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Review

A brief history of meat in the human diet and current health implications

Neil J. Mann*

Faculty of Veterinary and Agricultural Science, University of Melbourne Parkville, Victoria 3010, Australia

ABSTRACT

Anthropological investigations have confirmed many times over, through multiple fields of research the critical role of consumption of animal source foods (ASF) including meat in the evolution of our species. As early as four million years ago, our early bipedal hominin ancestors were scavenging ASFs as evidenced by cut marks on animal bone remains, stable isotope composition of these hominin remains and numerous other lines of evidence from physiological and paleo-anthropological domains. This ASF intake marked a transition from a largely forest dwelling frugivorous lifestyle to a more open rangeland existence and resulted in numerous adaptations, including a rapidly increasing brain size and altered gut structure. Details of the various fields of anthropological evidence are discussed, followed by a summary of the health implications of meat consumption in the modern world, including issues around saturated fat and omega-3 fatty acid intake and discussion of the critical nutrients ASFs supply, with particular emphasis on brain function.

1. Introduction

Contrary to views that humans evolved largely as a herbivorous animal in a 'garden of Eden' type of environment, archaeological and anthropological evidence indicates a very different reality, at least in the last three to four million years of evolutionary adaptation. It was in this time frame that our ancestral hominin line emerged from the receding African wetland forests to become bipedal, open grassland dwellers. In this new dryer environment digestible plant foods were less readily available than in the wetland forests, but grazing animals were abundant. The subsequent dietary change this demanded initially involved scavenging the remains of herbivore carcases, with an eventual shift to direct hunting over a time period of approximately 2 million years. This was accompanied by subsequent physiological and metabolic adaptations that culminated in modern humans (Mann, 2005). Numerous fields of scientific study have contributed to this understanding of human diet evolution and the central role of meat in the development of our species. Findings from each of these fields reveal a changing dietary pattern away from low-quality/highly fibrous, energy-poor plant stables to a growing dependence on more energy-rich animal foods, culminating in Palaeolithic *Homo sapiens* being top-level carnivores (Richards, Schulting, & Hedges, 2003).

2. Understanding human evolution through taxonomic classification

For the last hundred plus years modern humans have been classified as one of the great apes (family Hominidae), along with orangutans, gorillas and chimpanzees. Recent genetic studies however show two clear subfamilies: Ponginae (orangutans) and Homininae (humans and their ancestors, and chimps and gorillas). This latter group is further divided by phylogenetic studies into Hominins (humans and their ancestors), Pan (or chimpanzees and bonobos), and Gorillas (Cela-Conde, 2001).

In simplified terms a Hominin is a human or a human ancestor, which include all of the Homo species (*Homo sapiens, H. ergaster, H. rudolfensis*, includinging Neanderthals, Denisovans, and Flores), all of the Australopithecines (*Australopithecus afarensis, A. africanus, A. boisei*, etc.) and other ancient forms like *Paranthropus* and *Ardipithecus*. Since humans are more closely related to Pan than gorillas, it is likely that hominins and chimpanzees had a joint ancestor who probably lived between 4 and 8 million years ago, during the late Miocene (23 million years ago to 5 mya) in the wetland forests of Africa (Cela-Conde, 2001).

3. Climate change and shift in the hominin dietary pattern

Around 2.8 million years ago East African vegetation shifted from a wetland closed canopy dominated habitat to a drier open savanna grassland (De Menocal, 1995), due to changes in global climatic conditions, primarily drier conditions followed by worldwide expansion of the biomass of temperate climate (C4 photosynthetic) grasses at the expense of wetland forests (Gerling, Harris, Macfadden, Leakey, Quade, Eisenmann, and Ehlreinger, 1997). This was accompanied by a worldwide faunal change (Kohler, Moya-Sola, & Agusti, 1998) including the

* Corresponding author.
E-mail address: neil.mann@unimelb.edu.au.

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spread of large grazing animals. Thus, the foods available to our hominin ancestors in an open grassland environment were very different from those of the jungle/forest habitats that were home for many millions of years. Sometime after this climatic change during the mid Pliocene (5 mya to 2.5 mya), probably on multiple occasions, ancestral bipedal hominins of the Australopithecus line ventured out from the shrinking wetlands of east Africa into these expanding grasslands. This shift of habitat was accompanied by dietary changes and subsequent physiological and metabolic adaptations (Mann, 2010).

4. The anthropological and archaeological evidence of transition to meat consumption

The lines of investigation used by anthropologists to deduce the evolutionary diet of our evolving hominin ancestors include: (i) changes in cranio-dental features; (ii) fossil isotopic chemical tracer methods; (iii) comparative gut morphology of modern humans and other mammals; (iv) the energetic requirements of developing a large ratio of brain to body size; (v) optimal foraging theory; (vi) dietary patterns of surviving hunter-gatherer societies; (vii) specific diet related adaptations and (viii) fossil evidence of animal butchery.

A detailed account of the methodologies involved has been published previously (Mann, 2000, 2005). In brief cranio-dental changes included a decrease in molar teeth size, jaws/skull became more gracile, and shearing crests appeared on teeth, all indicative of less emphasis on grinding and more on biting and tearing of animal flesh (Speth, 1989). The C13/C12 isotope ratio in fossil remains is indicative of diet, and is a particularly good marker of the intake of broad-leaf plant material versus grasses. Examination of early hominin remains indicates that they ate large quantities of the C13-enriched foods (Sponheimer, & Lee-Thorp, 1999). As hominins in general have shown no capacity for digestion of grasses or teeth microwear patterns indicative of grass mastication (Puech, 1992; Grine, & Kay, 1988), these hominins were likely consuming grazing animals, which lived on such grasses. Similarly, the bone strontium:calcium ratio (Sr/Ca) ratio in mammals shows an inverse correlation with trophic level, with pure carnivores showing the lowest ratio. Early hominins had a Sr/Ca ratio midway between contemporary carnivore and herbivore species (Sillen, 1992; Sillen, & Lee-Thorp, 1994). These results alone would indicate that even very early Australopithecine hominins (2–3 mya) consumed a considerable proportion of meat in their diet (Sillen, & Armstrong, 1995), though at this stage it was not likely to be directly hunted, but rather scavenged from dead remains (Mann, 2005).

