More helpful with respect to cancer prevention might be to distinguish between avoidable factors and genetic background. In the interest of avoiding involuntary exposures, this means a focus on the possible role of work place exposures, food contaminants, pharmaceuticals, chemicals in consumer products, air, water, and soil, and physical factors such as radiation. While physical exercise and low alcohol consumption are well established beneficial factors, comparatively less attention has been paid to chemical exposures as avoidable factors.

Setting the scene: what is the contribution of non-genetic factors to breast cancer?

Due to the common genes that are shared by identical twins there is an increased likelihood that the twin of a person diagnosed with cancer will suffer from the same disease. Analyses of differences in the cancer incidence among twins can therefore be used to estimate the relative contributions of heritable and environmental factors to disease causation. Recent studies among Scandinavian twins have produced fascinating insights. For breast cancer in women it was found that heritability accounted for 27% of the variation in susceptibility to this form of cancer. Environmental factors that were shared by both twins explained 6%, and environmental factors

not common to the pair contributed 67%[8]. This means that the most important contributor to the causation of breast cancer is non-genetic or environmental, even under circumstances where the genetic background is very similar.

Studies of families with a heritable predisposition to breast cancer have produced similar results. Women who carry a mutated form of the tumour suppressor genes *BRCA1* and *BRCA2* suffer from a significantly higher risk of developing breast and ovarian cancer than women not afflicted by this genetic change. Among carriers of the altered genes who were born before 1940, the risk of developing breast cancer by the age of 50 is 24%. Interestingly, women harbouring the mutation, but who were born after 1940, have a much higher risk (67%) of being diagnosed with breast cancer at 50 years of age. Similarly, physical activity and leanness delay disease onset significantly in predisposed women when compared with carriers who were obese[9]. Together, these observations show that even in a genetic background that strongly predisposes to breast cancer, non-genetic factors can dramatically modulate risk. This lends further urgency to the question: What are these non-genetic factors?

The role of oestrogens

Prominent among non-genetic risk factors in breast cancer are the female sex hormones, oestrogens. Although essential for breast development, they also play an important role in the causation of breast cancer. This is not restricted solely to natural oestrogens. With the realisation that synthetic oestrogens (e.g. in certain pharmaceuticals) also contribute to risks, concerns are growing over other oestrogen-like chemicals present in the environment, in food or in cosmetics and personal care products.

Oestrogens and breast development

Mammary glands are composed of a tree-like ductal structure for the production and release of mothers' milk. These structures are not fully developed or functional at birth. Baby girls are born with a duct structure that extends only a small distance from the nipple. Until puberty, these ducts grow in proportion with the rest of the body, but during puberty they experience a massive growth phase. Essential for this growth are steroidal oestrogens, natural hormones produced by the ovaries.

Through specialised cellular receptors that regulate the expression of genes important in growth (oestrogen receptors α and β), oestrogens stimulate division of the cells in the blind ends of the ducts, the "end buds". This process leads to the elongation and branching of the duct system. With every secretion of oestrogens during ovulation, the entire structure becomes more elaborate and branched. The final phase of development occurs during pregnancy when there is a further massive branching of ducts and the entire system matures fully. After breastfeeding and weaning, many of the ducts grown in pregnancy are remodelled to resemble the state before pregnancy[10].

Natural oestrogens and breast cancer

Paradoxically, natural oestrogens are not only key players in breast development, but also contribute to breast cancer. It is thought that in promoting the growth of end buds, oestrogens may lead to an increase in cells that later in life become prone to cancerous growth. This is borne out by the observation that the majority of breast cancers derive from end buds of the ductal lobular units, which are the cells that contain oestrogen receptors and are most responsive to oestrogens in breast development. Consequently, most breast cancers are oestrogen receptor positive and rely on oestrogen for growth.

During the periods when the duct structures grow, especially during development and puberty, the breast is particularly vulnerable to cancer-causing influences[11]. Elevated levels of oestrogens during foetal life are also associated with breast cancer[12]. In the womb, the hormone influences the number of end buds in the primitive duct structure of the foetus: higher oestrogen levels induce the growth of more end buds, thereby enlarging the cell pool from which cancer cells derive[10].

The cyclical secretion of oestrogen during a woman's life is now recognised as a key determinant of breast cancer risk: the more oestrogen reaches the sensitive structures in the breast during her lifetime, the higher the overall risk. Thus, every year of delay in the onset of regular ovulations corresponds to a 5% reduction in breast cancer risk. Conversely, every year of delay in menopause increases the risk by 3%[13].

On the other hand, pregnancies have a strong protective influence. Each child birth is thought to decrease the risk of breast cancer by 7%, and this effect is even more pronounced before the age of 20[13]. The very high levels of oestrogen and other hormones that are secreted during pregnancy stimulate the full maturation of the duct system of the breast. It is thought that this leads to a reduction in the number of cells in the end buds that are vulnerable to cancer-causing factors, and thus to a decrease in cancer risk.

Oestrogens in contraceptives, Anti-miscarriage drugs and hormone replacement therapy

The cancer-promoting effects of oestrogens are not limited to natural hormones. External oestrogens administered as oral contraceptives, anti-miscarriage drugs or for the suppression of menopausal symptoms are also associated with breast cancer. The use of these therapies has increased enormously during the last decades. For example, hundreds of millions of women worldwide have taken oestrogen and progestin as oral contraception[13]. In 2003, one third of all women in Britain aged 50-64 used hormone replacement therapy (HRT)[13].

Combined oestrogen and progestin oral contraceptives lead to a slightly higher breast cancer risk among women who are current users of "the pill" and have been using it for more than 10 years, but there were no detectable increased risks more than 10 years after last use[13].

Between 1953 and 1971, approximately 300,000 women in the UK alone used the oestrogenic drug diethylstilbestrol (DES) to avoid miscarriages. Not only was the drug ineffective for its intended purpose, recent studies have shown that women whose mothers took DES face twice the normal breast cancer risk[14]. The risk is expected to grow further as these "DES daughters" reach menopausal age. These results highlight the risks that stem from exposure to oestrogens at the "wrong" stages of development in the womb.

What led to the widespread use of HRT was the idea that replacing oestrogen lost during menopause might prevent many symptoms of ageing in women, including coronary heart disease and osteoporotic bone fractures. Initially, HRT was "oestrogen-only", but in the early 1980s it became clear that oestrogen-only HRT promoted cancer of the womb (endometrial cancer). But endometrial cancer could be prevented if oestrogen was given in combination with progesterone. Although the cancer causing effects of this HRT combination therapy began to emerge already in the mid 1990's[15], combined oestrogen-progesterone HRT became the most widely prescribed regimen, in Europe and the USA.

The potential benefits and harms of HRT were tested in controlled clinical trials. In 2002, one of these trials, the Women's Health Initiative (WHI) trial, had to be stopped early because oestrogen-progesterone HRT led to increased risks of breast cancer among the participating women. These risks were considered to outweigh the benefits of this form of HRT in terms of reduced bone fractures and reduced colon cancer[16].

Coinciding with the completion of the WHI trial, the results of a very large UK observational study of women receiving mammography screening, the Million Women Study, were published. It showed that all forms of HRT, including oestrogen-only and oestrogen-progesterone, increased breast cancer risks. The study authors estimated that the use of HRT during the last decade in the UK alone had resulted in an extra 20,000 breast cancer cases[17]. A very recent US study found that postmenopausal women taking combined oestrogen and progestin hormone replacement therapy for three years or longer run four times the risk of developing lobular breast cancer. This is shorter than the time associated with an increased risk of other types of breast cancer[53].

Oestrogen-only HRT and breast cancer risks

After the early cessation of the oestrogen-progesterone arm of the WHI trial in 2002, an analysis of the effects of oestrogen-only HRT (also part of the WHI trial) continued and was completed in 2006[18]. In contrast to the UK Million Women Study and other published evidence it revealed decreased breast cancer risks in women who received oestrogen-only HRT. This finding was difficult to explain. Can it be taken to mean that oestrogens, when administered as a synthetic agent (as opposed to synthesised internally and released by a woman's ovaries) are not associated with breast cancer?

In a 2004 interim report, when the downward trend in breast cancer risks had already become apparent, the WHI Steering Committee exercised great care in interpreting their observations[19]. They acknowledged that the risk reduction was not anticipated and was in conflict with the results of other observational studies, most notably the Million Women Study conducted in the UK[17]. While it is clear that combined oestrogen-progesterone HRT has a stronger effect on breast cancer, the Million Women Study found a weaker, albeit significant contribution to risks also with oestrogen-only HRT. A recent meta-analysis of a large number of HRT studies and trials carried out worldwide also supports this notion. It showed that oestrogen-only HRT is associated with breast cancer[20]. Thus, the risks of synthetic oestrogens taken as drugs cannot be dismissed, and some have even argued that the risk reduction in the WHI trial is best interpreted as due to chance[21].

The recent down-turn in breast cancer incidence in the USA and other countries – a consequence of declining HRT use?

With news of a recent sharp decline in breast cancer incidence rates in the USA the association between HRT use and breast cancer has received renewed attention. Between 2002 and 2003 the reported down-turn was between 4% and 7% for women between 50 and 69 years of age[22, 23]. This drop coincided temporally with a pronounced decrease in HRT use. From 2002 onwards the dispensing of HRT in the USA declined by 30–40% [24]. Careful analysis revealed that the changes in breast cancer rates could not be explained by less frequent mammography screenings with a consequent reduction in diagnosed breast cancer cases[24, 25]. One recent Californian study could even demonstrate a quantitative link between changes in HRT use and incidence[25]. This analysis showed that, from 2001 to 2004, the incidence of breast cancer declined by 8.8% in regions with the smallest reductions in HRT prescriptions, by 13.9% in those with intermediate reductions, and by 22.6% in areas with the greatest reductions in combination HRT. The reductions in breast cancer were largely confined to women above the age of 50 and to patients with estrogen receptor positive tumours, both features that lend further support to the idea that changes in HRT use played a role.

A down-turn in breast cancer rates subsequent to reductions in HRT use was also observed in North Germany[26] and in Canada[27]. However, in The Netherlands, Norway and Sweden declines in HRT use were not accompanied by a drop in breast cancer incidence[28, 29]. In these countries, HRT use has been less intensive and was of shorter duration than among US women. Under such conditions decreases in breast cancer incidence are not expected to occur upon discontinuation of HRT[30]. Taken together, the available evidence strongly suggests that the sudden decline in HRT prescriptions may have led to the decrease in breast cancer, but additional, as yet unexplained factors might also have been at play. Very recent data from the USA show that the 2003 drop in breast cancer incidence did not continue in 2004[23].

Phytoestrogens and breast cancer

The possibility that plant-derived oestrogens, so-called phytoestrogens, may have protective effects on breast cancer has attracted considerable attention because of the relatively low incidence rates in East Asia. In these countries diets are rich in soy food, a source of phytoestrogens. Phytoestrogens have biological effects that could potentially reduce breast cancer risks, such as inhibition of surface receptors that tumour cells rely on for growth, that could potentially reduce breast cancer risks. However, these effects occur at pharmacological doses unattainable through consuming soy-rich diets[31]. On the other hand, there are concerns that phytoestrogens, through their ability to activate oestrogen receptors, may promote the growth of latent breast cancers.

The possible protective effects of phytoestrogens on breast cancer have been assessed in numerous epidemiological studies. Comparison of these studies is complicated because researchers used different measures of exposure to soy and phytoestrogens. In a recent meta-analysis of investigations conducted between 1978 and 2004, comparability was achieved by standardisation of phytoestrogen exposure in terms of soy protein intake. The authors came to the conclusion that soy intake is associated with a modest reduction in breast cancer risk[32]. Early life exposures to phytoestrogens may be important: The protective effects of soy-rich diets became more apparent in studies that included women whose consumption began in early childhood[32]. Overall, however, the differences between published studies introduced a great deal of uncertainty, and for this reason, the authors cautioned against over-interpretation and were hesitant to generalise their findings into clinical recommendations.

Some laboratory data suggest that phytoestrogens may promote breast cancer. Research demonstrating that one specific phytoestrogen, genistein, could stimulate the growth of oestrogen-responsive mammary tumours in a mouse model, raised considerable concern[33]. However, the relevance of these animal models for risk extrapolations to humans is the topic of considerable debate (summarised in[31]). For example, unlike women, mice are unable to synthesise sufficient amounts of oestrogen to promote mammary tumours. In summary, neither animal nor human data currently allow firm conclusions about the effects of phytoestrogens on breast cancer risk. Whether the continuous rise in breast cancer experienced by East Asian women since the early 1980s[34] is due to a withdrawal of phytoestrogens through adoption of a more "Westernised" diet is therefore also unresolved.

