SHORT REPORT

Docosahexaenoic Acid and Shore-Based Diets in Hominin Encephalization: A Rebuttal

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Abstract

Carlson and Kingston (2007) propose that pre-formed dietary docosahexaenoic acid (an omega-3 fatty acid in fish) did not have a significant role in hominin encephalization. Their position hinges on claiming that humans are able to make sufficient docosahexaenoic acid from the plant-based 'parent' omega-3 fatty acid – α-linolenic acid. They also suggest that hominin fish consumption occurred too late to have materially influenced encephalization. We quantify here a summary of the published data showing that humans cannot make sufficient docosahexaenoic acid to maintain normal infant brain development. We also provide evidence that the fossil record shows that some of the earliest hominins were regularly consuming fish. Hence, we reject Carlson and Kingston's (2007) position and reiterate support for the concept that access to shore-based diets containing docosahexaenoic acid was necessary for hominin encephalization beyond the level seen in the great apes.

Carlson and Kingston (2007) dismiss the concept that access to the pre-formed dietary omega-3 fatty acid - docosahexaenoic acid (DHA) - played a significant role in human brain evolution. They do not challenge the crucial role of DHA in mammalian brain development and function but propose that in order to fulfill its neurological functions, sufficient DHA can be made in the body from its plant-based precursor, α-linolenic acid (ALA). The assertion that sufficient DHA can be synthesized by humans and that dietary DHA would therefore not be important in human brain evolution has been recently made elsewhere (Langdon, 2006). However, the literature does not support the claim of human self-sufficiency for DHA synthesis, so we wish to set that record straight and reassert the view that a dietary source of DHA is now and always was important for human brain development, function and evolution (Crawford, 2006; Cunnane, 2005).

Insufficiency of ALA for normal brain development

Humans have the enzymes to make DHA from ALA, but the majority of studies on human adults show that this pathway is *extremely* inefficient, i.e. conversion is rarely >0.5% (Table). In practical terms, 0.5% conversion means that in order to obtain the average recommended DHA intake for North Americans (~300 mg/d), one would have to consume daily about 60 g of ALA. This amount of ALA exceeds actual ALA intake by 30-40 fold and is almost equivalent to the recommendation for total daily fat intake. An intake of 100 mg/d of DHA is considered to be too low by organisations such as the American Heart Association and the International Society for the Study of Fatty Acids and Lipids, but even if 100 mg/d of DHA was enough, it is still essentially impossible to regularly consume 20 g/d of ALA in order to convert 0.5% of it to DHA. Furthermore, the multiple desaturation-chain elongation steps needed to convert ALA to DHA require several nutritional co-factors, notably zinc, iron and

vitamin B_6. Insufficient intake of these nutrients compromises even further this already inefficient pathway making it completely unreliable (Cunnane 2005).

Two autopsy studies have compared brain DHA content in babies consuming or not consuming pre-formed dietary DHA. They address the real issue around conversion which is – how much newly made DHA gets into the infant brain? Both these studies show that brain DHA is about 50% lower in babies not receiving pre-formed dietary DHA (Cunnane et al., 2000; Farquharson et al., 1992; Makrides et al., 1994). Hence, during the first six months of life, the developing brain of infants not provided with pre-formed dietary DHA accumulates about 50% less DHA than when pre-formed DHA is provided. Despite its structural similarity to DHA and in contrast to the claim by Carlson and Kingston (2007), docosapentaenoic acid - the omega 6 fatty acid analogue of DHA - cannot prevent the functional deficit of low DHA, either in the brain or in the retina (Crawford, 2006).

In support of their position, Carlson and Kingston (2007) point to the Institute of Medicine's recommendations that ALA is a sufficient source of omega 3 fatty acids. However, they neglect to mention that the Food and Drug Administration of the USA now requires DHA to be included in infant formulas. In Europe, regulatory agencies equivalent to the FDA have required a dietary source of DHA for infants for at least a decade now. Virtually every professional association representing pediatricians strongly advocates the FDA's position about the need for preformed DHA in infant formulas. Hence, the Institute of Medicine's position is not supported by any other major regulatory agency in the USA or elsewhere.