4.1. Dietary requirements for encephalization

Humans have larger brain sizes than would be expected for their body size, a phenomenon described as “encephalisation” a topic reviewed in detail elsewhere (Aiello, & Wheeler, 1995; Mann, 2000). In hominins the increase in brain size has occurred predominantly in the last 2–3 million years. In fact since the time of A*ustralopithes aferensis* some 4 mya, brain size has increased threefold (Aiello, & Wheeler, 1995). What the driving force was for this dramatic increase can only be speculated, although many sound hypotheses based on socio-ecological factors have been put forward. Irrespective of the driving force for encephalisation, two critical requirements had to be met: (i) the brain's chemical requirement for long chain (LC) polyunsaturated fatty acids (PUFA), particularly arachidonic acid (AA, 20:4n-6) and docosahexaenoic acid (DHA, 22:6n-3) (Crawford, 1992) and (ii) the increased metabolic requirements of a larger brain (Martin, 1981; Mann, 2000). The fatty acids mentioned are the major structurally significant and biochemically active components of the brain gray matter of all mammalian species (Sinclair, & Crawford, 1975). The availability of these particular fatty acids may have provided a selective pressure acting to increase brain size, by simply supplying adequate dietary substrate to allow formation of brain tissue (Crawford, 1992). In this sense adequate can only imply an amount that allowed for gradual brain size increase. The metabolic requirement of the human brain per kilogram is approximately nine times higher than the human body as a whole (Aiello, & Wheeler, 1995). Kuzawa, Chugani, Grossman, Lipovich, Muzik, Hof, Wildman, Sherwood, Leonard, and Lange (2014) have used positron emission tomography (PET) and magnetic resonance Imaging (MRI) data to show this high energy demand of the human brain, reaching a maximum glucose usage of 65% of the bodies resting metabolic rate in early childhood. To sustain such a metabolically expensive large brain, there are two possible evolutionary adaptations that could be employed: (i) elevate the basal metabolic rate (BMR); or (ii) compensate for higher brain energy with lower mass-specific metabolic rates of other tissues.

Humans fit the general mammalian predictive value for basal metabolic rate (BMR), indicating no increase in basal metabolism sufficient to account for the additional metabolic expenditure of the enlarged brain (Aiello, & Wheeler, 1995). However, there is an evident reduction in the size of another metabolically active organ, that is the gastro-intestinal tract. When examining individual organs, the brain mass surplus (and energy requirement), however, is closely balanced by the reduction in size (and energy requirement) of the gastrointestinal tract. This finding is not surprising, considering the function of other metabolically expensive tissues, the gut is the only organ which can vary in size sufficiently to offset the metabolic cost of the larger brain.

Diets high in bulky food of low digestibility require relatively enlarged gut size with voluminous fermenting chambers (rumen and caecum). Diets consisting of higher levels of high quality foods are associated with a relatively smaller gut, with simple stomachs, reduced colon size, but proportionately long small intestine (Chivers, & Hladik, 1980) as seen in carnivores. This pattern is seen clearly in primates where higher food quality relates to smaller gut size and larger brain size, with humans being the extreme case (Aiello, & Wheeler, 1995), where large intestine volume is reduced and small intestine volume increased. For early hominins, with increasing brain size the problem would have been to provide adequate levels of high quality food to permit the necessary reduction in gut size (Aiello, & Dean, 1990). With the relatively poor macronutrient density of wild plant foods, particularly in the open woodlands and savanna, the obvious solution was to include increasingly larger amounts of animal-derived protein/fat rich foods in the diet (Speth, 1989).

4.2. Digestive system adaptation

Both pure herbivores (folivores and frugivores) and pure carnivores (such as felids) have physiological and metabolic adaptations suited to their diet (Morris, & Rogers, 1983; Milton, 1986).

Humans fit neither category, but are truly omnivores, falling between the largely frugivorous make-up of such anthropoid relatives as the chimpanzee and the adaptations of the true carnivores (Martin, 1992). A sacculated stomach or well-developed caecum and colon are associated with plant-based diets. The lower the plant quality (or the higher the fibre content), the more pronounced are these features. The ruminant animals show the greatest volume in the stomach region (rumen). Non-ruminant herbivores, such as the horse, have greatest development in the caecum and colon. Measures of relationship between gastrointestinal length or surface area, and body length or surface area give a good relative comparison of carnivore versus herbivore characteristics (Table 1). Carnivores tend to have a well-developed acid stomach and long small intestine. The human gut with its simple stomach, relatively elongated small intestine and reduced caecum and colon is suggestive of reliance on a high-quality diet in which meat is a predominant component. The size of the human gut relative to body size is also small in comparison with other anthropoids, with a much more pronounced small intestine similar to carnivores (Milton, 1986; Martin, 1992). Approximate relative proportions of gut volume for humans and some other primates are shown in Table 2.

Table 1
Quantitative comparison of length and surface area of gastrointestinal tracts of some animals and humans.

Species	Intestinal length: body length	Gastrointestinal surface area: body surface area
Cattle	20: 1	3.0: 1
Horse	12: 1	2.2: 1
Dog	6: 1	0.6: 1
Human	5: 1	0.8: 1

Adapted from Henneberg, Sarafis and Mathers (1998).

Table 2
Relative gut volume proportions for a selection of primate species (% of total volume).

Species	Stomach	Small intestine	Caecum	Colon
Gorilla	25	14	7	53
Chimpanzee	20	23	5	52
Human	17	67	NA	17

Adapted from Milton (1986); NA = not applicable.

4.3. Optimal foraging and meat intake

The subsistence patterns of hunter gatherers, early hominins and our palaeolithic ancestors can also be explained in mathematical terms of cost/benefit analysis. The major survival determinant is daily energy procurement (less energy expenditure), termed 'Theory of Optimal Foraging' (Pyke, Pulliam, & Charnow, 1977). The wild fruits, vegetables, foliage and tuberous roots available to hunter-gatherers and early hominins were generally fibrous and of low energy density (Southgate, 1991). The high energy/time spent in collection and preparation of such plant foods, particularly wild seed grains, is not well rewarded in terms of energy gain; hence these are not feasible as a primary energy source (Table 3). This explains why meat intake was essential despite often abundant plant food availability (Hawkes, Hill, & O'Connell, 1982).

4.4. Hunter-gatherers in recent times

The study of dietary patterns of surviving hunter-gatherer societies also gives us a clear picture of what our pre-agricultural ancestors (both early hominins and later homo species) consumed. Calculations have been made from Murdock's Ethnographic Atlas (Gray, 1999) of 229 hunter-gatherer societies for whom we have extensive dietary intake data, showing that the majority of such societies obtained between 56 and 65% of their subsistence (energy) from animal foods. The predicted macronutrient energy intake ranges were carbohydrate 22–40%, protein 19–35% and fat 28–47% (Cordain, Brand-Miller, Eaton, Mann, Holt, and Speth, 2000).

Table 3
Energy return rates from foraged and hunted foods by South American hunter-gatherers.

Food	Food type	Return rate (kcal/h)
Peccary	Animal	65,000
Antelope	Animal	16,000–32,000
Rabbit	Animal	13,500–15,400
Squirrel	Animal	5400–6300
Roots	Plant	1200–6300
Fruits	Plant	900–6000
Snakes	Animal	5900
Birds	Animal	4800
Seeds	Plant	500–4000

Adapted from Cordain, Eaton, Brand-Miller, Mann, and Hill (2002).