Growing concerns about chemicals with hormonal activity

There is convincing evidence that natural and synthetic oestrogens play a role in breast cancer. This has led to renewed concerns about chemicals with hormonal activities found in food, personal care products or as environmental contaminants. These substances include organochlorine pesticides such as DDT, polychlorinated biphenyls, polychlorinated dioxins and furans, plasticizers, UV-filter agents in sun creams, widely used preservatives and antioxidants such as parabens. Many of these agents were shown to behave like the female sex hormone oestradiol, although much higher concentrations are usually required to show effects of similar strength[35]. However, their high persistence, combined with their widespread presence in human tissues adds to fears regarding their potential role in the development of breast cancer. It appears plausible to suspect that these compounds too would be contributors to breast cancer risks, just like pharmaceutical oestrogens. What is the evidence for an involvement of synthetic and natural chemicals in breast cancer?

Synthetic chemicals and breast cancer

Studies carried out to examine whether specific persistent chemicals such as 1,1,1,trichloro-2-(p-chlorophenyl)-2-(o-chlorophenyl)ethane (o,p'-DDT), 1,1'-dichloro-2,2'-bis(p-chlorophenyl)ethylene (p,p'-DDE) and polychlorinated biphenyls (PCBs) are implicated in breast cancer could neither prove nor rule out a possible link. Some have prematurely concluded from these studies that there is no relationship between these chemicals and breast cancer risk[36, 37]. However, a variety of methodological limitations in these studies mean that we cannot conclude there is no relationship.

Some study outcomes indicate that women harbouring certain genetic changes in drug metabolising enzymes (cytochrome P450 1A1) may be at increased risk from PCB exposure[38]. The evidence concerning a possible link with dioxin exposure is suggestive. Young women exposed to the polychlorinated dioxin, tetrachlorodibenzo-p-dioxin (TCDD) during the 1975 Seveso accident north of Milan, Italy, suffered a two-fold increase in breast cancer risks[39]. However, these women sustained pronounced exposures resulting in quite high TCDD blood levels, not comparable with those found in other European women.

Epidemiological studies of the effects of plasticizers (e.g. phthalates), UV-filter agents, cosmetic ingredients (e.g. phthalates, parabens) or other widely used chemicals in consumer products are missing. Noteworthy are studies in occupational settings that show elevated breast cancer risks among women exposed to organic solvents for more than 10 years[40].

The uncertainty about an involvement of individual endocrine disrupting chemicals in breast cancer stems in part from the general features of investigations that aim to pinpoint specific risk factors as linked to cancer risks. To be identified as a determinant of risk, the effects of a specific chemical have to be quite pronounced. These difficulties are not limited to studies of the effects of chemicals. Investigations of the role of diet in breast cancer have also failed to show consistent and statistically significant associations between fruit and vegetable intake, or dietary antioxidants and breast cancer[6].

The pollutant "cocktail effect" and exposure timing

Despite these difficulties, evidence emerging from recent research shows that two important issues must be fully addressed to avoid wrongly dismissing a role for chemicals in breast cancer.

First, studies in humans have largely focused on single chemicals but have ignored the large number of agents that occur together in women's tissues and therefore may act in concert to contribute to breast cancer risks[41].

Second, to understand the role of chemicals in breast cancer, exposures during critical windows of vulnerability, including development in the womb, must be captured. Studies that only examine exposures at the time of breast cancer diagnosis or even decades later run the risk of overlooking disease-causing factors[42].

Breast cancer and the pollutant "cocktail effect"

Chemicals such as o,p'-DDT, p,p'-DDE and PCBs do not act in isolation in a woman's body, but in concert with natural oestrogens and a large number of other hormonally active chemicals and carcinogens. These include: chemicals released during the preparation of food (for example, during the grilling of meat)[43]; a growing plethora of man-made chemicals found as environmental pollutants (dioxins, certain PCBs and pesticides); those used in cosmetics (such as antioxidants, UV-filter agents, and some synthetic fragrances)[44]; those that leach from plastics (for example bisphenol A, nonyl phenol)[35]; and plant-derived oestrogens in certain foods.

The hormonal strength of many of these chemicals is considerably lower than that of natural or pharmaceutical

oestrogens. Nevertheless, laboratory experiments have shown that a sufficient number of such chemicals can significantly enhance the effects of natural oestrogens, even when they are present at levels that individually do not produce measurable effects[45]. There is now good evidence (reviewed in[46]) that combined exposure to hormonally active chemicals can produce additive effects at low doses. Whether the individual doses are effective on their own, is not the key determinant. What also drives the likelihood of mixture effects is the sheer number of chemicals present in a "pollution cocktail". Thus, in principle, combination effects will result from toxicants at or even below threshold doses, provided sufficiently large numbers of components sum up to a suitably high dose. Whether such "cocktail effects" are likely to arise in reality, depends on the nature of hormonally active chemicals, and their number. At present, information about these factors is patchy, but indications are that scores of chemicals may be involved[46] . The recent advances in our knowledge about determinants of mixture effects highlight that the focus of the previous human studies of the effects of chemicals on breast cancer was wrong. Instead of concentrating on a few, arbitrarily selected substances, the entirety of hormonally active chemicals must be considered.

A recent study among Spanish women suggests that cumulative exposure to hormonally active substances is significant. Breast cancer risk was associated with the body burden of lipophylic organohalogen oestrogenic chemicals, excluding the natural hormones[47, 48]. This is the first evidence that chemicals in our environment, with oestrogenic properties that are 'accidental', and not just natural hormones or pharmaceutical oestrogens may contribute to the development of breast cancer. Similar epidemiological studies should be repeated in other countries.

Breast cancer and exposure during periods of increased vulnerability

There are periods in a woman's life when the breast is particularly vulnerable to cancer-causing influences. One such period is puberty, when the breast experiences the first significant growth phase of the ductal system, the other is during development in the womb, when the breast tissue is laid down.

PUBERTY

The increased sensitivity of the breast tissue at this time of life was first noticed in the aftermath of the atomic bombs in Hiroshima and Nagasaki. As a result of the massive levels of radioactivity, breast cancer in Japanese women increased significantly, but only in women who were exposed during puberty or at an even younger age. Older women experienced far less pronounced breast cancer risks[11].

The importance of exposure to chemicals before or during puberty was very recently highlighted in a US study of breast cancer and DDT exposure at a young age. Previous investigations of a link between DDT and breast cancer have looked at exposures later in life, when the breast tissue is less vulnerable. However, it could be shown[49] that in women born after 1931, high levels of p,p'-DDT were associated with a 5-fold increased breast cancer risk. When DDT came into widespread use, these women were under 14 years of age, and mostly under 20 when DDT use in the USA peaked. Many women exposed to DDT in puberty have not yet reached the age of 50, when breast cancer becomes more common.

DEVELOPMENT IN THE WOMB

Another key period is during development in the womb, when the origins of the mammary gland ductal system are laid down. Elevated levels of natural oestrogens during this critical time are associated with increased breast cancer risks of daughters later in life[12].

The recent demonstration of elevated breast cancer risks in the daughters of women who took diethylstilboestrol (DES) to avoid miscarriages[14] shows that synthetic oestrogens can have similar effects. The risk is expected to grow further as these "DES daughters" reach menopausal age. It is thought that DES exposure of the developing foetus in the womb may have promoted the growth of ductal end buds, thereby enlarging the number of cells from which cancer can develop later in life.

Other studies with laboratory animals point in the same direction and suggest that exposure to man-made oestrogen-mimicking compounds in the womb can alter the development of the mammary tissue with possible consequences for breast cancer[50, 51].

Tumour growth is most pronounced when the cancer-causing agent is given to young animals in which the mammary gland is developing, whereas adult animals are almost immune[10]. Some hormonally active chemicals, such as dioxins, can increase the sensitivity of rats to other breast cancer-causing substances when given at critical times during development in the womb[42]. These observations highlight the importance of documenting exposure to potentially cancer-causing chemicals at the appropriate times. For human studies, this poses an enormous challenge: to prove or dismiss a link with breast cancer, exposure to chemicals must be recorded many years before the cancer becomes manifest.

Implications for safety testing of chemicals

Safety testing of chemicals in general faces two fundamental issues that greatly influence test outcomes: Timing and duration of exposure and choice of toxicological effect to be monitored. Recent research has shown that both these issues have not been adequately addressed during the evaluation of hormonally active chemicals.

The testing of chemicals for possible carcinogenic effects in laboratory animals is usually carried out after they are born, and does not encompass their development in the womb. Although there is evidence that exposure during development will increase the sensitivity with which cancer-causing agents can be detected, this is not incorporated in safety testing strategies.

Furthermore, a great deal of carcinogenicity testing focuses on the screening for chemicals that have the ability to cause gene mutations. However, many of the hormonally active chemicals shown to have profound developmental effects on breast cancer risks in animals are not mutagenic and will therefore be missed during screening exercises.

These two inadequacies have led researchers to question whether, in trying to identify cancer-causing chemicals, they are using the wrong tools, at the wrong times[42].

Conclusion

Although it is clear that many factors play a role in breast cancer, a contribution of environmental chemicals cannot be dismissed. Indeed, concerns are mounting although convincing evidence from human studies is missing due to methodological limitations. Nevertheless, in view of the proven contribution of natural and therapeutically used oestrogens, it is biologically plausible that less potent hormonally active chemicals may also contribute to risks, and the health experience of Spanish women supports this idea[47]. By adopting targeted research strategies, and with better use of animal studies on mammary carcinogens[52] and *in vitro* data, the issue should be pursued further with urgency.

There is also a need to act sooner to limit exposures. Preventative action should be based on evidence available from experimental laboratory studies, and should not wait for the outcome of human epidemiological studies because confirmatory data from epidemiological studies will take decades to materialise. Given the known role of oestrogens in breast cancer, it would be prudent to reduce exposures to chemicals that can mimic oestrogen. Consideration should therefore be given to amending current chemicals policy so that such chemicals are replaced with safer alternatives, where possible.

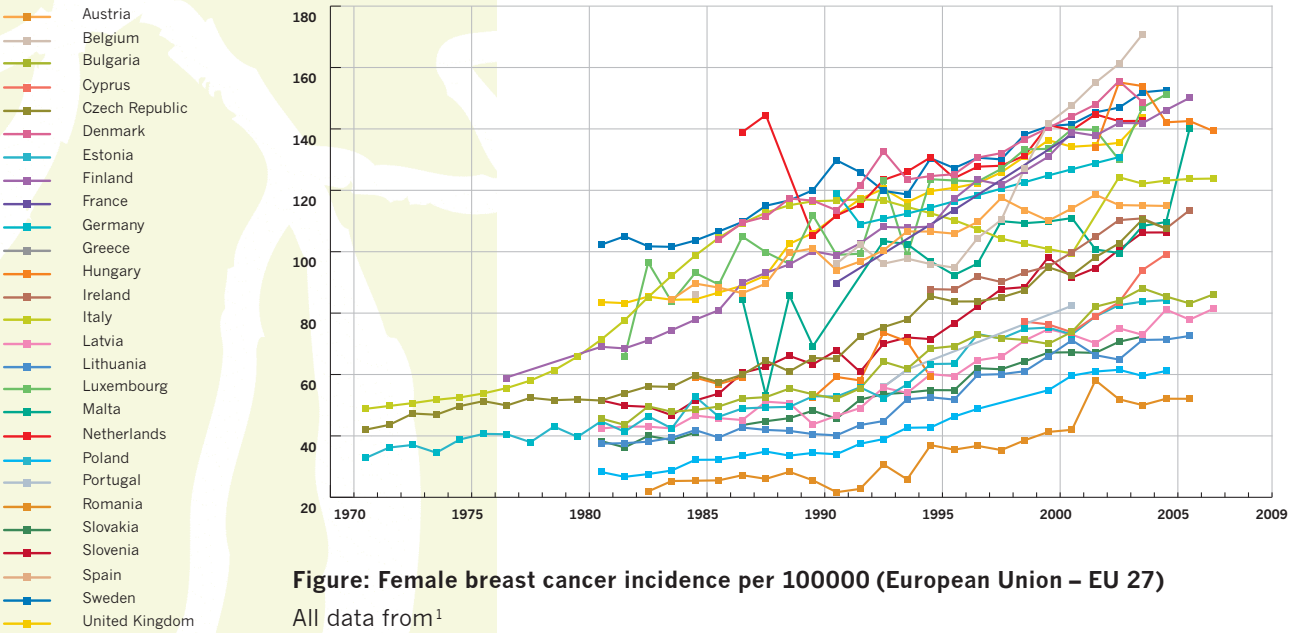

Figure: Female breast cancer incidence per 100000 (European Union – EU 27)

All data from[1]

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Major gaps in public health laws allow cosmetics companies to use almost any ingredient they choose — even lead, mercury, and placenta

Ingredients in any kind of product — from sunscreen and mascara to deodorant and baby shampoo — are sold to consumers with no restrictions and no requirement for safety testing. Check out EWG's list of ingredients and products you should avoid when making your next shopping decision.

Are ingredients in personal care products actually harmful?
While some companies make products that are safe to eat, other companies choose to use known human carcinogens or developmental toxins. Nearly all these chemicals can penetrate the skin, and some we ingest directly from our lips or hands. More than one-third of all personal care products contain at least one ingredient linked to cancer. When risky and unstudied chemicals are used in cosmetics, the stakes can be high — unlike trace contaminants in food or tap water, chemicals in cosmetics are base ingredients.

