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Chapter 7*

The potential of nutrition to promote physical and behavioural well-being

Bernard Gesch

Introduction

Good nutrition is considered to be seminal to our physical well-being throughout our lives. It is widely accepted as a major factor in the prevention of chronic disease. Since the brain also has to be nourished, there are increasingly good reasons to believe that the health benefits of good nutrition extend into positive effects on our behaviour. Yet it is curious that most of the authoritative reference works on diet and international dietary standards focus on health issues but hardly mention behavioural well-being. It is argued that such standards should be reassessed to take into account brain function and behaviour.

Food is a meeting point of the social and physical worlds. The availability of food, food types, and food choices will interact with a wide range of socio-economic factors. Yet, no species can flourish without food and water. This global necessity highlights vast inequalities. Many of the World’s population starve while at the same time we are witnessing an epidemic of obesity. This may be the visible tip of an iceberg and, to extend that analogy further, it is the 90% that goes unseen below the water that is the most dangerous. The impacts of various changes in modern diets such as increased intakes of salt, saturated fats, and refined sugars are already apparent in global health. This chapter will extend the discussion to examine the relationship with behavioural well-being.

The nutritional ascent of man

The human brain is exceptionally large for a terrestrial animal and has facilitated an enhanced capacity for adaptation and problem-solving (Crawford et al. 1999). Anthropological science has taught us how important nutrition was during the evolution of our brain (e.g. Martin 1983; Blumenschine 1991; Foley and Lee 1991). The brain is a fatty organ and it is believed that the availability of two highly

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unsaturated fatty acids, arachidonic acid (AA) and docosahexaenoic acid (DHA), from which our central nervous system is primarily constructed is likely to have been a limiting factor in brain size (e.g. Crawford et al. 1972; Broadhurst et al. 2002) and complexity (Fernstrom 1999). The omega-3 highly unsaturated fatty acids that are most important for brain function, eicosapentaenoic acid (EPA) and DHA, are only found in appreciable quantities in oily fish and seafood, primarily because these creatures consume the algae that synthesize EPA and DHA. The dietary omega-3 precursor of EPA and DHA is alpha-linolenic acid (ALA). It is found in dark green leafy vegetables and some nuts and seeds, but its conversion to DHA in particular may be limited (Burdge and Wootton 2002). AA on the other hand is an omega-6 fatty acid and this group is plentiful in modern diets. Omega-6 fatty acids are typically found in vegetable oils, but AA is found in meat and dairy produce. The fact that the savannah is not a rich environment for DHA has led scientists to reconsider whether Homo sapiens really did evolve on the plains (Broadhurst et al. 2002; Crawford et al. 1999) rather than near water in coastal or lakeside habitats (Stringer 2000; Tobias 2002; Morgan 1990). However, DHA could have come from eating the fresh brains of other terrestrial animals on the savannah. So, either humans used their stone tools to break open the strong skulls of freshly slaughtered beasts, or they could have simply reached down on the shore and popped fresh shellfish into their mouths; for an intelligent species the latter has greater elegance. Certainly, some of the earliest fossil remains of our species have been found on the shoreline of the Cape of Good Hope (Stringer 2000; Rightmire and Deacon 1991) and it is gaining credence in anthropology that it was around coastal and inland shorelines that our brain mass evolved to about twice the size of that of our predecessor, Homo habilis (Crawford et al. 1999; Broadhurst et al. 2002).

The evolution of such a metabolically expensive organ is remarkable because in the adult it consumes around 20% of available energy (Leonard and Robertson 1997) and receives 12% of basal cardiac output to supply it with nutrients (Shepherd et al. 1983). A recent article concluded that middle Palaeolithic people (Neanderthals) were less effective at exploiting coastal and lakeside food resources than Homo sapiens in the upper Palaeolithic era (Klein et al. 2004). This change in exploitation occurred as recently 50 000 years ago and it is argued this may have been the decisive change in our behaviour that facilitated our success over Neanderthals (Klein et al. 2004). Furthermore, it has been pointed out that, in coastal hunter–gatherer cultures, women tend to collect shellfish and this may have been true of our ancestors as well (Parkington 1998). A pregnant mother would thus have ready access to large quantities of the nutrients essential for the development of her fetus’s brain and central nervous system, which receives 70% of the energy supplied (Cunnane et al. 2000). Shellfish are also a rich source of protein and animal studies have demonstrated that lower maternal protein intakes reduce delivery of DHA from the mother to the fetus, which may impair the development
and function of the fetal brain (Burdge and Wootten 2002). Klein and colleagues (2004) suggest that the Palaeolithic evidence points to the collection of shellfish as the key source of nutrients rather than deep water fishing as there is a lack of evidence for fishing technology. Certainly, it appears to be the case that shellfish were plentiful in the areas where early fossils of our ancestors have been found (Stynder et al. 2001).

**Moving into the fast lane**

It should be recognized, however, that anthropology is an uncertain science involving the meticulous piecing together of evidence to form a picture, but many of the conclusions about the importance of nutrients on brain development are beginning to be supported by experimental evidence, indicating that nutrients rich in sea food are as relevant to brain development now as they appear to have been to our forebears (Uauy et al. 2001). Indeed, shellfish not only supply DHA and EPA but are also particularly rich in trace elements such as zinc, iron, and iodine when compared to deep-water fish. These nutrients are also essential for cognitive development, offering further support to anthropological evidence that these were the food sources that facilitated our brain development. Zinc, for instance, may also turn out to be limiting factor in brain development as intake positively correlates with fetal head circumference (Ward et al. 1990) and has been shown experimentally to alter essential fatty acid composition (Wauben et al. 1999; Ayala and Brenner 1983, 1987; Dieck et al. 2005). Animal studies have shown that low zinc (Oteiza et al. 1990) and also low protein intakes (Bennis-Taleb et al. 1999) impair brain development. Fortunately for us, our highly evolved brain appeared to pay off as evidence suggests our ancestors moved north and overwhelmed existing populations of Neanderthals (Klein et al. 2004). Indeed, contemporary findings from 10 of 13 randomized trials suggest that significant improvements occur in nonverbal intelligence when participants are given micronutrient supplementation (Benton 2001) but these effects depend on the quality of dietary baselines; broadly the worse the diet, the greater the effect. In a population of rural Thai children where zinc, iron, and iodine deficiencies are prevalent, supplementation of these nutrients treated the deficiency with concomitant improvements in cognitive functioning (Manger et al. 2004). Since nutrients appear to positively impact cognitive functioning nowadays, it is feasible that better nutrition raised our ancestors’ functioning to a new level.

**Food for thought**

An underlying question remains as to which is the causal factor. Thus, did our diet actually shape our behaviour, was it the other way round, or, indeed, a little of each? The environment in which a living organism evolved is likely to have been a determinant of which elements became essential for life. The phospholipid membrane structure of the brain, as well as the use of trace elements as enzyme
catalysts, is considered to have originated in a marine environment (Broadhurst et al. 2002; Nielsen 2000). This is consistent with evidence that is suggestive that a coastal diet provided a suitable nutritional environment to support the physical enlargement of our brains. While nutrition is widely accepted as influencing long-term health, we somehow manage to decouple that relationship from behaviour with the assumption that our behaviour is purely a matter of free will. This is despite the fact that we cannot by any means decouple nutrition from actual brain function.

Nevertheless, Derek Bryce-Smith in his Royal Society of Chemistry Lecture (Bryce-Smith 1986) reviewed evidence that poor nutrition or exposure to lead (a neurotoxin and nutrient antagonist) might influence behaviour, personality, and mentation. He reasoned that, since nutrients support the operation of our senses, an individual couldn’t normally sense a lack of nutrients or exposure to neurotoxins ambient in the environment. Hence, Bryce-Smith proposed that there could be potent effects on our behaviour that (unlike alcohol) act without our knowledge, e.g. an association between exposure to lead and violence (Needleman et al. 1996; Masters 2000; Stretesky and Lynch 2001). Essential nutrients are involved in protecting the body from the deleterious effects of neurotoxins. Furthermore, if an individual is unwittingly undermined by poor nutrition, those around them are unlikely to know about it either, and would tend to attribute any inappropriate behaviour to deficits in the perpetrator’s personality.

**Changing dietary baselines**

If this scenario is correct, our forebears’ diet may have been a recipe for success. Curiously then, there is surprisingly little overlap between estimates of what our ancestor ate and what we eat nowadays: the intakes of the essential omega-3 and omega-6 fatty acids were 1:1 rather than the 1:15 ratio found nowadays, perhaps the most extreme example of dietary change (Eaton and Konner 1995; Eaton and Eaton 2000; Eaton et al. 1997): Palaeolithic diets are considered to provide more fibre and less salt; carbohydrates would come chiefly from uncultivated vegetables, fruit, and perhaps honey rather than the grains and refined sugar consumed nowadays. The focus of further discussions will be on these dietary factors.