4.5. Adaptations to meat in the early hominin diet

Humans also exhibit a range of specific adaptations indicative of extensive reliance on animal foods in the diet. Similar to obligate carnivores, humans have an inefficient ability to chain elongate plant-rich 18-carbon fatty acids into the 20- and 22-carbon polyunsaturated fatty acids (PUFA) essential for cell membrane function and brain tissue (Emken, Adlof, Rohwedder, & Gulley, 1992), hence requiring direct consumption from animal tissue. Likewise, humans have inherited a decreased ability to synthesise the amino sulfonic acid, taurine (Chesney, Helms, Christensen, Budreau, Han, & Sturman, 1998), which is involved in numerous essential physiological functions. Studies on vegans reveal a plasma level of taurine 78% lower than in omnivores (Laidlaw, Shultz, Cecchino, & Kopple, 1988). The proposed rationale, as for obligate carnivores, is that there was reduced selective pressure to synthesise taurine in vivo because exogenous dietary sources of preformed taurine (found only in animal tissue) were being consumed for a lengthy time period.

Furthermore, haem and other porphyrine iron-rich compounds derived only from animal foods are absorbed by humans in preference to ionic forms of iron common in plants, whereas herbivorous animals cannot absorb these haem complexes and rely on absorption of the ionic form of iron (Bothwell, Charlton, 1982). Finally, mammalian hosts and their various parasites undergo close co-evolution. Cestodes of the family Taeniidae are parasites of carnivores, spread by eating meat. *Taenia saginata* and *T. solium* use humans exclusively as their host, indicating a substantial period of co-evolution and meat consumption by humans and their ancestors (Henneberg, Sarafis, & Mathers, 1998).

4.6. Archaeological evidence of animal butchering

Archaeological findings in the east African Rift Valley dating to 2.5–2.1 mya indicate macroscopic and microscopic features of stone tool-imparted damage caused by hominin butchery. These observations support the hypothesis that the earliest stone artifacts functioned primarily as butchery tools and also imply that scavenging of large ungulate carcasses may have been practiced by early Australopithecine hominins as early as 2.5 mya (Domínguez-Rodrigo, Pickering, Semaw, & Rogers, 2005). Other archaeological work has demonstrated that microscopically distinct wear-polishes on tools from five early Pleistocene archaeological sites in Kenya, dated to 1.5 mya ago, are indicative of cutting soft animal tissue (Keeley, & Toth, 1981). All indicating the consumption of ASFs was widespread over a long time period, even well before modern day humans appeared.

5. The dietary impact of the transition to agriculture and reduced meat intake

The dietary changes involved with the transition from hunting and gathering to agriculture have been extensively reviewed (Angel, 1984; Hillman, Colledge and Harris, 1989; Ulijaszek, 1991). This transition began in the Near East approximately 10,000 years ago with the growing of wild cereal crops as a response to population increase and/or scarcity of large mammalian wild game. The transition, however, was associated with physiological stresses, including reduced stature, osteomalacia, dental caries and various nutritional deficiencies, and infectious disease (Cohen, 1989).

The archaeological evidence indicates a shift from consumption of hoofed mammals (gazelle, antelope and deer), root plants, wild pulses, various nuts and fruit, to a more narrow diet of cultivated wheat, barley, oats, rice or corn, depending on location (Van Zeist, & Bakker-Heeres, 1979). This transition away from hunted animal foods also corresponded with a fundamental reversal of the high-protein, low-carbohydrate diet, along with the shift in fatty acid intake type. The omega-6: omega-3 intake ratio began itsrise from approximately 3:1 in pregaricultural times to > 12:1 in the current Western diet, where the

Table 4
Estimated daily palaeolithic intake of selected nutrients compared with current US and Australian intakes.

Nutrient	Palaeolithic intake[a]	US intake[a]	Australian intake[b]
Macronutrients (% energy)			
Protein	37	15	17
Carbohydrate	41	51	46
Fat	22	34	33
P: S ratio	1.4	0.4	0.4
Vitamins (mg/day)			
Riboflavin	6.5	1.3–2.1	2.1
Folate	0.36	0.15–0.21	0.27
Thiamine	3.9	1.1–1.8	1.7
Vitamin C	604	77–109	120
Vitamin A (RE)	2870	1170–1429	1165
Vitamin E	32.8	7–10	NR
Minerals (mg/day)			
Iron	87	10–11	14
Zinc	43.4	10–15	12
Calcium	1956	750	875
Sodium	768	4000	NR
Potassium	10,500	2500	3317
Fibre (g/day)	104	10–20	23

[a] Adapted from Eaton, Eaton & Konner (1997).
[b] Adapted from Mclennan and Podger (1998). Results shown are for combined men and women aged 25–44 years. NR = not recorded in Australian National Nutrition Survey 1995, RE = retinol equivalents. P:S = polyunsaturated: Saturated fat intake ratio.

n-6 PUFA from seed oils are now abundant in the diet and omega-3 fatty acids, particularly the LC forms from animals has fallen (Sincalir and O'Dea, 1990). Similarly, micronutrient intake levels dropped following the shift to agriculture, where a grain staple dominated, in place of a diet dominated by hunted animal foods and wild plants (Table 4).

It has been argued that the replacement of protein from meat and fish with high glycaemic index (GI) carbohydrates from starch and sugars may have implications for insulin resistance and the development of type 2 diabetes - a condition unknown in hunter-gatherer societies. There may be more certainty about the impact of reduced intakes of fish and meat on iron and zinc status. Humans evolved on dietary intakes of these minerals, which were several times the current intake level (Eaton, Eaton, & Konner 1997). Likewise, the low vitamin B12 status of vegetarians indicates a reliance on animal foods in the diet, that likely has a long historical basis (Mann, Li, Sinclair, Dudman, Guo, Elsworth, Wilson, & Kelly, 1999).

6. Health aspects of animal foods in the human diet

6.1. Animal foods as a rich source of nutrients

Meat in general is a quality dietary source of several micronutrients important for health and disease prevention, the most abundant being: vitamin B12, selenium, zinc, niacin, phosphorus and particularly iron in a bioavailable form. Although it should be noted that the levels of each nutrient mentioned do vary considerably between animal species and the feeding regimes they are subject to. Pork and poultry for instance are becoming more dominant in the food supply and may be low in several of these nutrients, depending on their own food regime. Animal foods (particularly red meat) are a well known source of haem iron, which is absorbed more efficiently than non-haem iron found in plants (Valenzuela, de Romaña, Olivares, Morales, and Pizarro, 2009). There is more than a seven fold difference in iron bioavailability between haem iron and non-haem iron, although non-haem iron absorption can be enhanced by simultaneous consumption of vitamin C, meat, poultry and fish proteins, or inhibited by phytate, polyphenols, calcium, eggs, soy protein and alcohol (Hunt, 2003).