10 **Why do you have progesterone listed as a possible carcinogen?**
Progesterone is "reasonably anticipated to cause cancer in humans" as judged by the federal government's National Toxicology Program (NTP). Within Skin Deep determinations on ingredient hazards were based not on our judgment, but on evaluations by government, industry, and academic experts. For progesterone, we find no independent, definitive assessments that conflict with NTP's cancer rating of the chemical. Some doctors continue to recommend progesterone cream to patients.

Although progesterone in personal care products is chemically identical to that produced in the body (Progesterone, CAS Registry No. 57-83-0), it is the disruption of the body's natural delicate hormonal balance by adding extra progesterone that can lead to health concerns. Please see the National Toxicology Program's 10th Report on Carcinogens (2002) for more information.

24 **My sunscreen says it has nano particles in it, should I be concerned?**
Environmental Working Group is among the many advocacy groups who have raised concerns about whether the rapidly expanding use of nanotechnology poses risks to human health or the environment. Many zinc and titanium sunscreens contain nanosize particles, **even when they are not on the label.**

Although we expected to reach a different conclusion at the outset of our sunscreen investigation, when we balanced all factors important in sunscreen safety, we found many zinc and titanium-based sunscreens that our analysis shows are among the safest and most effective sunscreens on the market. Our product ratings reflect our concern about the very real dangers of cancer and other health hazards from sun exposure, balanced against concerns about the potential health hazards of sunscreen ingredients. Read our full assessment of zinc and titanium sunscreens here.

Our study shows that consumers who use sunscreens without zinc and titanium are likely exposed to more UV radiation and greater numbers of hazardous ingredients than consumers relying on

zinc and titanium products for sun protection. We found that consumers using sunscreens without zinc and titanium would be exposed to an average of 20% more UVA radiation — with increased risks for UVA-induced skin damage, premature aging, wrinkling, and UV-induced immune system damage — than consumers using zinc- and titanium-based products. They contain four times as many high hazard ingredients known or strongly suspected to cause cancer or birth defects, to disrupt human reproduction or damage the growing brain of a child. They also contain more toxins on average in every major category of health harm considered: cancer (10% more), birth defects and reproductive harm (40% more), neurotoxins (20% more), endocrine system disruptors (70% more), and chemicals that can damage the immune system (70% more).

Zinc oxide and titanium dioxide are stable compounds that provide broad spectrum UVA and UVB protection, while the available studies consistently show very little or zero penetration of intact skin by these compounds, indicating that real world exposure to potential nano sized particles in these products is likely very low (Borm 2006). The sun protection benefits, in contrast, are very high.

EWG's rating of zinc and titanium-based products as among the safest and most effective sunscreens available in the U.S. today should not be interpreted as an endorsement of nano-materials in general. We remain deeply concerned about the overall safety and oversight of nanotechnology as well as impacts to workers and the environment.

Benefits:
◦ UV exposure is damaging to health, and Zinc and Titanium offer broad spectrum UV protection.
◦ Zinc and titanium are 2 of only 4 UVA blockers used in the US. Alternatives including Tinosorb S and Tinosorb M are available in Europe but not approved by FDA for use in the U.S. As it stands, the only other U.S.-approved sunscreen with UVA-I protection are avobenzone and Mexoryl SX, both of which are unstable in most sunscreen formulations (i.e., they break down in the sun).
◦ In 15 peer-reviewed studies, nanosize zinc and titanium were shown not to penetrate through unbroken skin at concentrations exceeding 1.5%. A recent review for the EU decision-making body found that, "There is currently little evidence from skin penetration studies that dermal applications of metal oxide nanoparticles used in sunscreens lead to systemic exposure" (Borm 2006).
◦ Zinc oxide poses a low level of concern based on currently available science: it is well studied and is a necessary nutrient to humans.

Concerns of zinc and titanium in sunscreens:
◦ No studies have tested nanosize zinc and titanium penetration through fragile or damaged skin. Conventional zinc is widely used on damaged skin including diaper ointment and burn treatment. However, other nano-scale particles have been shown to penetrate the skin, especially when it is repeatedly flexed.
◦ U.S. regulatory framework has lagged far behind industry in addressing the impacts of nanotechnology. Due to inadequate labeling requirements, consumers have no options for avoiding products containing nanoparticles.
◦ Nanoparticle production poses serious concerns for workers, especially particle inhalation, which available science indicates is likely the greatest human hazard for nanoparticle exposures. Occupational production is virtually unregulated in the U.S

◦ Nanoparticles, including zinc and titanium, are potentially toxic to the environment. Like all sunscreen ingredients their use in sunscreens results in releases of the chemicals through

production, users' contact with water, and as waste.
◦ Read our detailed summary of nanotechnology risks and benefits.

What to do:
◦ FDA needs to evaluate nanoparticles as distinct from larger particles in products.
◦ Manufacturers using materials with all or a fraction of the ingredient in the nano-scale range must clearly label their products with this information, to allow consumers the option of avoiding them.
◦ FDA must evaluate and approve new sunscreen chemicals that can protect from UVA and might offer fewer risks to workers and the environment.
◦ The safety of nano-scale zinc and titanium in sunscreen must be fully assessed.

It may smell great, but do you know what's in it? Fragrances are the great secrets of the cosmetics industry, in everything from shampoo to deodorant to lotion, and falling straight into a giant loophole in federal law that doesn't require companies to list on product labels any of the potentially hundreds of chemicals in a single product's secret fragrance mixture. **Fragrances can contain neurotoxins and are among the top 5 allergens in the world.** Our advice? Buy fragrance free. (See fragrance ingredient report.)

If fat scraped from the back of the hide of mink and emu isn't something you'd like to smear on your skin, you may want to avoid mink and emu oil, conditioning agents in sunscreen, shaving cream, hair spray and more. These are just two of many ingredients made from animal parts — you'll find a partial list here; use Skin Deep to find more. (See all animal part ingredients.)
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These tiny little inventions are touted as the next green revolution, **but we don't find much sexy or green about untested ingredients that can slide up the optic nerve to the brain or burrow inside red blood cells.** They're found in cosmetics in forms ranging from tiny wire cages called "buckeyballs" to miniscule bits of metals used as sunscreens. Good luck finding them, though — companies don't have to tell us that they're in our products, though we found that more than one-third of all products contain ingredients now commercially available in nano forms. And we did find them listed outright on the labels of some sunscreens (nano metals) and skin creams (buckeyballs). Buyer beware! (see all ingredients known to be nanoparticles or see all ingredients that may be lurking as nanoparticles.)

Phthalates

Pronounced "tha'-lates," these little plasticizer chemicals pack a punch to male sex organs. Whether it's sperm damage, feminization of baby boys, or infertility, a growing number of studies link phthalates to problems in men and boys. Pregnant women should avoid it in nail polish ("dibutyl phathalate") and everyone should avoid products with "fragrance" on the label, chemical mixtures where phthalates often hide. (See all phthalate ingredients.)

BUYER BEWARE:
• nailtiques Protein Formula 3
• Pinkie Swear Pinkie Sheer Nail Blush
• Sally Hansen Teflon Tuff Nail Color (Beige 40; Blackberry Wine; Clear; French White Tip; Sheer French Pink; Soft Blush Frost)
• Sally Hansen Teflon Tuff Teflon Tuff Base & Top Coat
>>see more

Surprised to learn that the same factories making gas for your car also make emollients for your face cream? Meet the workhorse chemicals of the cosmetics industry — petroleum byproducts, and the cancer-causing impurities that often contaminate them. These ingredients include carcinogens in baby shampoo (see new research on 1,4-dioxane) and petrochemical waste called coal tar in scalp treatment shampoos. We list a few products and their cancer-causing contaminants here, but use Skin Deep to find more. (See select petroleum byproduct ingredients.)

FYI: PETROLEUM IN PRODUCTS

- Avon ANEW ULTIMATE Skin Transforming Cream
- Africa's Best Kids Organics No-Lye Organic Conditioning Relaxer System with ScalpGuard (Kids Regular)
- Perfectone Permanent Color Creme, #5/0 Light Brown
- Avon becoming EVENING RETREAT moisturizers

Environmental Estrogens:The Invisible Threat That Surrounds Us

By Kimberly Pryor

Source: http://www.cpmedical.net/articles/environmental-estrogens-the-invisible-threat-that-surrounds-us

Daily, we eat and breathe substances shown to cause birth defects and cancer in animals. These environmental estrogens are everywhere. In the milk and water we drink, in the food we consume, in birth control pills, dental sealants, and plastics. Based on breast milk concentrations nationwide, it has been estimated that at least 5% and possibly more of the babies born in the United States are exposed to quantities of polychlorinated biphenyls (PCBs) sufficient to cause neurological defects.

1 Concern over environmental estrogens is so great that in 1999 the Environmental Protection Agency (EPA) initiated a screening and testing program to identify the potential endocrine-system impact of the 87,000 chemicals in commercial use. In addition, the Centers for Disease Control (CDC) and the National Institutes of Health (NIH) are examining blood and urine samples to quantify what risk Americans may face from exposure to approximately 50 environmental estrogens.

2 Meanwhile, what can we do to protect ourselves from these prolific chemicals? Scientists are exploring certain nutrients in vivo and in vitro to determine if they can guard against environmental estrogens. Before addressing which nutrients may act as an ecoestrogen shield, however, we must examine how and why these chemicals threaten both our longevity and our children's health. **Widespread Contamination** One particularly sobering example of the heart-breaking consequences of human interaction with environmental estrogens was illustrated in a 1984 study. The study involved 242 newborn infants whose mothers consumed, over six years, moderate quantities of PCB-contaminated Lake Michigan fish. The infants belonging to mothers who had consumed the fish weighed an average of 190 grams less at birth than controls. This level was comparable to the low birth weights of children whose mothers smoked during pregnancy. The PCB-exposed infants had smaller head circumferences and exhibited poorer neuromuscular maturity. Furthermore, the mothers who had consumed the most fish had the highest serum PCB concentrations, and their babies had the highest umbilical cord PCB levels. This was particularly disturbing considering the mothers ate as little as two salmon or lake trout meals per month.

3 A follow-up study indicated that, at six to seven months of age, the contaminated infants experienced delays in psychomotor development and poorer visual recognition compared with controls. At four years of age, the children exhibited short-term memory problems. During the testing, 17 of the children whose mothers' breast milk had the highest PCB concentrations became unmanageable and refused to cooperate. In another study comparing the same infants to children of mothers exposed to a PCB farming accident, both groups experienced growth retardation and neurological defects. These defects were directly dose- related to umbilical cord

serum PCB concentrations and levels in fetal blood.1 Many drainage basins are just as contaminated in other parts of the country as the Great Lakes. In the Central Valley of California, wildlife drink from agricultural drain canals containing estrogenic chemicals. In the farm communities of Southeastern Spain, fat samples from local children contained a total of 14 pesticides.

4-6 Banned in the U.S. since the early 1970s, synthetic estrogens such as DDT and PCBs continue to poison the environment, partially due to their ongoing use in developing countries and their ability to vaporize and drift across the globe.

7 In addition, ecoestrogens keep a tenacious grip on the planet, as DDT has a half-life of 57.5 years in temperate soils. Despite the ban on these two destructive chemicals, other estrogenic pesticides, plasticizers and chemicals continue to be used in the United States. This widespread contamination is particularly alarming given that PCBs, dioxins, DDT and a number of other pesticides — often called organochloride compounds — are lipid-soluble and find a home in fatty tissue in the body. In particular, these organochloride compounds are found in breast milk, with its high lipid content. The concentrations in embryos and fetuses parallel those in mothers. Infants, therefore, are at an increased risk.1 Humans also are exposed to estrogenic compounds through the consumption of sex-steroid-treated meat and dairy products. The Joint Food and Agricultural Organization/World Health Organization Expert Committee on Food Additives (JECFA) and the FDA claimed in 1988 that the estrogenic residues found in meat from treated animals posed no risk for consumers. One group of scientists who re-evaluated the JECFA conclusions, however, were particularly concerned with meat concentrations of the natural estrogen, estradiol. These scientists believed that these estrogenic residues could jeopardize the health of prepubertal children. In the scientists' opinion, JECFA's conclusions concerning the safety of hormone residues in meat “seem to be based on uncertain assumptions and inadequate scientific data.”

8 **The Danger Begins** In the early 1970s, scientists first realized that substances not intentionally made to act as hormones could unintentionally take on an estrogenic role. This realization came after a chemical spill of Kepone, a chemical used in manufacture of the pesticide Mirex™, resulted in lowered sperm counts in exposed men. Researchers confirmed that Kepone was a weak estrogen, although its chemical structure bore no resemblance to the natural hormone. Scientists soon realized that Kepone had plenty of company. They confirmed that DDT and other pesticides acted like endogenous estrogens or produced estrogenic breakdown products.

