Nutrition Discussion Forum

Dietary lipids and evolution of the human brain

That *Homo sapiens* has a brain that is larger than and functionally superior to those of existing primates and extinct hominoids is not in dispute, although the precise relationship of brain size to functional capacity may be less certain.

The intriguing question for biologists is the nature of the driving force(s) behind this evolution, and here we are definitely in disputed territory.

Broadhurst *et al.* (1998) provide their own speculative proposals for the main factors leading to the expansion of the hominoid cerebral cortex during the last one to two million years. Briefly, they suggest that such expansion has been nutrient-driven. The fully developed and functioning brain, they argue, requires a large amount of preformed long-chain polyunsaturated fatty acids (LCPUFA) of the *n*-3 and *n*-6 families, substances that can only practically be derived from the diet. The authors suggest that the most abundant source of these compounds was fish and argue that ready access to fish would have given early man a distinct advantage over animals that were predominantly or entirely herbivorous. They point out that the great East African Rift Valley, a focal point of hominoid evolution, was characterized by huge freshwater lakes that would have provided the nutrients that they claim were the 'driving force' for the increase in brain size.

How good a case has been made for this proposition?

The propositions in this article are not brand new. In a previous publication (Crawford & Marsh, 1989), Professor Crawford argued generally for a major role for food as a driving force for mammalian evolution. In their review in the January issue of *BJN* (Broadhurst *et al.* 1998), he and his colleagues focus more specifically on the role of LCPUFA in brain development. The authors begin (pp. 3–12) with a fascinating and wide-ranging assessment of the anthropological and paleontological aspects of hominoid evolution in East Africa. It is only on page 12 that they begin to focus on the nutritional and biochemical aspects of brain development, and it is here that my comments will be mainly concerned.

The authors present us with a series of facts that provide the basis for their hypothesis. More than half the weight of the brain is lipid, predominantly phospholipids and cholesterol. A large proportion of the phospholipid fatty acid is composed of the LCPUFA docosahexaenoic (DHA) and arachidonic (AA) acids in roughly equal amounts. Most brains have this composition, so that species differences are quantitative rather than qualitative. Brain contains little linoleic acid (LA) and α-linolenic acid (LNA), generally regarded as the precursors, by elongation and desaturation, of the LCPUFA. The authors cite limited evidence that LA and LNA elongation and desaturation is 'slow and inefficient' in brain, thereby strongly suggesting that access to adequate preformed LCPUFA is essential for proper brain growth and development. The freshwater fish lipid profile has a DHA : AA ratio that is closer to that of human brain than any other common food source. Add to these biochemical 'facts' some speculation about the availability to early hominoids of lake fish in the African Rift Valley at an appropriate span in evolutionary history and we have the basis for the hypothesis of LCPUFA-driven brain development in *H. sapiens*.

A first general comment is that the authors use a number of terms that they assume will be fully understood but do not clearly define. A prime example is 'brain-specific diet' or 'brain-specific nutrition'. The reader is left to presume that this refers to preformed LCPUFA, which provide the 'driving force' for nutrition-driven brain expansion. LCPUFA are found in cellular structures other than brain and cannot be 'specific' in the sense that they are unique to brain. Alternatively, if the word 'specific' is used to imply that it is only these compounds that provided the driving force, then the use of the term begs the question of whether the hypothesis can be sustained and should not strictly be used until the hypothesis can be tested adequately. Neither can 'specific' mean that only the brain of *H. sapiens* contains DHA and AA, since we are told clearly that the brain composition of all forty-two species so far examined is similar. The authors may regard this as a quibble on my part but if readers are to take the authors' ideas seriously, then the utmost rigour needs to be adopted in the use of terminology and the formulation of concepts.

Broadhurst and her co-authors have not considered brain components other than LCPUFA. An organ as large and as complex as the brain requires, in addition to LCPUFA, numerous other essential nutrients that must also be derived from the diet. Without some consideration for their potential role in brain evolution, is the term 'brain-specific' fully justified?

The authors' entire hypothesis rests firmly on the proposition that the brain of *H. sapiens* was able to develop its large size in relation to other hominids, primates and herbivores as a result of a more abundant supply of preformed LCPUFA than was available to other species. The notion that herbivorous animals could obtain their LCPUFA by processes of further elongation and desaturation of LA and LNA (which are abundant to them) is dismissed on the grounds that these metabolic pathways are 'slow and inefficient'. One criticism that may be levelled here is that few publications are cited to support this contention and the authors have a worrying tendency to cite references to conference proceedings and books rather than to peer-reviewed publications. At the present time, the evidence that rates of elongation and desaturation of LA and LNA *in vivo* limit the accumulation of LCPUFA in nervous tissue is at best limited.

