Nutrition Discussion Forum

Dietary lipids and evolution of the human brain

I have been asked to comment on an interesting topic that as a nutritional biochemist I know almost nothing about. With this firm subject matter appreciation and conceptual starting point, I have read the publication by Broadhurst *et al.* (1988) with considerable interest. For me it was almost a new speciation event!

Drs Broadhurst, Cunnane and Crawford have posed an intriguing hypothesis wherein they speculate that brain growth in early Homo occurred because of 'brain-specific' nutrition that had an enabling impact on the evolution of early man. The authors cite an association between 'precocious' cultures and the consumption of fish and shellfish. This association of the evolution of intelligent behaviours with consumption of polyunsaturated fatty acids has some merit, particularly as lack of consumption of these components reduces brain size and brain function. From recent research, it is likely that *n*-3 fatty acids may impact on human behaviours. Perhaps these behaviours were important to other cultural aspects of the speciation event?

I find it attractive to agree with the notion posed by Broadhurst *et al.* (1998), but would suggest that the scavenging early hominid needed something more as a single speciation event: perhaps we should look to a change in a gene locus that favoured survival between meals and during different seasonal fortunes. On a metabolic basis, I would suggest that this enabling gene could be one that favoured an efficient conservation of a reserve of extra energy for use later when the 'fishing and clamming' was poor. Perhaps these same genes also represent the evolutionary origins of our present day problems with surplus energy.

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The idea of a genetic basis favouring adipose storage and efficient energy metabolism to enable conservation of reserves during extended periods of energy deprivation or high energy expenditure is a valid point to introduce into Broadhurst *et al.* (1998). In general we agree with Dr Clandinin's comment, and would have discussed this aspect if space had permitted. However, a change in genetic locus favouring adiposity/metabolic efficiency is neither necessary nor sufficient to account for the phenomenal brain expansion of *H. sapiens.* Most mammals will gain body fat and even become obese when allowed free access to high-fat or high-sugar diets, especially when relatively sedentary in captivity. And, as noted in our text, all mammals have a doscosahexaenoic acid (DHA) : arachidonic acid (AA) ratio of approximately 1:1 in central nervous system (CNS) tissue, to the virtual exclusion of other polyunsaturated fatty acids (PUFA).

Despite these physiological similarities, no other mammals have anywhere near the intellectual capacity of humans. Further, very recent research indicates that past work has consistently overestimated the brain volume of hominid species (Conroy *et al*., 1998). If this turns out to be generally accepted, then the exponential increase in encephalization quotient (EQ) as discussed in our paper is even more dramatic, hence a nutritional explanation for our current EQ becomes even more plausible. There is neither enough time nor the requisite fossil evidence for a smooth Darwinian progression towards *H. sapiens*.

Also, for clarification in paleoanthropological terms, 'speciation event' defines what might be a fairly long and ill-defined stretch of time during which a new species apparently arose, and takes into account gradations between named species, and regional and temporal variations in the fossil record. It does not define an overall sharp 'single' speciation event.

The first evidence for modern or near-modern humans at various fossil localities appears from before 300,000 to after 100,000 years ago; however, the earliest occurrences are restricted to Africa. It is certainly possible that genetic modifications in lipid trafficking coupled with ready access to the essential nutrients, long-chain PUFA (LC-PUFA) included, helped various African Homo populations make the precocious leap to modernity.

Broadhurst *et al.* (1998) focus on how hominids with 1–2 million years of very slow, nearly stagnant intellectual growth could have made a quantum leap forward to modern humans. We argue that the evolution into modern humans could not have taken place in a random location on

the planet, and that it required an incidental abundance of balanced LC-PUFA in the diet before intellectual expansion. There must have been enough LC-PUFA available in the diet to (1) provide many generations of hominids with fuel for fetal/infant development as well as childhood and adult needs, (2) allow for the fact that substantial amounts of PUFA would almost certainly have been oxidized for energy requirements (Chen *et al.* 1995; Cunnane & Anderson, 1997), (3) both explain and allow for our inefficient conversion of linoleic acid to AA and α-linolenic acid to DHA.

Adipose storage and efficient energy metabolism have been considered genetically in the nutritional anthropology origins of non-insulin-dependent diabetes mellitus (NIDDM) (Broadhurst, 1997). In the current Western quest to 'eat as much as you want and not gain weight' it is often forgotten that the evolutionary purpose for overeating is to gain weight, and to a greater or lesser degree we are all adapted to do so, or we would never have survived in the past. Our ancestors gained weight voluntarily and lost it involuntarily, the exact opposite of what many of us attempt to do today. Dr Clandinin may be correct in that adiposity is carried to an extreme in humans, especially in females. After our energetically 'expensive' CNS was established, our continual requirement for adequate LC-PUFA to fuel it could be a reason why genotypes favouring adiposity were selected for and retained. Another reason may be that *H. sapiens* is indeed adapted to a littoral environment, since our subcutaneous layer of fat is strongly reminiscent of sea mammals, and is unlike the lean savannah land mammals (Crawford *et al.* 1993).