9 Wildlife seemed to be particularly vulnerable to environmental estrogens. In fact, problems with wildlife provided the first hint that environmental estrogens might also be causing problems in humans. After examining two- and four-year-old salmon in the Great Lakes, researchers discovered enlarged thyroids in every specimen. Of the male salmon, 40 to 80% also experienced a high rate of precocious sexual maturation. In addition, many of the salmon eggs did not hatch. These disastrous effects traveled up the food chain. In bald eagle nests, egg shells thinned and cracked, an effect attributed to DDT.1 Further support for environmental estrogens' destructive role arose when University of Florida researchers discovered reproductive abnormalities in females, and feminization of male alligators nesting at Florida's Lake Apopka. Lake Apopka is located adjacent to an EPA Superfund site contaminated with dicofol and DDT. Both of these

substances are known estrogen mimics. At six months of age, female alligators from Lake Apopka had plasma estrogen concentrations almost two times greater than normal females. In addition, the alligators suffered from abnormal ovaries, and an increased mortality rate. The plasma testosterone levels in male juvenile alligators from Lake Apopka were more than three times lower than control males in Lake Woodruff. Lake Apopka males also had poorly organized testes and abnormally small phalli. In two cases, Lake Apopka alligators without penises were identified as females, but were subsequently observed to have testes. Two animals with penis-like appendages were identified as males yet possessed ovarian tissue. The researchers concluded the reproductive abnormalities were likely due to the alligators' exposure to an estrogenic substance.

10 **Reproductive Abnormalities** Many synthetic chemicals serve as sex steroid imposters. They trick the body into believing they are natural, endogenous estrogens, which enables them to push the real hormone out of the way. As these imposters replace the endogenous estrogen, they are capable of sending the wrong signals to the chemical messenger pathways through which estrogens normally work. Although there is considerable debate over the extent of harm environmental estrogens cause, much evidence points toward a possible link between environmental estrogens and reproductive diseases and cancer. In 1948, doctors began prescribing the estrogenic Diethylstilbestrol (DES) to prevent miscarriages. Twenty-three years later, scientists discovered that some of the adolescent daughters whose mothers had taken DES developed adenocarcinoma, a rare form of vaginal cancer. The cells in the vagina or fallopian tubes of these female offspring were deformed and there were structural changes in the girls' uteri. Some men exposed in utero to DES experienced increased incidences of cryptorchidism, where one or both testicles had not descended into the scrotum, an important risk factor for testicular cancer. Abnormal congenital openings of the male urethra upon the undersurface of the penis, called hypospadias, and decreased semen volume and sperm counts, were also found in the DES-exposed men.

11 Breast cancer incidence has steadily climbed in the US, which has been attributed to the accumulation of estrogenic chemicals in the environment.

12-13 Many of these chemicals have caused cancer in animals and are suspected human carcinogens. Both DDT and PCBs have been shown to be tumor promoters and demonstrate estrogenic activity. According to Merriam Webster's Medical Dictionary, DDE is a persistent organochlorine produced by the metabolic breakdown of DDT. In 58 breast cancer patients, DDE levels were approximately 35% higher than in 171 matched, healthy controls.

14 Increased incidences of male reproductive disorders have accompanied the breast cancer rise. Testicular cancer, cryptorchidism and urethral abnormalities (hypospadias) — all conditions that arise at the fetal development stage — have more than doubled in the past 30-50 years, while sperm counts have declined by about half. Furthermore, testicular cancer is now a leading cause of death in young men.

15-20 **Impact of Synthetic Estrogens** The natural estrogen, estradiol, binds to extracellular proteins, and is less effective at entering the cells, whereas the synthetic estrogen DES, is attracted to the estrogen receptor, and more easily gains access to the cell. At equivalent

concentrations in the blood, more DES enters the cell than does the natural estrogen estradiol. As one researcher describes, “DES is a functionally more efficient estrogen than is the natural hormone.”10 Studies in rats have shown that estrogenic environmental toxicants dramatically affect fertility. Estrogenic chemicals have altered the tissue structure of the male animals' seminiferous tubules, with higher doses impairing testicular mass and sperm count. Estrogenic chemicals have also had toxic effects on both rat testes and epididymis. Researchers have speculated that the same effects might also occur in humans.

21 Other researchers have suggested that small amounts of many estrogenic chemicals may have as disastrous an effect as large amounts of any one chemical. Scientists also have connected reproductive disorders to populations where exposure to estrogenic agents are high.

22 Furthermore, another recent in vitro study determined that PCBs significantly increased MCF-7 human breast cancer cell proliferation, an estrogenic form of cancer. The addition of the drug hydroxytamoxifen, an estrogen antagonist, inhibited the increased cell proliferation associated with cancer.

23 Clearly, protection against these harmful environmental invaders is needed. Scientists have investigated the following nutrients for the role they may play in protecting humans against ecoestrogens. **Indole-3-Carbinol** Indole-3-carbinol inhibits cell proliferation in human MCF-7 breast cancer cells even more effectively than the drug tamoxifen. If tamoxifen can halt the activity of estrogenic chemicals such as PCBs, then I3C may do the same.

24 An antiestrogen, I3C is found in cruciferous vegetables (broccoli, cauliflower, brussels sprouts). It alters the way estrogen is metabolized in the body, from the “bad” pathway to the “good” pathway, as reported in the October 1999 issue of Vitamin Research News. The “tumor enhancer” metabolic pathway, 16 alpha-hydroxylation, is elevated in patients with breast and endometrial cancer and in those at increased risk of such estrogen-dependent cancers. When estrogen veers away from the 16-alpha pathway and instead takes the “tumor suppressor” metabolic pathway — called 2-hydroxylation — the incidence of cancer decreases. Healthy individuals not at risk for estrogen-dominant breast or endometrial cancer bypass the 16-alpha route and metabolize estrogen through the 2-hydroxylation pathway. Research has indicated that some organochlorine-based pesticides elevate estrogen excretion through the “tumor promoter” pathway in MCF-7 breast cancer cells, while phytochemicals like indole-3-carbinol (I3C) switch the elimination route to the “tumor suppressor” pathway.

25-26 In studying the effects of I3C and ICZ (an acid-derived condensation product of I3C) on the effects of estrogen metabolism, researchers concluded that I3C's antiestrogenic properties may help expel estrogenic contaminants from the body.

27 **Sperm Counts and Nutrients** Carnitine, arginine, zinc, selenium, vitamin B-12, and the antioxidants vitamin C, vitamin E, glutathione, and coenzyme Q10 have all been shown to improve sperm counts, sperm motility and male infertility. In one study of mice fed one of three different pesticides, 20 to 40 mg/kg body weight per day of vitamin C offered protection against the decreased sperm count and deformed sperm that developed in the animals treated with only the chemicals.

28-29 **Antioxidants and Ecoestrogens** Research indicates that oxidative damage may account for some of the toxicity of environmental estrogens. In mice, vitamins C and E have protected the liver against some of the damaging effects exerted by the estrogenic chemical dieldrin. It also has been shown that estrogenic chemicals such as PCBs increase the rate at which the body excretes ascorbic acid. Administering ascorbic acid to environmental-estrogen-exposed fish considerably neutralized the toxic effect of the chemical with a 10-fold decrease in the number of fish killed. Furthermore, ascorbic-acid-deficient guinea pigs have a harder time biodegrading pesticide residue and experience a greater accumulation of pesticide in tissue.

30-32 In rats and guinea pigs exposed to the potent environmental estrogen, PCB, the administration of 1000 mg/kg dietary vitamin E significantly reduced the amount of ascorbic acid excreted and the amount of thiobarbituric acid-reactive substances (TBARS), a significant marker of oxidation. In PCB-contaminated guinea pigs, feeding high levels of both ascorbic acid and vitamin E was more effective in reversing the PCB-induced severe growth retardation and in lowering the TBARS level than feeding the vitamins separately.

33-34 Furthermore, scientists have discovered that carotenes and carotenoids, including beta-carotene, were significantly lower in cancer patients compared to healthy controls. In postmenopausal women with breast cancer, serum xanthophyll (e.g. lutein) levels were significantly lower than among healthy controls. In premenopausal women, serum beta-carotene levels tended to be lower among breast cancer cases than among controls.

35 These results suggest that combination antioxidant nutritional formulas may offer significant protection against environmental estrogens. **Other Potential Protectors** High fiber intake may lower blood estrogen concentrations, particularly in premenopausal women. Also, selenium deficiency may be an indicator of environmental estrogens. Feeding PCBs to chicks has decreased the animal's ability to utilize selenium, leaving cell membranes vulnerable to the harmful effects of pesticide-induced peroxidation.

36-37 This selenium deficiency in PCB-treated animals suggests a possible need for additional selenium supplementation. **Conclusion** Environmental estrogen contamination is widespread. It is almost impossible to escape encounters with these chemicals. However, antioxidants, I3C and other nutrients can play a significant role in protecting us from such ecoestrogens.

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Hormones in Meat

Drugs In Our Meat – Shouldn't We Know?

Ever heard of the drugs oestradiol-17, zeranol, trenbolone acetate and melengestrol acetate? Probably not. That's because meat producers aren't required to tell you that these synthetic growth hormones - linked to increased risk of breast and prostate cancers in humans - are routinely injected or implanted into animals raised for meat in the U.S.

The European Commission has banned the use of these drugs in animals raised for human consumption in Europe, and forbids the import of meat containing these hormones from the US. But here in this country? The FDA not only allows these and other antibiotics and hormones to be routinely injected, implanted, or laced into farm or feedlot animals raised for meat, dairy or eggs, but it also doesn't require meat producers to tell you which drugs they use, or in what quantities.

While GMO labeling has taken center stage this past year in the fight for truth-and-transparency in labeling, there's another labeling battle looming: the labeling of meat, eggs and dairy products coming out of factory farms. These products routinely contain residues of dangerous antibiotics and hormones. The OCA plans to make the labeling of these products a priority in 2013.

Meanwhile, this week, the Government Accountability Project (GAP) announced it is suing the FDA - that agency that's supposed to be looking out for public health - because it won't release detailed information about which antibiotics are being used in what quantities in the animals raised for meat on our grocery shelves. We do know this: 80% of all antibiotics sold in the US are sold for use in animals raised for meat. Why? To make the animals grow faster and survive the hellish conditions in factory farms, or CAFOs (Confined Animal Feeding Operations). This means two things. Humans are indirectly consuming these antibiotics and hormones. And, as public health experts have warned repeatedly, the rampant and reckless use of antibiotics on factory farms is making antibiotics less and less effective as cures for diseases that affect humans.

Source: http://www.organicconsumers.org/

How to Clean without Using Harsh Chemicals

Saturday, October 16, 2010 by: Maddie Ellison, citizen journalist

(NaturalNews) More and more research is showing that the harsh chemicals in cleaning products are causing major health concerns for consumers. Studies show they can cause cancer and infertility, and new research is showing that some of those chemicals are even making us fat. You can have a clean home without using harsh chemicals.

There are many things you already own that can clean your home effectively. Baking soda isn't just useful in baking. Baking soda is a mild abrasive that can clean just about everything. You can buy it in large boxes in the laundry aisle of your favorite store or in hardware stores.

Here are some tips to using baking soda:
* Clean the inside and outside of your refrigerator with a solution of 3 tablespoons of baking soda dissolved in 1/2 cup of warm water.
* Baking soda is great for removing red wine and coffee stains. Try soda water first and if that doesn't work pour baking soda on the stain, rub it in, and then brush it off.
* Use a baking soda paste (baking soda with a small amount of water mixed with it) to remove mold and mildew from shower curtains and shower walls. Use an old tooth brush to rub it over the stain or to rub the grout between tiles.
* To keep your drain smelling fresh pour 1/2 cup of baking soda down your kitchen drain followed by 1/2 cup of vinegar. Then pour in some boiling water. The combination of the three will break down any fatty acids that are clogging your drain and it will make the drain smell fresher.

Another common household item that is a great all natural cleaner is vinegar. Vinegar not only cleans up grease it's also a great deodorizer. Here are some ways you can use vinegar to clean your home:

* Soak your shower head in vinegar to remove lime scale.
* Spray a mixture of equal parts water and vinegar on windows and mirrors and wipe clean with crumpled up newspapers.
* For stubborn marks in your toilet, sprinkle your toilet bowl with baking soda and then pour vinegar on top of it. It will bubble (think volcano science projects). Use your toilet brush to brush down the sides of your toilet bowl.

A quick search on the internet will give you many more ideas of how you can make your own cleaners. When you make your own cleaners you not only save money, but you also keep your family and pets safe.

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How to Protect Yourself from these Five Pervasive Toxins

Posted By Dr. Mercola | August 25 2010 |

A growing body of research links five of the most commonly used chemicals in the world to a host of ailments, including cancer, sexual problems and behavioral issues. Here's what CNN suggests you can do about them:

1. **BPA — Bisphenol A**

   BPA is used to make lightweight, clear, heat-resistant plastic. It's also used in epoxy resins.

   A growing body of research suggests that BPA poses a potential cancer risk and may disrupt the extremely sensitive chemical signals in your body called the endocrine system.

   To avoid it, buy stainless steel bottles and glass food storage containers. Switch to fresh or frozen vegetables instead of canned. If you buy plastic, check for the number on the bottom — if there is a number 7, assume the container contains BPA unless it explicitly says otherwise.

2. **Phthalates**

   This family of chemicals softens plastics. Phthalates are considered endocrine disrupters. Research has also shown phthalates disrupt reproductive development. Avoid shampoos, conditioners and other personal care products that list "fragrance" as an ingredient.

