

## NUTRITION AND THE EPIGENOME

Unlike behavior or stress, diet is one of the more easily studied, and therefore better understood, environmental factors in epigenetic change.

The nutrients we extract from food enter metabolic pathways where they are manipulated, modified and molded into molecules the body can use. One such pathway is responsible for making methyl groups - important epigenetic tags that silence genes.

Familiar nutrients like folic acid, B vitamins and SAM-e (S-Adenosyl methionine, a popular over-the-counter supplement) are key components of this methyl-making pathway. Diets high in these methyl-donating nutrients can rapidly alter gene expression, especially during early development when the epigenome is first being established.

Take a detailed look at the nutrients that affect our epigenome and the foods they come from: ▶

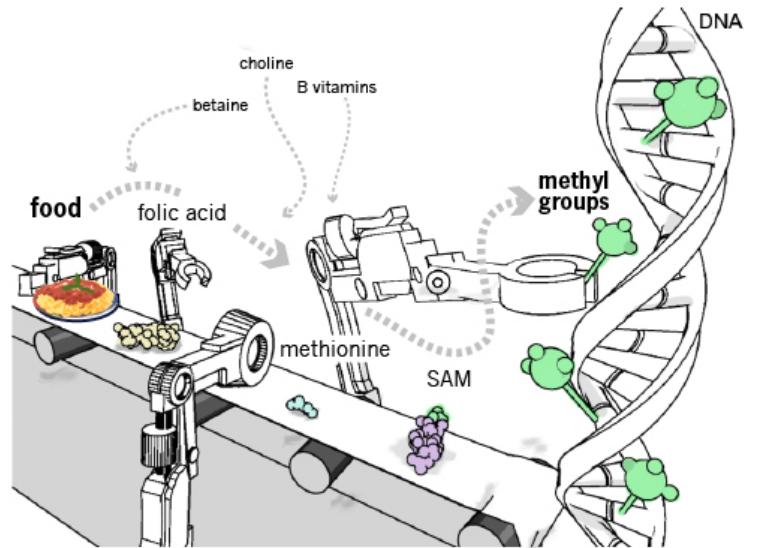


### Eating For Two

Experiments in mice show just how important a mother's diet is in shaping the epigenome of her offspring. Both mice and people have a gene called agouti. When a mouse's agouti gene is completely unmethylated it has a yellow coat color, is obese and prone to diabetes and cancer. When the agouti gene is methylated (as it is in normal mice) the coat color is brown and the mouse has a low disease risk. Fat yellow mice and skinny brown are genetically identical. You can think of the fat yellow mice as looking different because they have an epigenetic "mutation."

When researchers fed pregnant yellow mice a methyl-rich diet, most of the resulting pups were brown and healthy and stayed that way for life. These results indicate that an individual's adult health is heavily influenced by early prenatal factors. In other words, our health is not only determined by what we eat, but also what our parents ate.

**These Two Mice are Genetically Identical and the Same Age**

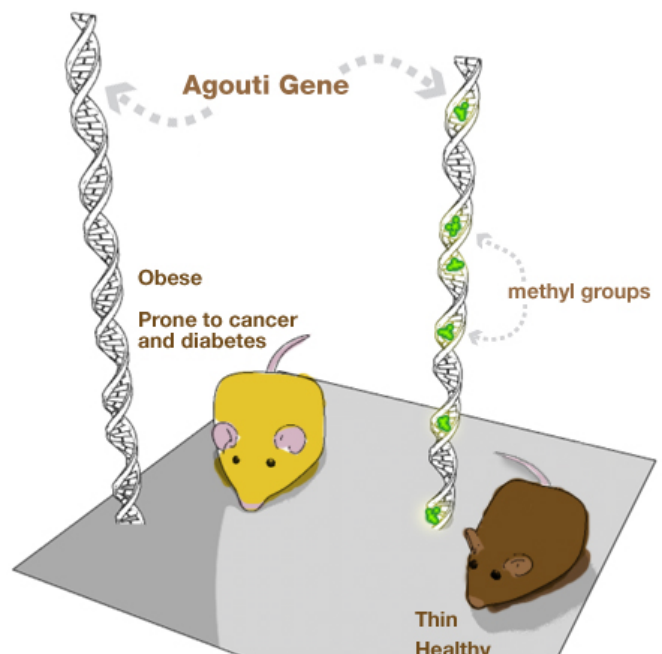


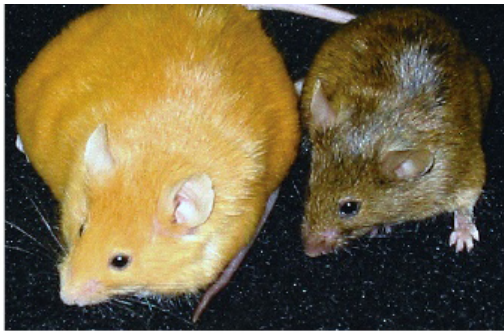
**Nutrients from our food are turned into methyl groups along a pathway:** the pathway is made up of many players that manipulate molecules into methyl groups and ultimately put them on our DNA.

