Out of the Box

Often when I Google, or just turn on a light, I marvel that so much of everyday life now was until recently dreams and visions that were – as freaks used to say – out of sight. This column suggests some far-out ideas. I agree with some of them.

For example, think planetary health, and it’s obvious that there are too many of us. Instead of the current 6.5 billion global population set to inflate to 9 million by 2050, a sustainable number might be the 1 billion of 1800 or the 1.5 billion of 1900, with births and deaths balanced. Consider the biosphere of which we are a part, and it’s evident that the multiplication of the human species, enabled by the public health developments always trumpeted as a triumph of civilisation, is a catastrophe. This month I go further out, with the idea that we are also too old.

Drugs and bugs

The pages of my paperback copy of Medical Nemesis have become brown: I bought it in 1979. When I first read it I thought: come on, I also like to push a point, but this goes too far. For example: ‘The pain, dysfunction, disability, and anguish resulting from technical medical intervention now rival the morbidity due to traffic and industrial accidents . . . and now make the impact of medicine one of the most rapidly spreading epidemics of our time’. Too much, Ivan!

Ivan Illich may have been on something in his then Mexican retreat, but he was also on to something. Take car crashes and just one killer disease caused by drug intervention: gut superinfection with Clostridium difficile, a bacterium discovered in the mid-1930s, coincident with the use of the first antibacterial drugs. In the UK, around 3300 people are killed on the road every year. In England C. difficile was registered as a cause of 2155 deaths in 2004, and in 2005 accelerated past the number of road deaths, totalling 3697. C.diff colitis mostly kills elderly hospital patients5. But it’s not a gentle way to go: the colon becomes inflamed, ulcerated and scarred, and may rot and crumble.

At the time I wrote my book Superbug about the consequences of overuse and abuse of antimicrobial drugs5, the total number of cases of C.diff colitis in Britain had risen from 121 in 1982 to 1643 in 1992; the Public Health Laboratory Service stated ‘there is no doubt that C. difficile will be a major problem in the 21st century’. Indeed. Here we are, and in 2006 the number of cases in England was over 50 0007. In the USA and Europe the estimate was 350 000 in 20028; a safe current guess is half a million.

Peter Borriello, an authority on C. difficile, is now director of the Centre for Infections at the UK Health Protection Agency. Interviewed for Superbug he said: ‘What is so interesting about C. difficile is that it will only infect following compromise of the normal gut flora, classically with antibiotics, or with anything else having antimicrobial activity. I could give you 10 to the power of 12 [10 million million] organisms of C. difficile and nothing would happen. I could give you one of a number of antibiotics first and then give you 10 organisms of C. difficile and you would go down with diarrhoea. With C. difficile the colonisation resistance is everything or nothing. We don’t know why this is’.

I think we do know. The explanation is three-dimensional new nutrition science stuff, with acknowledgements to Jeff Leach in Louisiana. First, we do indeed know that colonisation resistance – the power of commensal ‘friendly’ flora to maintain gut microbial ecology and to keep down bad guy bugs – is damaged and can be destroyed by antibiotics.

How? By analogy take the invasion of Iraq, the country of the Garden of Eden. Iraq is the human being, the Saddam Hussein regime guts-ache, Iraqi civilians the friendly flora, the Bush II regime the expert, and the US forces the antibiotics. Once upon a time in Iraq there was a population of men, women and children, and also some big bad guys who kept on causing trouble. Then Iraq was zapped, the bad guys were wiped out, together with hundreds of thousands of innocent people9, and the survivors were thrown into confusion. What then happened was – and is – destruction and disruption of balances of culture, religion, politics and economics, insurrection of all sorts of bad guys, and the implosion of the nation. The expert then perpetrates more heavy-duty zapping, which creates even more virulent resistance. By analogy, that’s what happens when antibiotics are relied upon for gut disorders. Governments contemplating invasions should be advised by microbiologists.

We don’t like to think about problems like global overpopulation and disruption, where there seems to be no solution in sight. But the problem of iatrogenic gut diseases can be addressed, and not only by the sane use of antimicrobial drugs. Here comes the second part of the story, and the psychedelic concept. The bacterial species that have evolved with us, and live in our guts, amount to 12 [10 million million] organisms of C. difficile and nothing would happen. I could give you one of a number of antibiotics first and then give you 10 organisms of C. difficile and you would go down with diarrhoea. With C. difficile the colonisation resistance is everything or nothing. We don’t know why this is’.

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and, as Jeff Leach says, ‘Fiber is not food for us, it’s food for the bacteria that live in our gut’10.

As we know, dietary fibre is stripped from refined foods, and so typical industrialised diets are fibre-depleted. Consequently, it is commonly recommended that on average fibre consumption should be increased by say 50%, to around 25 grams a day11. Jeff Leach says that any such recommendation is footling. His evidence is palaeolithic poo12. Rummages in coprolites of Stone Agers who for example lived on the shores of Lake Galilee and in the Trans-Pecos region of west Texas, reveal remarkably bio-diverse diets, and a dietary fibre intake of up to and often way above 100 grams a day.