3. **PFOA — Perfluorooctanoic acid (also called C8)**

   PFOA is used to make Teflon and other nonstick and stain- or water-repellent products. PFOA causes cancer and developmental problems. You can reduce your potential exposure by using stainless steel or cast iron cookware. If you use nonstick cookware, do not overheat it — this releases toxic gas.

4. **Formaldehyde**

   Formaldehyde is an ingredient in resins that act as a glue in the manufacture of pressed wood products. It is a known human carcinogen, causing cancers of the respiratory or gastrointestinal tract.

   Buying furniture free from formaldehyde eliminates much of the exposure you face from the chemical. If you have wood products containing formaldehyde, increase ventilation, reduce humidity with air conditioning or dehumidifiers and keep your home cool.

5. **PBDEs — Polybrominated diphenyl ethers**

   PBDEs are a group of chemicals used as flame retardants. Toxicology tests show PBDEs may damage your liver and kidneys and affect your brain and behavior. Try to find products without PBDE flame retardants and be sure to sweep up dust.

Sources:

» CNN May 31, 2010

Dr. Mercola's Comments:

Thanks to the spoils of the industrial revolution, your body is now home to a growing cocktail of chemicals.

Intermingling with your red and white blood cells, your endocrine system, brain, tissues and other organs are chemicals used to make epoxy resins, non-stock cookware, flame-resistant upholstery and plastic -- clearly substances that have no business taking residence in a living, breathing creature such as yourself.

Your Body Probably Contains Over 200 Chemicals

A typical American comes in regular contact with 6,000 chemicals and an untold number of potentially toxic substances on a less frequent basis. There are about 75,000 chemicals regularly manufactured and imported by U.S. industries, so you could potentially be exposed to any number of them.

Given the vast amounts of chemicals in the environment, it's not too surprising that the CDC's Fourth National Report on Human Exposure to Environmental Chemicals found an average of 212 chemicals in Americans' blood or urine.

Likewise, an Environmental Working Group study found that blood samples from newborns contained an average of 287 toxins, including mercury, fire retardants, pesticides, and Teflon chemicals, and this is from exposures they received *before birth*.

When it comes to the potentially hazardous chemicals you and your family are exposed to as you go about your daily lives, it can easily feel overwhelming. There are chemicals literally everywhere, but rather than feeling burdened by the thought I encourage you instead to focus on simple steps you can take to reduce your risk.

A good starting point, as CNN as suggested above, is to focus on avoiding some of the *most pervasive*, and most toxic, chemicals that are virtually guaranteed to be in your home right now.

Five Top Common Chemicals to Avoid ...

The five chemicals listed by CNN are definitely worthy of eliminating from your life as much as possible, and given that they are among the most widely used chemicals around, doing so will make a serious positive impact on your chemical exposure.

They gave a great summary above, but I'll touch on them again briefly here:

- **BPA:** BPA is one of the world's highest production-volume chemicals and is widely used in the production of plastics, canned foods and soda cans, food packaging, baby bottles and toys and more.

  The chemical can lead to heart disease, diabetes and liver problems in adults, and previous research has linked BPA to serious developmental and reproductive problems.

  You can find 10 tips to minimize your BPA exposure here.

- **Phthalates:** Phthalates, or "plasticizers," are a group of industrial chemicals used to make plastics like polyvinyl chloride (PVC) more flexible and resilient. They're also one of the most pervasive of the endocrine disrupters.

  These chemicals have increasingly become associated with changes in development of the male brain as well as with genital defects, metabolic abnormalities and reduced testosterone in babies and adults.

  You can help reduce your exposure by using the tips in this past article.

- **PFOA:** Teflon-coated cookware is the primary source of dangerous perfluorinated chemicals (PFOAs). Teflon pans quickly reach temperatures that cause the non-stick coating to begin breaking down, releasing toxins that have been linked to cancer, birth defects and thyroid disease into the air in your kitchen.

  I highly recommend you throw away this type of non-stick cookware immediately and replace it with either ceramic or glass. My personal choice is ceramic cookware, because it's very durable and easy to clean, and there's absolutely no risk of exposure to harmful chemicals.

- **Formaldehyde:** Formaldehyde, most commonly known as embalming fluid, serves a number of purposes in manufactured products. It is actually frequently used in fabrics to give them a variety of "easy care properties" as well as being a common component of pressed-wood products.

  Formaldehyde has been shown to cause cancer in animals, and may cause cancer in humans. Other common adverse health effects include fatigue, skin rashes, and allergic reactions. Choosing all natural materials for your clothing and furniture can help cut down on your exposure.

- **PBDEs:** These flame-retardant chemicals have been linked to altered thyroid levels, decreased fertility and numerous problems with development when exposure occurs in utero. PBDEs are commonly found in household items like upholstery and television and computer housings. Fortunately, several states now ban the use of PBDEs, so there is some progress toward reducing exposure.

  Another common source of PBDEs is your mattress, and since you can spend up to a third of your life in bed, this is a significant health concern. Mattress manufacturers are not required to label or disclose which chemicals their mattresses contain. Look for 100 percent wool, toxin-free mattresses.

  Another viable option is to look for a mattress that uses a Kevlar, bullet-proof type of material in lieu of chemicals for fire-proofing. Stearns and Foster uses this process for their mattresses, which is sufficient to pass fire safety standards.

What Else Can You do to Reduce Unnecessary Chemical Exposure to Your Family?

Rather than compile an endless list of what you should *avoid*, it's far easier to focus on what you should do to lead a healthy lifestyle with as minimal a chemical exposure as possible:

1. As much as possible, buy and eat organic produce and free-range, organic foods to reduce your exposure to pesticides and fertilizers.
2. Rather than eating conventional or farm-raised fish, which are often heavily contaminated with PCBs and mercury, supplement with a high-quality purified krill oil, or eat fish that is wild-caught and lab tested for purity.
3. Eat mostly raw, fresh foods, steering clear of processed, prepackaged foods of all kinds. This way you automatically avoid artificial food additives, including dangerous artificial sweeteners, food coloring and MSG.
4. Store your food and beverages in glass rather than plastic, and avoid using plastic wrap and canned foods (which are often lined with BPA-containing liners).
5. Have your tap water tested and, if contaminants are found, install an appropriate water filter on all your faucets (even those in your shower or bath). My personal favorite, and the one I personally use, is a high-quality reverse osmosis (RO) filter. You just need to add a few minerals back to the water, but RO reliably removes virtually every possible contaminant that could be in the water.
6. Only use natural cleaning products in your home.
7. Switch over to natural brands of toiletries such as shampoo, toothpaste, antiperspirants and cosmetics. The Environmental Working Group has a great safety guide to help you find personal

care products that are free of phthalates and other potentially dangerous chemicals. I also offer one of the highest quality organic skin care lines, shampoo and conditioner, and body butter that are completely natural and safe.

8. Avoid using artificial air fresheners, dryer sheets, fabric softeners or other synthetic fragrances.
9. Replace your Teflon pots and pans with ceramic or glass cookware or a safe nonstick pan.
10. When redoing your home, look for "green," toxin-free alternatives in lieu of regular paint and vinyl floor coverings.
11. Replace your vinyl shower curtain with one made of fabric, or install a glass shower door.

It is important to make these positive and gradual steps toward decreasing your chemical risk through healthy lifestyle choices. While you make the switch to remove and reduce chemicals around your home, remember that one of the ways to significantly reduce your toxic load is to pay careful attention to what you eat.

Organically-grown, biodynamic whole foods are really the key to success here, and, as an added bonus, when you eat right, you're also optimizing your body's natural detoxification system, which can help eliminate toxins your body encounters from other sources.

Male fish now exhibiting female traits due to toxic chemicals and pharma runoff

Sunday, September 12, 2010 by: David Gutierrez, staff writer

(NaturalNews) More than 80 percent of male bass in the Potomac River on the U.S. Atlantic coast are producing eggs or showing other female traits, the nonprofit Potomac Conservancy has warned, in a call for more research into the causes of intersex fish.

In a recent U.S. Geological Survey (USGS) report, intersex fish were found in a third of all 111 sites tested across the United States, including in major waterways such as the Mississippi River and the Rio Grande. The phenomenon occurred in 16 different species, but was most common in male smallmouth and largemouth bass.

Researchers agree that the phenomenon is almost certainly caused by the presence of pollutants in the water, including endocrine- (hormone) disrupting chemicals and the residue of pharmaceutical products.

"We have not been able to identify one particular chemical or one particular source," said USGS biologist Vicki Blazer. "We are still trying to get a handle on what chemicals are important."

Among the chemicals likely to be contributing to the problem, Blazer cited birth control pills and other hormone-containing drugs, antibacterial products including tissues, personal care products (especially those containing fragrances), flame retardants, pesticides and fertilizers.

"In fertilizer [and pesticides] there's natural estrogen and testosterone and other things ... so if we can hopefully pinpoint some of those mixtures or individual chemicals that then perhaps we could manage better," Blazer said.

It has been hard to narrow down the list of major contributors, however. For example, Blazer tested fish up- and downstream of sewage treatment plants to see if the factories might be major sources of endocrine-disrupting pollutants. She found no difference in rates of sexual abnormalities.

The Potomac Conservancy has called for more research into the problem.

"We've got to figure out what the heck is going on here," said the group's president, Hedrick Belin. "And we've got to figure it out sooner rather than later because it's clear the longer this mystery continues it's only going to lead to bad things yet to be discovered."

Because the hormonal systems of all vertebrates are strikingly similar, anything that has an impact on

fish living in water is likely to have an effect on humans drinking it, as well. Yet figuring out the specific effects of tainted water on people may prove difficult.

"Because fish, of course, are in the water all the time," Blazer said. "But what's in your drinking water, what you might be exposed to through skin and food and everything else, is another issue for people."

Even if researchers eventually figure out which chemicals are the major contributors to sexual deformity in fish, that may shed little light on the question.

"It's going to be a lot harder to get to how these chemicals affect people because of course you can't experiment on people," Blazer said.

Approximately 4.5 million residents of the Washington D.C. area get their drinking water from the Potomac.

According to the Potomac Conservancy, individuals can help reduce watershed pollution in part by making more careful purchasing decisions. Consumers should reduce their use of toxic chemicals such as pesticides, and look for more natural cosmetics and other products.

"The chemicals that are in personal care products such as some of the antimicrobials, fragrances, are endocrine disruptors," said Blazer. "So being smart about the kinds of products you're buying -- because they are available in things that are fragrance-free, antimicrobial-free, things like that -- are things that individuals can do."

Conservancy supporter Rep. James P. Moran of Virginia has urged people to always take old or unused drugs back to a pharmacy for disposal.

"Don't flush pharmaceuticals down the toilet," he said. "They don't disappear when you flush them."

The Potomac Conservancy is also working on a campaign to get pharmaceutical technologies to dispose of drugs more safely, and calling for better water filtration technology.

"We need to get these toxins out of our river water," Belin said.

Sources for this story include: http://www.guardian.co.uk/environme... http://www.wvpubcast.org/newsarticl... http://www.washingtonexaminer.com/l... http://sundaygazettemail.com/News/2....

Mercury Levels in Fish

Tuna is an excellent source of the brain and mood-boosting omega-3s, but also can pack in hefty doses of neuro-toxic mercury that damage mental and heart function. Researchers at Columbia University in New York report that some tuna is safer than others. Using genetic analysis to identify the species of fish in 100 samples of sushi-grade tuna, from 54 restaurants and 15 supermarkets across the country, the researchers found that mercury levels increased with increasing size of the fish from yellowfin to bluefin. Some samples exceeded the 1 ppm level that allows the US Food and Drug Administration to remove a food from the market. The levels were high enough in most samples for the researchers to recommend that health agencies consider adding bigeye and bluefin tuna to lists of fish that should be avoided, especially in children and pregnant or breastfeeding women.

Researchers at the University of Nevada investigated mercury levels in canned tuna and found that average mercury concentrations in all brands studied exceeded the 0.5ppm limit and up to 7% of the samples had levels higher than the 1ppm. They conclude that light tuna, which is mostly skipjack, is the best choice, since its mercury content is closer to 0.28ppm compared to 0.5ppm in chunk white tuna, which is mostly albacore.

Lowenstein J, Burger J, Jeitner C, et al: DNA barcodes reveal species-specific mercury levels in tuna sushi that pose a health risk to consumers. Biology Letters 2010;April 21st.

Gerstenberger S, Martinson A, Kramer J: An evaluation of mercury concentrations in three brans of canned tuna. Environmental Toxicology and Chemistry 2010;29:237-242.

Folic Acid Helps Prevent Cancer

Poor diet is a major risk factor in more than a third of all cancer cases. Folate, found in leafy green vegetables, maintains the cells' genetic code and a deficiency of this B vitamin is associated with the development of several cancers, including cancer of the colorectum, breast, ovary, pancreas, brain, lung and cervix. Researchers at the University of Aberdeen report that people who habitually consume the highest level of folate, or with the highest blood folate levels, are at reduced risk for colon polyps or cancer. Folate maintains genetic stability by regulating DNA biosynthesis, repair, and methylation, while folate deficiency induces and accelerates carcinogenesis by interfering with each of these processes. The researchers go on to say that suboptimal folate status in humans is widespread, even with folic acid fortification of processed grains.

