Science Communication Competition

The Science Communication Competition, launched in the Society's Centenary year, is now an annual event. It aims to find young talented science writers and give them the opportunity to have their work published, both online and in *The Biochemist*. The competition attracted lots of entries, and the overall winner for 2013 was Nicole Antonio from the University of Bristol, whose article follows.

Why eating crap could save your life

Nicole Antonio
(University of Bristol, UK)

Sadly, I don't mean eating crap like pizza and chips, washed down with a fizzy drink and a deep-fried Mars bar – that will certainly not save your life. I'm talking about poo. Real, stinking, human excrement. Your faeces is composed not only of what you had for dinner last night – don't pretend you haven't seen the unnervingly whole-looking pea or piece of sweetcorn floating in the toilet bowl – but also consists of other organisms, microbes to be precise. It may surprise you to learn that *over half* of the dry weight of your faeces is actually microbial. The average adult human is made up of approximately 10 trillion cells, yet is associated with over 100 trillion bacteria, collectively called your 'microbiome'. Therefore by number (but fortunately not by mass), you're actually more microbial than human! Bacteria cover every inch of your skin and every orifice, but the vast majority live in your gut. These bacteria help digest your food, protect against nasty, disease-causing, bacteria and in return get a nice, warm, nutrient-rich environment in which to live. Until they come out the other end that is.

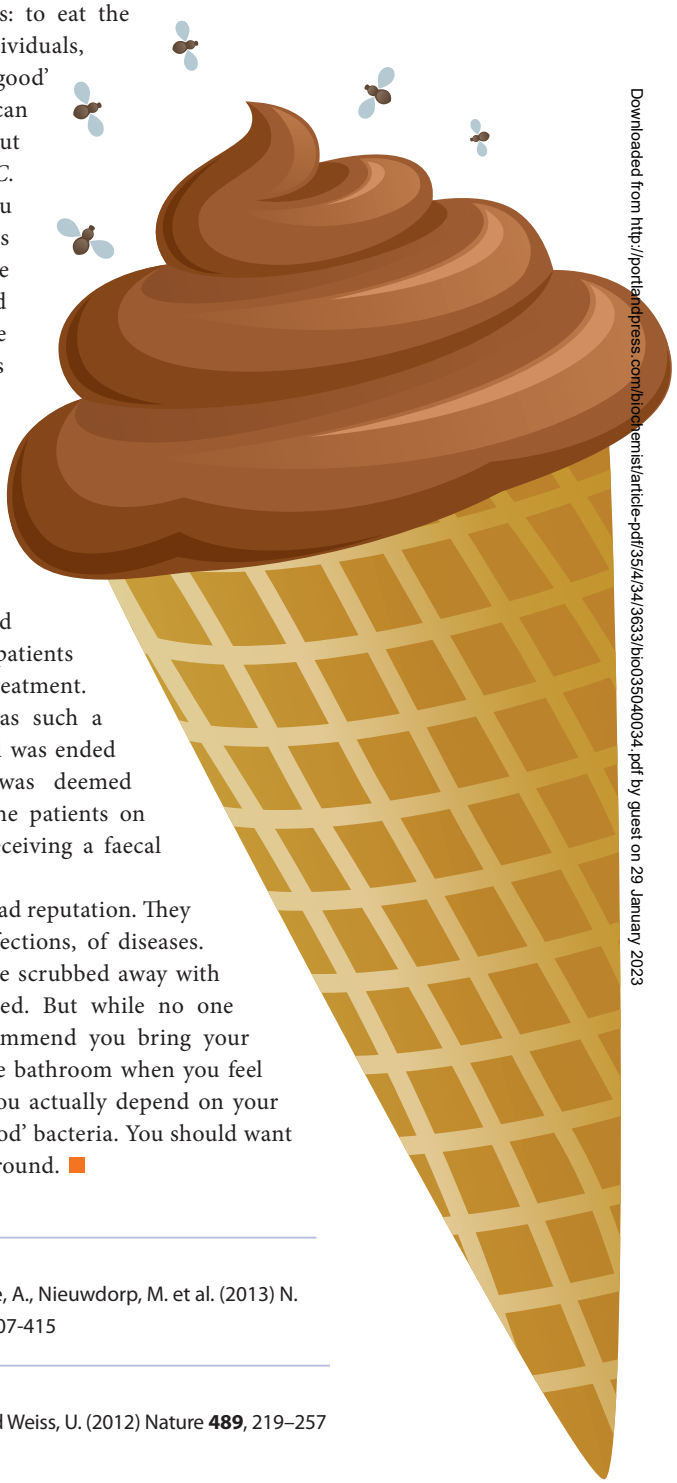
In the womb, your existence began completely sterile. However, during birth, opportunistic bacteria from your mother very quickly colonized you, and from that moment onwards, you have technically never been alone. The human microbiome is a complex ecosystem with a diverse range of different bacterial species. While you can't imagine David Attenborough commissioning a series on them any time soon, scientists are now attempting to sequence the genomes of every one of these bacterial species – our so-called 'second genome' – in an ambitious 5-year programme called the Human Microbiome Project (www.hmpdacc.org).