This, he proposes, is why many findings on dietary fibre and colon cancer are equivocal: these come from studies of populations consuming industrialised diets all of which are way, way below the amounts of fibre to which the human species is adapted by evolution. ‘Until we have better understanding of the diversity and quantity of fermentable substrate that entered our ancestral bowels . . . the possible and important protective role of fibre in aetiology of colorectal cancer may not be forthcoming’12.

Meanwhile the guts of populations that consume fibre-depleted diets are full of starved bacteria, always vulnerable to foreign pathogenic microbial species. These insurgents enter, proliferate, and cause havoc. Some strip the mucosal lining of the gut wall, cause inflammatory bowel diseases, and make us more vulnerable to local and systemic disease caused by toxins and carcinogens in foods such as processed meat that would do no harm to a healthy intact gut full of well-fed commensal bugs. Antibiotics accelerate this process which, once the gut wall is penetrated, is liable to be irreversible.

Are we too old?

So there we are. The proposal that humans – or rather, the gut flora with which we have evolved – are not adapted to fibre-depleted diets beyond dispute. Further, the evidence from Stone Age turds, that humans are evolved to consume diets that to us are almost unimaginably chewy and bulky, is impressive.

But there is a problem here. As far as we know, palaeolithic people usually did not survive into what we call later middle age, let alone old age. After all, why should the human species be evolved to continue to live after the age when children are grown? Where is the selective advantage? Sure, all communities need some wise and experienced elders. But we know that national economies now are brought down by the growing number of unproductive pensioners, who stick around and chew the fat, boring on about how much better things were in the old days. Likewise there must have been limits to the number of palaeolithic crumblies who could be supported, as they gnawed the fibre and demanded prime warm cave space.

Here’s the next idea. Almost all of us who are in ‘the Third Age’ are past our biological best-before date, the time of life when nature intended our entire bodies to be consumed by bugs. I agree that the usual recommendations for dietary fibre and indeed healthy foods are likely to be way under what we need for real well-being, and in my own diet I go for what I reckon is around double the Cummings–Bingham top quantile13. But I am not persuaded that compositional analysis of palaeolithic poo reveals our standard.

I pause for a reply, from Boyd Eaton14 or Jeff Leach. Meanwhile, it seems to me that the Gaian attitude to modern later middle-aged people may well be one of existential indifference or impatience: past the age of 50, say, we are relics. The implications of this idea are intriguing. What are the right food systems and diets for those of us who from the evolutionary point of view are meant to be dead?

What goes up . . .

Last month I reported that the British now are confused about food and its effects. There is more, published in the British national press during just one week. Super-size school uniforms are now being produced, including shirts with 17½ inch (44.5 cm) necks and trousers with 42 inch (107 cm) waists15. Young obese girls may become sexually mature at the age of 10, which predicts a surge of pre-teen pregnancies. Three-year-olds are now being treated for obesity. A University of London study proposed that schoolchildren be compelled to wear trainers in playgrounds, in case they felt like running around16.

Help might be at hand, for ‘Scientists find the gene that makes you fat’. This front-page lead news story17 was about ‘the discovery of an “alphabet soup” of genes that influence obesity’, one of which, an ‘FTO variant’, apparently predisposes to weight gain. ‘In the future, when scientists have found additional obesity genes, it may be possible to offer advice based on a person’s genetic make-up’; and weight loss ‘may be achieved with drugs targeted at the molecular pathways the gene influences’. Geneticist Professor Andrew Hattersley of the Peninsula Medical School in Exeter, one of the principal investigators, was excited. The story went round the world. Quoted in India, Dr Hattersley said: ‘This tells us that it’s wrong to assume that all those who are fat are the greediest or the laziest18. Anoop Misra, director of the diabetes centre at the Fortis Hospital in New Delhi, dryly pointed out that while obesity has increased 500% in urban India in the last 30 years, ‘we have the same genes today as we had in the mid-1970s’.

Meanwhile, in Europe 231 million people went on some sort of diet in 2002, contributing to an industry overall then worth £93 billion a year19. Dr Hattersley’s variant FTO
gene was not the biggest story that week. This was ‘Weighty case against diets’\(^\text{20}\): the results of a meta-analysis of 51 studies of the eventual effects of low-calorie diets, done by a UCLA team. Now at any time, one in four people in Britain is trying to lose weight. The average woman is estimated to lose and gain 25\(^\text{2}\) stone [360 pounds] during her lifetime\(^\text{21}\), and weight cycling, also known as yo-yo dieting, seems to increase the risk of cardiovascular disease\(^\text{22,25}\) and overall mortality\(^\text{24}\).

Traci Mann, the chief UCLA investigator, said: ‘The majority of people regained all the weight, plus more’. Most of the dieters ‘would have been better off not going on the diet’. She added: ‘My mother has been on diets and says what we are saying is obvious’. Her mother is right: in Europe, 1% of dieters achieve permanent weight loss\(^\text{19}\). The UCLA story also went round the world. In Brazil Janet Tomiyama, a colleague of Dr Mann, was quoted as saying ‘dieting consistently predicts future weight gain’\(^\text{25}\) or, to be precise, greater weight gain compared with non-dieters, as shown by eight of nine prospective studies specifically addressing this point\(^\text{20}\).