Duthie S: Folate and cancer: how DNA damage, repair and methylation impact colon carcinogenesis. Journal of Inherited Metabolic Diseases 2010;June 11th.

HOT TOPICS: Short bursts of vigorous exercise help offset the pro-aging effects of stress on body cells, possibly by protecting the tiny shoelace-like endings on DNA strands that if allowed to unravel promote cell aging and cell death, state researchers at the University of California, San Francisco. *PloS One 2010;5:e10837.*

Mothers who consume high glycemic diets (processed foods, potatoes, refined grains, sugar) during pregnancy might increase the chances of giving birth to babies with neural tube defects, according to a study from Boston University, while high glycemic diets also are linked to an increased risk for uterine fibroids. *American Journal of Epidemiology 2010;171:407-414/American Journal of Clinical Nutrition 2010;March 3rd.*

Scientists deliver wake-up call:
"Reduce chemical exposure to reduce breast cancer"

Brussels 2 April 2008 – Around 1 in 10 women in Europe will develop breast cancer. Bringing down this figure cannot be achieved without reducing exposure to certain chemicals, according to a report launched in the European Parliament today. (1) (2)

Entitled "Breast cancer and exposure to hormonally active chemicals", the report was written by Professor Andreas Kortenkamp, head of the Centre for Toxicology, School of Pharmacy, University of London.

Prof. Kortenkamp coordinated a major cluster of international research projects on endocrine (hormone) disrupting chemicals between 2002 and 2007. These projects received more than 20 million Euros in EU funding. (3)

"Good laboratory and epidemiological evidence exists suggesting that man-made chemicals which mimic oestrogen contribute to breast cancer," says Professor Kortenkamp.

"We will not be able to reduce the risk of breast cancer without addressing preventable causes, particularly exposure to chemicals."

Since 2005, 200 international scientists, including Professor Kortenkamp, have signed the Prague Declaration, which expresses scientific concerns related to the risks posed to health by chemicals in everyday use, particularly those that interfere with the human hormone system. (4)

The new report represents an appraisal of existing research and calls for action to reduce people's exposures to the chemicals that disrupt hormones and mimic oestrogen. The Health and Environment Alliance (HEAL) and CHEM Trust commissioned the research review as part of the Chemicals Health Monitor project (5) at a time when breast cancer is reaching epidemic proportions.

Over the past 20 years, breast cancer has increased dramatically throughout Europe, with incidence in some countries increasing by more than 50% or even doubling in the last 20 years. (6)

Elizabeth Salter Green Director of CHEM Trust says: "There is a misconception that breast cancer is an inherited disease and therefore inevitable. This is a quite simply a myth, most cases of breast cancer are acquired over a women's lifetime, and so most are preventable." (7)

European Parliamentarians could play a pivotal role in reducing breast cancer. "They need to wake up to what needs to be done," Ms Salter Green says. "Unfortunately, good science and its expert interpretation by scores of scientists throughout the EU have not yet been taken up and turned into effective policy action."

Génon Jensen, HEAL Executive Director urges MEPs not to miss the current policy opportunities that exist. They include specific action in three legislative areas: first, the EU chemical safety regulation, known as REACH; second, pesticide reform; and third, the directive on cosmetics that is currently under review. Ms Jensen says: "Several man-made chemicals should be removed from the market and replaced with less harmful substitutes."

"Such action could reduce the suffering of millions of women, and finally deliver the reduction in exposure that dozens of eminent EU scientists working at the cutting edge of research called for in the Prague Declaration nearly three years ago," Ms Jensen concludes.
-ends-

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Notes for journalists

1. The report is entitled "Breast cancer and exposure to hormonally active chemicals: An appraisal of the scientific evidence". It is a background briefing paper by Professor Andreas Kortenkamp, Head of Centre for Toxicology, School of Pharmacy, University of London, UK, April 2008, 16 pages. It is available from 01.01 hours (CET) on Wednesday, 2 April 2008 at www.chemicalshealthmonitor.org
2. Invitation and launch announcement - The presentations, debate and lunch take place in the Members' Restaurant (private salon), European Parliament, 12.30-14.30. It is hosted by MEP Avril Doyle. Details of the meeting at: www.chemicalshealthmonitor.org Avril Doyle is a member of MEPs Against Cancer, which has a membership of over 60 MEPS representing most EU countries: http://www.mepsagainstcancer.org
3. More information about report author, Professor Andreas Kortenkamp is available at http://www.pharmacy.ac.uk/andreas_kortenkamp.html
4. The Prague Declaration is available at http://www.ehponline.org/docs/2007/10517/suppl.pdf
5. Chemicals Health Monitor project website at http://www.chemicalshealthmonitor.org/
6. As shown in the latest available data up to 2005 in World Health Organisation (WHO)/ Europe (2007), European health for all database (HFA-DB), World Heath Organisation Regional Office for Europe. Data-base online at http://data.euro.who.int/hfadb/
   Breast cancer incidence graph can be accessed online at www.chemicalshealthmonitor.org
7. The widely held view that breast cancer is exclusively a genetic disease is inaccurate. Eight out of 9 women who develop breast cancer do not have an affected mother, sister, or daughter, according to the report "Factors affecting the risks of breast cancer" available on meeting report page at www.chemicalsmonitor.org.

Health and Environment Alliance (HEAL) *aims to raise awareness of how environmental protection and sustainability improves health and to empower the health community to contribute their expertise to policy making. Since its inception, HEAL's membership has grown to include a diverse network of more than 50 citizens', patients', women's, health professionals' and environmental organizations across Europe which together have a strong track record in increasing public and expert engagement in both EU debates and the decision-making process. Website: www.env-health.org*

CHEM Trust *is a UK charity whose aim is to protect humans and wildlife from harmful chemicals particularly hormone disruptors, the cocktail effect of chemicals and the role of chemical exposures in early life. CHEM Trust is working towards a goal where chemicals play no part in causing impaired reproduction, deformities, disease or deficits in neurological function. CHEM Trust is committed to engaging with medical, scientific and patient communities to raise the level of dialogue on the role of chemicals in chronic disease, and the wider implications this may have for disease prevention strategies. Website: http://www.chemtrust.org.uk/*

Chemicals Health Monitor Project (CHM) *was launched by HEAL, CHEM Trust, Collaborative on Health and Environment and others in March 2007. It aims to improve public health by ensuring that key scientific evidence on the links between chemicals and ill-health are translated into policy as quickly as possible. Key documents about the campaign and information about the project can be found at: http://www.chemicalshealthmonitor.org*

The Project Co-ordinator is ***Hana Kuncova*** *e-mail: hana@env-health.org*

Study suggests link of cleaners to breast cancer by Drew Kaplan July 23, 2010

A survey of Massachusetts women has found a potential link between the use of household cleaners and air fresheners and breast cancer.The study included interviews with 787 women who had breast cancer and 721 who did not. Researchers asked all the women about pesticide use but found little association.But when about 400 women in each group were asked about cleaning products, researchers found a potential connection.In fact, breast-cancer risk was highest among women who reported the most use of cleaning products and air fresheners; it was double the risk for those who reported low use of the products. Most study participants were white and middle-aged and were part of the Cape Cod Breast Cancer and Environment Study, which had financial support from the state of Massachusetts.

The results are published in the journal Environmental Health.

The connection was drawn mostly between mold and mildew cleaners and air fresheners. Surface and oven cleaners were not associated with increased risk. Chemicals of concern include synthetic musks, phthalates, 1,4-dichlorobenzene, terpenes, benzene and styrene and some antimicrobial agents, said Julia Brody, the lead researcher and executive director of the Silent Spring Institute.

Studies of this nature come with an inherent weakness, called recall bias. The researchers acknowledged that women who have cancer and believe in an association with cleaners might be more likely to report high use of them.

That said, the study adds weight to previous animal research showing that the same chemicals cause mammary-gland cancer in animals and disrupt the endocrine system, contributing to tumor growth, Brody said.

Much about the causes of breast cancer remains unknown. Many patients believe in environmental links, but they are notoriously difficult to prove.

“Although there seems to be an association between cleaning products and cancer, that’s a long way from saying, ‘Cleaning products cause breast cancer,’” said Dr. Charles Shapiro, director of breast medical oncology at the Ohio State University Comprehensive Cancer Center.

“I wouldn’t take too much from it,” Shapiro said, noting that the study was relatively small and that it’s impossible to draw definitive conclusions about cause and effect. He also cautioned that what is found in the laboratory in animal models doesn’t necessarily play out among humans.

“The take-home, if any, is if you’re worried about it, try to avoid those products,” he said.

Sandra Steingraber, a New York ecologist, cancer survivor and author of Living Downstream, said she’d advise everyone to stop using chemical cleaners. She uses vinegar and baking soda to clean her house.

“I just see this as such an easy problem compared to a lot of things,” said Steingraber, who is on the faculty at Ithaca College.

The new research, she said, “points to the really vexing problems of trying to make correlations between past exposures and present disease rates.”

Steingraber said: “Clearly, the conversation is shifting now. We can’t just sort of look at the murky evidence on cancer and the environment and sort of set it aside because it’s too inscrutable.”

http://www.dispatch.com/live/content/national_world/stories/2010/07/20/study-suggests-link-of-cleaners-to-breast-cancer-update.html?sid=101

Teflon non-stick Death by Tammera J. Karr, PhD

Link: http://yourwholenutrition.com/blog/eat-for-health/teflon-non-stick-death/

If you are the cook in your family, you know how it feels to be in someone else's kitchen or at a motel with a kitchenette and not have, those essential utensils to cook with that you are accustomed to. While in *sin city,* we were fortunate to stay in a hotel that was not a casino and did have a kitchen in our room. While this made our stay more bearable, the kitchenette was filled with Teflon coated pans and plastic serving utensils.

For home cooks like me, it was a visit to the haunted kitchen – the Spector's of Teflon poisoning, radiation from the microwave, poisons in the water, the noise, smoke and lights…eeehhhhhh For those who are informed or nuts depending on how you look at it – it is scary what consumers are ingesting, and exposing themselves, pets and children to, not to mention the poor canaries.

Yup, the canary in your kitchen works just like those sent into a mine. When toxic chemicals are released from Teflon/non-stick cookware, bird lovers all over the country have reported their tweeties face planting in the bottom of the cage – dead as a door nail.

"The federal government announced in 2006, enough health concerns have been raised to virtually eliminate continued exposure to the key chemical used to make Teflon." Evidence is piling up that emissions from the production of synthetic compounds in non-stick cookware, cleaning products, and a host of other common products may cause cancer and other health problems.

"Better things for better living — through chemistry." From the 1940s to the 1980s, E.I. DuPont de Nemours and Co. wooed customers with that slogan, one of the most memorable in American advertising. But today, two groups of DuPont products developed during that era — fluorotelomers and fluoropolymers — are showing how chemical-dependent "better living" can come at a high price.

DuPont and other companies use those synthetic compounds to make an extraordinarily wide range of products, including nonstick cookware (e.g, Teflon), grease-resistant food packaging (e.g., microwave popcorn and pizza boxes), stain-resistant fabrics and carpets (e.g., Stainmaster), shampoos, conditioners, cleaning products, electronic components, paints, firefighting foams, and a host of other artifacts of modern life.

Teflon is a $2 billion-a-year business and one of the country's best-known products. DuPont once called it the housewife's best friend. However, like many "better things" produced by industrial chemistry, these products can have disastrous side effects.

In 2006, the federal government said DuPont had voluntarily agreed to eliminate by the year 2010 any new emissions of the key Teflon chemical from its factories.[1]

Really did you hear about this in the news, get a recall postcard or see any warning labels?

Non-stick surfaces are metal pans (such as aluminum) coated with a synthetic polymer called polytetrafluoroetheylene (PTFE), also known as Teflon, a DuPont brand trademark.

Toxic fumes from the Teflon chemical released from pots and pans at high temperatures may kill pet birds and cause people to develop flu-like symptoms (called "Teflon Flu" or, as scientists describe it, "Polymer fume fever"). Some early studies have suggested that higher PFOA blood

levels in humans may be linked with higher than normal cholesterol levels, thyroid disease, and reduced fertility.[2]

Readers this is a real horror story, not a modern myth – how many Teflon pans do you own or coated product do you use, your kids and grand children? All for the sake of convenience, we have taken into our homes monsters.

Manufacturers' labels often warn consumers to avoid high heat when cooking on Teflon. But EWG-commissioned tests conducted in 2003 showed that in just two to five minutes on a conventional stove top, cookware coated with Teflon and other non-stick surfaces could exceed temperatures at which the coating breaks apart and emits toxic particles and gases.[3]

When reading through cancer risk information on the *American Cancer Society's* website the following information caught my attention: Teflon itself is not suspected to cause cancer. PFOA may be more of a health concern because it can stay in the environment and in the human body for long periods of time. It seems to be present at very low levels in just about everyone's blood. It's not clear how people are exposed to it, although it has been detected at low levels in some foods and drinking water systems and in household dust.