There are, however, many other factors in addition. We can be sure that our ancestors did not eat a highly processed diet. The Palaeolithic diet would probably have provided more phytochemicals, which we are beginning to appreciate are also important for health (Eaton and Eaton 2000). Palaeolithic diets would, by current standards, be considered organic, additive- and pesticide-free (Curl et al. 2003; Egger et al. 1985; Schab and Trinh 2004). Our forebears are likely to have expended more energy in physical activity (Eaton and Eaton 2003), and we are well aware that sedentary lifestyles are associated with obesity and undesirable consequences

While we cannot be certain of the Palaeolithic diets and estimates have been revised over the years, it would nevertheless be prudent to test experimentally the relative merits of modern diets with such differing dietary baselines—particularly as it is argued that our genes are attuned to the Palaeolithic diet (Cordain et al. 2000). How quickly can we expect our brain to adapt to its new nutritional environment? Perhaps our forebears were so closely engaged in the collection of food that they never took food security for granted. Indeed, it is useful to reflect if it is the hallmark of an intelligent primate that we are only now recognizing the seminal role of the aquatic environment for our well-being, just as concern is growing about the exhaustion of fish stocks through wasteful overexploitation (Clover 2004) and the contamination of fish stocks (Hightower and Moore 2003) through ill-considered treatment of the world’s oceans by industrialized nations.

**Organized food production**

We may be tempted to look back to our hunter–gatherer origins through rose-tinted glasses, but the increased security of food availability from agriculture and industrialization is likely to have facilitated the expansion of our population. If food shaped our behavioural strategies about 50 000 years ago, then it seemed to happen again around 10 000 years ago when we began organizing our society around agricultural food production, which played an important role in shaping our present social structures and higher population densities (Crawford et al. 1999).

Curiously, despite the likely advantages of better food security, it is argued that this change in food harvesting may have resulted in a decline in life span from that of the hunter–gatherer (Angel 1984). This could well be related to increased population density and the spread of infectious diseases. It is also possible that another element was the change in diet. Comparisons of active wild animals with farmed animals illustrate important changes in body tissue composition. Wild animals contain a higher percentage of protein than farmed animals (15% versus 10%) and a lower percentage of fat (5% versus 30%). Fat provides 9 calories for every gram consumed, so the wild animal would provide only 45 calories as fat to every 270 calories as fat from the extensively reared animal (Crawford 1968). The types of fats also differ (e.g. Ledger, 1968; FAO 1994). In wild animals the ratio of non-essential to essential fatty acids is typically 3 to 1, whereas in modern beef the same ratio is reported to be 50 to 1 (Crawford 1968). Animal studies have also shown that both nutrient restriction and overnourishment during pregnancy suppress placental cell proliferation and vascularity, which has an important impact on fetal development and thereby on neonatal mortality and morbidity (Redmer et al. 2004). Indeed, estimates of life span based on that of modern hunter–gatherers are around 40 years of age, whereas life expectancy from the onset of agriculture was
estimated to be relatively stable at around 20–25 years until the onset of industrialization in about 1800 (Eaton et al. 2002a, b).

The dawn of dietary complacency

A major benefit of industrialization appears to have been the extension of life expectancy. This is generally attributed to better food security and sanitation. There is, however, a possible downside, as incidence of degenerative disease increased. Longer life expectations and lack of exercise doubtless contributed to this, but the link with dietary change is also strong. As our social structures became more sophisticated, Westernized countries became increasingly distant from our food supplies. Most recently, with the globalization of markets, we may have surrendered control to the food industries. In the process, we seem to have forgotten that food and water are the physical basis for our survival.

With the onset of industrialization from around 200 years ago, it seems, from the perspective of what we eat, that a miracle must have occurred, because, irrespective of how much salt, saturated fat, hydrogenated fats, and refined sugar we started eating, we seemed to assume it had no implications for our well-being. Was this the ascent of man from primordial soup to the couch potato? Can it really be assumed that our nutrition was crucial in our evolution but has no relevance now? Or could something as simple as paying more attention to what we eat potentially be a major resource in promoting human well-being?

Diet and physical well-being

Food inequality in developing countries

No species can flourish without adequate food and water. Sadly, for many of the world’s population, this is the cruel reality as, according to the UN Food and Agriculture Organization (FAO 2003) report, there were 840 million people in the world who did not have enough to eat. The laudable target set at the millennium World Food Summit to halve global hunger by 2015 is already off target and, according to the FAO, the present rate of decline of hunger will need to be increased 12-fold to reach that target (FAO 2003). Indeed, the overall decrease in global hunger is largely due to improvements in China. The scale of the Chinese achievement obscures the fact that hunger has got worse in 47 countries (Short 2002).

Diet-related low birthweights affect approximately 30 million children born each year, which has implications for their mental and physical development as well their survival (WHO 2002; de Onis et al. 1998) and chronic diseases later in life (e.g. Barker 1995; Barker et al. 1989, 2001). For the two-thirds of the world’s absolute poor who still do not have food security, there is an irrefutable case that better food would promote their physical and social well-being. The hunger of these people puts our global economic culture into perspective. How precious even the most
humble offer of food and water must appear when faced with such tragic circumstances. The World Health Organization (WHO) has published perhaps the definitive studies on changes in global dietary practices and, according to the WHO (2003, section 2.2),

Hunger and malnutrition remain among the most devastating problems facing the majority of the world’s poor and needy people, and continue to dominate the health of the world’s poorest nations. Nearly 30% of humanity is currently suffering from one or more of the multiple forms of malnutrition (WHO 2000). The tragic consequences of malnutrition include death, disability, stunted mental and physical growth, and as a result, retarded national socioeconomic development. Some 60% of the 10.9 million deaths each year among children aged under five years in the developing world are associated with malnutrition.

If the anthropological perspective on our dietary origins is correct, it could be predicted that patterns of nutritional deficiencies seen nowadays would be related to our movement away from our historic coastal and lakeside food sources. It is noteworthy that the most important nutrient deficiencies globally are nutrients that are typically richest in shellfish and seafood. Iodine deficiency is estimated to affect more than 700 million people and is considered the greatest single preventable cause of brain damage and poor cognitive function (WHO/UNICEF 1999; Driutskaya and Riabkova 2004). It is estimated that 250 million young children are affected by subclinical vitamin A deficiency, which is considered the greatest preventable cause of childhood blindness and increased risk of infections and death (WHO/UNICEF 1995). Over 2000 million people are estimated to suffer from anaemia (WHO/UNICEF 2001). Unfortunately, dietary standards have only recently considered deficiencies in fatty acid intake and its sequelae.

In addition to food insecurity, many developing countries are experiencing rapid changes from traditional diets. These changes typically reflect trends towards Western dietary practices of high saturated fat and energy-dense foods. Increasingly, malnourishment persists with rapidly emerging incidence of chronic disease attributed to modern diets—so much so that by 2020 it is projected that, in the developing countries, diet will account for 71% of mortality from ischaemic heart disease and 75% of deaths due to stroke. Diabetes is projected to increase from 84 million in 1995 to 228 million in 2025 (Aboderin et al. 2001). On a global basis, 60% of the burden of chronic diseases will occur in developing countries.

If there are ways of ensuring that the typical circumstances of an entire population can be induced to favour distinctly positive health, surely finding a more equitable method of food distribution must be among them.

**Food excess in Western countries**

Western diets in particular have increasingly focused on energy-dense foods like saturated fats and sugar. According to the WHO (2003), ‘the increasing Westernization, urbanization, and mechanization in most countries around the World is associated with changes in diets towards one of high-fat, high-energy foods and sedentary lifestyles.’
Obesity

Obesity is a function of both energy intake and exercise but the evidence linking it to energy-dense foods is compelling. For instance, a meta-analysis of 16 studies testing high-fat versus low-fat diets concluded that a 10% reduction in fat intake over typically 2 months resulted in a loss of around 3 kg in body weight (Astrup et al. 2000). Similarly, consumption of energy-dense sugary drinks is associated with increased weight when examined by cross-sectional, longitudinal, and cross-over studies (Tordoff and Alleva 1990; Harnack et al. 1999; Ludwig et al. 2001). China has just reported that the numbers of obese people in its population has doubled to 60 million between 1992 and 2002, almost equalling the numbers in America in 10 short years. Obesity and depression have been reported as a major factor in maternal death in Britain (Brettingham 2004). Even economically poor countries are witnessing a disturbing increase in incidents of childhood obesity (de Onis and Blössner 2000). The best known comorbidities of obesity are diabetes and heart disease.

Diabetes

Diets high in saturated fats are an important risk factor for diabetes (Tuomilehto et al. 2002; Knowler et al. 2002). Australian Aboriginals have an inherent tendency to insulin dependence but this only seems to manifest itself when they consume Western diets (O’Dea 1991).

Cancers

WHO (2003) estimates that only a negligible proportion of the world’s population consumes the recommended intakes of fruit and vegetables, which is an important part of dietary variation. Diet is estimated to account for 30% of cancers in industrialized countries (Doll and Peto 1996) and 20% in developing countries (Willet 1995). Epidemiological studies provide circumstantial evidence that incidence of cancers follow the nutritional transitions of Latin America, Africa, North America, and Asia (Popkin et al. 1993).