... won’t come down

So diets don’t work. But the UCLA study points to a more radical conclusion. The Daily Telegraph got the point. The headline of a prominent article by William Leith was ‘Health warning: all diets make you fat’\(^\text{27}\). He explained, from his own experience of yo-yo dieting, recorded in his confessions\(^\text{28}\). ‘What happens when the body is given less food than it needs? In the short term, it lives off its own reserves of fat. It gets thinner, but another mechanism comes into play – it also gets better at getting fat. When you diet... your body thinks you are unable to find food. You think: diet. Your body thinks: famine... And crucially, the more diets you go on – the more faminises your body is exposed to, in other words – the better you become at getting fat’.

Well, I thought, as I skimmed the story, I couldn’t have put it better myself. Then I realised that William Leith was indeed, with all due credit, paraphrasing Dieting Makes You Fat, my first book, co-authored with Hetty Einzig\(^\text{29}\). Forgive me for quoting myself\(^\text{30}\): ‘The body is evolved to adapt to periods of energy restriction as if these are periods of scarcity or famine, by means of mechanisms that after the restriction is over, trigger hunger, inhibit satiety and preferentially conserve body fat... Indeed, it is hard to see how Homo sapiens could have evolved and survived without some such adaptive mechanisms’.

If you disagree with this thesis, let’s hear from you. Andrew Prentice, Gail Goldberg, Susan Jebb and colleagues have challenged one of its proposals, that weight cycling depresses metabolic rate, in a short-term study\(^\text{31}\). But I aver that now the force is with me.

The idea that weight cycling is not only futile, but also may well increase the risk of disease and death, has interesting implications. Obesity as conventionally defined – a BMI of 30 plus – certainly is a cause of a number of serious chronic diseases, as well as a source of misery and disadvantage. Further, even if at least in high-income countries overweight short of obesity is not in itself as troublesome as more zealous IOTF members claim\(^\text{32}\), it does predict later obesity. But given that diets do not work and that weight cycling is risky, fat people mainly interested in avoiding heart attacks may be best advised to stay fat.

Do not burst a blood vessel, dear IOTF reader. A rewrite of Cannon’s Paradox will explain how able-bodied overweight people who want to lose fat can do so reliably and safely. I am now trying it on myself. Every day when I get up, I... No, you will have to wait... Seriously though, on a population basis adult obesity is intractable. The only countries within which average BMI decreases, are those beset by famine, debt, invasion, or blockade. The solution is to jump generations. The focus of public health nutritionists interested in overweight and obesity should not be with adults, but with infants and young children and, much better, with adults preparing to be parents, for the sake of their unborn children.

Bibles and garlic

Here is a plug for the two-volume, magazine-format, 2153-page The Cambridge World History of Food\(^\text{35}\). Its space on my shelves is next to Alan Davidson’s elegant 1073-pager The Penguin Companion to Food\(^\text{34}\), which I also constantly consult. I well remember seeing them displayed together in Waterstone’s in London’s Gower Street, guessing correctly that their heft would tip me into overweight at Heathrow check-in on my way back to Brazil, but buying them both.

The History is made up of a series of 170 reviews, altogether somewhat like long-gone editions of the Encyclopaedia Britannica whose entries reflected the personalities and prejudices of a cornucopia of contributors. It is easy to bitch about shortcomings. Thus, of the editorial board of 39 all but seven are from the USA, which is dozy. Also, coverage of industrial food systems (as distinct from jaded topics like labelling and additives) is sketchy, compared with the excellent essays on ancient and traditional systems evidently of more interest to Kenneth Kiple, one of the two general editors. (Yes, coprolites are covered.) Further, the contributions on nutrients are competent but mundane, and those on nutrition and chronic diseases are best overlooked.

By contrast, the essays on the history of food and food culture are sound to splendid, although that on South America is cursory. Colin Spencer has contributed a masterpiece on the British Isles, and the Caribbean – Dr Kiple’s special subject – is comprehensively covered. The History is an indispensable source for information on staple foods. Ellen Messer of the World Hunger Program
on maize (corn) is particularly measured, though for political savvy I recommend Michael Pollan’s fulmination against the destruction of US agriculture by gross overproduction of cheapened corn\textsuperscript{35}. The two essays on breastmilk and artificial infant feeding by Antoinette Fauve-Chamoux, and on infant and young child nutrition by Sara Quandt, can remind readers what nonsense it is to pluck food and nutrition out of its historical context. And during this browse now, as I complete this column, I learn that \textit{suam}, the Chinese word for garlic, is a single character, ‘which often indicates the antiquity of a word’; and that in his \textit{Natural History}, Pliny the Elder includes 61 garlic-based remedies. Now you too know.

Geoffrey Cannon
GeoffreyCannon@aol.com

References

2 Hall C. Superbug kills almost as many as die on roads. \textit{The Guardian}, 7 April 2006.