HOW TYPE-2 DIABETES BECAME AN EPIDEMIC

Since 1980, the number of people around the world afflicted with diabetes has quadrupled. How did this ancient disease suddenly become the twenty-first century plague?

The disease of *diabetes mellitus* (DM) **has been recognized for thousands of years. The ancient Egyptian medical text, *Ebers Papyrus*, written around 1550 BC, first described this condition of "passing too much urine." Around the same time, ancient Hindu writings discussed the disease of *madhumeha*,** (diabetes in ayurveda)**, which loosely translated means "honey urine." Afflicted patients, often children, were mysteriously inexorably losing weight. Attempts to stop the wasting were unsuccessful, despite continual feeding, and the disease was almost uniformly fatal. Curiously, ants were attracted to the urine, which was inexplicably sweet.**

By 250 BC, the Greek physician Apollonius of Memphis had termed the condition *diabetes*, which, by itself, connotes only excessive urination. Thomas Willis added the term *mellitus*, meaning "from honey" in 1675. This descriptor distinguishes diabetes mellitus from a different, uncommon disease known as *diabetes insipidus*. Most commonly caused by traumatic brain injury, diabetes insipidus is also characterized by excessive urination, but the urine is not sweet. Fittingly, *insipidus* means "bland."

Colloquially, the non-specific term diabetes refers to diabetes mellitus, since it is far, far more common than diabetes insipidus. In its untreated form, excessive urine production is accompanied by almost complete wasting away of all tissues. Patients cannot gain weight, no matter what they eat. Life with diabetes is short, disgusting, and painful. Once affected, patients follow a predestined, fatal course.

Tasting the urine of the stricken patient for sweetness was the classic diagnostic test for diabetes. In 1776, the English physician Mathew Dobson (1732 – 1784) **identified sugar as the substance causing this characteristic sweet taste. The sweetness was found not only in the urine, but also in the blood. Slowly, an understanding of diabetes was unfolding, but a solution was still out of reach.**

In 1797, the Scottish military surgeon John Rollo became the first physician to formulate a treatment that carried any reasonable expectation of success. He had observed substantial improvement in a diabetic patient eating an all-meat diet. Given the uniformly grim prognosis of diabetes, this approach was a breakthrough. This extremely low carbohydrate diet was the first diabetic treatment of its kind.

By contrast, French physician Pierre Pioorry (1794 – 1879) **advised diabetics to eat large quantities of sugar to replace what they lost in their urine. While the logic seemed reasonable at the time, it was not a successful strategy. This outcome foretold the grim shades of the highly ineffective advice to follow a high-carbohydrate diet in the treatment of type-2 diabetes.**

Apollinaire Bouchardat (1806 – 1886)**, sometimes called the founder of modern diabetology, established his own therapeutic diet based on the observation that periodic starvation during the Franco-Prussian War of 1870 reduced urinary glucose. His book laid out his comprehensive dietary strategy, which forbade all foods high in sugars and starches.**

In 1910, Sir Edward Sharpey-Schafer, regarded as the founder of endocrinology, proposed that the deficiency of a single hormone he called insulin was responsible for diabetes. The word *insulin* came from the Latin *insula*, which means "island," as this hormone is produced in cells called *the islets of Langerhans* in the pancreas.

By the turn of the twentieth century, prominent American physicians Frederick Madison Allen (1879 – 1964) **and Elliott Joslin** (1869 – 1962) **became strong proponents of intensive dietary management for diabetes, given the lack of other useful treatments.**

Dr Allen envisioned diabetes as a disease in which the overstrained pancreas could no longer keep up with the demands of an excessive diet. To give the pancreas a rest, he prescribed the "Allen starvation treatment," which was very low in calories (1000 calories/day) **and very restricted in carbohydrates** (<10g/day)**. Patients were admitted to a hospital and given only whiskey and black coffee every two hours from 7 a.m. to 7 p.m. This regime continued daily until the sugar disappeared from the urine. Why was whiskey included? It was not essential but was administered simply because it "keeps the patient comfortable while he is being starved."**

The response of some patients was unlike anything seen previously. They improved instantly and almost miraculously. Others, however, starved to death, which was euphemistically called *inanition*.

A lack of understanding of the difference between type-1 and type-2 diabetes severely hampered the usefulness of Allen's treatment. Type-1 diabetic patients were usually dramatically underweight children. Whereas type-2 diabetic patients were mostly overweight adults. This ultra-low calorie diet could be deadly for the very malnourished type-1 diabetic.

Given the otherwise fatal prognosis of untreated type-1 diabetes, this was not the tragedy it may at first have appeared to be. Allen's detractors pejoratively called his treatments *starvation diets*, but they were widely considered the best therapy, dietary or otherwise, until the discovery of insulin in 1921.

Dr. Elliot P. Joslin wrote a textbook *The Treatment of Diabetes Mellitus* and it was considered the bible of diabetes care. Joslin himself is likely the most famous diabetologist in history. Although Joslin had lost many patients to diabetes, he had also saved many by applying Dr. Allen's treatments. In 1916, he wrote: *"Temporary periods of under-nutrition are helpful in the treatment of diabetes will probably be acknowledged by all after these two years of experience with fasting."* He felt the improvements were so obvious to everybody involved that studies would not even be necessary to prove the point.