Heart disease

Evidence of the role of saturated animal fats in cardiovascular disease is strong and has been demonstrated in animal experiments, epidemiological studies, and clinical trials in various populations (Kris-Etherton P et al. 2001). Heart rate variability is considered to be a good indicator of heart condition where beat-to-beat variations of heart-beat are measured using spectral estimation techniques (DePetrillo et al. 1999). Lowered heart rate variability has been associated with major depressive disorders (Carney et al. 1995; Tulen et al. 1996), minor depression (Carney et al. 1995), and ‘depression self-rating’ in non-depressed subjects (e.g. Krittayaphong et al. 1997).
Processed foods

Other aspects of modern processed diets have also been found to be highly questionable from a health perspective such as trans fatty acids from the industrial hardening of oils and increasingly from high-temperature fried foods. This process introduces hydrogen into polyunsaturated oils, resulting in a more extended fatty acid carbon chain similar to that of saturated fatty acids. This de-odorizes the oil and improves shelf life: qualities that are attractive to the food industry. Unfortunately, there is no evidence to suggest any health benefits from consuming trans fatty acids—quite the contrary. Since the early 1990s evidence has suggested that trans fats adversely affect fetal and infant growth by interfering with the biosynthesis of AA and DHA, the main structural fats in the central nervous system (Koletzko 1992; van Houwelingen and Hornstra 1994) and more recently in preschool children (Innis et al. 2004). Trans fats have also been shown to increase the rate of heart disease in large cohort studies (Oomen et al. 2001; Willett et al. 1993). The US Food and Nutrition Board (2002) famously suggested a ‘tolerable upper intake level’ of zero! High sodium intake is also a matter of concern in Western diets as a major risk factor associated with high blood pressure. This relationship has been demonstrated experimentally, by epidemiological studies, controlled clinical trials, and in population studies on restricted sodium intake (Gibbs et al. 2000).

The positive benefits for health of better diets

The positive flip side of this evidence implies that eating a better diet would be protective from these chronic diseases and certainly that would be the position argued by WHO. A meta-analysis of 32 trials (Cutler et al. 1997) concluded that a daily reduction of sodium intake reduced systolic and diastolic blood pressure in those with high blood pressure. For instance, studies that reintroduced dietary factors that would be plentiful in the Palaeolithic diet, such as fish oils and vitamin E, found a reduction in mortality by cardiovascular disease of 30% after a 3.5 year follow-up (GISSI–Prevenzione investigators 1999). Fish consumption correlates as a protective factor against chronic diseases in China (Wang et al. 2003). Double-blind placebo-controlled clinical trials of omega-3 fatty acids have demonstrated improved heart rate variability of adult subjects given fatty acids during 24-hour monitoring (Christensen et al. 1996, 1997, 1999). There is evidence that fish oils provide a protective effect in certain groups of cancers such as breast cancers (Palakurthi et al. 2000), an illness that is more common in industrialized countries. Numerous studies have shown that the consumption of fruit and vegetables, and in some cases pulses, is protective against cancers (World Cancer Research Fund 1997). Zinc supplementation has been shown in a double blind trial to reduce childhood mortality in small for gestational age infants (Sazawal et al. 2001).
A need for balance

If we are seeking to raise many of the world population from the shadow of chronic disease to distinctly positive health, then it is arguable that finding a dietary middle-way between the extremes of starvation and excess should be a priority.

Healthy food choices

At the same time that there is concern that we are eating too much sugar and saturated and trans fats, we also do not seem to be eating enough of the healthy foods such as fruits and vegetables. According to the US Centers for Disease Control (CDC 2002), for instance, 79% of US high school students had eaten less than the recommended five servings of fruits and vegetable a day.

Even the nutritional qualities of the healthy staple foods may also have altered. A comparison (Mayer 1997) of the nutritional values of fruit and vegetables first published in 1936 in the seminal work, *The nutritive value of fruits, vegetables and nuts* (McCance et al. 1936), with revised figures published in the fifth edition (Holland et al. 1991) showed there was significantly less calcium, magnesium, copper, and sodium in vegetables and magnesium, iron, copper, and potassium in fruit. The reason given for the update was because ‘the nutritional value of many of the more traditional foods has changed’. A comparative study highlighted changes to national diets in the UK in the last 50 years. A comparison of diets consumed by 4599 4-year-old children in 1950 was made with 493 children from the 1992/93 National Diet and Nutrition Survey. The authors (Prynne et al. 1999) concluded that the post-war diet, with its reliance on staple foodstuffs such as bread and vegetables, might well have been beneficial to the health of young people. Thus, the increase in food choice we hold dear nowadays may not necessarily be beneficial from a nutritional perspective. With the availability of such a variety of healthy foods to choose from today it is intriguing to consider why we do not manage to make better food choices. A possible answer is that, typically, it is the energy-dense foods that are often heavily promoted so perhaps this is driven by conscious choices. After all, the food industry has invested a great deal of effort in packaging foods to be attractive (Nestle 2003). Nestle (2002) characterises the food industry claims as ‘there is no such thing as good or bad food [except when their products are considered good]’. Alternatively, it has been proposed that energy density influences energy intake due to weak satiety signals that fail to compensate for very energy-dense foods (Prentice and Jebb 2004). It could be a matter of taste. Our sense of taste is influenced by zinc status for instance (Bryce-Smith and Simpson 1984). The addition of sugar, salt, and fats results in very stimulating foods that may be more palatable if the sense of taste is less sensitive. A survey found that only 56% of the US population had adequate zinc intakes (Breifel et al. 2000). According to the International Bibliographic Information on Dietary
Supplements (IBIDS) database of the Office of Dietary Supplements (ODS) at the National Institutes of Health, 98% of zinc is removed when sugar is refined, a common feature of modern dietary practices.

Another possibility is that high fat and sugar diets influence our brain chemistry directly and may have addictive qualities. While researching factors in obesity, animal studies suggest that mu opioid stimulation enhances the appetite for high fat and sugar consumption (Zhang and Kelley 2000) and vice versa. High glucose intakes of 25% added to feeding chow (Colantuoni et al. 2001) have been shown to stimulates mu-1 and dopamine D1 receptors much like drugs of abuse. It is argued that there may be a genetic adaptation towards energy-dense foods that developed at a time before we had unlimited supplies of these foods (Kelley et al. 2002). When animals are intermittently withdrawn from sugar intakes of 25% they exhibit signs of withdrawal (Colantuoni et al. 2002). So far, such findings do not seem to have been replicated on human populations. Nevertheless, breakfast cereals can contain 40% sugars and some drinks contain 100% of calories as free sugars.

This brings us back to the disparity between the estimates of the Palaeolithic diet where free sugar intakes are estimated to be far lower than in modern times. If our physiology and genetic make-up is more fully adapted to the diet of the last 50 000 years rather than that of the last 200 years as has been suggested (Cordain et al. 2000), we need to take notice, because our dietary baselines seem to have shifted considerably. These changes may map out into our behaviour. Following the publication of the US Food and Nutrition Board statement (2002) suggesting that we minimize consumption of trans fats, evidence has emerged from an animal study that dietary trans fatty acids may act on the endogenous neurotransmitter levels during brain development (Acar et al. 2002). Since these same neurotransmitter pathways are also implicated in mental illnesses such as schizophrenia (Davis et al. 1991), we are presumably left to guess if the introduction of trans fats has similarly affected human brains and if there are any consequent mental health implications. The fact is we don’t know and it should be a concern that evidence emerges after the widespread introduction of such dietary changes. Are the improved keeping qualities of hydrogenated oils really a good trade-off for the functionality of our brains?

While the adoption of such unhealthy food products is doubtless profitable, perhaps it would be a good idea to systematically test new food types for their implications on our brains before introducing them.

**Diet and mental health**

It is curious that many of the authoritative reference works on diet and international dietary standards focus on the health issue but hardly mention mental health. Unless we still believe that mind and body are separate, we might expect that what we eat would influence brain function if only because of its high metabolic activity. The blood–brain barrier protects the brain’s nutrient supply, but it is not invulnerable.
Animal studies suggest that the brain’s composition does not change as quickly in response to diet as the liver for instance, but smaller changes were noted nevertheless (Crawford et al. 1976). Many of the nutrients supplied to the brain are classed as essential. Indeed, diseases of nutritional deficiencies such as pellagra can present as mental confusion and delusions that can give the appearance of mental illness. Increasingly, there are good reasons to believe that the health benefits of good nutrition extend into effects on mental health. This brief discussion will touch on some of the evidence that nutrients can also positively impact on mental illness, perhaps holding out the possibility of a degree of prevention in the future.

Patterns of mental illness appear to be similar to the cross-national patterns of chronic disease in that, broadly, they are increasing in prevalence and appear to follow modern dietary practices, but it is recognized that we are only gradually beginning to appreciate what is a very complex picture.