### Diet During Early Development Can Cause Changes Lasting Into Adulthood

Your mother's diet during pregnancy and what you're fed as an infant can cause critical changes that stick with you into adulthood. Animal studies have shown that deficiency of methyl-donating folate or choline during late fetal or early postnatal development causes certain regions of the genome to be under-methylated for life.

For adults, a methyl deficient diet still leads to a decrease in DNA methylation, but the changes are reversible with resumption of a normal diet.





**While pregnant, both of their mothers were fed Bisphenol A (BPA) but DIFFERENT DIETS:**

The mother of this mouse received a **normal mouse diet**

The mother of this mouse received a diet **supplemented** with choline, folic acid, betaine and vitamin B12

## Of Toxins and Supplements

Chemicals and additives that enter our bodies can also affect the epigenome. Bisphenol A (BPA) is a compound used to make polycarbonate plastic. It is in many consumer products including water bottles and tin cans. When controversial reports questioning the safety of BPA came out in 2008, some merchants ceased to carry products made with BPA.

When pregnant yellow agouti mothers were fed BPA, more yellow, unhealthy babies were born than normal. Exposure to BPA during early development had caused decreased methylation of the agouti gene.

However, when BPA-exposed, pregnant yellow mice were fed methyl-rich foods, the offspring were predominantly brown. The maternal nutrient supplementation had counteracted the negative effects of exposure.

## Don't Count Dad Out

So if a pregnant mother's diet can affect the child's epigenetic outcome, can dad's diet do the same? Quite possibly, according to scientists who delved into the well-kept, historical records of annual harvests from a small Swedish community.

These records showed that food availability between the ages of nine and twelve for the paternal grandfather affected the lifespan of his grandchildren. But not in the way you might think.

Shortage of food for the grandfather was associated with extended lifespan of his grandchildren. Food abundance, on the other hand, was associated with a greatly shortened lifespan of the grandchildren. Early death was the result of either [diabetes](#) or [heart disease](#). Could it be that during this critical period of development for the grandfather, epigenetic mechanisms are "capturing" nutritional information about the environment to pass on to the next generation?



Food abundance for the grandfather was associated with a reduced lifespan for his grandchildren.

## A Bee's Royal Diet

Royal jelly is a complex, protein-rich substance secreted from glands on the heads of worker bees. A larva destined to become a queen is fed large quantities of royal jelly inside a specially constructed compartment called a queen cup.

The larvae that develop into workers and queens are genetically identical. But as a result of the royal jelly diet, the queen will develop functional ovaries and a larger abdomen for egg laying, while worker bees remain sterile. She'll also develop the necessary behaviors to act as queen, such as killing rival queens, making communication sounds known as "piping," and going on "mating flights." The queen is fed royal honey exclusively for the rest of her life.

In a recent series of experiments, scientists determined that royal jelly silences a key gene (*Dnmt3*), which codes for an enzyme involved in genome-wide gene silencing. When *Dnmt3* is active in bee larvae, the queen genes are epigenetically silenced and the larvae develop into the default "worker" variety. But when royal jelly turns *Dnmt3* off, certain genes jump into action that turn the lucky larvae into queens.



**Queen Bee Larvae:** Queens are raised in specially-constructed cells called "queen cups," which are filled with royal jelly.

## The Emerging Field of Nutrigenomics

As we better understand the connections between diet and the epigenome, the opportunity arises for clinical applications. Just as mapping our gene variations gives us a window into our personalized medical needs, so might a profile of one's unique epigenome.

Formed through a lifetime of experiences beginning in the womb, our epigenome may provide a wealth of information about how to eat better. Enter the future field of nutrigenomics, where nutritionists take a look at your methylation pattern and design a personalized nutrition plan. While we're not quite to that point yet, your doctor can already tell a lot about the your disease risk by looking at your [family health history](#).

## References

McGowan P.O., Meaney M.J., Szyf M. (2008). [Diet and the epigenetic \(re\)programming of phenotypic differences in behavior](#). Brain Research, 1237: 12-24 (subscription required).

Kaati G., Bygren L.O., Pembrey M., Sjöström M. (2007). [Transgenerational response to nutrition, early life circumstances and longevity](#). European Journal of Human Genetics, 15: 784-790.

Dolinoy D.C., Weidman J.R., Waterland R.A., Jirtle R.L. (2006). [Maternal Genistein Alters Coat Color and Protects Avy Mouse Offspring from Obesity by Modifying the Fetal Epigenome](#). Environmental Health Perspectives, 114:567-572.

Dolinoy D.C., Huang D., Jirtle R.L. (2007). [Maternal nutrient supplementation counteracts bisphenol A-induced DNA hypomethylation in early development](#). PNAS, 104: 13056-13061.

Kucharski R., Maleszka J., Foret S., Maleszka R. [Nutritional Control of Reproductive Status in Honeybees via DNA Methylation \(2008\)](#). Science, 319: 1827-1830 (registration required).

[Disclaimer](#) [Privacy Policy](#) [Permissions](#)

[Cite this page](#)

©2012 | 383 Colorow Dr, Salt Lake City, Utah 84108, (801) 585-3470