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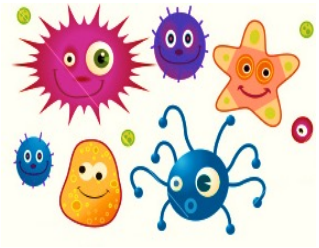
ONE minute medicine

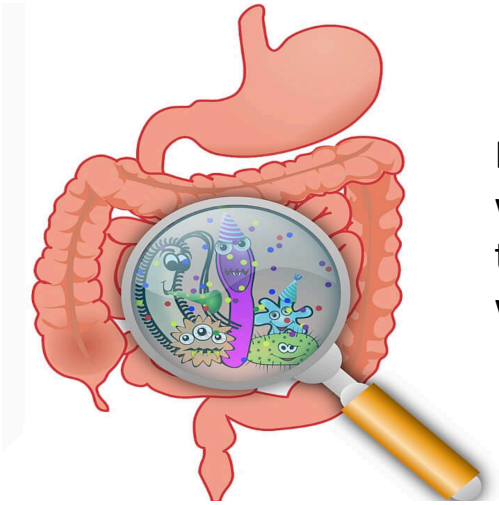


100 trillion friends you didn't know you had

The human gut contains tens of trillions of bacteria; many more of our cells are bacterial than human. We support these hikers because we need gut bacteria to digest our own food.

These resident bugs are categorised into three main groups, called bacteroidetes, firmicutes and actinobacteria, which break down parts of our food that would otherwise be wasted. The bacteroidetes even make vitamin K, a necessary nutrient we can't make for ourselves. Our bacteria help us extract a lot more energy from food than our digestive enzymes can do on their own. In a fascinating experiment, researchers produced "germ-free mice" without any gut bacteria and raised them in an isolation chamber.

When the germ-free mice received transplanted gut bacteria from a normal mouse — yes, that's a polite way of saying a poop transplant — they start eating 27 percent less food, the same as a normal mouse, while increasing their body fat by 60 percent in two weeks.



But when germfree mice were transplanted with gut bacteria from an obese mouse, they gained about twice as much weight as when the bacteria came from a lean mouse!

How bacteria help us digest

The bacteria types common in obesity lead to weight gain because they are especially good at helping us get more energy from food, helping break down complex carbohydrates like cellulose, xylan and pectin. For people who eat a lot of fruits and vegetables, that improvement in digestion may provide an extra 140 to 180 calories per day. Gut bacteria influence the absorption of nutrients in a variety of ways: for instance, by slowing the movement of food through the gut to allow more complete extraction of nutrients, and by increasing the production of an enzyme that moves glucose from the small intestine into the blood. They also suppress an enzyme called lipoprotein lipase that limits the ability of fat cells to take up fatty acids and triglycerides from the blood; suppressing this enzyme leads to an increase in fat storage.

This mechanism seems to be particularly important for obesity disorder purposes, when in germ-free mice gained only 10 percent of their weight instead of 60 percent after they receive the gut bacteria transplant.

Our gut bacteria also reduce the use of fat for energy in the liver and muscles.

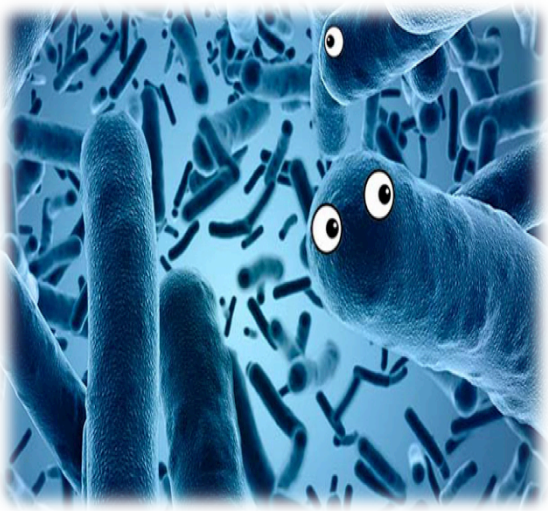
Gut bacteria: the newest weight-loss secret?