Aside from vitamins and minerals, animal foods also contain a number of bioactive nutrients and antioxidants that may affect health, including: (i) Creatine which is found in high amounts in meat, and is essential as an energy source for muscles (Gualano, Roschel, Lancha-Jr, Brightbill, and Rawson, 2012); (ii) Taurine, which is an antioxidant amino acid, found in fish and meat. It is formed in small quantities in the body and may be beneficial for heart and muscles (Wójcik, Koenig, Zeleniuch-Jacquotte, Pearte, Costa, & Chen, 2013); (iii) Glutathione, an antioxidant, present in high amounts in meat, particularly grass-fed animals (Descalzo, Rossetti, Grigioni, Irurueta, Sancho, Carrete, & Pensel, 2007); (iv) Conjugated linoleic acid (CLA): A family of ruminant trans fats that may have various beneficial health effects when consumed in normal amounts from foods, such as lamb (Koba, & Yanagita, 2014). Red meat is the richest meat source of CLA (main CLA isomer being c9,t11), which has many potential health benefits in cancer, coronary heart disease and diabetes (Mulvihil, 2001).

In addition, animal foods (particularly red meat) contain the amino acid beta-alanine, which the body uses to produce carnosine (a dipeptide of beta-alanine and histidine), a substance that is important for muscle function (Sale, Saunders, & Harris, 2010). High levels of carnosine in human muscles have been associated with decreased fatigue and improved exercise performance (Derave, Ozdemir, Harris, Pottier, Reyngoudt, Koppo, Wise, & Achten, 2007). Other studies have shown that carnosine has a number of antioxidant properties that may be beneficial to health (Choi, Kwon, Kwon, & Kang, 1999). Adhering to diets that are low in beta-alanine, such as vegetarian and vegan diets, decrease levels of carnosine in muscles over time (Everaert, Mooyaart, Baguet, Zutinic, Baelde, Achten, Taes, De Heer, & Derave, 2011). Meat is also a significant source of dietary choline (an important precursor of membrane phospholipids and neurotransmitters), containing approximately 78 mg/100 g (Zeisel, & Niculescu, 2006), behind milk, liver and eggs (Williams, 2007). Although choline can be made in the body, dietary essentiality has been demonstrated and the Australian Nutrient Reference Values recommend an adequate intake (AI) of 550 mg/d for men and 425 mg/d for women (NHMRC, 2006).

6.2. Meat and heart disease

Coronary heart disease and cardiovascular disease (CVD) in general are major causes of premature death in western societies. CVD is not one disease, but rather a group of various adverse conditions involving the heart and blood vessels leasing to heart attacks, strokes, and hypertension. There have been mixed results from observational studies on the link between meat and heart disease. Some studies have found an increased risk from eating high amounts of both processed meat (often with high NACl and nitrate and frequently higher levels of fat) and unprocessed meat (Bernstein, Sun, Hu, Stampfer, Manson, & Willett, 2010), whereas others have found no effect at all (Wagemakers, Prynne, Stephen, & Wadsworth, 2007). Thus there is no hard evidence supporting the link between meat consumption per se and CVD, as observational studies only reveal an association (correlation), but cannot prove a direct causal relationship. Several theories have been proposed to explain this association of high meat intake with heart disease, the main one being the amount and type of fat (saturated) associated with meat. However, meat has changed composition considerably over the last 25 years and Australian meat in general is not as high in fat and saturated fat as US red meat (Mann, 2005), upon which most negative health aspects have been based. Obviously though, a high intake of meat means less intake of other foods, such as heart-healthy fish, fruit and vegetables, an aspect of dietary displacement that can lead to various disease states. Also high meat intake in observational studies is linked with various unhealthy lifestyle factors, such as; lack of physical activity and smoking, along with association of meat products with a nutritionally imbalanced Western diet and overeating in general. Hence to relate the disease outcomes to meat intake itself is misleading and fails to acknowledge the general less healthy lifestyle of high meat consumers relative to more vegetarian orientated individuals

who have made conscious diet and healthy lifestyle choices (Bedford, & Barr, 2005). The most popular theory relating CVD and red meat is still the diet-heart hypothesis. Many people believe that meat may cause heart disease because it contains high amounts of cholesterol and saturated fat, impairing the blood lipid profile (de Souza, Mente, Maroleanu, Cozma, Ha, Kishibe, Uleryk, Budylowski, Schünemann, Beyene, and Anand, 2015). However, most lipid researchers now agree that dietary cholesterol is not a risk factor for elevated blood cholesterol and CVD (Eckel, & Jakicic, 2014). Also, the role of saturated fats in the development of heart disease is not entirely clear. Many cohort studies have not been able to link saturated fat with increased risk for heart disease (Siri-Tarino, Sun, Hu, & Krauss, 2010).

Dietary intervention studies feeding elevated quantities of lean red meat to human subjects have however, shown consistent reductions in serum total and LDL cholesterol levels (O'Dea, Traianedes, Chisholm, Leyden, and Sinclair, 1990; Morgan, Sinclair, & O'Dea, 1993), a key indicator of reduced CVD risk. Short term human feeding trials over 2–3 week periods (then cross over) with individual saturated fatty acids show that only three fatty acids actually raise serum low density lipoprotein (LDL) cholesterol levels (lauric 12:0, myristic 14:0, palmitic 16:0), (Derr, Kris-Etherton, Pearson, & Seligson, 1993), all of which are relatively low in the lean muscle meats (Williams and Droulez, 2010). In itself, lean meat does not have adverse effects on the blood lipid profile. In Spanish studies, lean lamb for instance has been shown to have similar, minimal effects on blood lipids as fish or white meat, (Mateo-Gallego, Perez-Calahorra, Cenarro, Bea, Andres, Horno, Ros, and Civeira, 2012).

