

# Type 2 diabetes and obesity—what do we really know?

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Social and economic factors have led to a dramatic rise in type 2 diabetes and obesity around the world. In a review in *Science*, Mark McCarthy, professor at the University of Oxford, UK, and Paul Franks,

professor at Lund University, Sweden, examine the knowledge of the actual causes and the interplay between genetics and lifestyle factors.

By studying how our genes express themselves in response to [environmental factors](#) and changes in lifestyle, we will better understand how health recommendations and treatments can be tailored to each individual.

"Environmental factors that disturb cellular and physiological processes and have an effect on the individual's predisposition to diseases, such as type 2 [diabetes](#), are likely to do so through active, and reactive, modulation of genome function. There is a compelling rationale behind this concept, but the details about how these processes work remain poorly defined", says Paul Franks.

"However, there is emerging evidence that epigenomic changes such as DNA methylation and histone modifications, which affect the ways in which genes are transcribed and translated into proteins, are important features of these processes", he continues.

Previous research has largely focused on dietary components and which diet would be best to lower the risk of obesity and type 2 diabetes, but so far there is no clear evidence from epidemiological or [clinical trial data](#) that a specific diet is optimal for long-term weight-loss or lowering the risk of diabetes. Some types of dietary fat may be harmful while others may in fact be protective. The researchers also concluded that the widely established view that vitamin D supplementation lowers blood sugar levels and the risk of type 2 diabetes is unlikely to be accurate.

In recent years, there has been an explosion of interest in the role of the gut microbiome in the development of type 2 diabetes and obesity.

"Several studies have detected differences in the composition of the [gut](#)

[microbiome](#) between healthy people and those with obesity and type 2 diabetes, but the cause and effect remain unclear", says Mark McCarthy, and continues:

"In addition, both high and [low birth weight](#) have been linked to type 2 diabetes later in life. It seems as if these associations reflect the complex interplay of genetic variation in both the foetus and the mother, along with the impact of the intrauterine environment."

The major challenge is getting closer to a mechanistic understanding of why type 2 diabetes and obesity occur and why they have become so much more common in the last 40-50 years.

"We have only a superficial understanding of that. It often gets blamed on our western lifestyle but the specific components of modern life that are most damaging remain unclear. We also know that this occurs on the background of hundreds of genetic differences that influence predisposition. The genetic findings are increasingly allowing us to understand the mechanisms involved, and to start to connect the genetic and environmental contributions", says Mark McCarthy.

## **Facts/Type 2 diabetes**

- The rising prevalence of type 2 diabetes and obesity constitute major threats to human health around the world
- About ten per cent of the global population already have type 2 diabetes, or are likely to develop it, and 40 per cent of adults are overweight or obese
- The heritability for [obesity](#) and type 2 diabetes is 70 and 35 per cent respectively
- The western lifestyle represents a culprit with a combination of excess calories, physical inactivity, sleep deprivation, endocrine disorders and smoking

- More research is needed to figure out which of the elements of our lifestyle are important to define more effective interventions

**More information:** "Exposing the exposures responsible for type 2 diabetes and obesity," *Science*, [science.sciencemag.org/cgi/doi ... 1126/science.aaf5094](https://science.sciencemag.org/cgi/doi/10.1126/science.aaf5094)

Provided by Lund University

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