KIDS OBESITY:
IS IT PROGRMMED IN THE GENES?

Early Exposure to common chemicals may be
Programming kids to be FAT.

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Institute for Sound Health & Integrative Medicine

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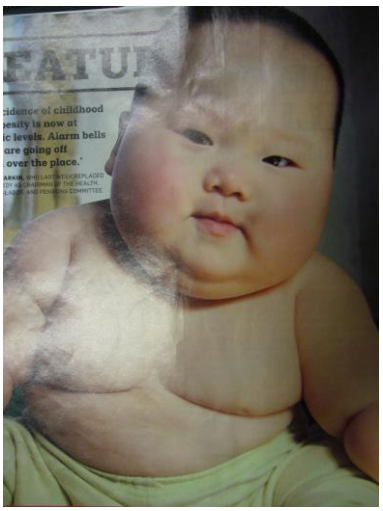
Healthy Ever After Children's Research Initiative™

The Mission:
Documenting causal relationship of obesity in babies and children's Metabolic disorders and Environmental toxicology.

The following article is the groundbreaking science behind this Initiative.

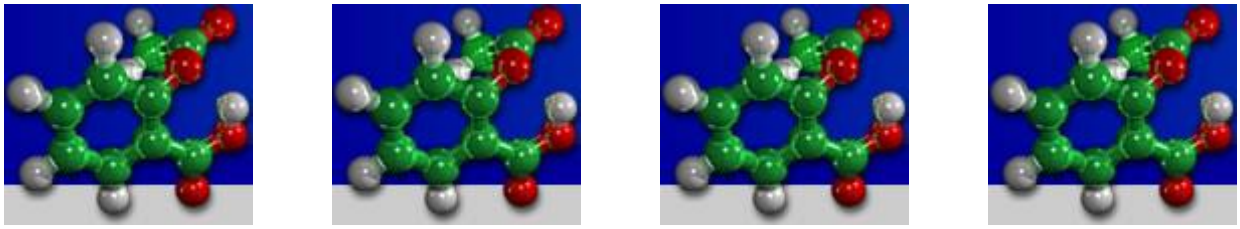
It's easy enough to find culprits in the nation's epidemic of obesity, starting with tubs of buttered popcorn at the multiplex and McDonald's 1,220-calorie deluxe breakfasts, and moving on to the couch potato-fixation of America. Potent as they are, however, these causes cannot explain the ballooning 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."***

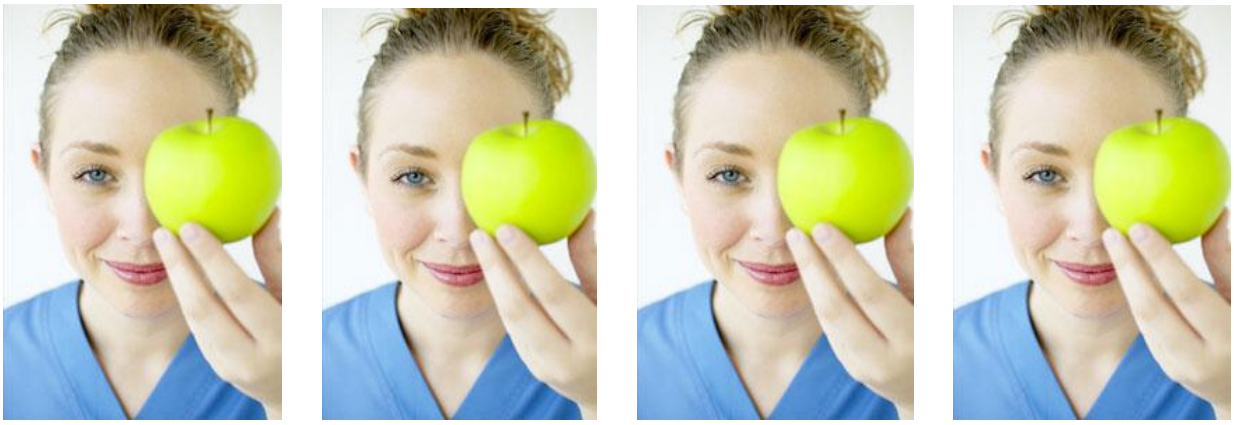
The search for the non-obvious has led to a familiar villain: early-life exposure to traces of chemicals in the environment. Evidence has been steadily accumulating that certain hormone-mimicking pollutants, ubiquitous in the food chain, have two previously unsuspected effects. They act on genes in the developing fetus and newborn to turn more precursor cells into fat cells, which stay with you for life. And they 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 explanation 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.