6.3. Saturated fat and meat intake

Saturated fatty acids have been linked with CVD via elevating serum cholesterol levels since the Seven Countries Study of the 1950"s and 1960"s (Keys, 1970). Ruminant animals in general can produce substantial amounts of saturated fatty acids due to biohydrogenation of dietary lipids in the rumen (Noble, 1981). Although the SFAs, lauric, myristic and palmitic acids are considered to be detrimental to human health due to their association with increased LDL cholesterol levels (Hu, Stampfer, Manson, Ascherio, Colditz, Speizer, Hennekens, & Willett, 1999), shorter chain forms and stearic acid (C18:0) are considered neutral (Kelly, Mann, Turner, & Sinclair, 1999; EFSA, 2010). The major monounsaturated fatty acid (MUFA) found in meat, oleic acid (C18:1cis) has also been shown to be either neutral or mildly hypolipidaemic in terms of reducing blood cholesterol and triglycerides (EFSA 2010), while the range of polyunsaturated fatty acids (PUFA) found in meat are also mildly hypolipidemic and essential to growth, development, and disease prevention in animals and humans (Simopoulos, 1999; Ponnampalam, Giri, Pethick, & Hopkins, 2014). In the past forty years numerous fatty acid feeding trials have been conducted in human subjects and algorithms developed relating specific fatty acid consumption to changes in both serum total and LDL cholesterol. A range of these algorithms are detailed below:

$$\Delta\text{LDL} - \text{Chol} = 2.6\,\Delta(\text{C14: 0}) + 2.9\,\Delta(\text{C16: 0}) - 0.5\,\Delta(\text{C18: 0}) - 0.7\,\Delta(\text{PUFA})$$

(Derr, Kris-Etherton, Pearson, & Seligson, 1993).

$$\Delta\text{LDL} - \text{Chol} = 0.0378\,\Delta(\text{12: 0} + \text{14: 0} + \text{16: 0}) + 0.0018\,\Delta(\text{18: 0}) - 0.0178\,\Delta(\text{MUFA}) - 0.0248\,\Delta(\text{PUFA})$$

(Yu, Derr, Etherton, & Kris-Etherton, 1995)

$$\Delta\text{LDL} - \text{chol} = 0.01\,\Delta(\text{12: 0}) + 0.071\,\Delta(\text{14: 0}) + 0.047\,\Delta(\text{16: 0}) + 0.043\,\Delta(\text{TRANS F}) + 0.025\,\Delta(\text{TRANS V}) - 0.0044\,\Delta(\text{18: 1}) - 0.017\,\Delta(\text{18: 2, 18: 3})$$

(Müller, Kirkhus, & Pedersen, 2001).

Where (Δ) the change in LDL cholesterol is expressed in mmol/L and the change (Δ) in intake of fatty acids is expressed in energy% (percent of calories), F = Fish; V = Industrially produced trans fat,

Various versions of these algorithms have been developed as shown above, with slight differences. However several general trends have been observed: (i) The saturated fatty acids: lauric, myristic and palmitic have been associated with elevated LDL cholesterol levels. (ii) The shorter chain saturates (C6-C10) and stearic acid, have usually shown no effect, though in some cases stearic acid has been associated with a slight LDL cholesterol lowering effect. (iii) Industrially produced trans fatty acids are hypercholesterolemic, though ruminant trans fatty acids show little effect on LDL cholesterol levels. (iv) Monounsaturated fatty acids generally have been shown to have a neutral effect on serum cholesterol. (v) Polyunsaturated fatty acids in general have a small cholesterol lowering effect.

Because the main lipid present in lean meat is structural membrane bound phospholipid, the proportion of saturated fatty acids tends to be less than the combined MUFA and PUFA, furthermore almost half the saturated fatty acid in lean meats is stearic acid, with only low levels of both myristic and palmitic acids in membrane phospholipids and the absence of lauric acid. Hence from a cholesterol-heart disease risk view point, 'lean' meats do not present a risk and never have, despite many years of commentary by poorly informed "experts" to the contrary. Apart from the lobby groups with vested interests or passionate "beliefs" regarding the evils of meat, this common message has been based on epidemiological studies where it is almost impossible in dietary data collection tools to separate lean meat from meat products and processed meat, which can often be associated with high fat levels, either intramuscular (marbled through the meat - generally from over feeding with grain) or as attached fat or in some meat products added fat from either an animal or vegetable source rich in saturated fat. Unfortunately these studies have to by necessity, use quite blunt tools in determining food intake, usually in the form of self administered food recalls or dietary intake records that are poor at both measuring consumption quantity, but more importantly, in identifying foods in detail. Furthermore such studies can only be correlational and cannot show cause and effect, even though in the community at large that is how they are interpreted and communicated.

It is undeniable however that storage animal fat, in the form of triglycerides (both intramuscular and attached) is high in energy content compared with protein or carbohydrate and can be high in saturated fatty acids and myristic and palmitic acids in particular (approximately 13 g/100 g fat, [NUTTAB 2010]). The avoidance of visible animal fat is a wise health choice, although some intramuscular fat (marbling) is an advantage in terms of the eating quality of the meat (Pethick, Ball, Banks, & Hocquette, 2011). Hence this becomes a balancing act between health aspects of meat and eating quality, a balance that should involve absolute minimal levels of attached fat (through trimming) and a conservative level of intramuscular fat.

It is also relevant when considering the contribution of meat to total fat and saturated fat in the diet, to put it into perspective by looking at relative contribution levels from a range of foods. In the Australian situation it has been shown that lean red meat cuts in general are only a minor contributor of total and saturated fatty acids in the diet (Mann, Ashton, O'Connell, Sinclair, & Kelly, 2006). Lean red meat was shown to contribute ~4.2% of total fat and approximately 1.6 g of saturated fat into the daily diet of Australian adults. Hence there is no evidence to suggest moderate consumption of lean meat is likely to raise the risk of heart disease.

Other foods such as spreads and oils (20.2%), fast foods and take away foods (18.2%) contributed far higher quantities of fat and saturated fat to the diet (Mann, Ashton, O'Connell, Sinclair, & Kelly, 2006). One of the major contributors of fat/oil in the Australian diet is palm oil, used widely in processed food formulation and deep frying of take-away food products. It contains approximately 41% by weight, myristic and palmitic acid (NUTTAB 2010), in comparison to lean meats < 1%

and lean meat/fat composites at < 14%. A typical potato crisp product for instance when prepared with palm oil would have a total of 15 g of the three hypercholesterolemic saturated fatty acids combined (NUTTAB 2010 - Potato crisp plain, unsalted) in a 100 g sample. This is > 15 times the level encountered in a similar weight of lean meat. Thus four to five potato crisps with a combined weight of 6–7 g would contain the same level of these saturated fatty acids as 100 g of lean meat.

