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The REAL Cause of the Global Obesity Epidemic. Are Toxic Chemicals Making Us Fat?

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World Wide Obesity Epidemic

Some 68% of all Americans are overweight, and obesity has almost doubled in the last couple of decades worldwide. As International Business Tribune reports:

> Studies conducted jointly by researchers at Imperial College London and Harvard University, published in the medical journal The Lancet, show that obesity worldwide almost doubled in the decades between 1980 and 2008.
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> 68 per cent of Americans were found to be overweight while close to 34 percent were obese.

Sure, people are eating too much and exercising too little (this post is not meant as an excuse for lack of discipline and poor choices). The processed foods and refined flours and sugars don't help. And additives like high fructose corn syrup – which are added to many processed foods – are stuffing us with empty calories.

But given that there is an epidemic of obesity even in 6 month old infants (see below), there is clearly something else going on as well.

Are Toxic Chemicals Making Us Fat?

The toxins all around us might be making us fat.

As the Washington Post reported in 2007:

> Several recent animal studies suggest that environmental exposure to widely used chemicals may also help make people fat.
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> The evidence is preliminary, but a number of researchers are pursuing indications that the chemicals, which have been shown to cause abnormal changes in animals' sexual development, can also trigger fat-cell activity — a process scientists call adipogenesis.
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> The chemicals under scrutiny are used in products from marine paints and pesticides to food and beverage containers. A study by the Centers for Disease Control and Prevention found one chemical, bisphenol A, in 95 percent of the people tested, at levels at or above those that affected development in animals.

These findings were presented at last month's annual meeting of the American Association for the Advancement of Science. A spokesman for the chemical industry later dismissed the concerns, but Jerry Heindel, a top official of the National Institute of Environmental Health Sciences (NIEHS), who chaired the AAAS session, said the suspected link between obesity and exposure to "endocrine disrupters," as the chemicals are called because of their hormone-like effects, is "plausible and possible."

Bruce Blumberg, a developmental and cell biologist at the University of California at Irvine, one of those presenting research at the meeting, called them "obesogens" — chemicals that promote obesity.

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Exposed mice became obese adults and remained obese even on reduced calorie and increased exercise regimes. Like tributyltin, DES [which for decades was added to animal feed and routinely given to pregnant women] appeared to permanently disrupt the hormonal mechanisms regulating body weight.

"Once these genetic changes happen in utero, they are irreversible and with the individual for life," Newbold said.

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"Exposure to bisphenol A is continuous," said Frederick vom Saal, professor of biological sciences at the University of Missouri at Columbia. Bisphenol A is an ingredient in polycarbonate plastics used in many products, including refillable water containers and baby bottles, and in epoxy resins that line the inside of food cans and are used as dental sealants. [It is also added to store receipts.] In 2003, U.S. industry consumed about 2 billion pounds of bisphenol A.

Researchers have studied bisphenol A's effects on estrogen function for more than a decade. Vom Saal's research indicates that developmental exposure to low doses of bisphenol A activates genetic mechanisms that promote fat-cell activity. "These in-utero effects are lifetime effects, and they occur at phenomenally small levels" of exposure, vom Saal said.

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Research into the impact of endocrine-disrupting chemicals on obesity has been done only in laboratory animals, but the genetic receptors that control fat cell activity are functionally identical across species. "They work virtually the same way in fish as they do in rodents and humans," Blumberg said. "Fat cells are an endocrine organ."

Ongoing studies are monitoring human levels of bisphenol A, but none have been done of tributyltin, which has been used since the 1960s and is persistent in the marine food web. "Tributyltin is the only endocrine disrupting chemical that has been shown without substantial argument to have an effect at levels at which it's found in the environment," Blumberg said.

Concern over tributyltin's reproductive effects on marine animals has resulted in an international agreement discontinuing its use in anti-fouling paints used on ships. The EPA has said it plans next year to assess its other applications, including as an antimicrobial agent in livestock operations, fish hatcheries and hospitals.

Bisphenol A is approved by the Food and Drug Administration for use in consumer products, and the agency says the amount of bisphenol A or tributyltin that might leach from products is too low to be of concern. But the

> National Toxicology Program, part of the National Institutes of Health, is reviewing bisphenol A, and concerns about its estrogenic effects prompted California legislators to propose banning it from certain products sold in-state, a move industry has fought vigorously.