Genetically modified foods BOND to cells differently and block normal uptake of minerals. Maladaptive metabolism then increases risk for allergies, asthma, Autism ASD spectrum, and diabetes. Obesity follows in a high percentage..

The new thinking about obesity comes at a pivotal time politically. As the debate over health care shines a light on the country's unsustainable spending on doctors, hospitals, and drugs: the obese make tempting scapegoats.

About 60 percent of Americans are overweight or obese, and their health-care costs are higher: $3,400 in annual spending for a normal-weight adult versus $4,870 for an obese adult, mostly due to their higher levels of type 2 diabetes, heart disease, and other conditions. If those outsize costs inspire greater efforts to prevent and treat obesity, fine. But if they lead to demonizing the obese—caricaturing them as indolent pigs raising insurance premiums for the rest of us—that's a problem, and not only for ethical reasons: it threatens to obscure that one potent cause of weight gain may be largely beyond an individual's control.



That idea did not have a very auspicious genesis. In 2002 an unknown academic published a paper in an obscure journal. Paula Baillie-Hamilton, a doctor at Stirling University in Scotland whose only previous scientific paper, in 1997, was titled "Elimination of Firearms Would Do Little to Reduced Premature Deaths," reported a curious correlation***. Obesity rates, she noted in The Journal of Alternative and Complementary Medicine, had risen in lockstep with the use of chemicals such as pesticides and plasticizers (now known as "ENDOCRINE DISRUPTORS") over the previous 40 years.*** True enough. But to suggest that the chemicals caused obesity made as much sense as blaming the rise in obesity on, say, hip-hop. After all, both of those took off in the 1970s and 1980s.

What's really in the hotdog? What's really in the bun? Are they GMO, Organic or Kosher?

Despite that obvious hole in logic, the suggestion of a link between synthetic chemicals and obesity caught the eye of a few scientists. For one thing, **there was no question that exposure in the womb to hormone like chemicals can cause serious illness decades later.** Women whose mothers took the anti miscarriage, estrogen like drug DES during pregnancy, for instance, have a high risk of cervical and vaginal cancer. In that context, the idea that exposure to certain chemicals during fetal or infant development might "program" someone for obesity didn't seem so crazy, says Jerrold Heindel of HIEHS. In 2003 he therefore wrote a commentary, mentioning Baillie-Hamilton's idea, in a widely read TOXICOLOGY journal, bringing what he called its "provocative hypothesis" more attention. He underlined one fact in particular. When many of the chemicals Baillie-Hamilton discussed had been tested for toxicity, researchers focused on whether they caused weight loss, which is considered a toxic effect. They overlooked instances when the chemicals caused weight gain. But if you go back to those old studies, Heindel pointed out, you see that a number of chemicals caused weight gain—and at low doses, akin to those that fetuses and newborns are exposed to, not the proverbial 800 cans of diet soda (the now dis-credited saccharin study) a day. Those results, he says, had "generally been overlooked.

***All sugar is not Equal*.**

High Fructose Corn Syrup is metabolized differently, causing weight gain. It is in soft drinks, cookies, crackers, cereals, and most boxed cake mixes. ---- What's in Your Cupboard?