6.4. Meat and cancer

Cancer is now one of the world's most common causes of death, rivalling CVD as the highest killer in western societies (Cancer Council of Australia, http://www.cancer.org.au/about-cancer/what-is-cancer/facts-and-figures.html). A number of observational studies indicate that eating large amounts of red meat may increase the risk of colon cancer over time (Norat et al., 2005), however certainly not all studies support this view (Flood, Velie, Sinha, Chaterjee, Lacey, Schairer, & Schatzkin, 2003). Once again the studies involved are observational studies, which cannot prove that meat intake actually causes cancer, but rather establish or identify a possible relationship. As cancer lacks reversible biomarkers, doing dietary intervention trials in humans, as we do with CVD risk studies, is not possible. However from animal studies we know that several substances found in, or associated with red meat may possibly increase the risk of cancer in humans, whether these substances actually have the same affect in humans is not clear and whether the quantities consumed from meat reach dosage levels indicated in animal studies as 'of concern', is still doubtful. These substances include: Heterocyclic amines (HCA), a class of cancer-causing substances (Tasevska, Sinha, Kipnis, Subar, Leitzmann, Hollenbeck, Caporaso, Schatzkin, and Cross, 2009) formed when meat is exposed to very high temperatures, such as during frying, baking or grilling (Knize, & Felton, 2005). HCAs can be found in relatively high amounts in well-done and overcooked meat, which may increase the risk of various cancers, including colon cancer, breast cancer, and prostate cancer (Zheng, & Lee, 2009). Although there is no clear-cut proof that meat intake causes cancer, it seems sensible to avoid eating high amounts of overcooked meat.

7. Pasture feeding and fat profile in meat

In Australia today consumers trim most external fat from meat cuts, with subsequent nutritional analyses showing meat cuts to be relatively lean, with total fat content < 7% raw weight and often as low as 2% of raw weight, (Williams, Droulez, Levy, and Stobaus, 2007). An important contributor to the leanness of muscle meat in Australian animals (particularly cattle and sheep) is that almost all animals are pasture (grass) fed for most of their lives, although most are given short periods of grain finishing before slaughter (Williams, 2007). On average saturated fatty acids make up approximately, 40% of total fatty acids in the lean component and 48% in the fat component of lamb, with similar proportions of palmitic and stearic acids (Williams, 2007). PUFAs range from 11% to 29% of total fatty acids in the lean portion of meats, mostly linoleic acid (18:2n6) an omega-6 PUFA, with traces of other omega-6 PUFA such as arachidonic acid and variable but small amounts of omega-3 PUFA, including alpha-linolenic acid (18:3n3) and the LC omega-3 fatty acids: EPA, Docosapentaenoic acid (DPA, 22:5n3) and DHA. Pasture fed red meat is a better source of omega-3 fats than grain fed, and this explains the better fatty acid ratio in Australian red meat compared to that in the US, where there is extensive grain feeding (Marmer, Maxwell, & Williams, 1984; Sinclair and O'Dea, 1987; Mann, 2005). As red meat is consumed by Australians in greater proportions than fish, it actually makes a greater contribution to intake of LC omega-3 PUFA, in the Australian diet (Meyer, Mann, Lewis, Milligan, Sinclair, & Howe, 2003; Howe, Meyer, Record, & Baghurst, 2006; Williams, 2007).

The importance of omega-3 PUFA in the human diet should not be underestimated in terms of health outcomes (Simopoulos, Leaf, & Salem, 1999). They are formed *in vivo* in the ruminant animal via sequential chain elongation and desaturation metabolic steps (Barcelo-Coblijn, & Murphy, 2009), from the precursor omega-3 fatty acid, alpha-linolenic acid (ALA, 18:3n3) found in the chloroplasts of green foliage (Bolton, Wharfe, & Harwood, 1978). Hence the poor levels of these compounds in grain fed animals (Mann, 2005), as ALA is not found to any extent in seed grains, thus, meat from grain-fed animals does not provide omega-3 fatty acids but it is rich in omega-6 fatty acids, particularly linoleic acid (Delport, & Schönfeldt, 2007).

8. Animal foods and brain health

Research in brain development, function and disease states is revealing the important role of a range of nutrients, not historically strongly linked with the brain. Four critical nutrients gaining prominence in this area of research are either obtained only from animal foods or are more bioavailable when consumed in animal foods, though it must be remembered that there is considerable variability in the level of these nutrients in different types of animal foods, turkey and chicken being good examples of meats with relatively low iron content for example when compared with red meats. These four nutrients are: zinc, iron vitamin B12 and the LC omega-3 PUFA, EPA and DHA.

8.1. Zinc and Iron

Zinc deficiency in animals for instances can lead to: (i) structural malformations in the brain, (ii) changes in enzymes and proteins important for neurotransmission and (iii) behavioral problems, including reduced attention, activity, memory and ability to learn. In humans, low maternal zinc intake during pregnancy and lactation are associated with neonates (6 months) having: (i) less focused attention and (ii) decreased motor function. Zinc supplementation in low birth weight infants leads to increased activity and functionality, while in older children there is evidence of improved neuropsychological functions (Bhatnagar, & Taneja, 2001). Although zinc is widely distributed in foods, its absorption from animal foods is greater than from plants particularly due to its association with proteins in the animal tissue and due to inhibition by phytates and some other constituents of plants (Zheng, Mason, and Rosenberg, 1993; Kristensen, Hels, Morberg, Marving, Bugel, & Tetens, 2006). It has also been established that zinc status is lower in people with depression compared to controls, with severity of symptoms worsening with lower levels of serum zinc. In women with iron deficiency, serum zinc levels have also been found to correlate with mood (Gibson, Heath, & Szymlek-Gay, 2014). In a study examining zinc status and cognitive function of pregnant women in Ethiopia, results of the Raven's Coloured Progressive Matrices (RCPM) Test in third trimester pregnant women showed a clear and significant correlation of RCPM score and serum zinc levels (Stoecker, Abebe, Hubbs-Tait, Kennedy, Gibson, Arbide, Teshome, Westcott, Krebs, & Hambidge, 2009). In a study in children in rural Kenya the dietary micronutrients iron and zinc were found to be associated with higher cognitive function gains. This study was a two year randomised longitudinal feeding trial, with four dietary supplementation arms (control [no food supplement], vegetarian supplement, milk supplement, meat supplement). Iron intake correlated with RCPM score and zinc intake was associated with higher gains in Digit Span-Total Test. The meat supplemented group had the greatest increase in RCPM scores ($p < .01$) (Gewa, Weiss, Bwibo, Whaley, Sigman, Murphy, Harrison, & Neumann, 2009).

An excellent review and evaluation of iron studies related to mental health by Grantham-Mcgregor and Ani (2001) outlined a range of findings on the effect of iron deficiency on children's cognition and behaviour. These included: (i) Most correlational studies show an association between iron deficiency anemia (IDA) and poor cognitive and

motor development, along with behaviour problems. (ii) Longitudinal studies indicate that children with IDA in infancy continue to have poor cognition, behaviour problems and learning difficulties into middle childhood. (iii) Short term iron treatment trials for toddlers (< 2 years old) show little benefit. (iv) For children > 2 years old, the evidence is more convincing for iron treatment.