Depression
A multinational epidemiological study that examined incidents of depression and schizophrenia found strong correlations with national dietary practices (Peet 2004). The most striking finding was that sugar consumption was strongly associated with worse outcomes in schizophrenia and prevalence of depression, while fish consumption was inversely associated with depression. Like any epidemiological data, these findings have to be interpreted with care. Such data cannot demonstrate a causal relationship but the strength of the correlations does, nevertheless, strongly suggest a need for follow-up clinical trials. Evidence is converging from treatment studies to indicate that the same nutrients that would have been plentiful in estimated Palaeolithic diets are protective against depression. The fish oil EPA was found highly effective in a case of severe treatment-resistant depression (Puri et al. 2001, 2002) and randomized controlled trials have confirmed that it can significantly reduce symptoms of unipolar depression (Nemets et al. 2002; Peet et al. 2002) and similar benefits have been found for omega-3 treatment in bipolar mood disorder (Stoll et al. 1999). Peet demonstrated that a one-gram dose of EPA was more effective than a larger dose (Peet and Horrobin 2002) in treating depression in patients who remained depressed despite adequate medication. This suggests that there are optimal dosages, which is reassuring from an evolutionary perspective as moderate intakes are more easily achievable on a daily basis. In addition to consuming less omega-3 fatty acids nowadays, it has also been noted that we may not eat sufficient green vegetables, a good dietary source of folate (Gerrior and Zizza 1994). There is strong evidence of the protective relationship between folate intake and depression, as set out in a Cochrane review (Taylor et al. 2003). A number of clinical studies have found low zinc status in depressed patients compared to controls (e.g. Maes et al. 1994, 1997; McLoughlin and Hodge 1990). Furthermore,
the severity of unipolar depression was negatively correlated with the serum level of zinc. A pilot randomized controlled trial of 14 patients with unipolar depression on antidepressant medication found that those who received 25 mg zinc daily significantly augmented the reduction in Beck Depression Inventory (BDI) scores by 40% after 12 weeks of treatment when compared with placebo supplementation (Nowak et al. 2003). The authors appropriately argued that this should be followed up with larger trials.

**Schizophrenia**

There is a history of treating schizophrenia with various dietary interventions but, sadly, this has received little attention until comparatively recently. Within schizophrenia there is considerable heterogeneity, so there is a need for caution in generalizing about this condition but abnormalities of fatty acid and phospholipid metabolism have been consistently reported, including excessive activity of PLA2 enzymes, increased lipid peroxidation, and nuclear magnetic resonance (NMR) spectroscopy evidence of increased brain membrane phospholipid turnover (Peet et al. 1999). Controlled trial treatments with omega-3 fatty acids have been shown to be effective in reducing hallucinations and delusions as well as anhedonia, inattention, or lack of volition, in both medicated and unmedicated patients (Puri et al. 1998; Peet et al. 2001). In patients given 2 g/day EPA there were improvements on the positive and negative affect scale (PANAS) and its subscales, but there was also a large placebo effect in patients on typical and new atypical antipsychotics and no difference between active treatment and placebo. In patients on clozapine, however, there was little placebo response, but a clinically important and statistically significant effect of EPA on all rating scales. This effect was greatest at 2 g/day rather than at a higher dose of EPA (Peet et al. 2002). This suggests that there are optimal dosages and that nutrients can augment the effectiveness of drugs. Clinical improvements following treatment with EPA have also been shown to correlate with improved blood fatty acid status, reduced membrane phospholipid turnover, and reversal of cerebral atrophy (Puri et al. 2000; Richardson et al. 2000). Negative results have been reported in only one of five controlled trials to date where a 3 g/day intervention was used (Fenton et al. 2001). These data on fish oils indicate that it is EPA rather than DHA that is effective but it has to be recognized that these are relatively small studies and more large-scale clinical trials are required, particularly as this is a low-risk approach.

Overall, these findings are highly encouraging but a great deal more research is needed in this area, particularly focusing on the possibility that a better diet is protective from such illness. The World Health Organization is predicting a 50% rise in child mental disorders by 2020. This strongly suggests that the relationship between diet and mental health should be added to formal considerations of dietary adequacy.
Nutrition and behavioural well-being

It may be possible to extrapolate from the health benefits of diet to our behaviour. The primary concern will be to focus on the possibility that diet can influence behaviour directly and hence that dietary interventions may even be possible for behavioural disorders.

Childhood developmental disorders

Evidence is emerging that diet may be linked with childhood developmental disorders. Many of the features associated with attention deficit hyperactivity disorder (ADHD), dyslexia, dyspraxia, and autistic spectrum disorders are consistent with lack of or imbalances in highly unsaturated fatty acids (Richardson and Ross 2000). Fatty acid abnormalities could also help to account for some of the key cognitive and behavioural features of these conditions, such as anomalous visual, motor, attentional, or language processing, as well as some of the associated difficulties with mood, appetite or digestion, temperature regulation, and sleep (Richardson and Puri 2000). Reviews of clinical and experimental studies support the idea that lack of highly unsaturated fatty acids (HUFA) may play a role in these overlapping developmental conditions (Richardson and Ross 2000). Thus, physical signs of fatty acid deficiency, such as excessive thirst, frequent urination, rough, dull, or dry hair and skin, and soft or brittle nails, have been clearly linked with ADHD, dyslexia, and autistic spectrum disorders, as have reduced blood concentrations of HUFA and iron (Konofal et al. 2004).

There are relatively few controlled trials to test the effects of fatty acids on ADHD. One of the first involved evening primrose oil (providing the omega-6 fatty acid, gamma-linolenic acid (GLA)), but this showed little clear benefit (Aman et al. 1987; Arnold et al. 1989). With the accumulation of evidence that it is the omega-3 fatty acids that may be washed out in modern diets, emphasis shifted to fish oils (providing both EPA and DHA), which have been demonstrated to reduce behavioural and learning difficulties in both ADHD and dyslexia (Burgess 1998; Richardson and Puri 2002). However, supplementation with pure DHA was twice found to be ineffective in ADHD (Voigt et al. 2001; Hirayama et al. 2004). A further pilot randomized study of effects of fatty acids on children with inattention, hyperactivity, and other disruptive behaviours included biochemical analyses that demonstrated that both the omega-3 fatty acids and vitamin E were correlated with reductions assessed by the disruptive behaviour disorders (DBD) rating scale. Vitamin E is commonly added to fish oil as an antioxidant but was not always declared as an active constituent in earlier studies using EPA and DHA. Thus, the results of this pilot study suggest the need to declare it as an active ingredient and for further research with both n-3 fatty acids and vitamin E in children with behavioural disorders (Stevens et al. 2003). Zinc has been shown to be an effective adjunct to methylphenidate in treating ADHD at 15 mg/day in a randomized
study of 44 children (Akhondzadeh et al. 2004). Zinc at 40 mg/day has been shown to be an effective monotherapy for ADHD in a randomized controlled trial of 400 Turkish children (Bilici et al. 2004). Although this dosage is above physiological requirements, Turkish children of lower socio-economic status have been shown to have significantly lowered zinc status (Tanzer et al. 2004).

**Linkage of heart and brain function**

These findings are consistent with evidence from the treatment of depression and schizophrenia that EPA, not DHA, may be the key omega-3 fatty acid in functional disturbances of attention, cognition, or mood. This in itself is surprising as DHA, not EPA, is found in the brain. One alternative is that EPA may improve brain blood flow through inhibition of cyclooxygenase and vasoconstrictive eicosanoids (Ellis et al. 1992). More recently it has been found that the phospholipase A2–arachidonic acid pathway and 20-hydroxyeicosatetraenoic acid production may have a regulatory role in cerebral blood flow (Mulligan and MacVicar 2004). This blood flow carries the brain’s energy supply, which may constitute a limiting factor to the brain’s information-processing capacity (Peppiatt and Attwell 2004).

Certainly there is a body of literature that suggests that heart function affects brain function and behaviour; this will be discussed below. It is equally possible that the therapeutic effects of fatty acids are influenced by the dietary intakes of other nutrients such as vitamin E (Stevens et al. 2003) or zinc as these nutrients appear to interact with fatty acid metabolism (Bekaroglu et al. 1996; Wauben et al. 1999).

These data nevertheless suggest that there is a plausible relationship between diet and a range of developmental conditions with clearly described associated behaviours. More research is required to delineate that relationship. If diet is a protective factor from these disorders then it can only be hoped that such requirements are eventually taken into account when assessing standards of dietary adequacy.