Scientists in Japan, whose work Heindel focused on, were also finding that low levels of certain compounds, such as bisphenol A (the building block of hard, polycarbonate plastic, including that in baby bottles), had surprising effects on cells growing in lab dishes. Usually the cells become fibroblasts, which make up the body's connective tissue. These pre-fibroblasts, however, are like the kid who isn't sure what he wants to be when he grows up. With a little nudge, they can take an entirely different road. They can become adipocytes—FAT CELLS. **And that's what the Japanese team found: bisphenol A, and some other industrial compounds, pushed pre-fibroblasts to become fat cells. The compounds also stimulated the proliferation of existing fat cells.** "The fact that an environmental chemical has the potential to stimulate growth of "pre-adipocytes" has enormous implication," Heindel wrote. If this happened in living animals as it did in cells in lab dishes, "the result would be an animal with the tendency to become obese.

It took less than two years for Heindel's "IF" to become reality. For 30 years his colleague Newbold had been studying the effects of estrogens, but she had never specifically looked for links to obesity. Now she did. 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."

According to the Washington Post: Food dyes are a serious problem.

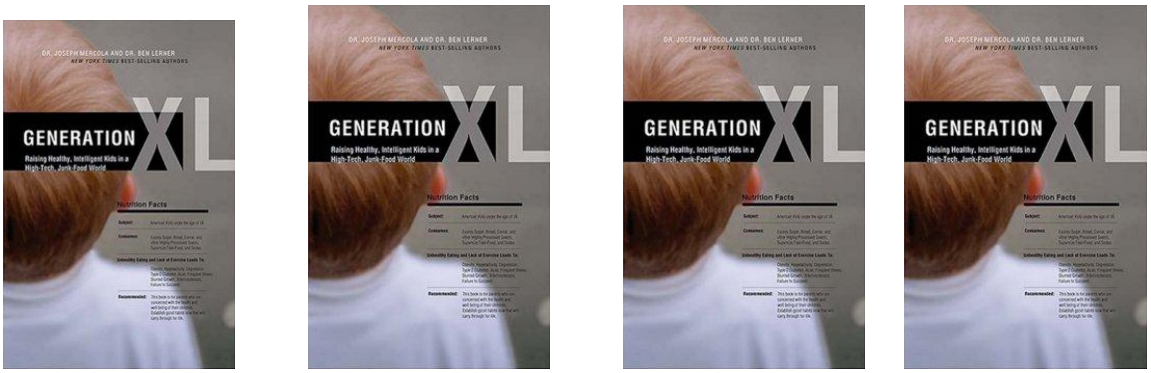
"Beyond the behavioral problems and cancer risks, the greatest hazard that dyes pose for children may also be the most obvious: They draw kids away from nutritious foods and toward brightly colored processed products that are high in calories but low in nutrients, such as fruit-flavored drinks and snack foods. Those types of foods are a major force in America's obesity epidemic."

On the other side of the country, Bruce Blumberg of the University of California, Irvine, had also read the 2002 Baillie-Hamilton paper. He wasn't overly impressed. "She was peddling a book with questionable claims about diets that 'detoxify' the body," he recalls. "And to find a correlation between rising levels of obesity and chemicals didn't mean much. There's a correlation between obesity and a lot of things." Nevertheless, her claim stuck in the back of his mind as he tested environmental compounds for their effects on the endocrine (hormone) system. "People were testing these compounds for all sorts of things, saying, 'Let's see what they do in my experimental system,'" Blumberg says. "But cells in culture are not identical to cells in the body. We had to see whether this occurred in live animals."

In 2006 he fed pregnant mice **tributyltin, a disinfectant and fungicide used in marine paints, plastics production, and other products, which enter the food chain in seafood and drinking water.** "The offspring were born with more fat already stored, fatter cells, and became 5 to 20 percent fatter by adulthood," Blumberg says. Genetic tests revealed how that had happened. The tributyltin activated a receptor called PPAR gamma, which acts like a switch for cells' fate: in one position it allows cells to remain fibroblasts, in another it guides them to become fat cells. (It is because the diabetes drugs ACTOS and AVANDIA activate PPAR gamma that one of their major side effects is obesity.) The effect was so strong and so reliable that Blumberg thought **compounds that reprogram cells' fate like this deserved a name of their own: OBESOGENS**. As later tests would show, tributyltin is not the only obesogen that acts on the PPAR pathway, leading to more fat cells. So do some phthalates (used to make vinyl plastics, such as those used in shower curtains, and, until the 1990's, plastic food wrap), bisphenol A, and perfluoroalkyl compounds (used in stain repellents and nonstick cooking surfaces.)