Fredrick Banting, Charles Best, and john Macleod made the breakthrough discovery of insulin at the University of Toronto in 1921. They isolated insulin from the pancreases of cows and, with James Collip, found a way to purify it enough to administer it to the first patient in 1922. Leonard Thompson, a fourteen-year-old boy with type-1 diabetes, weighed only sixty-five pounds when he started insulin injections. His symptoms and signs rapidly disappeared, and he immediately regained a normal weight. They quickly treated six more patients with equally stunning success. The average lifespan of a ten-year-old at diagnosis increased from about 16 months to thirty-five years!

Eli Lilly and Company partnered with the University of Toronto to commercially develop this revolutionary new drug insulin. The patent was made freely available so the entire world could benefit from the medical discovery of the century. By 1923, 25,000 patients were being treated with injected insulin, and Banting and Macleod received the Nobel Prize for Physiology or Medicine.

Euphoria ensued. With the momentous discovery of insulin, it was widely believed diabetes had finally been cured. British biochemist Fredrick Sanger determined the molecular structure of human insulin, which garnered him the 1958 Nobel Prize in Chemistry and paved the way for the biosynthesis and commercial production of this hormone. Insulin's discovery overshadowed the dietary treatments of the previous century, essentially throwing them into disrepute. Unfortunately, the story of diabetes did not end there.

It soon became clear that different types of diabetes mellites existed. In 1936, Sir Harold Percival Himsworth (1905 – 1993) **categorized diabetics based on their insulin sensitivity. He'd noted that some patients were exquisitely sensitive to the effects of insulin, but others were not. Giving insulin to the insulin-insensitive group did not produce the expected effect: instead of lowering blood glucose efficiently, the insulin seemed to have little effect. By 1948, Joslin speculated that many people had undiagnosed diabetes due to insulin resistance.**

By 1959, the two different types of diabetes were formally recognized: type-1, or insulin-dependent diabetes, or type-2, or non-insulin-dependent diabetes. These terms were not entirely accurate, as many type-2 patients are also prescribed insulin. By 2003, the terms insulin-dependent and non-insulin-dependent were abandoned, leaving only the names *type-1* and *type-2* diabetes.

The names juvenile diabetes and adult-onset diabetes have also been applied, to emphasize the distinction in the age of patients when the disease typically begins. However, as type-1 is increasingly prevalent in children, these classifications have also been abandoned.

THE ROOTS OF THE EPIDEMIC

In the 1950s, seemingly healthy Americans were developing heart attacks with growing regularity. All great stories need a villain, and dietary fat was soon cast into that role. Dietary fat was falsely believed to increase blood cholesterol levels, leading to heart disease. Physicians advocated lower-fat diets, and the demonization of dietary fat began in earnest. The problem, though we didn't see it at the time, was that restricting dietary fats meant increasing dietary carbohydrates, as both create a feeling of satiety (fullness)**. In the developed world, these carbohydrates tended to be highly refined.**

By 1998, the United States government had formed a committee to look into the issue of hunger and malnutrition across the country and recommend solutions to these problems. A report released in 1977, called *Dietary Goals for the United States*, led to the 1980 *Dietary Guidelines for Americans*. These guidelines included several specific dietary goals, such as raising carbohydrate consumption to 55% – 60% of the diet and decreasing fat consumption from approximately 40% of calories to 30%.

Although the low-fat diet was originally proposed to reduce the risk of heart disease and stroke, recent evidence refutes the link between cardiovascular disease and total dietary fat. Many high-fat foods, such as avocados, nuts, and olive oil, contain mono- and poly-unsaturated fats that are now believed to be heart-healthy.

Similarly, the link between natural saturated fat and heart disease has been proven false. While artificially saturated fats, such as trans-fats, are universally accepted as toxic, the same does not hold true for naturally occurring fats found in meat and dairy products, such as butter, cream, and cheese—foods that have been part of the human diet fort time beyond memory.

As it turns out, the consequences of this newfangled, unproven, low-fat, high-carbohydrate diet were unintended: the rate of obesity soon turned upwards and has never looked back.

The 1980 Dietary Guidelines spawned the infamous food pyramid in all its counterfactual glory. Without any scientific evidence, the formerly "fattening" carbohydrate was reborn as a healthy whole grain. The foods that formed the base of the pyramid—foods we were told to eat every single day—included breads, pastas, and potatoes. These were the precise foods we had previously avoided in order to stay thin. They are also the precise foods that provoke the greatest rise in blood glucose and insulin.

Obesity increased immediately. Ten years later, diabetes began its inevitable rise. Age-adjusted prevalence is still rising precipitously. In 1980, an estimated 108 million people world-wide suffered with diabetes. By 2014, that number had swelled to 422 million. Even more concerning is the fact that there seems to be no end in sight.