**Sugar and behaviour**

If the amount of energy supplied to the brain puts a limit on its information-processing capacity, it might be deduced that high sugar consumption would be helpful to brain performance. There have been a number of randomized studies conducted to assess the behavioural and cognitive effects of sugar. An experiment using a counterbalanced Latin square design involving 48 children, compared the behavioural effects of sugar, aspartame, and saccharine as a placebo in blind dietary sequence changes every 3-weeks. The study concluded, ‘Even when intake exceeds typical dietary levels, neither dietary sucrose nor aspartame affects children’s behaviour or cognitive function’ (Wolraich et al. 1994). A study of 48 children may not, however, be a strong basis on which to form such robust conclusions. Small studies do not have the numbers for appropriate randomized control for broader extrapolation and may lack the power to detect smaller effects that may be cumulative in the
population at large. A literature review acknowledged that ‘Sugar clearly does not induce psychopathology where there was none before, but it may on occasion aggravate an existing behaviour disorder’ but again the largest study quoted was 76 selected children (Kinsbourne 1994). To further illustrate the complications of dietary studies, the Wolraich study used vitamin C and riboflavin to assess compliance in the active cells at dosages comparable to those reported in the literature to improve cognition and behaviour (e.g. Benton et al. 1995; Heseker et al. 1995). A recent dose-ranging study of 67 adult students (Flint and Turek 2003) with normal glucose metabolism suggests that large doses of glucose that increase blood glucose levels do not influence attention, but that a moderate dose (100 mg/kg) of glucose selectively impairs measures of impulsivity or disinhibition. Furthermore, the glycaemic index (broadly how quickly the carbohydrate can be utilized) of carbohydrate sources also appears to influence cognitive effects in human and animal studies (Benton et al. 2003). Refining of sugar removes many of the micronutrients required in carbohydrate metabolism; hence the term empty calories. It has been argued that the lack of these micronutrients is the reason that behaviour might be affected by sugar consumption (Schoenthaler 1994). Given the pervasive use of refined sugar in modern diets and its interactions with health concerns, there is a strong case for large randomized studies to investigate sugar and behaviour in more detail.

**Mood**

While many of us might be ostensibly healthy, we may wonder if taking better care of our diet would enhance our mood. Researchers have examined the effects of diet on mood in apparently healthy subjects but, as with other areas reported, the effects may depend on the dietary baseline, i.e. the quality of the exiting diet. Such factors may be regional. Intakes of many trace elements are influenced by the soil types from which the food originates; for example, UK soil is low in selenium. In a study of 11 healthy men who where fed either a low or a high selenium diet for 15 weeks, raising selenium intakes beyond the typical US recommended dietary allowance (RDA) of 70–356 mg had no measured effect on mood but low baseline erythrocyte selenium correlated with changes in hostile–agreeable and depressed–elated scales using the profile of mood state (POMS)-B10 assessment subscales (Hawkes and Hornbostel 1996). The authors concluded that, because US dietary selenium baselines are higher than in the UK, these findings were consistent with a UK double-blind experimental study (Benton and Cook 1991). Here 50 subjects significantly decreased ratings of anxiety, depression, and tiredness over 5 weeks with 100 mg of selenium per day. This was particularly true of those subjects who had estimated intakes of 28–62 mg per day, while those in the higher intake ranges of 63–280 mg per day showed less change in mood. It is noteworthy that the lower range is more typical of UK dietary selenium intakes (MAFF 1997).
Similarly, low iron status is relatively common and may contribute to lethargy. Studies of athletes suggest that the lack of energy that results from iron deficiency anaemia can be a serious disincentive to exercise (e.g. Nielson and Nachtigall 1998). It should be noted that nutritional regimes are routinely applied with training to enhance athletic excellence. Other factors of the diet that have been considered in relation to mood are the intakes of carbohydrates. Benton and Donohoe (1998) assert that carbohydrate intake is associated with improved mood, while poor mood stimulates the eating of ‘comfort foods’ such as chocolate. Carbohydrate metabolism predicted results in tests of memory and cognition in healthy subjects (Donohoe and Benton 2000). The specific mood response to carbohydrate intake is complex as this has been shown to be influenced by many aspects of the dietary baseline: when food is consumed; the time delay between meals; the amount consumed; the fibre content; the glycaemic index of the food; the level of accompanying protein (Benton 2002; Benton et al. 2003).

The relationship between diet and mood has so far not benefited from the considerable international investment to investigate the relationship between diet and health. Mood reflects how well we feel in ourselves and hence the quality of life experienced. This would also be a worthy area to add to the considerations of dietary adequacy.

**Diet and antisocial behaviour**

A question remains, however, as to whether diet might affect behaviour to which we assign free will. This is a common underlying assumption in the criminal justice system, so this discussion will focus on criminal and antisocial behaviour.

Firstly, let us define what we mean by crime and the broader category of antisocial behaviour. Central to any form of criminal justice is the notion that culpability can be attributed. Culpability is distinct from simply establishing guilt as the degree of individual liability is judged in relation to an action. The classical form of justice assumes that man is an agent of free will and can choose to commit an offence; hence culpability can be fully attributed. Crime is judged in relation to a body of criminal law that sets out offences, that is, acts for which a legal penalty will apply. Straightforwardly, a crime is deemed to have been committed when one of these laws is judged to have been broken. When other forms of social rules are judged to have been broken, such as within an institution, it will be described as antisocial behaviour. While this serves as a useful distinction, it is recognized that in both cases it is assumed that the perpetrator would know that such behaviours attract a sanction, albeit of differing severity.

A dramatic illustration of a possible relationship between diet and behaviour is the following diet consumed by a persistent criminal offender. He had been sentenced by UK courts on 13 occasions for stealing trucks in the early hours of
the morning and on the last three occasions he was imprisoned. This was all he ever ate.

- Breakfast: Nothing (asleep).
- Mid-morning: Nothing (asleep).
- Lunch time: 4–5 cups of coffee with milk and 2.5 heaped sugars.
- Mid-afternoon: 3–4 cups of coffee with milk and 2.5 heaped sugars.
- Tea: Fries, egg, ketchup, and 2 slices of white bread; 5 cups of tea or coffee, with milk and 2.5 heaped sugars.
- Evening: 5 cups of tea or coffee, with milk and 2.5 heaped sugars; 20 cigarettes; £2 worth of sweets, cake, and (if money available) 3–4 pints of beer.

The court was exasperated by his behaviour because on release from prison he had chosen to steal a truck to return home! Notice that there are no fruits or green vegetables whatsoever. There are no obvious sources of omega-3 fatty acids. There are at best modest sources of protein and fibre. Much of the calories come from sugary drinks and cake. Consider how you might feel if you lived on his diet for 3 months. With the approval of the court he was given dietary education to improve his eating habits. He eventually trained as a chef. His probation officer reported that he had not re-offended in 15 years.

**Nutritional status of offenders**

National dietary surveys have not to date differentiated between offenders and non-offenders, so the possibility remains that the diets of offenders are somehow atypical. The most accurate method is to determine this is by assessments of nutritional status from blood. A study that compared the nutritional status of offenders with that of non-offending controls found no significant differences in nutritional status. However, there were differences in sugar metabolism (Gans *et al*. 1990). This needs wider population assessments, as such conclusions cannot be taken as typical given regional variations in diet. It would be a reasonable point to make that there must be criminals who consume a healthy diet. Perhaps so, but since national dietary standards were never established with brain function or behaviour in mind, we really don’t know yet if a diet is adequate from the perspective of the brain.

**Reinterpreting crime trends**

If nutrition plays a role in our behaviour, then effects from nutrition would not only have to be in force within individuals but presumably should be capable of helping shape patterns of social behaviour. In the case of crime, these changes are considerable over the last century. Using the UK as an example, in 1900 there were 77 934 offences reported which rose to 5 170 843 in 2000, albeit reaching a peak in 1992, followed by a decline during the 1990s and then a levelling off.
Even when adjusting for population, notifiable offences (broadly more serious) rose almost 10-fold per person in the UK from the 1950s to the mid-1990s (Home Office 1997). Rapid changes in reported crime, as illustrated in Fig. 7.1, would be difficult to explain in terms of genetics for instance. There is no doubting the relevance of other changes such as socio-economic factors, legislation, and police practice in these trends. Nevertheless, the scale of change over the decades is considerable. Taking the foregoing into account, a scholarly review of such trends (Rutter et al. 1998) commented, ‘The overall rate of crime has risen severalfold over the last fifty years, a rise so rapid that it can only be due to some impact in the environment. Clearly if society has been so spectacularly successful in causing the levels of crime to increase there must be the potential for the right sort of interventions to be equally effective in causing it to decrease!’