Mercury makes up more that 50% of "Silver Fillings" and is a Bio Hazard outside of the Body and a Neurotoxin inches from your brain when placed in your teeth. This is an endocrine disruptor for Mother and fetus which contribute to the metabolic creation of obesogen cells.

THE MORE PESTICIDES CHILDREN WERE EXPOSED TO AS FETUSES, THE GREATER THEIR RISK OF BEING OVERWEIGHT AS TODDLERS.



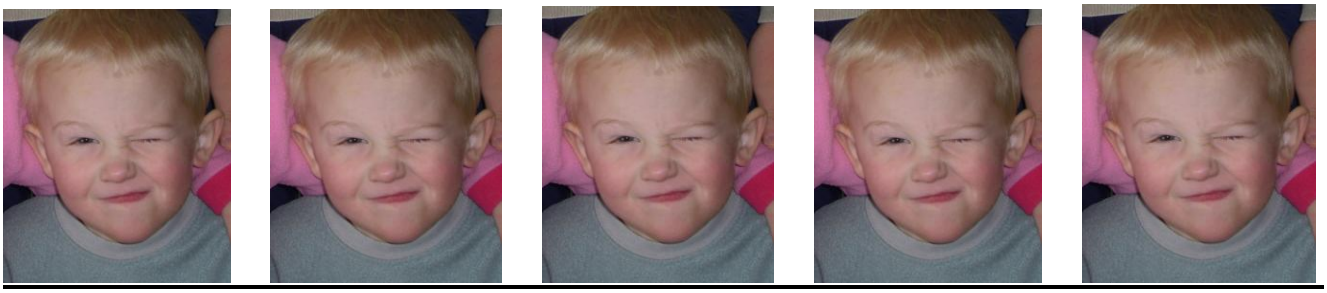
Hospital costs for obesity-related ailments in children have more than TRIPLED over the last twenty years, with 30 percent of boys, and a staggering 40 percent of girls, at risk of developing type 2 diabetes-an obesity-related condition-sometime during their lives.

Programming the fetus to make more fat cells leaves an enduring physiological legacy. "The more adiposities, the fatter you are," says UCSF's Lustig. **But adipocytes 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 adipocytes, an animal is doubly cursed: it is hungrier more often, and the extra food it eats has more places to go—and remain.**

Soy is not a health food. Soy is not a health food. Soy is not a health food.

Within a year of Blumberg's ground-breaking work, it became clear that altering cells' fate isn't the only way obesogens can act, and that exotic pollutants aren't the only potential obesogens. In 2005 Newbold began feeding newborn rats genistein, an **estrogen like compound found in soy,** at doses like those in soy milk and soy formula. By the age of 3 or 4 months, the rats had higher stores of fat and a noticeable increase in body weight. And once again, mice fed genistein did not eat significantly more – not enough more, anyway, to account for their extra avoirdupois, suggesting that the compound threw a wrench in the workings of the body's metabolic rate. "The only way to gain weight is to take in more calories than you burn," says Blumberg. "But there are lots of variables, such as how efficiently calories are used." Someone who uses calories very efficiently, and burns fewer to stay warm, has more left over to turn into fat.

"One of the messages of the ***obesogens research is that prenatal exposure can reprogram metabolism so that you are predisposed to become fat," says Blumberg.***



The jury is still out on whether soy programs babies to be overweight – some studies find that it does, other studies that it doesn't – but Newbold didn't want her new grandchild to be a guinea pig in this unintentional experiment. When her daughter mentioned that she was planning to feed the baby soy formula, as about 20 percent of American mothers do, Newbold said she would cover the cost of a year's worth of regular formula if her daughter would change her mind. (She did.) As a scientist rather than a grandmother, however, Newbold hedged her bets. "Whether our results can be extrapolated to humans," she said in 2005, "remains to be determined."