Iron status has also been found to be a significant factor in cognitive performance in women of childbearing age, affecting processing speed and accuracy of cognitive function (Murray-Kolb, and Beard, 2007). Other studies have also shown links between diet, iron deficiency (and zinc) with brain function in various population groups, some important findings include: (i) Low consumption of foods rich in bioavailable iron (Fe) and zinc (Zn) such as meat, particularly red meat, and high consumption of foods rich in inhibitors of Fe and Zn absorption, such as phytate, certain dietary fibres and calcium, cause iron and zinc deficiencies. Unfortunately food composition tables in general fail to acknowledge an acceptance for a higher factor for heme iron bioavailability and rate its value with inorganic iron, an area that needs to be addressed with a concerted effort by meat scientists. Neuropsychologic impairment is one of several potential outcomes of these deficiencies (Sandstead, 2000). (ii) Iron deficiency anemia is a significant factor in cognitive performance in adult populations, and can be partially reversed by iron supplementation (Khedr, Hamed, Elbeih, El-Shereef, Ahmad, and Ahmed, 2008). (iii) Iron deficiency is widespread and associated with cognitive defects, while zinc deficiency has been linked to low activity and depressed motor development in children. While vitamin B_{12} deficiency has been linked with cognitive problems in the elderly. All three deficiencies are more prominent in those consuming diets poor in animal source foods (Black, 2003).

8.2. Vitamin B12

Vitamin B12 is the generic descriptor for a group of cobalt containing corrinoid compounds. The natural form hydroxycobalamin, is produced by bacteria, usually in the gut of animals. However it is readily converted in the body to the cyanocobalamin form. In the case of ruminant animals, it is absorbed from the gut and present in the tissues, particularly the organs (Gille, & Schmid, 2015). Contrary to many populist articles, it cannot be produced by plants, fungi or even animals as they all lack the necessary enzymes for its formation, hence vitamin B12 tends to be deficient in many vegetarians, particularly vegans, unless supplemental forms or fortified foods are consumed (Pawlak, Parrott, Raj, Cullum-Dugan, & Lucus, 2013).

Active forms of vitamin B12 are converted into a range of coenzyme forms in the human body, which play essential roles in numerous critical metabolic pathways. Methylcobalamin for instance functions as a coenzyme for methionine synthase and hence plays a critical role in conversion of homocysteine into methionine by transfer of a methyl group from methyl-tetrahydrofolate (Chen, Crippen, Gulati, & Banerjee, 1994). While 5′-deoxyadenosylcobalamin functions as a coenzyme for methylmalonyl-CoA mutase, involved in amino acid and odd-chain fatty acid metabolism in mammalian cells (Fenton, Hack, Willard, Gertler, & Rosenberg, 1982). Vitamin B12 is particularly critical for the synthesis of fatty acids in nerve cell myelin and, in conjunction with folate, for DNA synthesis. Adequate intake of vitamin B12 is also essential for normal blood function and neurological function. Vitamin B12 deficiency has also been connected to reactions related to methylmalonyl-CoA mutase, which is needed to convert methyl-malonyl CoA into succinyl-CoA, a key molecule in the TCA cycle. Failure of which leads to elevated levels of methyl-malonic acid (MMA), a myelin destabilizer. Similarly failure of the methylcobalamin coenzyme role in methionine formation leads to elevated homocysteine levels, which has a range of negative health implications (Mann, Li, Sinclair, Dudman, Guo, Elsworth, Wilson, and Kelly, 1999). A range of mental health related issues have been linked to vitamin B12 deficiency, including: fatigue, depression, poor memory, mania (Sethi, Robilotti, & Sadan, 2005) and psychosis (Masalha, Chudakov, Muhamad, Rudoy, Volkov, & Wirguin, 2001). A broad range of brain function studies have been conducted showing the critical role of adequate vitamin B12 levels in the body.

As early as the 1990's studies were showing the effect of vitamin B12 on brain myelination. MRI scanning of the brain was showing severe brain atrophy with signs of retarded myelination, the frontal and temporal lobes being most severely affected, in patients with insufficient intake of vitamin B12 (Lövblad, Ramelli, Remonda, Nirkko, Ozdoba, and Schroth, 1997). Animal studies at the time were also showing clear impairment of the methyl-cobalamin dependent methionine synthetase reaction as the important basic defect in a range of nervous system diseases. Accumulation of adenosyl-homocysteine was common and resulted in a fall in the adenosyl-methionine: adenosyl-homocysteine methylation ratio, and this change was believed to be the cause in defective methylation and demyelination in the nervous system (Metz, 1992). Human studies also showed inhibition of the vitamin B12-dependent enzyme, methionine synthase, leads to a rapid fall in s-adenosylmethionine (SAM) in the brain, leading to reduced neurotransmitter production and reduced methylation of nerve myelin (Weir, & Scott, 1995). In older adults low vitamin B12 status correlates strongly with rapid cognitive decline (Clark, Birks, Nexo, Ueland, Schneede, Scott, Molloy, and Evans, 2007). In adolescents there is evidence of impaired cognitive performance in individuals with marginal cobalamin status (Louwman, van Dusseldorp, van de Vijver, Thomas, Schneede, Ueland, Refsum, & van Staveren, 2000). In a study by Tangney, Tang, Evans, & Morris (2009), serum vitamin B12 concentrations was a more important risk factor for cognitive decline when compared to serum homocysteine concentrations, particularly in older populations, with the combination of the two showing the greatest effect on decline in cognitive function. In studies involving clinical investigation, MRI scanning and cognitive evoked potential, vitamin B12 deficiency has been linked to cognitive impairment and several neurological syndromes (Kalita, & Misra, 2008). In fact neurological complications appear to be an early manifestation of vitamin B12 deficiency (Atanassova, Chalakova, Goranov, Ilieva, Sotirova, & Massaldjieva, 2004). Vitamin B12 status below the normal range has also been shown to be associated with severity of white-matter lesions, especially periventricular lesions. Given the absence of an association with cerebral infarcts, it is hypothesised that this association is explained by effects on myelin integrity in the brain rather than through vascular mechanisms (de Lau, Smith, Refsum, Johnston, & Breteler, 2009). Vitamin B12 treatment has been shown to improve frontal lobe and language function in patients with cognitive impairment, but shows only minor success in reversing dementia (Eastley, Wilcock, and Bucks, 2000). In foetal and early childhood development vitamin B12 has been shown to be critically important for brain development and function. Maternal vitamin B12 status in pregnancy also strongly influences cognitive function in offspring (Bhate, Deshpande, Bhat, Joshi, Ladkat, Watve, Fall, de Jager, Refsum, & Yajnik, 2008).