The possible effects of PFOA on cancer risk in humans are not completely understood. Studies in lab rodents have found exposure to PFOA increases the risk of certain tumors of the liver, testicles, mammary glands, and pancreas in these animals.[4]

Although DuPont has never conceded that PFCs might cause health or environmental problems, the company has bowed to relentless and rising public pressure in recent years and moved to rein in its emissions. But whatever action is taken at this point, a class of molecules that did not exist on the planet before the 20th century is now here to stay.

What are my choices?

Stainless steel is a terrific alternative to a non-stick cooking surface. Most chefs agree that stainless steel browns foods better than non-stick surfaces.

Cast iron remains a great alternative to non-stick cooking surfaces. Lodge, America's oldest family-owned cookware manufacturer refers to its cookware as "natural non-stick." Cast iron is extremely durable and can be pre-heated to temperatures that will brown meat and will withstand oven temperatures well above what is considered safe for non-stick pans.

Glass pans and baking dishes conduct heat efficiently and are easy to clean.

Stoneware is also very popular for backing on to achieve that perfect crust on breads and pizzas.

Beware of the scary monsters in your kitchen.

[1] http://eartheasy.com/article_teflon_toxicity.htm
[2] http://www.cancer.org/cancer/cancercauses/othercarcinogens/athome/teflon-and-perfluorooctanoic-acid–pfoa
[3] http://www.ewg.org/healthyhometips/dangersofteflon
[4] http://www.cancer.org/cancer/cancercauses/othercarcinogens/athome/teflon-and-perfluorooctanoic-acid–pfoa

The Alarming Reason Why More Girls are Starting Puberty Early

By Mercola

U.S. girls are entering puberty at a younger age than in the past. More than 10 percent of white 7-year-old girls have breast changes that signal the start of puberty, according to a study conducted in the mid-2000s. This is up from just 5 percent in the early 1990s.

The average age of a girl's first period also declined, from an average of 14 or 15 years in the past to about 12 years today.

CNN reported:

"Experts aren't sure what's behind the increase in earlier puberty, but it's likely due to a combination of factors, including the childhood obesity epidemic and substances in the environment.

Early puberty in girls is a growing public health concern because studies have shown that girls who start puberty earlier are more likely to develop breast and uterine cancer later in life …

Early development in girls has been linked with poor self-esteem, eating disorders, and depression, as well as cigarette and alcohol use and earlier sexual activity."

As for what's causing the increasing in early puberty, chemicals in the environment, obesity, and above-average weight gain during infancy are all being considered as possible factors.

Sources:

- CNN.com August 10, 2010
- Time August 9, 2010
- Pediatrics August 9, 2010; [Epub ahead of print]

Dr. Mercola's Comments:

You're hearing quite a bit about precocious puberty these days, and I believe the data is indicative of a public health disaster yet to come.

Why?

Because the earlier you enter puberty, the longer you're exposed to elevated levels of the female hormone estrogen, which is a risk factor for certain cancers such as breast cancer. Time will tell whether or not estrogen-related cancer rates will rise dramatically as the current generation of youngsters enters adulthood and middle age, but there's certainly that risk.

The study published in *Pediatrics* at the beginning of this month compared results from two previous studies that used breast development to gauge the beginning of puberty. One was conducted in the early 1990s and the other about five years ago.. In that time span, the rate of white 7-year-old girls entering puberty **doubled**, from five percent to more than 10 percent.

Black and Hispanic girls are, on average, maturing at an even faster rate, with nearly 25 percent of black girls and 15 percent of Hispanic girls entering puberty by age seven!

The average age of the first period has declined as well. In the 19th century the onset of menstruation occurred around the age of 15. Now the average age of the first period is around 12.

There's no denying that something odd is afoot. The question is, will our government and health agencies do anything about it? So far, efforts to reduce the public's exposure to the most obvious contributors have been successfully suppressed by industry interests...

What's Causing Precocious Puberty?

Scientists have brought forth a number of potential explanations, and in all likelihood it's a combination of factors, such as:

- Hormones in food
- Pesticides in produce
- Obesity (which exposes girls to more estrogen because estrogen is stored in fat tissue)
- Phthalates in plastics and cosmetics

It's now a well-established fact that commonly used plastic chemicals, such as bisphenol A and phthalates, disrupt the human endocrine-system and affect your hormones, which control development and function in your body.

There's also mounting evidence that these chemicals can cause developmental harm in fetuses and children, either through active exposure during pregnancy and/or while nursing, or due to the preexisting toxic load of the mother.

Unfortunately, it's near impossible to actually study and test these factors because we're surrounded by so many estrogen-like chemicals, there are virtually no 'clean' control populations with which to compare!

It is truly a sad testament to the level of chronic toxicity we humans now live in.

However, although we may not be able to determine the exact extent of damage caused by each individual factor, you can protect your health, and the health of your family, by paying closer attention to your lifestyle choices in general.

Your Food Choices are of Paramount Importance

Obesity, hormones and pesticide residues in your food, and precocious puberty can be viewed as a linked triad.

Overweight children have elevated levels of insulin, an increased ability to convert hormones into estrogen, and an increased ability to store environmental toxins, all of which may contribute to early puberty. But obesity is not a cause of early puberty in and of itself.

It is, however, directly linked to diet, and the *primary reason* why diet may be a driving factor behind the early puberty phenomenon is the excessive use of hormones and other estrogen-mimicking chemicals in livestock and dairy production.

The US FDA currently allows six different kinds of steroid hormones to be used in food production.

1. Estradiol -- natural female sex hormone
2. Progesterone -- natural female sex hormone
3. Testosterone – natural male sex hormone
4. Zeranol – synthetic growth promoter
5. Trenbolone acetate– synthetic growth promoter
6. Melengestrol acetate– synthetic growth promoter

Federal regulations allow these to be used to 'beef up' cattle and sheep, but not poultry or pigs.

In addition to these types of growth hormones, most conventional meats are also heavily contaminated with pesticides, courtesy of the cattle's grain- and corn-based diet.

Avoid Non Organic Meats and Dairy as They are Loaded With Puberty Inducing Chemicals

This is why I only promote eating organically raised and grass-fed meats.

Despite the difficulty testing the impact of factors such as hormones in food on precocious puberty, studies have been able to discern a distinct dose relationship.

A recent study published in the journal *Public Health Nutrition*, for example, found that 49 percent of girls who ate meat 12 times a week at the age of 7 had reached puberty by the age of 12 1/2, compared with 35 percent of those who ate meat four times a week or less.

This at least will give you an inkling of how hormone-laced meat alone can alter your child's development.

But meat is not the only food group capable of wreaking havoc on your child's delicate hormonal balance.

Dairy is another major source.

The genetically engineered recombinant bovine growth hormone (rBGH or rBST) is permitted to be used on dairy cows to increase milk production, despite the fact that rBGH-containing milk contains high levels of a natural growth factor (IGF-1), which has also been incriminated as a major cause of breast, colon, and prostate cancers.

Unfortunately, labeling is not required by law. Some brands will state that their milk is "rBGH-free" however, and organic dairies also do not use rBGH. Either of these are certainly preferable to milk that contains this dangerous hormone, but I still don't recommend drinking any kind of pasteurized milk, organic or otherwise.

You can avoid *both* the risks of rBGH and pasteurization by only drinking raw milk, preferably from a trusted local farmer. This is really the only milk worth drinking if you want to protect your health and certainly that of your growing children.

The So-Called 'Health Food' that Wreaks Havoc on Your Hormones

I'm talking about unfermented soy in its many varieties.

Yes, this so-called "health food" is anything but healthy, especially for infants and children. Sadly, some misinformed moms feed their vulnerable babies soy infant formula, which exposes their child to the equivalent of five birth control pills' worth of estrogen every day!

It's also important for pregnant women to avoid eating non-fermented soy, as a high estrogenic environment in utero may increase their child's subsequent breast cancer risk.

Also keep in mind that soy is present in virtually every processed food and that over 95 percent of soy is GMO, which has its own set of health risks.

Americans are consuming soy in unprecedented quantities. Limiting or eliminating processed foods from your family's diet would clearly be one of the best health investments you could ever make, for a number of reasons besides reducing your soy intake.

The Harmful "Yin and Yang" of the Gender-Bender Chemicals

We now know that plastics contain a number of estrogen-mimicking chemicals that easily leach out, contaminating everything it touches, such as food and beverages.

Two of the most studied chemical types in this group are bisphenol A (BPA) and phthalates, which could be viewed as the yin and yang of harmful plastic chemicals.

BPA acts as a synthetic estrogen (female hormone) while phthalates have increasingly become associated with reduced testosterone (male hormone) in both babies and adults, along with developmental changes in the male brain, genital defects, and metabolic abnormalities.

BPA is typically found in more rigid plastic products and containers (as well as the lining of cans), while phthalates are a typical ingredient in softer, more pliable plastics.

These two are some of the most pervasive 'gender-bender' chemicals we know of. BPA, for example, has been detected in the umbilical cord blood of 90 percent of newborn infants tested.

If you look around your house, you're likely to find either or both of these chemicals in abundance, unless you've gone really "green."

Avoiding BPA-containing plastic products is an important step to limit your BPA exposure. Remember, this chemical is typically found in food and beverage containers, and has been shown to leech into whatever food or beverage it comes in contact with. Ridding your household of these items is therefore especially important if you're pregnant, planning a pregnancy, or have young children.

For more information about BPA and tips on how to avoid it, please review this previous article.

Avoiding phthalates is a bit trickier as these chemicals can be found in an even more diverse array of products – everything from toys, to floors, detergents, personal care products and cosmetics, just to name a few.

For more on phthalates and potential sources to avoid, please see this link.

Tips to Avoid the Most Pervasive Gender-Bender Chemicals

There are about 75,000 chemicals regularly manufactured and imported by U.S. industries – the vast majority of which have never been tested for safety. Rather than compile an endless list of what you should *avoid*, it's far easier to focus on what to do to minimize your exposure.

Here are 11 measures you can implement right away to protect yourself and your children from common toxic substances that could cause precocious puberty and long-term health problems:

1. As much as possible, buy and eat organic produce and free-range, organic meats to reduce your exposure to added hormones, pesticides and fertilizers.
2. Eat mostly raw, fresh foods. Processed, prepackaged foods (of all kinds) are a major source of soy and chemicals such as BPA and phthalates.
3. Store your food and beverages in glass rather than plastic, and avoid using plastic wrap and canned foods (which are often lined with BPA-containing liners).
4. Use glass baby bottles and BPA-free sippy cups for your little ones.
5. Make sure your baby's toys are BPA-free, such as pacifiers, teething rings and anything your child may be prone to suck on.
6. Only use natural cleaning products in your home to avoid phthalates.

7. Switch over to natural brands of toiletries such as shampoo, toothpaste, antiperspirants and cosmetics. The Environmental Working Group has a great safety guide to help you find personal care products that are free of phthalates and other potentially dangerous chemicals.
8. Avoid using artificial air fresheners, dryer sheets, fabric softeners or other synthetic fragrances, many of which can also disrupt your hormone balance.
9. Replace your Teflon pots and pans with ceramic or glass cookware. Teflon contains PFOA, which has been linked to thyroid disease.
10. When redoing your home, look for "green," toxin-free alternatives in lieu of regular paint and vinyl floor coverings.
11. Replace your vinyl shower curtain with one made of fabric.

Theo Colburn's book *Our Stolen Future* is a great source for further investigation as it identifies the numerous ways in which environmental pollutants are disrupting human reproductive patterns. I believe it is one of the best resources on this topic and highly recommend it.

The Soap You Should Never Use -- But 75% of Households Do

By Dr. Mercola

Link: http://articles.mercola.com/sites/articles/archive/2012/08/29/triclosan-in-personal-care-products.aspx

Triclosan, a high production volume ingredient used as a bactericide in personal care products such as toothpaste, deodorant, and antibacterial soap, has been linked to heart disease and heart failure in a new study.

Yet the U.S. Food and Drug Administration (FDA) states that "Triclosan is not currently known to be hazardous to humans."[1]

What this means is that until action is taken to get this common additive out of your toiletries, you could be applying a chemical with proven toxicity to your body multiple times a day …

Triclosan Interferes With Muscle Function

Tricolsan impairs muscle function and skeletal muscle contractility, researchers report in a new study done at the University of California Davis. Although the study was done in mice, researchers said the effects of the chemical on cardiac function were "really dramatic."

After mice were exposed to one dose of triclosan, heart muscle function was reduced by 25 percent, and grip strength was reduced by 18 percent. Fish were also exposed to triclosan – about the equivalent dose as would be accumulated in a week in the wild – and this led to poorer swimming performance. Researchers also exposed individual human muscle cells (from heart and skeletal muscles) to a triclosan dose similar to everyday-life exposure, and this, too, disrupted muscle function and caused both heart and skeletal muscles to fail.

The study's lead author noted:[2]

"Triclosan is found in virtually everyone's home and is pervasive in the environment. These findings provide strong evidence that the chemical is of concern to both human and environmental health."