The task is formidable: 242 men and women, aged between 18 and 40, donated samples from up to 18 different body sites including the airways, skin, mouth, gut and vagina, plus stool and saliva samples. If even the slightest indication of gum disease or yeast infection was detected, those individuals were excluded; the project aims to define *healthy* microbiome communities. The investigators resampled the subjects three times during the course of the study to determine the stability of the microbiome, generating a total of 11 174 samples. This vast number of samples is currently being analysed in various laboratories across the world.

We are already aware that the microbiome not only influences our healthy physiology, but also plays a prominent role in disease; bacteria are thought to explain why individuals react differently to various drugs and why some are more susceptible to certain infectious diseases while others appear immune. They are thought to contribute to chronic diseases and conditions like irritable bowel syndrome, diabetes, asthma, neurological disorders and cancer. Recent research has even shown that dental plaque bacteria entering the bloodstream can cause blood clots and therefore lead to an increased risk of a heart attack or stroke. Although it isn't fully understood yet, we know that obese individuals have a distinct microbial communities compared with lean individuals, and that this is not simply due to differences in diet – it is possible that the presence of certain bacteria help to absorb fat, leading to an increased chance of obesity. Ultimately, completion of the Human Microbiome Project will unleash the potential to improve health by deliberately altering bacterial populations. Just imagine: a future of yoghurt drinks that could help you lose weight.

All this leads me on to why on earth you'd want to eat crap. A species of bacteria called *Clostridium difficile* is one of the nasty guys that you've probably heard about. A superbug in the same league of MRSA, *C. difficile* is an opportunistic bacterium that causes severe diarrhoea and can be life-threatening. When patients are admitted to hospitals and prescribed antibiotics, these wipe out not only the disease-causing critters, but also the good bacteria of their microbiome. Due to *C. difficile*'s remarkable resistance to antibiotics, it can flourish in the absence of any competition and rapidly seizes control of the gut. In 25% of patients, further antibiotic treatment does not cure the infection, and new treatment options are desperately needed.

A new study by Els van Nood and colleagues published in the *New England Journal of Medicine*[1], describes a revolutionary, and quite counter-intuitive treatment for those with unresponsive *C. difficile* infections: to eat the faeces of healthy individuals, providing the 'good' bacteria that can re-colonize the gut and out-compete *C. difficile*. Before you ask, these patients aren't given a plate and a fork and asked to tuck in; instead the 'faecal transplant' is administered via a tube directly into the small intestine. In all, 90% of patients given a faecal transplant recovered compared with 20–30% of the patients on the existing treatment. This was regarded as such a success that the trial was ended prematurely – it was deemed unethical to keep the patients on the placebo from receiving a faecal transplant too.



Bacteria have a bad reputation. They are the cause of infections, of diseases. They are things to be scrubbed away with bleach, to be avoided. But while no one would actually recommend you bring your knife and fork to the bathroom when you feel ill, just remember you actually depend on your 100 trillion or so 'good' bacteria. You should want those guys to stick around. ■

Reference

1. van Nood, E., Vrieze, A., Nieuwdorp, M. et al. (2013) N. Engl. J. Med. **368**,407-415

Further reading

Lupp, C., Skipper, M. and Weiss, U. (2012) Nature **489**, 219–257