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Diabetes Mystery: Why Are Type 1 Cases Surging?

Researchers are baffled by the worldwide increase in type 1 diabetes, the less common form of the disease

By Maryn McKenna | January 24, 2012


Image: Getty Images

When public health officials fret about the soaring incidence of diabetes in the U.S. and worldwide, they are generally referring to type 2 diabetes. About 90 percent of the nearly 350 million people around the world who have diabetes suffer from the type 2 form of the illness, which mostly starts causing problems in the 40s and 50s and is tied to the stress that extra pounds place on the body's ability to regulate blood glucose. About 25 million people in the U.S. have type 2 diabetes, and another million have type 1 diabetes, which typically strikes in childhood and can be controlled only with daily doses of insulin.

For reasons that are completely mysterious, however, the incidence of type 1 diabetes has been increasing throughout the globe at rates that range from 3 to 5 percent a year. Although the second trend is less well publicized, it is still deeply troubling, because this form of the illness has the potential to disable or kill people so much earlier in their lives.

No one knows exactly why type 1 diabetes is rising. Solving that mystery—and, if possible, reducing or reversing the trend—has become an urgent problem for public health researchers everywhere. So far they feel they have only one solid clue.

"Increases such as the ones that have been reported cannot be explained by a change in genes in such a short period," says Giuseppina Imperatore, who leads a team of epidemiologists in the Division of Diabetes Translation at the U.S. Centers for Disease Control and Prevention. **"So environmental factors are probably major players in this increase."**

A Challenge of Counting

Type 1 and type 2 diabetes share the same underlying defect—an inability to deploy insulin in a manner that keeps blood sugar from rising too high—but they arise out of

almost opposite processes. Type 1, which once was known as juvenile diabetes, is an autoimmune disease in which the body attacks its own cells—namely, the beta cells of the pancreas—destroying their ability to make insulin. In type 2, formerly known as adult-onset diabetes, tissues that need insulin to take up glucose (such as the liver, muscles and fat) become resistant to insulin's presence. The insulin-producing cells respond by going into overdrive, first making more of the hormone than normal and then losing the ability to keep up with the excess glucose in the blood. Some people end up unable to make insulin at all.

The first strong signal that the incidence of type 1 diabetes was on the rise came in 2006, from a World Health Organization project known as DIAMOND (a combination of words in several languages for worldwide diabetes). That survey, which looked at 10 years of records from 112 diabetes research centers in 57 countries, found that type 1 had risen an average of 5.3 percent a year in North America, 4 percent in Asia and 3.2 percent in Europe.

Statistics from Europe—where the single-payer health care systems that care for residents throughout their lives generate rich stores of data—back up that first finding. In 2009 researchers from a second project called EURODIAB compared diabetes incidence across 17 countries and found not only that type 1 was rising—by 3.9 percent a year on average—but also that it was increasing most quickly among children younger than five. By 2020, they predicted, new cases of type 1 diabetes in that age group will nearly double, from 3,600 children to an estimated 7,076 children.

Most assessments of diabetes in the U.S. have been more partial and local. There is one comprehensive national surveillance project, the federally funded SEARCH for Diabetes in Youth study, which published data in 2007. Because that was an initial report, however, researchers could not compare it with earlier years. Still, when looked at against the findings of other studies, it suggests a rising tide. For example, the 2007 study found higher rates of type 1 in the U.S. than did the WHO's worldwide study of the year before. In addition, the SEARCH study results were sharply higher than regional studies from the 1990s in Alabama, Colorado and Pennsylvania.

Competing Hypotheses

The challenge for explaining the rising trend in type 1 diabetes is that if the increases are occurring worldwide, the causes must also be. So investigators have had to look for influences that stretch globally and consider the possibility that different factors may be more important in some regions than in others.

The list of possible culprits is long. Researchers have, for example, suggested that gluten, the protein in wheat, may play a role because type 1 patients seem to be at higher risk for celiac disease and the amount of gluten most people consume (in highly processed foods) has grown over the decades. Scientists have also inquired into how soon infants are fed root vegetables. Stored tubers can be contaminated with microscopic fungi that seem to promote the development of diabetes in mice.

None of those lines of research, though, have returned results that are solid enough to motivate other scientists to stake their careers on studying them. So far, in fact, the search for a culprit resembles the next-to-last scene in an Agatha Christie mystery—the one in which the detective explains which of the many suspects could not possibly have committed the crime.

The last scene in the drama, unfortunately, still has not been written. Currently the suspects getting the closest scrutiny are infections with bacteria, viruses or parasites. The presumptive etiology: a version of the "hygiene hypothesis" that links clean modern lifestyles and allergies.

The **hygiene hypothesis** proposes that early exposure to infections or soil organisms teaches the developing immune system how to maintain itself in balance and so keeps it from reacting in an uncontrolled way later in life when it encounters allergens such as dust and ragweed. Living hygienically, it goes on to say, has deprived children of those early exposures, fueling an epidemic of allergies. The diabetes version of the hygiene hypothesis proposes that when the immune system learns not to overreact to allergens, it also learns to tolerate compounds from the body's own tissues—and therefore prevents the autoimmune attack that destroys the ability to make insulin.

Some circumstantial evidence supports that proposal. Children with multiple siblings—who might bring infections home from day care or school—are less likely to be hospitalized for type 1 diabetes (a proxy measure for incidence). The disease is also less common in children who attend day care themselves, and it is more common in specially bred mice that do not encounter infections because they are raised in a sterile environment.

By themselves, however, those findings do not make the case. Christopher Cardwell, a lecturer in medical statistics at Queen's University Belfast, has conducted meta-analyses of associations between type 1 and birth order, maternal age at birth, and birth by cesarean section, all of which affect the organisms to which young children are exposed. "All of these seemed to be associated," he says, "but they all were in my opinion fairly weak associations. None were of a magnitude that could explain the increasing incidence over time."

Back to Fat

Recently the search for a cause behind the rise of type 1 diabetes has taken an unexpected turn. Some investigators are reconsidering the role of an old adversary: being overweight or obese.

That suspicion might seem counterintuitive given that diabetes dogma holds that being overweight tugs the body toward producing large amounts of insulin (as in type 2), not too little insulin. But some contend that the stress of producing all that extra insulin can burn out the insulin-producing beta cells of the pancreas and push a child whose beta cells are already under attack into developing type 1 diabetes.)This idea, called the

accelerator or overload hypothesis, proposes that “if you have a kid who is chubby, that extra adiposity is going to challenge the pancreatic beta cells,” says Rebecca Lipton, an emeritus professor at the University of Chicago. “In a child who has already started the autoimmune process, those beta cells are just going to fail more quickly, because they are being forced to put out more insulin than in a thin child.”

Overweight makes a logical perpetrator. People are packing on the pounds in rich countries and poor ones. Of course, investigators want to do more than just to explain the rise of type 1 diabetes; they want to prevent it. Unfortunately, if excess weight is a major contributor to the problem, that task will not be easy. No one, so far, has been able to slow the global obesity epidemic. (By 2048, according to researchers from Johns Hopkins University, all American adults will be at least overweight if present trends continue.) Until societies can ensure that most children (not to mention adults) are more physically active, eat healthfully and maintain a normal weight, diabetes researchers will be in the position of detectives who, having solved a murder, realize they can do nothing to prevent the next one.