Similarly, the Daily Beast noted in 2010:

> [Bad habits] cannot explain the ballooning of one particular segment of the population, a segment that doesn't go to movies, can't chew, and was never that much into exercise: babies. In 2006 scientists at the Harvard School of Public Health reported that the prevalence of obesity in infants under 6 months had risen 73 percent since 1980. "This epidemic of obese 6-month-olds," as endocrinologist Robert Lustig of the University of California, San Francisco, calls it, poses a problem for conventional explanations of the fattening of America. "Since they're eating only formula or breast milk, and never exactly got a lot of exercise, the obvious explanations for obesity don't work for babies," he points out. "You have to look beyond the obvious."
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> The search for the non-obvious has led to a familiar villain: early-life exposure to traces of chemicals in the environment. Evidence has been steadily accumulating that certain hormone-mimicking pollutants, ubiquitous in the food chain, have two previously unsuspected effects. They act on genes in the developing fetus and newborn to turn more precursor cells into fat cells, which stay with you for life. And **t**hey may alter metabolic rate, so that the body hoards calories rather than burning them, like a physiological Scrooge. "The evidence now emerging says that being overweight is not just the result of personal choices about what you eat, combined with inactivity," says Retha Newbold of the National Institute of Environmental Health Sciences (NIEHS) in North Carolina, part of the National Institutes of Health (NIH). "Exposure to environmental chemicals during development may be contributing to the obesity epidemic." They are not the cause of extra pounds in every person who is overweight—for older adults, who were less likely to be exposed to so many of the compounds before birth, the standard explanations of genetics and lifestyle probably suffice—but environmental chemicals may well account for a good part of the current epidemic, especially in those under 50. And at the individual level, exposure to the compounds during a critical period of development may explain one of the most frustrating aspects of weight gain: you eat no more than your slim friends, and exercise no less, yet are still unable to shed pounds.
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> Newbold gave low doses (equivalent to what people are exposed to in the environment) of hormone-mimicking compounds to newborn mice. In six months, the mice were 20 percent heavier and had 36 percent more body fat than unexposed mice. Strangely, these results seemed to contradict the first law of thermodynamics, which implies that weight gain equals calories consumed minus calories burned. "What was so odd was that the overweight mice were not eating more or moving less than the normal mice," Newbold says. "We measured that very carefully, and there was no statistical difference."
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> `Programming the fetus to make more fat cells leaves an enduring physiological legacy. "The more [fat cells], the fatter you are," says UCSF's Lustig. But [fat cells] are more than passive storage sites. They also fine-tune appetite, producing hormones that act on the brain to make us feel hungry or sated. With more [fat cells], an animal is doubly cursed: it is hungrier more

> often, and the extra food it eats has more places to go—and remain.
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> In 2005 scientists in Spain reported that the more pesticides children were exposed to as fetuses, the greater their risk of being overweight as toddlers. And last January scientists in Belgium found that children exposed to higher levels of PCBs and DDE (the breakdown product of the pesticide DDT) before birth were fatter than those exposed to lower levels. Neither study proves causation, but they "support the findings in experimental animals," says Newbold. They "show a link between exposure to environmental chemicals ... and the development of obesity." [See this for more information on the potential link between pesticides and obesity.]
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> This fall, scientists from NIH, the Food and Drug Administration, the Environmental Protection Agency, and academia will discuss obesogens at the largest-ever government-sponsored meeting on the topic. "The main message is that obesogens are a factor that we hadn't thought about at all before this," says Blumberg. But they're one that could clear up at least some of the mystery of why so many of us put on pounds that refuse to come off.

Consumption of the widely used food additive monosodium glutamate (MSG) has been linked to obesity.

Pthalates – commonly used in many plastics – have been linked to obesity. See this and this. So has a chemical used to make Teflon, stain-resistant carpets and other products.

Most of the meat we eat these days contains estrogen, antibiotics and powerful chemicals which change hormone levels. Modern corn-fed beef also contains much higher levels of saturated fat than grass-fed beef. So the meat we are eating is also making us fat.

Arsenic may also be linked with obesity, via it's effect on the thyroid gland. Arsenic is often fed to chickens and pigs to fatten them up, and we end up ingesting it on our dinner plate. It's ending up in other foods as well.

A lot of endocrine-disrupting pharmaceuticals and medications are also ending up in tap water.

Moreover, the National Research Council has found:

> The effects of fluoride on various aspects of endocrine function should be examined further, particularly with respect to a possible role in the development of several diseases or mental states in the United States.

Some hypothesize that too much fluoride affects the thyroid gland, which may in turn lead to weight gain.

Antibiotics also used to be handed out like candy by doctors. However, ingesting too many antibiotics has also been linked to obesity, as it kills helpful intestinal bacteria. See this and this.

Moreover, many crops in the U.S. are now genetically modified. For example, 93 percent of

soybeans grown in the US are genetically engineered, as are:

- 86% of all corn
- 93% of canola
- 93% of cottonseed oil
- Between 2008 and 2009, 95% of all sugarbeets planted were genetically engineered to be able to tolerate high doses of the pesticide Roundup

Some allege that Roundup kills healthy gut bacteria, and that genetically modified crops cause other health problems.