One element of this might be that it is our brain’s environment that is being impacted. We have after all already considered a range of changes that have occurred in modern diets. There is evidence that violence is influenced by genetic susceptibility factors (e.g. Caspi et al. 2002). These effects may themselves be influenced by diet as there is a growing awareness of nutrient-gene interactions. Zinc for instance has been shown to be involved in the regulation of gene expression (Dieck et al. 2003; Blanchard et al. 2000) and maintaining the configuration of mammalian gene transcription proteins (e.g. Hanas et al. 1983; Vallee et al. 1981; Dieck et al. 2005). In addition, exposure to lead (a nutrient antagonist) correlates with increased delinquency (Needleman et al. 1996) and this may have increased in the UK during this period until the introduction of unleaded fuel in the early 1990s (e.g. Thomson et al. 1989). It has to be recognized, however, that such historical data have limitations and the connection with changing social patterns going back many decades is speculative. There is, however, more direct evidence that raises the possibility that such influences could have increased their grip at a time when social behaviour has deteriorated.
Evidence linking diet and antisocial behaviour

A comparison of conventional and nutrition education approaches to rehabilitating 102 offenders over 12 months in the community found that the re-offending rates (11.9%) of the group given nutrition education were almost a third of that of the controls (33.8%) receiving conventional probation programmes (Schauss 1978). An experimental study of 3399 imprisoned juveniles, where refined and sugary foods, snack foods, and drinks available to the inmates were replaced with unsweetened fruit juices and popcorn, found that, over a 12-month period, there were 21% fewer serious antisocial acts, a 25% reduction in assaults, a 100% reduction in suicides, and a 75% reduction in the use of restraints (Schoenthaler 1983). This study lacked randomized allocation or placebo control. Schoenthaler subsequently conducted a placebo-controlled double-blind randomized experimental trial using nutritional supplements based around the US RDA of minerals and three times the US RDA of vitamins on 62 13–17-year-old male and female incarcerated juveniles. The active group committed 28% (15–41%, 95% confidence interval (CI)) fewer rule violations compared to controls \( (p = 0.005) \) (Schoenthaler et al. 1997). It was reported that this effect was most marked in 16 subjects who had significantly improved their blood vitamin status during the trial from a low baseline of the following vitamins: C, thiamin, niacin, pantothenic acid, pyridoxine, and folate. A further randomized placebo-controlled study of 468 school children aged 6–8 years was conducted using a food supplement formulation of 50% of the US RDA of vitamins and minerals. Of these children, only 80 had a school discipline record at baseline. It was reported that antisocial incidents fell significantly \( (p < 0.02) \) by 47% (29–65%, 95% CI) for those 40 children on active supplementation who had a discipline record at baseline (Schoenthaler and Bier 2000). There are more appropriate statistical methods to model these data to deal with the floor effect using a Poisson regression. However, the broad conclusion that significant improvements in behaviour can also be seen in younger children from a simple dietary adjunct should be followed up. Hamazaki et al. (1996) found that Japanese university students undertaking exams while receiving supplemental DHA on a randomized double-blind basis did not show an increase in hostility during a frustration test, while those on placebo increased hostility. This effect was however, not observed under non-stressful conditions (Hamazaki et al. 1998). A randomized placebo-controlled pilot study of 30 women with borderline personality disorder found that EPA significantly reduced ratings of aggression and depression compared to controls (Zanarini and Frankenburg 2003). While these experimental studies offer broadly encouraging results, they need to be replicated with larger randomized designs.

With the co-operation of the UK Home Office an empirical study was conducted to test if poor nutrition is a cause of antisocial behaviour (Gesch et al. 2002). The study was simply designed to provide a powerful test for a general effect on behaviour, where statistical power was reported as 92% to detect a change in the
A prison was chosen as all sources of food are known. It was found that, typically, prisoners did not make appropriate food choices resulting in many of their dietary intakes falling below UK government dietary standards (Eves and Gesch 2003). Using a double-blind placebo-controlled randomized stratified design, 231 young adult prisoners (18–21 years of age) were studied to see whether nutrient supplementation had any influence on proven disciplinary offences committed by the prisoners. The active nutritional supplements provided broadly the daily adult requirements of vitamins, minerals, and omega-3 and -6 essential fatty acids (see Gesch et al. 2002). On an intention to treat basis compared to placebo, those who received active nutritional supplements committed an average of 26.3% (8.3–44.33%, 95% CI) fewer offences ($p = 0.03$, two-tailed). This analysis included everyone recruited to the trial including those who had participated for as little as 3 days but the average was 5 months (see Fig. 7.2).

Having rejected the null hypothesis on an intent to treat basis in a between-groups comparison, a more sensitive test for the actual effect of treatment (in this analysis, each individual becomes their own control as we have established that both groups are matched at baseline by rates of offending) was conducted for the effect on those taking active supplements for a minimum of 2 weeks ($n = 172$) (see Fig. 7.3). The result was an average 35.1% reduction of offences (95% CI 16.3–53.9%, $p < 0.001$, two-tailed), whereas those taking placebos remained within standard error, proving no evidence of change. The greatest reduction occurred for the most serious incidents (including violence) dealt with by governor reports. Based on an analysis of 338 governor reports, the active group committed 37% (11.6–62.4%, 95% CI) fewer governor reports ($p < 0.005$, two-tailed), whereas the placebo group remained within standard error ($p > 0.1$, two-tailed) at 10.1% fewer governor reports ($-16.9–37.1\%$, 95% CI), again providing no evidence of change.
Thus, with placebos there was no evidence for a reduction in offending over time spent in the prison, while, in marked contrast, those receiving active nutritional supplements committed significantly fewer disciplinary offences over the same period.

The differences in the rate of offending in the prison could not be explained by ethnic or social factors, or variations in the administration of governor reports, or opportunity to offend as they should have been controlled for by the randomized design: the compliance of both randomly allocated groups was closely matched as were all baseline measures of behaviour and diet, so it had to be the nutrients in the capsules that caused the change in behaviour. The issue then is the nutritional quality of the existing dietary baseline. The dietary baseline had been modified (the independent variable) using nutritional supplements. The supplements were really an analogue for a better diet but they have the advantage that the nutritional value is precisely known and allowed for the use of placebo. Ideally, such improvements in nutritional intake should come from the diet (for a more detailed discussion, see Eves and Gesch 2003). The dosages provided were physiological rather than high dose and might be readily achievable with even a modest increase in expenditure on diet coupled with dietary education.

Given the wide implications, these findings need to be widely replicated. The participants did not guess accurately what sort of capsules they had been given either, so the importance of Bryce-Smith’s idea becomes clear; here we have a potent effect on behaviour that can be measured but not sensed. The findings are all the more surprising because this age group of prisoners has proved to be notoriously resistant to behavioural improvements (Kershaw 1999). A limitation of the study was that biochemical measures were not available; they will be required in any replication to explore the utilization of nutrients and also mediating mechanisms. Thus, having empirically demonstrated an effect on antisocial behaviour, we
are only at the start of understanding the potential of this intervention. It is quite possible that, with a better understanding of the range and balance of nutrients required, the protective effect could be further improved.

**Health implications of an adequate diet**

It should come as no surprise that ensuring the prisoners’ diets reached UK government dietary standards did not result in adverse reactions. Indeed, it could be argued that the real experiment was the ongoing default position where prisoners were not reaching these dietary standards. The only referral for a possible adverse event was a young man who was surprised that his evacuations had increased in frequency from once a fortnight to once every 2 days: the prison doctor rightly considered this an indicator rather than an adverse event! Here we have criminals in a maximum-security institution not appreciating what their bodies are telling them. Some of the participants had not heard of vitamins, let alone knew which foods contained them; they were not even remotely equipped to make healthy food choices. Being pragmatic, a combination of supplementation to reinstate nutrients first coupled with dietary education may be prudent in such populations. Notwithstanding the seriousness of their crimes, many of them took the trouble to express gratitude for being involved in the trial.

**Implications of a protective effect on behaviour**

*These findings suggest that diet can affect behaviour to which we assign free will.* Indeed, it is difficult to see how free will could be exercised without involving brain function. Furthermore, these effects may be far more potent than we realized. This raises the question as to what life would have held for these 231 young men if they had grown up with better nourishment. The only studies so far published that provide any indication are longitudinal studies conducted in Mauritius. An experimental enrichment programme, comprising nutrition, education, and physical exercise for 83 children aged 3 to 5 years, yielded significantly lower scores for schizotypal personality and antisocial behaviour at age 17 years and for criminal behaviour at age 23 years compared with 355 matched controls (Raine et al. 2003). The beneficial effects of the intervention were greatest for children who showed signs of malnutrition at 3 years of age. A second birth cohort of 1795 were assessed for signs of malnutrition at age 3 years. Cognitive measures were assessed at ages 3 and 11 years, while antisocial, aggressive, and hyperactive behaviour was assessed at ages 8, 11, and 17 years. The authors concluded that there was a dose–response relationship between the degree of malnutrition and degree of externalizing behaviour at ages 8 and 17 (Liu et al. 2004). They argue that these findings suggest that reducing early malnutrition may help reduce later antisocial and aggressive behaviour. Given the implications, this needs to be replicated. It may be simplistic, but if there are fewer crimes committed there will also be fewer victims.
Observational evidence

The experimental studies above are supported by observational studies that indicate relationships between antisocial behaviour and a number of nutrients but it is recognized that these relationships will require more research to delineate. Violent offenders were found to have lowered levels of omega-3 and omega-6 but raised omega-9 essential fatty acids compared to age-matched non-offending controls (Corrigan et al. 1994). A cross-national epidemiological analysis found a significant inverse correlation between seafood consumption and incidence of homicides (Hibbeln 2001). Hibbeln et al. (1998) found an inverse relationship between omega-3 plasma essential fatty acids and levels of the metabolites of serotonin and dopamine taken from cerebrospinal fluid in violent offenders, but this was not found with non-offending controls. Stevens et al. (1996) found an increased number of behavioural problems in 6–12-year-old boys with lower plasma concentrations of omega-3 fatty acids. Virkkunen et al. (1987) reported abnormalities in plasma phospholipid concentrations of fatty acids in offenders with a history of alcohol abuse. Violent offenders were found to have significantly lower cholesterol levels than non-violent subjects who were matched by age, sex, alcohol indices, and education (Golomb et al. 2000). Walsh et al. (1997) reported significantly elevated copper/zinc ratios in assaultive young males compared to non-assaultive controls. Plasma homocysteine has been positively associated with ratings of hostility and anger (Stoney and Engebretson 2000). Regulation of homocysteine is dependent on folate, cobalamin (vitamin B12), and pyridoxine (vitamin B6). Depressed patients, for instance, were found to have raised homocysteine levels and lowered cobalamin and folate (Bottiglieri et al. 2001). There may also be a case for addressing individual dietary requirements. A review of 207 subjects selected from 258 consecutive cases suggested that self-reports of violence were significantly reduced compared to baseline by individualized nutritional therapy. Such data are difficult to interpret without controls but the authors appropriately argued for the need to follow up with randomized trials to test efficacy (Walsh et al. 2004).