Triclosan May Also Alter Hormone Regulation

This ubiquitous chemical is a chlorinated aromatic compound and is used to help reduce or prevent bacterial contamination. It's commonly added to many antibacterial soaps and body washes, toothpastes and certain cosmetics, as well as furniture, kitchenware, clothing and toys.

It would be wise to seriously question purchasing ANY product that contains triclosan as an ingredient on the label, not only because of the new muscle function finding discussed above, but also because of its potential impact on hormones.

A *Toxicological Sciences* study found that triclosan affected estrogen-mediated responses, and many chemicals that imitate estrogen are known to increase breast cancer risk.[3] Triclosan also suppressed thyroid hormone in rats, and this is only one study in an accumulating body of research showing this chemical to be a potent endocrine disrupter.

Past research has also shown:

- Exposure to triclosan disrupts thyroid hormone-associated gene expression in frogs, even at low levels (triclosan exposure at 0.15 *parts per billion* was enough to disrupt a hormone-signaling system in frogs)[4]
- Triclosan decreases circulating concentrations of the thyroid hormone thyroxine (T4) in rats[5]

Even the FDA states that "animal studies have shown that triclosan alters hormone regulation" and that "other studies in bacteria have raised the possibility that triclosan contributes to making bacteria resistant to antibiotics."[6] Although they still maintain that triclosan is not known to be hazardous to humans, they are conducting a review of the chemical, the results of which they expect to release to the public in the winter of 2012.

The U.S. Environmental Protection Agency (EPA), which regulates triclosan as a pesticide, has also announced it will undertake a comprehensive review of triclosan beginning in 2013, and notes they will "pay close attention to the ongoing endocrine research and will amend the regulatory decision if the science supports such a change." Unfortunately, what this means for you for now is that essentially *nothing* is being done right now to get this chemical out of your hand soap, body wash and toothpaste.

Triclosan Was First Registered as a Pesticide

If you need more indication that triclosan is probably not the best ingredient to be brushing your teeth with or rubbing onto your underarms, consider that it was first registered with the EPA in 1969 … as a pesticide.

Today it is still registered as a pesticide, although aside from this and its uses in personal care products, it's also widely used for industrial uses, for instance it is incorporated in conveyor belts, fire hoses, dye bath vats, or ice-making equipment as an antimicrobial pesticide, as well as added to adhesives, fabrics, vinyl, plastics (toys, toothbrushes), polyethylene, polyurethane, polypropylene, floor wax emulsions, textiles (footwear, clothing), caulking compounds, sealants, rubber, carpeting, and a wide variety of other products.[7]

Triclosan is Already Found in 75% of Americans

As it stands, this chemical has already been detected in the urine of three-quarters of the U.S. population,[8] which means urgent action is clearly needed. Last year, House Rules Committee Chairwoman Louise M. Slaughter and two colleagues called on the FDA to enact a ban on triclosan, noting that "triclosan is clearly a threat to our health" and citing the following reasons for the proposed ban:[9]

- *"The presence of triclosan in the human body and its impact on our 'body burden'*
- *Bacterial resistance to antibiotic medications and antibacterial cleaners*
- *The potential for endocrine disruption as a result of triclosan bioaccumulation in the body*
- *Wastewater contamination*
- *The threat of destroying ecological balance*
- *The fact that triclosan is no more effective than soap and water"*

This last point is an important one, as the "benefit" of adding an antimicrobial product like triclosan to your hand soap is that it should kill off more germs, and theoretically keep you healthier. On the contrary, there is little or no evidence that these triclosan-containing antibacterial products outperform the good-old-fashioned techniques like washing with soap and water. There is, however, evidence that *plain soap is more effective* than its antibacterial counterparts. Researchers noted:[10]

"The lack of an additional health benefit associated with the use of triclosan-containing consumer soaps over regular soap, coupled with laboratory data demonstrating a potential risk of selecting for drug resistance, warrants further evaluation by governmental regulators regarding antibacterial product claims and advertising."

It's Easy to Opt Out of Triclosan-Containing Products

The decision to stop using products that contain triclosan is an easy way to positively impact your and your family's health. There is simply no reason to ever purchase any product with triclosan in it. Triclosan is clearly listed on product ingredient labels, so you can easily check to see if it's there before deciding on a purchase. Remember, this chemical is not only in soaps but also body washes, toothpaste, shampoo, and 140+ other personal care and home products. Unfortunately, triclosan is now also contaminating rivers, streams and sewage sludge that is applied to agricultural fields, so there is a chance you're getting exposed from environmental sources as well.[11]

Aside from reading labels, if a product claims to be "antibacterial," there's a good chance it contains triclosan, so this can be used as a warning label of sorts if you're looking to avoid this chemical.

BREAST CANCER ACTION

What You Should Know About the Environment and Breast Cancer

Powerful evidence from experimental, body burden and ecological research indicates that there is a connection between chemicals and breast cancer. While we pursue the research that will lead to even more definitive answers, we can and should reduce our exposure to substances we believe cause cancer.

Why we believe there is a link between the environment and breast cancer:

- **70% of the people with breast cancer have none of the known risk factors.** The so-called known risk factors, like late menopause, having children late in life, and family history are present in only 30% of breast cancer cases.[1]
- **Non-industrialized countries have lower breast cancer rates than industrialized countries.** Breast cancer rates are highest in North America and northern Europe and lowest in Asia and Africa.[2]
- **People who move to industrialized countries from countries with low rates develop the breast cancer rates of the industrialized country.** For example, Japan has a lower rate of breast cancer than the United States. Japanese women who move to the United States have increased breast cancer rates (and their daughters even more so) that approach those of U.S.-born women.[3]

What we know:

- **Estrogen stimulates breast cell growth.** Excess amounts of estrogen are thought to contribute to breast cancer risk. Some chemicals, such as the pesticide dieldrin, organochlorines, and certain plastics additives, act like estrogen in the body.[4]
- **Production and use of harmful chemicals are on the rise.** Of an estimated 85,000 synthetic chemicals in commercial use today, more than 90% have never been tested for their effects on human health.[5] Meanwhile, production of these chemicals is rising at least 3.3% per year.[6] Use of carcinogenic pesticides in California rose 127% between 1991 and 1998.[7]
- **Ionizing radiation is a proven cause of breast cancer.** The effect of exposures to low levels of radiation can build up in the body and do harm. This does not mean that you should never get another x-ray, but you should be mindful of your exposure to radiation.

What needs to be done:

- **Take a "better safe than sorry" approach.** We, as a society and as individuals, must adopt a precautionary principle approach to public health—acting to reduce our exposure to known and suspected carcinogens now based on the weight of evidence indicating that they are harmful to our health.
- **Companies must be made accountable for the products they make.** Rather than waiting until a product is proven to be harmful, companies that produce (and profit from) chemicals should be required to prove that they are safe before they can market them.
- **Research into environmental links to diseases should be a priority.** In order to stop cancer where it starts, we need to spend as much time and money researching causes as we spend looking for cures.

Notes:

1 US General Accounting Office, "Breast Cancer 1971-1991: Prevention, Treatment & Research," GAO/PEMD-92-12, 1991.

2 Horn-Ross PL and Kelsey JL, "Breast Cancer: Magnitude of the Problem and Descriptive Epidemiology," Epidemiologic Reviews, 15:7-16 (1993). Pisani, P., "Breast Cancer: Geographic Variations and Risk Factors," Journal of Environmental Pathology, Toxicology and Oncology, 11:313-316 (1992).

3 Stanford JL, Herrinton LJ, Schwartz SM, Weiss NS, "Breast Cancer Incidence in Asian Migrants to the United States and Their Descendants," Epidemiology, 6(2):181-3 (1995 Mar).

4 National Academy Press, Hormonally Active Agents in the Environment, ISBN-0309-06419-8, 1999.

5 Bennett M, Davis BJ, The Identification of Mammary Carcinogens in Rodent Bioassays. Environmental and Molecular Mutagenesis. In press, 2002.

BREAST CANCER ACTION

What You Should Know About the Environment and Breast Cancer

6 Steingraber, S., Living Downstream: A Scientist's Personal Investigation of Cancer and the Environment, p. 281, New York: Vintage Books, 1998.

7 Kegley, Susan, Ph.D., "Hooked on Poison: Pesticide Use in California, 1991-1998," Californians for Pesticide Reform, 1999.

Glossary:

Cancer: a term for diseases in which abnormal cells divide without control.

Ionizing radiation: radiation that changes molecules when it passes through them, creating electrically charged particles (ions). Ionizing radiation is found in x-rays and nuclear waste.

What You Can Do:

Avoid exposures to toxins. If you can afford it, buy organic food. Use non-toxic cleaning and pest-control products Learn more at http://www.bcaction.org/Pages/GetInformed/NonToxicHome.html Chemicals in plastics are more likely to seep out when plastics get hot, so don't use plastic containers to microwave food. Stay away from unnecessary x-rays.

Get informed! Read Living Downstream by Sandra Steingraber, Silent Spring by Rachel Carson. Subscribe to BCA's newsletter, Rachel's Environment & Health News, and the Green Guide (contact information is below).

Get active! Push for more research into environmental links to cancer, and better regulation of the toxins we know or suspect are doing harm. Join BCA or the other environmental organizations listed below.

Cancer Organizations

Charlotte Maxwell Complementary Clinic
(free therapies for low-income women with cancer)
510-601-7660; www.charlottemaxwell.org

Women's Cancer Resource Center, Berkeley
510-420-7900; www.wcrc.org

Environmental Health and Justice Organizations

Californians for Pesticide Reform
415-981-3939; www.igc.org/cpr

Center for Environmental Health
510-594-9864; www.cehca.org

Communities for a Better Environment
510-302-0430; www.cbecal.org

Environmental Research Foundation
(publishers of Rachel's Environment & Health Weekly)
888-2RACHEL; www.rachel.org

Greenaction
415-252-0822; www.greenaction.org

Health Care Without Harm
703-237-2249; www.noharm.org

Natural Resources Defense Council (NRDC)
212-727-2700; www.nrdc.org

Silent Spring Institute
(Research)
617-332-4288; www.silentspring.org

[Updated October 2004]

Breast Cancer Action
55 New Montgomery Street
Suite 323
San Francisco, CA 94105

877-2STOPBC
877-278-6722
415-243-9301
415-243-3996 Fax

Email: info@bcaction.org
www.bcaction.org

..... article from "The Scotsman" ...

Women warned not to wear perfume during pregnancy

Published Date: 31 August 2008

By Kate Foster

PREGNANT women have been advised to avoid using perfumes or scented body creams after research suggested the products can cause unborn boys to suffer infertility or cancer in later life.

Research on rats carried out by Professor Richard Sharpe has found that the reproductive system of male foetuses can be damaged as early as at eight weeks' gestation by chemicals including those found in many cosmetics.

The damage can result in in fertility or testicular cancer – both growing medical problems across the world – said Sharpe, principal investigator at the Medical Research Council's Human Sciences Unit.

Sharpe, who will unveil his findings at a major conference on fertility in Edinburgh this week, has discovered a "time window" at 8 to 12 weeks' gestation – before some women even know they are pregnant – during which certain hormones in the foetus are activated and the male reproductive system is established.

Sharpe has found that future problems with male fertility including undescended testicles, low sperm count and the risk of testicular cancer could be determined at this time if these hormones, such as testosterone, do not work properly.

Experiments on rats have confirmed that if the hormones are blocked the animals suffered fertility problems.

Sharpe told Scotland on Sunday: "We have found the male programming window, which occurs far earlier in foetal development than was previously thought, before the reproductive organs fully develop. This is when the androgens such as testosterone in the foetus are at their most active.

"If the male foetus does not receive enough androgens it may not realise its full

reproductive potential, including the size of the penis and testes, undescended testes or the sperm count. The chances are, something will be wrong with the reproductive system. It may be one thing or several things.

"Women could stop using body creams and perfumes. Although we do not have conclusive evidence that they do harm, there are components about which there are question marks; for example it could be certain combinations of chemicals. If you are thinking about how a baby might be exposed, that's one way, and it's something positive you can do. It might have no consequence, but it's something positive women can do for their baby."

Sharpe will reveal his findings this week at the Simpson Symposium in Edinburgh, a gathering of fertility experts organised by Edinburgh University.

Up to 8% of boys are thought to be born with undescended testicles, which is the most common birth defect in boys and is linked to infertility. The condition is also a risk factor for developing testicular cancer later in life.

Sperm quality and number have declined in the last 30 years. About one in seven couples in the UK will have difficulty conceiving at some time. About one third of cases are due to problems in the man.

Testicular cancer is also increasing worldwide by between 1% and 6% a year. The annual number of new cases of testicular cancer in the UK grew from 850 in 1975 to 1,889 in 2004.

However, campaigners urged women not to panic over the suggestion until further studies are conducted.

Susan Seenan, spokeswoman for the charity Infertility Network UK,

said: "A lot of women will not even know they are pregnant at this stage, or how far along they are. I would be very concerned about alarming women until these tests have been done on humans. We welcome any new research in infertility but we would like to see a lot more research in this area before the findings on animals can be said for humans."