Pesticide Exposure Linked to Obesity, Type 2 Diabetes, and Metabolic Disease in Seniors

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by **Beyond Pesticides**
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(*Beyond Pesticides*, February 27, 2024) Popular culture and official policy continue to ignore a blatant source of the rise in obesity: chemical exposures, including pesticides. A study, "Associations of chronic exposure to a mixture of pesticides and type 2 diabetes mellitus in a Chinese elderly population," contributes to the now-massive trove of evidence linking pesticides to diseases and shows that by the time people reach retirement age they are suffering from a heavy burden of contamination that raises their risk of complex disease.

Since the 1960s, obesity in both adults and children has nearly tripled. More than half of U.S. adults were either obese or severely obese by 2018, according to data from the National Health and Nutrition Examination Study. The 55-year trend line is decidedly upward. More women than men are obese, and black women suffer the most, but men are racing to catch up. Between 1999 and 2018, Mexican American men shot up from the lowest percentage of obesity to nearly the highest.

Obesity is a milestone on the road to Type 2 diabetes, heart disease, high blood pressure, kidney failure, joint replacement, and more. The causes of obesity are severely misunderstood. Most people believe that discipline and

willpower are what keep a person from being fat, even if they have “fat genes.” The medical opinion is “calories in, calories out” – obesity, genetic or not, can be staved off with diet and exercise. But despite decades of advice, sweat, tears, and billions of dollars spent on ineffective diet pills and menus, obesity is a global emergency. If popular attitudes and medical theories were correct, obesity would be far less common and more easily controlled. It is not. Therefore, beliefs and advice are incorrect—or at least incomplete.

The researchers from the Shanghai Municipal Center for Disease Control and Prevention identified 39 pesticides in the study population. Women had slightly higher levels and a stronger correlation between obesity, pesticide burden and type 2 diabetes than men. The most significant contributors were β-Hexachlorocyclohexane (β-BHC) and oxadiazon.

β-BHC is a byproduct of technical grade lindane production and common near lindane factories. For example, in 2005 an Italian biomonitoring program found β-BHC levels 20 times higher than the legal limit in cows’ milk. The subject cows’ water came from a river which had been polluted by waste from a lindane facility. Lindane is available in the U.S. only as a treatment for head lice and not for any agricultural uses. It has been listed as a Persistent Organic Pollutant under the Stockholm Convention since 2009. The International Agency for Research on Cancer classifies it as a possible human carcinogen; it has been linked to aplastic anemia and breast cancer and is an endocrine disruptor. Oxadiazon is a herbicide and likely human carcinogen used in the U.S. on golf courses, parks, athletic fields, playgrounds, cemeteries and some horticultural contexts but which is not registered for any food uses.

The β-BHC and oxadiazon associations with type 2 diabetes in the Chinese senior study are “pronounced among elderly women,” according to the authors. They are also linear, meaning that for each increment of pesticide body burden, the risk of diabetes rises a comparable amount. These data, the authors

write indicate “that it is an urgent need to take practical measures to control these harmful pesticides.”

Although β-BHC and oxadiazon now have limited uses in the U.S., the study found levels in the Chinese seniors of many pesticides that are still used in the U.S. in agricultural, horticultural, residential, and other applications. These include atrazine, acetochlor, metolachlor, and permethrin, to name a few, all of which have been reported to disturb lipid functions. A 2020 review of agrochemicals affecting obesity discusses more obesogenic pesticides registered in the U.S.

A concurrent publication by most of the same authors as the 2024 Chinese pesticide study reviewed evidence for environmental obesogens’ disruption of lipid metabolism. This review notes that, “Currently, more than 50 types of chemicals with high human exposure levels have been identified as environmental obesogens that can interfere with lipid metabolism and induce obesity. Experimental studies have shown that the lipid metabolism interference effects of obesogens have multiple targets, including nuclear receptors [thyroid, steroid, vitamin D3, and retinoid receptors], transcription factors [wide number of proteins that initiate and regulate the transcription of genes], cytokines [proteins important to cell signaling], and hormones. The interfering factors of environmental obesogen-induced obesity include transgenerational effects, susceptibility [developmental] windows, gender differences…and diet habits…”

Lipids are fat-soluble compounds that are essential for cells’ structural integrity along with numerous other functions in organisms from bacteria to humans. But when fat consumption exceeds the body’s need for lipids, humans make more fat cells or expand existing cells. When these storage options are full, lipids begin leaking into other tissues such as the kidneys and pancreas, contributing to a wide variety of serious diseases.

