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The changing flora of obesity

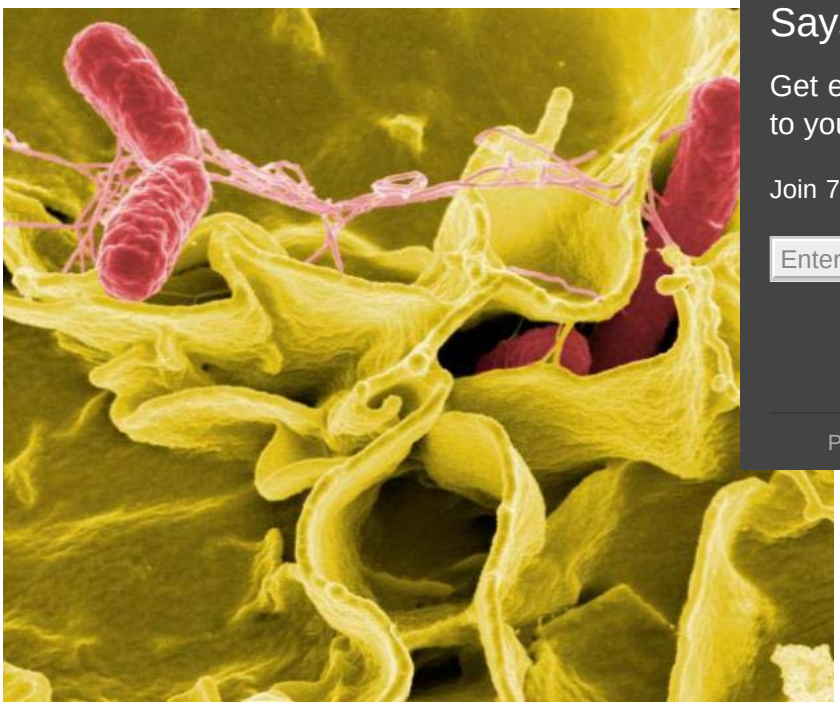
By Lauren Hoskin

It has long been known that human bodies do not exist as a single organism. In fact, we go to bed each night with 100 trillion microbes tucked up cosily inside our intestines. What we are just beginning to find out however, is just how important these microbes are in regulating our nutritional intake.

The gut microbiota is a diverse community consisting mainly of bacteria. Estimates suggest that it comprises somewhere between 500 and 1000 species, mostly *Bacteroides, Bifidobacteria, Ruminococcus* and *Prevotella*.

This cooperative community has many functions, the primary role being to ferment otherwise indigestible carbohydrates, thus extracting energy. It also helps to absorb nutrients, produce vitamins, train our immune systems and prevent excess growth of deadly bacteria. In fact, it is so useful, it is often considered

as an organ in itself.



Past studies have shown that nutritional differences can have huge impacts on the microbiota and vice versa. One study investigated the microbiota of children from a rural village in Burkina Faso and discovered that it contained an exceptional abundance of the bacteria *Prevotella*, which European children completely lacked.

Prevotella was shown to be particularly competent at digesting cellulose, which suggests that the microbiota of this community has coevolved with their diet rich in plant fibres. It has also been shown that ‘germfree’ rats, those raised in a microbe-free environment, metabolised 8.1% less of their dietary calorific intake than normal mice.

Recent research revealed a substantial difference between the gut microbiota of twins when one was obese and the other slim. When these twins’ faecal microbiota was extracted and transplanted into germfree mice, the mice were shown to develop the body composition of their human counterparts, with a high increase in body mass and adiposity. In essence, the mice with the microbes from the obese twin became obese too.

Furthermore, when the obese mice were cohoused with the lean mice, they eventually established a lean-like microbiota and were prevented from developing obesity. However, this change only occurred in mice that were on a diet representative of a healthy human one; rich in fruit and vegetables and low in saturated fats, demonstrating that body mass cannot be altered simply by a microbiota transplant!

This research is an important landmark since it suggests that the diet and microbiota are in constant communication and that interactions between the two can greatly alter the host body. This includes the indication that the gut microbiota could be an important contributor to diseases such as obesity. It also means that in the future, mice could prove a useful tool for screening the impact of different nutrients on both the gut microbiota and the development of obesity.

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