A tendency to store fat, release insulin, and desaturate/elongate only the minimum amount of dietary 18-carbon PUFA that is metabolically necessary were probably positive factors for humans adapted to Paleolithic foods and constant physical activity, but may be maladaptive today. These are adaptations to a diet (a) low in fat and often energy, (b) relatively high in *n*-3 PUFA and LC-PUFA, (c) raw and unprocessed, and (d) free from high-density carbohydrates, often such that dietary fat rather than carbohydrate was an absolute requirement for energy (Broadhurst, 1997). For example, the incidence of NIDDM in the USA is greater among minorities (Asian, Black, Hispanic, Native American) than Caucasians, even when diet and lifestyle factors are taken into account (Carter *et al.* 1996). In addition, the incidence of NIDDM among Africans, Mexicans, Puerto Ricans, Koreans, Japanese, Chinese, and Filipinos (groups for which adequate data exist) residing in the USA is greater than the incidence in their respective countries of origin (Carter *et al.* 1996). A widely accepted explanation for these observations is genetic 'thriftiness', or a prevalence of genotypes which are adapted to sporadic wild food supplies, and heavy physical labour (Neel, 1962; Ravussin & Bogardus, 1990). As a group, Caucasians have spent the longest time on an agricultural diet, and do tend to metabolize alcohol, dairy products, wheat and other glutinous grains, and evidently sugars, better than other groups. Not coincidentally, Caucasians have the lowest intrinsic incidence of NIDDM (2–3%) and a lower incidence of central obesity. Central obesity is associated with an increased risk of cardiovascular disease and NIDDM, and is very common in aboriginal populations, many of whom were still hunter–gatherers 100 years ago (Szathmary 1994; Kissebah & Hennes, 1995).

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The recent review article on diet and brain evolution by Broadhurst *et al.* (1998) is quite interesting. It basically suggests, once again, that fats, especially long-chain fatty acids, are necessary for brain ontogeny and phylogeny. Unfortunately, their main point related to nutrition and brain evolution omits numerous previous authors, albeit in more concise form (e.g. Chamberlain, 1994*a*,*b*, 1996, 1997). Their concluding tenet that ''brain-specific nutrition affects brain evolution'' is 'old hat', and is apparently gathering a historical following in its rediscovery.

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Professor Chamberlain has commented that our ideas are not new, and that we have practiced 'selective overlooking'. In support of his letter he has provided several personal letters, meeting abstracts and the reference for one published article (Chamberlain, 1996). Professor Chamberlain has referenced prior work by Professor Michael Crawford in both Chamberlain (1996) and Chamberlain (1997). In his letter to the *British Journal of Nutrition* it appears that he has overlooked the fact that Michael Crawford is an author of our paper. Professor Crawford has been writing and lecturing about the relationship between PUFA nutrition and brain evolution for nearly 30 years. By no means do we claim that our basic idea is new. However, no previous authors have put forth a strong multi-disciplinary approach which provides much-needed *quantitation* to this concept. Previous authors have not provided supporting numbers or facts which nutritionists can use to evaluate this idea, such as a percentage of energy from fish which would be reasonable for the hominid diet; a discussion of meat and, especially, fish and shellfish scavenging; a table of the fat contents of fish in the Rift Valley; or a profile of how large and unique the Rift Valley lakes were during our evolution. Many previous authors have not provided an up-to-date discussion of long-chain PUFA metabolism in humans either; in fact they have tended to wave away the argument by referencing Michael Crawford!

The article which we have authored is a review, so it is clearly designed to discuss an established concept as opposed to new data and/or ideas. However, Broadhurst *et al.* (1998) is not a comprehensive review of the so-called 'aquatic ape theory', but rather a discussion designed to help those in the nutritional sciences field understand the very deep ramifications of dietary long-chain PUFA. As it stands we owe a great deal to the editorial staff of the Journal for allowing us to exceed the standard page length. It was not possible to review, discuss, or develop all the contributions published in anthropological journals which might seem relevant to this topic. In order to reduce page length, we severely limited abstracts and papers published before 1990 in our references, and even applied these limitations to our own publications. We note also that many anthropological papers discussing the ecological approach to evolution were reviewed during the research for Broadhurst *et al.* (1998) which, at first glance, appeared to support our thesis, but upon closer examination seem to miss the point by suggesting that *first* the brain evolves to be relatively large and complex, *then* a high-quality food source is located to sustain the exceptional needs of the large brain. Our thesis basically relies on the provision of lacustrine/marine foods by simple gathering and scavenging to enlarge the brain *first*; *then* the larger and more complex brain can seek high quality food with increasing success and sophistication. As we have explained in our text, the true situation is likely to lie between these end members, and there will be feedback loops affecting other organ systems such as the gut (Aiello & Wheeler, 1995), reinforcing the growth of intelligence. However, an underlying theme of our work which has not been captured by previous authors is that geological, paleoanthropological, and nutritional evidence independently agree that evolution into genus Homo (and possibly modern *H. sapiens*) could not have occurred in a random location on the planet.

We therefore thank Professor Chamberlain for bringing his and some other contributions to the attention of the readers.

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