There may even be behavioural implications for trace elements not yet accepted as essential for human health. A study found significantly fewer homicides, suicides, and rapes in counties of Texas with naturally occurring levels of lithium in drinking water in the range 70–170 μg/L compared to rates in those counties with little or no lithium; these dosages are far lower than those used in medicine. Similarly, violent offenders were found to have less lithium in their scalp hair than controls. These studies simply demonstrate correlations but a double-blind randomized trial of 24 former drug addicts supplemented with 400 μg lithium for a month found significant improvements in mood, reflecting changes in happiness, friendliness, and energy. The author argued a case for the essentiality of lithium in humans at dosages of 1000 μg a day (Schrauzer 2002).
Physiological markers

Physiological markers linked to nutrition also seem to be predictive of antisocial behaviour (Christensen et al. 1996). It has already been reported that the output of the heart supplies the brain with energy, the amount of which may be the limiting factor on information-processing, and one might presume has an impact on behaviour. This appears to be so. Low heart rate variability is thought to be a sensitive marker of low autonomic nervous system function (Akselrod and Gordon 1985) and is considered to measure autonomic adaptation; as such it is considered representative of more basic brain functioning. Wadsworth (1976) reported a stronger correlation between delinquency and lowered resting pulse rate than between delinquency and social deprivation. Children aged 6–7 years with mental health symptoms could be distinguished from controls by low reactivity in parasympathetic and sympathetic systems (Boyce et al. 2001). In a 15-year longitudinal study of the siblings of juvenile delinquents, abnormalities in autonomic function identified at age 15 years predicted with 75% accuracy which subjects would be incarcerated by the age of 29 (Raine et al. 1995). Low heart rate variability also predicted psychopathology and hostility in a 2-year longitudinal analysis of urban boys at risk of delinquency (Pine et al. 1998). Similarly, heightened autonomic nervous system responsiveness was associated with a reduced likelihood of criminal convictions in subjects at social high risk for criminal behaviour (Brennan et al. 1997).

Physiological mechanisms

The research suggests that nutrition influences behaviour, but the mechanisms by which this occurs are not fully understood. It is, however, widely accepted that antisocial behaviours are influenced by the regulation of serotonin (e.g. Linnoila et al. 1983; Stanley et al. 2000; Virkkunen et al. 1995), so this will be used as an illustration. Serotonin is derived from the dietary amino acid tryptophan. To demonstrate that dietary tryptophan depletion results in increased aggressive responses, researchers had to control for other possible explanations such as food depletion causing an increase in aggression (Bjork et al. 1999). This illustrates that nutrients tend to interact with many mechanisms operating in parallel (see also Stacey et al. 1994). Providing piglets with supplemental AA and DHA (the two fatty acids considered so influential in our brain development) significantly increased concentrations of serotonin, tryptophan, and dopamine in the frontal cortex of their brains (de la Presa Owens and Innis 1999). Vitamin E deficient diets have been shown in animal studies to lower serotonin in rat striatum (Castano et al. 1993). Zinc is known to modulate the function of a number of neurotransmitter gated ion channels, including recombinant murine 5-hydroxytryptamine (3A) (5-HT(3A)) receptors, and it potentiates serotonin-induced responses (Hubbard and Lummis 2000). Furthermore, animal studies have shown that low iron and zinc intakes predispose towards aggression (Halas and Reynolds 1977; Munro 1987). Pyridoxine has
been demonstrated in monkey brains to have a regulatory effect on the conversion of 5-hydroxy-L-tryptophan (5-HTP), a precursor of serotonin (Hartvig et al. 1995). Oral doses of pyridoxine were found to influence blood serotonin and pyridoxal phosphate levels in hyperactive patients (Bhagavan et al. 1975). It is not suggested that these studies offer proof of mechanisms that are at work in antisocial behaviour, as this will require a great deal more research, but they do illustrate the potential for plausible physical pathways to be identified by which levels of nutrients could affect levels of antisocial behaviour.

Mediation of dietary effects on antisocial behaviour

How such effects are mediated is another important question. The picture is far from clear at this stage but inhibitory mechanisms are considered fundamental components of the attentional and higher cognitive processes that form the key dysfunctions of ADHD, for example, which is a common precursor of antisocial personality disorder (ASP; e.g. Taylor 1998; Rubia et al. 1998). Impulse control is a current focus of interest for how nutrients might impact behaviour. Adults with ASP, conduct disorder, and aggressive impulsiveness have been shown to have neuropsychological deficits in frontal-lobe-mediated paradigms (Pennington and Bennetto 1993; Brower and Price 2001). Moreover, low executive functioning has been shown to correlate with measures of physically aggressive behaviour (Giancola et al. 1998; Foster et al. 1993; LaPierre et al. 1995). Violent offenders have also been found to be deficient in shifting attention from one category to another when compared to normal control subjects (Bergvall et al. 2001). Poor development of self-directedness and cooperative traits in forensic populations may be associated with deficits in neuropsychological functioning (Bergvall et al. 2003).

Replication

Large \((N = 700)\) clinical trials are planned within the UK and Dutch prison systems to investigate the relationship between a participant’s nutritional status (measured from blood samples) and changes in specific nutrient groups with the following outcomes, antisocial behaviours, violence, drug abuse, self-harm, impulse control, attention, planning, interpersonal relating, and food choice, with concomitant implications for reassessing dietary standards and our understanding of such behaviours in the community.

Integrating nutrition with existing risk factors for antisocial behaviour

There do appear to be interactions between our social and physical environment as Bryce-Smith (1986) predicted. While this may seem daunting initially, there are many positive aspects to this, as diet could provide an additional means to promote
behavioural well-being at a time when criminal justice resources are under stress. These findings also may improve our understanding of established socio-economic risk factors such as low income, family interactions, and peer-groups that correlate with offending (Blackburn 2002). There will also always be a biophysical component to any social situation, as we all need to eat. This will interact with both individual and environmental theories of crime (Blackburn 2002). There is a great deal of research into ‘risk factors’ that affect the behaviour of juveniles (Smith 1995; Rutter et al. 1998) including, for example, the breakdown of families (Heiss 1995). However, one of the social functions of families is to provide food, so it would be illuminating to investigate the extent to which diets interact with such breakdowns. It is well established that diet is related to income (e.g. WHO 2003). If we no longer produce our own food, the costs per calorie of healthy foods like fruits and green leafy vegetables are typically much higher because they contain so few calories. The healthy energy-dense foods like lean meats and fish are much more expensive than more highly refined energy-dense foods derived from saturated fats, white flour, and refined sugars. These cheap, energy-dense foods are likely to seem more attractive and affordable if you have a limited income. Those on low incomes are likely to face the dual pressures of socio-economic and physiological stress as a consequence of not easily affording adequate diets. The cycle is therefore self-reinforcing. If we grow up with friends in this environment, this may conceivably increase the wayward influence of peer pressure as the behaviour of our friends is more likely to be undermined by poor nutrition. We will presumably also be subject to the vagaries of a poor social environment. However, this would not explain why, in general, more boys get into trouble than girls and this reaches a peak in late adolescence. One might wonder how diet could be involved in these trends. The link is speculative at this stage but, for instance, differences have been found in DHA metabolism between males and females (Burdge and Wootton 2002; Burdge et al. 2003). A factor in the peak age of offending may turn out to be the more pronounced growth spurt in young males, which brings with it a range of physiological changes such as rapid growth that may place the brain under increased nutritional stress (Gesch 2002). If this scenario is correct, it highlights the importance of providing healthy meals to schoolchildren to help break this self-reinforcing cycle. The British Government recently concluded that just 6% of schoolchildren make healthy food choices at school meals and, according to the Times Educational Supplement, 14 April 2004, ‘The Government has been forced to pump £342 million into school behaviour improvement programmes.’ In view of the foregoing, these two facts may not be coincidence. Would it not be prudent to apply the precautionary principle and to try our best to ensure that all our children at least reach existing dietary standards? Large studies of the effects of diet in schools are being planned.
Implications in criminal justice

