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Diet and ageing

A dog's life

Gut bacteria may help to explain why a Spartan diet increases lifespan

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IT IS now generally accepted that eating less makes animals live longer. That has been demonstrated in creatures ranging from worms to mammals. Exactly why it should be so remains, however, hotly debated. So Jeremy Nicholson of Imperial College, London, and his colleagues set out to shed some light on the matter. Their results have just been published in the *Journal of Proteome Research*.

One theory of ageing suggests senescence is a result of damage caused to body cells by reactive molecules called free radicals. These molecules are created as a side effect of the release of energy from glucose. If that were true, a lower metabolic rate might slow the process down. The question is: does eating less result in a lower metabolic rate? The answer that Dr Nicholson and his colleagues have come up with is that it does—in dogs, at any rate.

They have come to that conclusion by drawing on data from a 15-year experiment conducted by Purina, an American dog-food company. This experiment involved taking 48 Labrador pups from seven litters just after they were weaned. The dogs were put in pairs, with partners chosen to be siblings of the same sex and similar weaning weight. One pup was allowed to eat as much as it wanted, while its partner received 25% less than the amount the first pup had consumed on the previous day. After three years, the regime was changed so that the first dog of each pair was fed the diet deemed necessary to keep it at a healthy weight while its partner received 25% less than that. The experiment concluded that, on average, the dogs fed less food lived almost two years longer than those fed more—adding dogs to the list of animals that benefit this way.

What enabled Dr Nicholson to build on this result was that Purina's researchers had collected and frozen samples of their dogs' urine at periodic intervals. He and his colleagues were able to use this liquid gold to track the metabolism of each animal throughout its life.

They were particularly interested in two sorts of molecules. The first were derivatives of creatine, a substance that helps to supply energy to muscles. As the dogs grew, the levels of creatine derivatives in their urine increased. Later, as they became elderly, those levels fell. No surprise there. But throughout their lives, the dogs that were fed well had more creatine derivatives in their urine than their calorie-deprived counterparts. This, the researchers suggest, shows that the dieting dogs' muscles were less active and that those animals had thus

used less energy than their well-fed confrères. Their overall metabolism, in other words, had been depressed.

Why that might be was hinted at by a second sort of molecule—a group of compounds called aliphatic amines. These chemicals (which, incidentally, give urine its aroma) are made when bacteria munch on a chemical called choline that is part of an animal's food. The reason for Dr Nicholson's interest was that choline is essential for metabolising fat, but dogs cannot synthesise it themselves. Aliphatic amines gave him some indication of how much choline the dogs were able to absorb.

Choline is made available for absorption from the intestine by the activities of the gut bacteria that are liberating it for their own purposes. The amount of aliphatic amines in urine is thus an indirect measure of how much choline is available. As in the case of creatine, this differed between the two groups. Dogs on the restricted diet had lower levels of the amines in their urine than did their well-fed counterparts—implying that less choline was being made available. And if less choline were available, that would limit a dog's ability to metabolise fats, and thus restrict its metabolic rate.

The apparent drop in choline levels was much greater than could be accounted for by a relative lack of food, so Dr Nicholson suspects that the restricted diet was also causing the composition of the dogs' gut flora to change in a way that did not favour choline-munching bugs.

That result, if true, echoes one published a few months ago by Jeffrey Gordon of Washington University, in St Louis. He showed that putting obese people on a diet changes the mix of their gut bacteria. In that case the consequence is a change in the metabolism of carbohydrates rather than fats. Nor was there a direct link with longevity of the sort implicit in Dr Nicholson's work. Nevertheless, the parallel is intriguing—and yet another incentive to cut down on the calories.

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