8.3. Omega-3 fatty acids

As outlined previously, the omega-3 fatty acids are a family of PUFAs of varying chain length and functionality in the human body. Plants are a useful source of the principle shorter chain fatty acid ALA, which has numerous health effects in the human body. However, the longer chain forms of omega-3 fatty acids EPA and DHA, which are critical agents in terms of cell membrane structure and precursors for a range of vasoactive and inflammatory related eicosanoids, are only found in marine organisms and some land herbivores (Lane, Derbyshire, Li, & Brennan, 2014). A related long chain omega-3 fatty acid is docosapentaenoic acid (DPA, 22:5n3), which is found in grass fed red meat in higher levels than EPA and DHA (Mann, 2005). Limited research data is available on this fatty acid, though it may have beneficial effects on platelet function (Mann, O'Connell, Baldwin, Singh &

Meyer, 2010). There is also a growing body of evidence indicating both independent and shared effects of EPA, DPA and DHA (Dyall, 2015). The chain elongation, desaturation pathway common in most animals, should theoretically allow the conversion of ALA to EPA and DHA, however in humans this pathway is very inefficient and hence little plant derived ALA finds its way to the LC forms of omega-3 fatty acids (Baker, Miles, Burdge, Yaqoob, & Calder, 2016). Consumption of red meat as well as fish has been shown to raise tissue levels of both EPA and DHA (Mann, Pille, Johnson, Warrick, Sinclair, Reder, & Lorenz, 1997; Li, Mann, Sinclair, Turner, Ball, Kelly, & Wilson, 1999).

The scientific literature contains abundant evidence of the critical role of adequate intake of LC omega-3 fatty acids, particularly EPA and DHA in brain development and function, in both animal and human studies. Animal studies, in mice and primates, show that DHA-depleted diets impair learning and memory, and that re-feeding DHA reverses these impairments (Reisbick, Neuringer, Hasnain, & Connor, 1994; Gamoh, Hashimoto, Hossain, & Masumura, 2001). Feeding rats diets high in DHA improves both working memory (short-term memory) and reference memory (longer-term) in both old and young animals (Gamoh, Hashimoto, Sugioka, Shahdat Hossain, Hata, Misawa, and Masumura, 1999). Studies in human infants show a strong correlation between the intake of omega-3 oils, especially DHA, and cognitive function, visual acuity, and overall brain development (Birch, Garfield, Castaneda, Hughbanks-Wheaton, Uauy, & Hoffman, 2007). In a review by Dangour and Uauy (2008), on LC omega-3 fatty acids for optimal function during brain development and ageing, they highlighted evidence of several aspects of the importance of these fatty acids: (i) Provision of EPA and DHA in preterm babies improved retinal electrical responses and brain cortex related visual acuity maturation, similar to human milk fed infants; (ii) Results in young children suggested that neurodevelopment and cognitive abilities are also enhanced by early provision of EPA and DHA through breast milk or DHA-fortified foods; (iii) Cross-sectional surveys reported that higher EPA and DHA consumption was associated with reduced risk of impaired cognitive function; (iv) Prospective cohort studies indicated that increased fish consumption was associated with decreased risk of dementia in older people. In a psychiatric study conducted in the US military, Sublette, Hibbeln, Galfalvy, Oquendo, and Mann (2006) found that suicide risk was linked to low levels of the LC omega-3 fatty acids levels in the blood and cell membranes.

Total EPA and DHA concentrations have also been found to be associated with benefits for cognition in the elderly (Whalley, Deary, Starr, Wahle, Rance, Bourne, & Fox, 2008). Dementia has also been associated with low plasma LC omega-3 fatty acid concentrations (Cherubini, Andres-Lacueva, Martin, Lauretani, Iorio, Bartali, Corsi, Bandinelli, Mattson, & Ferrucci, 2007). In the Atherosclerosis Risk in Communities (ARIC) study, it was shown that an increase of one standard deviation in dietary LC omega-3 fatty acids (% of energy) and balancing LC n-3/n-6 decreased the risk of 6-year cognitive decline in verbal fluency (Beydoun, Kaufman, Sloane, Heiss, & Ibrahim, 2008). In a middle aged population group LC omega-3 PUFA consumption was associated with a reduced risk of impaired cognitive function (Kalmijn, van Boxtel, Ocké, Verschuren, Kromhout, & Launer, 2004). In a study on cognitive ageing and childhood intelligence it was found that total erythrocyte LC omega-3 fatty acids and the ratio of DHA to arachidonic acid was associated with better cognitive function in late life (Whalley, Fox, Wahle, Starr, & Deary, 2004). Finally, associations in conjunction with intervention trials showing enhanced cortical visual acuity and cognitive outcomes in preterm and term infants fed DHA, suggest that perinatal deficits in brain DHA accrual may represent a preventable neurodevelopmental risk factor for the subsequent emergence of psychopathology (Mcnamara and Carlson, 2006).

Apart from the studies on these four specific nutrients rich in animal foods (iron, zinc, vitamin B12 and LC omega-3 fatty acids, there have been numerous observational studies in developing countries on animal source foods in general that show diet quality in terms of consumption level of animal source foods is positively associated with cognitive development in children. A 21 month, four arm supplemented feeding trial in Kenya with grade 1 level students ($n = 555$), showed significant positive effects on cognitive performance outcomes by children in the meat supplemented arm relative to other groups (Whaley, Sigman, Neumann, Bwibo, Guthrie, Weiss, Alberz, and Murphy, 2003). While an Egyptian study in school aged children showed a diverse group of nutrients predicted developmental outcomes related to verbal ability, activity level and behaviour. Common to all the nutrients investigated was the fact that they were most available in foods of animal origin (Wachs, Bisry, Moussa, Yunis, McCabe, Harrison, Sweifi, Kirksey, Galal, Jerome, & Shaheen, 1995).

9. Summary and conclusions

Once our pre-agricultural, hominin ancestors' left the wetland jungles of Africa for a drier savannah grassland existence some 3–4 million years ago, they lived a hunter-gatherer existence with an eclectic food intake pattern, where animal foods became the dominant source of not only energy, but also protein, LC fatty acids, vitamin B12, iron and zinc. Adaptations to such a dietary pattern accumulated in our bodies over time, and a certain level of dependence developed for at least some animal food in the diet to provide protein and specific micronutrients, although the driving requirement for ASF for energy in a hunter-gatherer existence is no longer relevant in modern society where food energy is plentiful. It can be argued that the modern Western divergence from our evolutionary dietary pattern involving high meat intake to a more grain and processed food based diet, forms the basis of lifestyle diseases that we now face (Cordain, Brand-Miller, Eaton, Mann, Holt, & Speth, 2000; Cordain, Eaton, Brand-Miller, Mann, Sebastian, Lindeberg, & O'Keefe, 2005). In more recent times research has shown that particular issues arise around brain functionality when animal foods are absent from the human diet and this is primarily evident in children and the elderly. Thus, there is no historical or valid scientific argument to preclude lean meat from the human diet, and a substantial number of reasons to suggest it should be a central part of a well-balanced diet (Mann, 2000). A detailed review of the hominin transition to animal food consumption and its role in the development of our species can be explored in Larsen (2003).

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