And Cornell University's newspaper – the Cornell Sun – reports that our intestinal bacteria also substantially affect our ability to eliminate toxins instead of letting them make us fat:

> Cornell scientists researching the effects of environmental toxins to the onset of obesity and Type II Diabetes, discovered that—unlike other factors such as eating too many unhealthy foods—the extent of damage caused by pollutants depends not on what a person puts into her mouth, but on what is already living within her gut.
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> Prof. Suzanne Snedeker, food science, and Prof. Anthony Hay, microbiology, researched the contribution that microorganisms in the gut and environmental toxins known as "obesogens" have on ever rising obesity levels. Their work, which was published last October in the journal Environmental Health Perspectives, reported a **link between composition of gut microbiota, exposure to environmental chemicals and the development of obesity** and diabetes. The review, "Do Interactions Between Gut Ecology and Environmental Chemicals Contribute to Obesity and Diabetes?" combined three main ideas: predisposed gut microbe composition can increase an individual's risk of obesity and Type II Diabetes, gut microbe activity can determine an individual's metabolic reaction to persistent pollutants such as DDT and PCB and certain pharmaceuticals can also be metabolized differently depending on the community of microbes in the gut.
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> The microbe community influences many metabolic pathways within the gut, Snedeker said. **Our bodies metabolize chemicals, but how they are metabolized, and how much fat is stored, depends on gut ecology**. Microbes are responsible not only for collecting usable energy from digested food, but also for monitoring insulin levels, **storage of fat and appetite**. Gut microbes also play an integral role in dealing with any chemicals that enter the body. According to Snedeker, differences in gut microbiota can cause drugs like acetaminophen to act as a toxin in some people while providing no problems for others. While pharmaceutical and microbe interactions are well understood, there is little information in the area of microbe response to environmental toxins.
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> She said, there are more than three dozen chemicals called obesogenic compounds, that can cause weight gain by altering the body's normal metabolic responses and lipid production.
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> "It seems probable that gut microbes are affecting how our bodies handle these environmental chemicals," Snedeker said. According to Snedeker, enzymes that are influenced by interactions of gut microbes break down approximately two-thirds of the known environmental toxins. Therefore, differences in the gut microbe community strongly affect our bodies' ability to get rid of environmental pollutants. Obesogens can alter normal metabolic behavior by changing the levels of fat that our bodies store. Snedeker and Hay

> suggested that **the microbes in the gut of humans determine the way in which these chemicals are metabolized and thus could contribute to obesity**.
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> Snedeker and Hay concluded that **although high levels of obesogenic chemicals are bound to cause some kind of disruption in the gut microbe community responsible for breaking these chemicals down, the degree of the disturbance is dependent upon gut microbial composition**. In other words, **the amount of weight an individual is likely to gain when exposed to environmental toxins, or her risk of acquiring Type II Diabetes, could depend on the microorganism community in their gut**.

No, Everything Won't Kill You

In response to information about toxic chemicals in our food, water and air, many people change the subject by saying "well, everything will kill you". In other words, they try to change the topic by assuming that we would have to go back to the stone age to avoid exposure to toxic chemicals.

But this is missing the point entirely. In fact, companies add nasty chemicals to their products and use fattening food-producing strategies to cut corners and make more money.

In the same way that the financial crisis, BP oil spill and Fukushima nuclear disaster were caused by fraud and greed, we are daily exposed to obesity-causing chemicals because companies make an extra buck by lying about what is in their product, cutting every corner in the book, and escaping any consequences for their health-damaging actions.

In fattening their bottom line, the fat cats are creating an epidemic of obesity for the little guy.

What Can We Do To Fight Back?

Eating grass-fed meat instead of industrially-produced corn fed beef will reduce your exposure to obesity-causing chemicals.

Use glass instead of plastic whenever you can, to reduce exposure to pthalates and other hormone-altering plastics.

Try to avoid canned food, or at least look for cans that are free of bisphenol A. (For example, the Eden company sells food in bpa-free cans.) Buy and store food in glass jars whenever possible. And wash your hands after handling store receipts (they still contain bpa).

Eat yogurt or other food containing good bacteria to help restore your healthy intestinal flora. If you don't like yogurt, you can take "probiotic" (i.e. good bacteria) supplements from your local health food store.

And don't forget to tell your grocery store that you demand real food that doesn't contain bpa, pthalates, hormones, antibiotics or other junk. If we vote with our pocketbooks, the big food companies will get the message.

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