Although researchers understand some of the details of how gut bacteria influence like trying to restore a damaged habitat, with potential interactions between species that are difficult to predict.

Different species of bacteria compete with one another, so one dominant type might be able to elbow out any replacement that doctors put in.

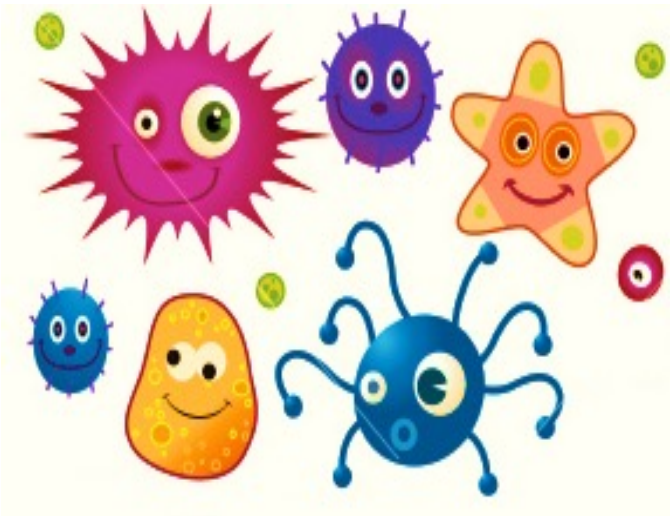
A clinical trial is under way in people, but it will be a while before microbiome therapy for obesity is ready for action. A critical difference between animal research and clinical therapy is that doctors can't transplant bacteria into germ-free people; researchers have to contend with the species that are already present in the gut.

Antibiotics might be making us fat



When researchers give mice low-dose antibiotics from birth, they end up with a higher proportion of firmicutes and fewer bacteroidetes (the pattern associated with obesity) and have more fat tissue than their littermates without antibiotics. In mice receiving antibiotics for four weeks in infancy, their gut bacteria population gradually goes back to normal, but then in adolescence they develop obesity and metabolic problems, including accumulation of fat in the abdomen and the liver. Putting antibiotic treated mice onto a high-fat diet produces the strongest effects of all, especially in males, which are more susceptible to diet induced obesity.

A similar relationship may occur in people. For example, among Canadian babies who did not receive antibiotics in their first year of life, 18.2 percent were overweight or obese at age 12. In contrast, 32.4 percent of children exposed to early antibiotics became overweight or obese. After adjustment for potential confounding factors like birth weight and the mother's weight, the relationship remained statistically significant in boys but not girls. Boys were more than five times as likely to become overweight at seven years old.



However, the opposite result was found for children of overweight mothers, who were half as likely to be overweight at seven if they had received antibiotics, suggesting that the effect of these drugs may vary depending on the child's initial gut bacteria population.

Similarly, among more than 27,000 Danish children born to normal-weight mothers, antibiotics in the first six months of life predicted a 54 percent higher chance of being overweight at seven years old. However, the opposite result was found for children of overweight mothers, who were half as likely to be overweight at seven if they had received antibiotics, suggesting that the effect of these drugs may vary depending on the child's initial gut bacteria population. For now, we can take a couple of lessons from this research.
Parents should minimise antibiotic use in children, especially in the first year of life, because changes in gut bacteria at that age can have lasting consequences.

The average child in the United States receives ten to twenty courses of antibiotics before age 18, increasing the risks of asthma, allergies and inflammatory bowel disease, in addition to obesity and diabetes.

We should think twice about blaming people for their weight. The composition of our gut bacteria profoundly affects our bodies but as yet no-one knows for sure how to use that knowledge to combat obesity.

References

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Farzad Heidari PhD