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In comparing brain development in different species of animals, the focus needs to be on the total amount of DHA/AA available to the animal (whether derived preformed or by metabolism from LA/LNA) because the proportions of the fatty acids are very similar in the brains of all species. Is the size of a herbivore's brain, which is smaller than a human being's, limited by availability of LCPUFA? I am not alone in rejecting this idea (Pond & Colby, 1990).

Many present-day human beings (and presumably many latter-day individuals also) are strict vegans. It is not immediately apparent that such people are intellectually inferior to their omnivorous counterparts. The authors have not cited evidence to support such a difference and I am unaware of any such evidence. Likewise, for many years of the 20th century in industrialized countries, generations of human babies have been reared primarily and often exclusively on infant formulas that contain minute quantities, if any, of LCPUFA. Is there documentation to demonstrate that such babies developed into adults who were or are intellectually inferior to their primarily breast-fed counterparts? The authors may wish to make a distinction between availability of LCPUFA as an evolutionary driving force over a million or so years and their availability to present-day individuals who are genetically programmed to have a larger brain than other species. Nevertheless, for the individual, the problem of accumulating sufficient LCPUFA to fulfil that potential must still apply.

When one looks at herbivores such as cows, one is struck by the impressive way in which the animal conserves its precious stock of essential fatty acids (EFA), not just in the brain but in all membranous structures of its large body. Huge amounts of EFA are accumulated despite this animal's apparently suicidal habit of destroying a large proportion of its dietary intake of EFA by hydrogenation in its rumen. During evolution it has adapted so as to conserve whatever EFA is available. It should not be surprising that *H. sapiens*, in whom wastage due to biohydrogenation does not occur, is even more successful in such conservation. Furthermore, even though desaturation and elongation may be inefficient, they may be active enough to ensure that, in combination with efficient conservation, supplies meet needs. Might it not be that other environmental pressures provided the driving force for brain size development in *H. sapiens* and that mechanisms for conservation of the necessary LCPUFA adapted to cope with this increased size?

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References

Broadhurst CL, Cunnane SC & Crawford MA (1998) Rift Valley lake fish and shellfish provided brain-specific nutrition for early Homo. *British Journal of Nutrition* **79**, 3–21.

Crawford M & Marsh D (1989) *The Driving Force: Food, Evolution and the Future*. London: Heinemann.

Pond C & Colby R (1990) The fats of evolution. *New Scientist* 27 January, 66–77.

Dietary lipids and evolution of the human brain – Reply by Broadhurst et al.

In his letter, Professor Gurr all but rejects our concept that prolonged access to a rich dietary source of long-chain polyunsaturates (LCPUFA) was a central feature of human brain evolution (Broadhurst *et al.* 1998). Instead, he favours an explanation involving 'other environmental pressures' but provides no suggestions as to what they would be. He complains about a 'worrying tendency to cite references to conference proceedings' but goes on to cite a book review about an earlier, broader discussion of diet and human evolution (Crawford & Marsh, 1989) as his only evidence to justify his hesitancy; surely if we are in 'disputed territory', there would be a few peer-reviewed publications in addition to a book review to establish that dispute.

The concept proposed by Crawford & Marsh (1989) was that limited availability of two LCPUFA, docosahexaenoate (DHA; 22:6*n*-3) and arachidonate (AA; 20:4*n*-6), places severe constraints on early brain development, and probably was a significant determinant of human brain evolution. Later, we extended this concept to include the potential importance of trace elements and energy availability and proposed that a shore-based environment rich in shellfish was probably sufficient to accommodate the nutritional requirements for human brain evolution; initially, fishing would not have been possible nor was it necessary (Cunnane *et al.* 1993). In the present review (Broadhurst *et al.* 1998), we focus on the plausibility of the Rift Valley providing not only the paleoanthropological and geological evidence but also the nutritional/ecological evidence in a fresh-water, proto-oceanic environment. We also try to link this evidence to modern nutritional studies to provide a viable explanation for the emergence of human intellectual capacity and its ongoing vulnerability.

We concede that the term 'brain-specific nutrition' is ambiguous. No nutrients are truly brain-specific; even DHA is more specific to the eye than to the brain. The term emerged from recognizing that (i) the growing brain is especially sensitive to the supply of LCPUFA, (ii) unlike other organs, the brain does not accumulate appreciable quantities of 18-carbon PUFA, and (iii) the brain is vulnerable to the absence of other nutrients such as Zn, Cu, I and Fe that are involved in PUFA metabolism; like LCPUFA, abundance and bioavailability of Zn, Cu and I are greater from fish, shellfish and meat than from terrestrial plants.

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