

Could the food we eat affect our genes? Study in yeast suggests this may be the case

February 11 2016



Fruits & Vegetables. Credit: Global Panorama

Almost all of our genes may be influenced by the food we eat, according to new research published in the journal *Nature Microbiology*. The study, carried out in yeast – which can be used to model some of the body's fundamental processes – shows that while the activity of our genes influences our metabolism, the opposite is also true and the nutrients available to cells influence our genes.

The behaviour of our [cells](#) is determined by a combination of the activity of its genes and the chemical reactions needed to maintain the cells, known as [metabolism](#). Metabolism works in two directions: the breakdown of molecules to provide energy for the body and the production of all compounds needed by the cells.

Knowing the genome – the complete DNA 'blueprint' of an organism – can provide a substantial amount of information about how a particular organism will look. However, this does not give the complete picture: genes can be regulated by other genes or regions of DNA, or by 'epigenetic' modifiers – small molecules attached to the DNA that act like switches to turn genes on and off.

Previous studies have suggested that another player in gene regulation may exist: the [metabolic network](#) – the biochemical reactions that occur within an organism. These reactions mainly depend on the nutrients a cell has available – the sugars, amino acids, fatty acids and vitamins that are derived from the food we eat.

To examine the scale at which this happens, an international team of researchers, led by Dr Markus Ralser at the University of Cambridge and the Francis Crick Institute, London, addressed the role of metabolism in the most basic functionality of a cell. They did so using [yeast cells](#). Yeast is an ideal model organism for large scale experiments as it is much simpler to manipulate than animal models, yet many of its important genes and fundamental cellular mechanisms are the same as or very similar to those in animals and humans.

The researchers manipulated the levels of important metabolites – the products of metabolic reactions – in the yeast cells and examined how this affected the behaviour of the genes and the molecules they produced. Almost nine out of ten genes and their products were affected by changes in [cellular metabolism](#).

"Cellular metabolism plays a far more dynamic role in the cells than we previously thought," explains Dr Ralser. "Nearly all of a cell's genes are influenced by changes to the nutrients they have access to. In fact, in many cases the effects were so strong, that changing a cell's metabolic profile could make some of its genes behave in a completely different manner.

"The classical view is that genes control how nutrients are broken down into important molecules, but we've shown that the opposite is true, too: how the nutrients break down affects how our genes behave."

The researchers believe that the findings may have wide-ranging implications, including on how we respond to certain drugs. In cancers, for example, tumour cells develop multiple genetic mutations, which change the metabolic network within the cells. This in turn could affect the behaviour of the [genes](#) and may explain why some drugs fail to work for some individuals.

"Another important aspect of our findings is a practical one for scientists," explains says Dr Ralser. "Biological experiments are often not reproducible between laboratories and we often blame sloppy researchers for that. It appears however, that small metabolic differences can change the outcomes of the experiments. We need to establish new laboratory procedures that control better for differences in metabolism. This will help us to design better and more reliable experiments."

More information: Mohammad Tauqeer Alam et al. The metabolic background is a global player in *Saccharomyces* gene expression epistasis, *Nature Microbiology* (2016). [DOI: 10.1038/nmicrobiol.2015.30](https://doi.org/10.1038/nmicrobiol.2015.30)

Provided by University of Cambridge

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