Another challenge to the simplistic calories-in/calories-out model came just this month. **The time of day when mice eat, scientists reported, can greatly affect weight gain.** Mice fed a high-fat diet during their normal sleeping hours gained more than twice as much weight as mice eating the same type and amount of food during their normal waking hours, Fred Turek of Northwestern University and colleagues reported in the journal *Obesity.* And just as Newbold found, the two groups did not differ enough in caloric intake or activity levels to account for the difference in weight gain. Turek **suspects that one possible cause of the difference is the disruption in the animals' circadian rhythms.** Genes that govern our daily cycle of sleeping and waking "also regulate at least 10 percent of the other genes in our cells, including metabolic genes," says Turek. "Mess up the cellular clock and you may mess up metabolic rate." That would account for why the mice that ate when they should have slept gained more weight: the disruption in their clock genes lowered their metabolic rate, so they burned fewer calories to keep their body running. Studies in people have linked eating at odd times with weight gain, too.

Is GMO Wheat or Gluten in your grains causing you weight gain?
Do you CRAVE what you are allergic to?

Mice are all well and good, but many a theory has imploded when results in lab animals failed to show up in people. Unfortunately, that is not the case with obesogens***. In 2005 scientists in Spain reported that the more pesticides children were exposed to as fetuses, the greater their risk of being over-weight 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," said Newbold. ***They "show a link between exposure to environmental chemicals . . . and the development of obesity."***

Genetically modified corn is in most animal feed, and high fructose corn syrup. How it is changing metabolic pathways in every family and every household?

Given the ubiquity of obesogens, traces of which are found in the blood or tissue of virtually every American, why isn't everyone overweight? For now, all scientists can say is that even a slight variation in the amounts and timing of exposures might matter, as could individual differences in physiology. "Even in genetically identical mice," notes Blumberg, "you get a range of reactions to the same chemical exposure." More problematic is the question of how to deal with this cause of obesity. If obesogens have converted more precursor cells into fat cells, or have given you a "thrifty" metabolism that husbands calories like a famine victim, you face an uphill climb. "It doesn't mean you can't work out like a demon and strictly control what you eat," says Blumberg, "but you have to work at it that much harder." He and others are quick to add that obesogens do not account for all cases of obesity, especially in adults. "I'd like to avoid the simplistic story that chemicals make you fat," says Blumberg. For instance, someone who was slim throughout adolescence and then packed on pounds in adulthood probably cannot blame it on exposure to obesogens prenatally or in infancy: if that were the cause, the extra fat cells and lower metabolic rate that obesogens cause would have shown themselves in childhood chubbiness.

Plastics leach into water and are endocrine disruptors which cause metabolic syndrome.

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. (Please contact www.YourHealthYourChoice.us for research sources)

Question: **What specific areas of research have been studied at Your Health Your Choice Foundation since 1997?**

Answer**: Neurology and Neuro-toxicology and related Immunology, Homotoxicology and Environmental toxicology, Dental-Brain toxicology studies, Endocrinology, Allergy and Electro-Magnetic Polarity imbalances, Severe digestion Dysbiosis and Malabsorption and its relationship to brain function, Enzyme Metabolic Imbalance research, Chinese Herbology & Acupuncture Energy Medicine, Rain Forest Herbology, Psychological and Organic Brain development and Maturation when impeded by toxicity, and finally, CCBT Sound Entrainment therapy based on the 45 year research of Dr. Alfred Tomatis MD, for Brain Neuro-plasticity possibilities during childhood maturation and development.**

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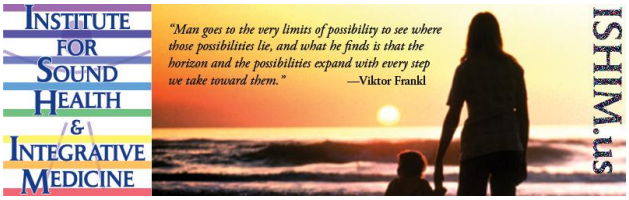

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