It is not suggested that nutrition is the only explanation of antisocial behaviour, only that it might form a significant part. Unlike in medicine, however, most of the ‘risk factors’ targeted in criminal justice interventions are typically correlations, which leaves questions of volition, culpability, prevention, and rehabilitation open if cause and effect are not formally demonstrated with appropriate randomized experimental designs. This leaves crucial questions unanswered. What causes someone to commit a crime or behave in an antisocial manner when others do not? How do you predict or prevent offending unless you know what causes it? How do you accurately focus resources unless you know about causes? For instance, the public costs of testing cognitive skills approaches in English prisons was reported in the London Times (18 November 2003) to cost £150 000 000 and this approach was found to be ineffective (Cann et al. 2003). In contrast, The Economist of 29 June 2002 reported the cost of the nutritional approach to be as little as 0.2% of that expended on custody. Faced with escalating costs, there is a climate of increasing honesty in the US that federal criminal justice policies are not underpinned by rigorous evidence, with statements such as ‘The effectiveness of most crime prevention strategies will remain unknown until the nation invests more in evaluating them’ (Sherman et al. 1997), ‘It’s easy to fool yourselves about efficacy if you haven’t done a proper clinical trial’ (Marshall 2000), and ‘Progress is often thwarted by Government programmes and strategies that are not based on rigorous evidence’ (Baron et al. 2003). The report by the US Department for Justice and Collation for Evidence-Based Policy (Baron et al. 2003) recommended that in future interventions should be evaluated by randomized designs as the sole basis for strong evidence of efficacy with which to underpin criminal justice policy. Evidence is, after all, only as good as the means used to obtain it! Ironically, the finding that a better diet can positively impact on behaviour exceeds the standards suggested as a strong basis of evidence to underpin criminal justice strategy but this approach is not yet assimilated into criminal justice thinking. These studies will need to be widely replicated and, if so, we may have finally demonstrated a causal factor in antisocial behaviour. Further studies are also planned in the UK to test effects of nutrition on crime in the community. If the finding that diet can positively influence behaviour directly is widely replicated, it questions the underlying assumptions of the classical form of justice where it is assumed that behaviour is entirely a matter of free will. The picture will then be more complex but the solution perhaps more straightforward. Some of these factors will act in ways that we can see and some of them will not, so we will need a broader interpretation of the causes of antisocial behaviour in criminal justice, where physical and social functioning are both considered relevant to culpability.

Awareness offers hope because, if this scenario is correct, this process can be reversed if we choose to nourish our children rather than condemn them for influences on
their behaviour that we have not taken into account. In doing so we may need to choose if we prefer to lock up ever more of our nation’s children or to nourish them properly. Indeed, one of the difficulties in criminal justice is knowing when to intervene. Pre-emptive involvement in the criminal justice system runs the risk of being prejudicial to those who might not offend and has been shown to accelerate criminal careers through association, while intervening too late can also result in escalation. In marked contrast, how is providing a healthy diet going to be prejudicial at any stage? The Dutch Ministry of Justice is actively investigating this approach and made a telling comment: ‘There are only positive side-effects to providing offenders with a better diet!’

According to a UK Home Office report (Brand and Price 2000), ‘The total cost of crime to England and Wales in 1999/2000 is estimated at around £60 billion, although this figure is still far from comprehensive, as it does not include important costs such as fear of crime or quality of life impacts.’ If diet could achieve even a modest impact on these costs the benefits could be vast. There is strong case to conduct further large randomized trials to explore if diet can reduce crime in the community and antisocial behaviours in prisons and other institutions such as schools.

**Discussion**

**The global implications for well-being**

A range of evidence has been reviewed that suggests that the dietary requirements for good health are also supportive of positive social behaviour. These findings need to be widely replicated, as potentially there are global implications. The interventions that have been reviewed simply provide nutrients at physiological dosages that are already known to be essential for health. Providing a healthy diet does absolutely no harm, yet we have arrived at a situation where many of the human population do not obtain even this. Millions starve while millions suffer from diseases of excess and yet we spend untold amounts on medical treatment to treat illness and prisons to curb behaviour. What if much of this suffering could be prevented?

Good nutrition is essential irrespective of race, age, legislative boundaries, if you are in prison, or in a rich or developing country. This is a basic need that unites us all: hence it is not where you eat that is important but what you eat. Humans are, after all, both social and physical beings—the hardware and software of life, so to speak. Nutrition can impact on both aspects and thus spans both nature and nurture. How we interact with food is likely to be a complex function of choice, family, peer-group, physical activity, economics, food distribution, and physiology. Food plays an important role in our social structures. Much of traditional family routine is based around meals. A mother’s love, courtship, and friendship are often expressed through giving food. We incorporate food into medicine and our
religious practices such as offerings to deities or sacrament. Yet for every social situation there will always be a biophysical analogy. Irrespective of what we eat, getting food into one’s mouth will be socially mediated but one’s mouth is the gateway to the physical world. Crucially, our physiological responses to our diet are likely to be the most finite component of this equation, as they seem to be primarily derived from our evolution. This may explain why longitudinal studies can link a child’s diet (or, for instance, childhood exposure to lead) with crime as an adult. This is not a reinterpretation of crude determinism because this is a feedback cycle where choice will inform what we put into our mouths, while the nutrients in the food will support the operation of the senses that inform our choice (for instance the retina is 30% AA and DHA). It is worth reiterating that some of these factors will act in ways that we can see and some of them will not, so we need a broader interpretation of what promotes well-being, where physical and social functioning are both considered relevant. The brain needs to be nourished in two ways: love, nurturing, education as well as the nutrients for the brain to function properly. Not one, not the other, but both. That applies to both the rich and poor nations.

If we really are what we eat then changing our diet will change us.

We seem to have made major changes to modern diets in a relatively short space of time with little or no systematic examination for potential impacts on brain function or behaviour. Hence, evidence of such impact is only emerging on a retrospective basis. More should be done to prospectively assess such impact. This not only relates to the food industries. Such a basic issue also seems to have passed largely unnoticed between the boundaries of academic disciplines and the authorities that construct dietary standards. There is a need for contiguous scientific disciplines to cooperate to investigate the broader picture. Because nutrition is fundamental for life and also the composition of our brains, its effects are likely to be pervasive. The health consequences of modern diets are already visible but we are only beginning to appreciate them in brain chemistry, mental health, cognition, and behaviour. We are likely to interpret behavioural problems in terms of what we can see, so it is feasible that, on a societal scale, the effects of poor diet are liable to be greatly amplified, as greater numbers of social interactions are subject to these hitherto unnoticed influences on our behaviour. These effects may even shift the socially acceptable norms of behaviour without our knowledge. It may sound far-fetched but few would have predicted the potency of effect shown in the prison study. There is even research being planned to explore the role of nutrition in artistic expression. Physiological factors like nutrition may also turn out to be important in understanding the tragic and irrational acts that litter news broadcasts and seem to defy a rational explanation. They may add to, rather than supplant existing explanations. Compared to the myriad of socio-economic problems found in deprived areas, nutrition may actually be one of the more straightforward factors
to change. An intriguing aspect of our British prison study is that the prisoners received three meals a day and, despite making poor food choices, their diets were possibly better than those consumed by many young men of the same age in the community. Yet the improvement in behaviour from boosting their diets was highly significant. We do not know if the improvement in behaviour would have come from raising the prisoners to existing dietary standards or because some would have exceeded them slightly. We have limited knowledge of what the optimum ranges of nutrients are from a behavioural perspective because dietary standards have not really considered this. The bottom line here is that dietary standards need to be reassessed to take into account mental health, behavioural, developmental, and cognitive parameters. It is recognized that this will be a major undertaking involving extensive research. However, an implication is that, for a great number of people, their ability to behave sociably could be improved by what they eat, without them even being aware of it. It is hard to imagine anything that might impact on the future well-being of our species so directly.

Research is emerging that suggests that good nutrition is potentially inexpensive, humane, and effective at promoting health as well as social behaviour. Most importantly, people’s positive potential might be realized if such an approach is taken at a time when global resources are under stress. To make an analogy, no amount of energy spent on software will resolve a hardware problem. Nature offers us a clue about the way forward by providing natural foodstuffs that grow in harmony with their environment and invariably contain a range of nutrients in dosages to which our metabolism is attuned. It should come as no surprise, therefore, that many vitamins, minerals, and essential fatty acids seem to promote aspects of well-being whether it be health or behaviour—particularly those nutrients that modern dietary practices have rendered less available. If nutrition was truly seminal in raising our ancestors to a new level of functioning, then let us rethink our present-day attitude to food. We need to develop our understanding of the dosage and range of nutrients required to positively impact on the human condition. It may be a recipe that goes beyond individual well-being; it may be a recipe for peace.

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