Research on environmental contributions to obesity was pioneered by Bruce Blumberg, who recounts how he discovered the effects of tributyltin (TBT) in his 2018 book with Kristin Loberg, *The Obesogen Effect: Why We Eat Less and Exercise More but Still Struggle to Lose Weight*. TBT refers to a family of tin compounds used to keep marine snails off ship hulls (a use now banned), to prevent fungal growth in wood and textile production, as a stabilizer in polyvinyl chloride products, and other uses. It bioaccumulates and can take 30 years to break down. Blumberg's presentation at Beyond Pesticides' 2018 36th National Pesticide Forum, is available on YouTube.

Dr. Blumberg, a professor of developmental and cell biology at the University of California Irvine and a molecular biologist by training, was curious about Japanese research showing that TBT could change fish from female to male, so he looked for cellular receptors that TBT could bind to. He found that TBT did not activate sex hormone receptors as expected; instead, it activated the process that leads to fat cell development. He showed that frog embryos exposed to TBT converted their testes to fat, that mice exposed to TBT in the womb had larger fat deposits as adults, and that this predisposition affected later generations. Subsequent research into the term Blumberg coined, obesogens, has expanded knowledge of these phenomena.

One of the widely-studied culprits is the notorious organophosphate chlorpyrifos. It has a painful and ragged history of regulation by EPA, which itself has repeatedly opined that it is toxic to human health. Currently, as BP reported last November, chlorpyrifos residues are still permitted in food owing to a shoddy and biased court-ordered instruction by the Eighth Circuit Court of Appeals.

The organophosphate insecticide chlorpyrifos does its damage in varied ways. Beyond Pesticides covered a 2019 study finding that it promotes obesity development even at low doses. The study found that chlorpyrifos prevented "diet-induced

thermogenesis" in brown adipose tissue at concentrations "as low as 1 part per million." Brown fat is considered better than white fat, and it burns calories to keep the body at an even temperature in cold conditions.

An earlier study by some of the same authors of the 2024 pesticide-diabetes research showed that chlorpyrifos also contributes to obesity by causing leaky gut and inflammation; when they transferred chlorpyrifos-altered microbes to unexposed mice, those mice added fat and lost insulin sensitivity –major factors in type 2 diabetes induction.

Despite reduced usage, TBT keeps on giving – and demonstrating that even at individually low doses, and even when a chemical has been banned or restricted, it can remain in the environment and combine with other toxic chemicals to cause harm. A 2019 study showed that "Combined exposure [to TBT and the "forever chemical" perfluorooctane sulfonate (PFOS)] significantly promoted the fat accumulation in newly hatched [fish] larvae, even when the doses of TBT and PFOS were both at the levels that did not show obesogenic effect. The interactive effect of TBT and PFOS could aggravate the total obesogenic effect of their mixtures, indicating a synergistic interaction."

There are ways to fight back against the onslaught:

- Eating organic food reduces risk of metabolic diseases including diabetes, which strongly suggests that pesticides have a direct link to diabetes. See Beyond Pesticides' 2020 blog post, "Food For Thought: Eating Organic Reduces Risk of Type 2 Diabetes." In a post last October, "Organophosphate Pesticides and the Link to Respiratory, Metabolic, and Heart Disease," we noted that "Replacing dietary exposure to food grown in chemical-intensive agriculture with organic consistently reduces pesticide levels in one's body…maintaining lower levels of conventional, synthetic pesticides is likely

to reduce the risk of developing chronic diseases like type 2 diabetes. In addition to positive impacts on the human microbiome, organically grown food (i.e., milk, meat, strawberries, tomatoes, and a range of other foods) contain a much more diverse bacterial community than their chemically grown counterparts."

- Schools should switch to organic foods. See our "Call on USDA to Provide Organic School Lunches to Fight Childhood Obesity."
- See our Pesticide-Induced Diseases: Diabetes for more information.

The body of research now available also supports the very recent admission by some health professionals that obesity is not caused by poor character, laziness or lack of willpower. The review of environmental obesogens and their role in metabolic diseases cites approximately 50 studies reporting specific obesogenic effects of more than 50 chemicals. Obesity has multiple determinants, but absent willpower is not one of them. Unfortunately, the medical establishment is still focused on mechanisms, such as brain activity, that cause people to eat too much, and suggest that high-calorie food is too easily available. These are probably factors, but the message that environmental obesogens are a dire emergency has not yet been received. The prevailing concept is that too much food is the problem, when it's perhaps not the amount of food, but the pesticide load of the food, that is an essential cause of the slow-motion global pandemic of obesity and diabetes.

All unattributed positions and opinions in this piece are those of Beyond Pesticides

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