Archeological evidence for aquatic resource use by early Homo

Carlson and Kingston (2007) note that the earliest tools used to catch fish (bone harpoon points, etc.) are found in fossil beds of anatomically modern hominins. The implication is that sufficient encephalization to evolve tool making skills occurred before

substantial consumption of fish. While tool making for fishing is, indeed, relatively recent, evidence that much earlier hominins fished *without using tools* has in fact been presented (Stewart, 1994; Stewart, 2006). Three lines of evidence at Early Pleistocene archaeological sites suggest abundant and intentional hominin fish intake: (i) very biased skeletal representations, (ii) presence of only one taxon (*Clarias)*, and (iii) at an Olduvai Gorge site, presence of identifiable cutmarks on *Clarias* cranial bones. These sites also contain early tools and other faunal remains. Archaeological sites in middle – late Pleistocene Egypt contain mounds of tens of thousands of *Clarias* bones procured by hominins when the fish were spawning.

Clarias is a common, slow-moving, shallow water catfish that could easily caught by hand a million years ago, much as is still done today in these areas. Accounts also exist of baboons, hyenas and other carnivores catching and eating *Clarias* while on spawning runs. Not only are *Clarias* sufficiently abundant and accessible in freshwater lakes and rivers but with about 230 mg of DHA per 100 g tissue, they are an excellent source of pre-formed DHA. Hence, contrary to Carlson and Kingston (2007), there is good fossil evidence that some early hominins fished frequently and by hand, thereby gaining access to DHA and other brain-selective nutrients long before extensive encephalization or development of tools to help in this process.

More to fish and shellfish than DHA

In addition to the importance of fish and shellfish as sources of pre-formed DHA for neurological development, there is a broader perspective to the role shore-based foods in human brain evolution that relates to the developing brain's susceptibility to other nutrient deficiencies (Crawford, 2006; Cunnane, 2005). Like DHA, iodine and iron are needed for energy metabolism and normal human brain development. Endemic clinical and sub-clinical

iodine deficiency is present in about 20% of humans worldwide. The global problem of iodine deficiency primarily affects people not regularly consuming fish or shellfish. In people not regularly consuming 'shore-based' foods (fish, shellfish, turtles, frogs, plants, etc.), iodine adequacy depends heavily on iodized table salt, without which clinical hypothyroidism, subnormal cognitive development and cretinism would still be the public health nightmare they were prior to iodization of table salt.

The scale and impact of endemic iodine deficiency is rivalled only by iron deficiency. Unlike iodine, iron is not yet legislated into the food supply but great efforts are being made to find a simple, cheap, reliable way to provide iron supplements where they are needed. Iron and other key metal nutrients needed for brain development and function (zinc, copper, selenium) are more bioavailable from fish and shellfish than from plant-based diets where their absorption is impaired by phytates and other 'antinutrients'. Plant-based diets rich in staples like cassava or soy are not only a very poor source of iodine but they also contain goiterogens which inhibit iodine absorption. Hence, though it is not impossible, it is much more difficult to obtain adequate amounts of brain-selective minerals from an exclusively plant-based diet than it is from one containing shore-based foods including fish. In other words, it is not only a source of pre-formed dietary DHA that would have facilitated brain evolution in hominins consuming fish and shellfish; side-stepping the vulnerability to cognitive deficits exposed by plant-based diets chronically deficient in iodine and iron was at least as important (Crawford, 2006; Cunnane, 2005).

ALA is ubiquitous but marked encephalization is rare

The ubiquitous presence of ALA in edible plants remarked by Carlson and Kingston (2007) stands in stark contrast to the rare encephalization of the mammalian brain beyond the level seen in the great apes. Only two species have brains substantially encephalized over

those of the great apes – humans and dolphins. Dolphins are marine carnivores with a high intake of DHA and other brain selective minerals. We contend that despite the abundance dietary ALA, negligible access to pre-formed dietary DHA and brain-selective minerals is an important metabolic and structural constraint on primate brain development and evolution. By routinely exploiting a shore-based habitat and diet during the past two million years, hominins were able to break through these constraints on primate brain size and function. This exploitation of shore-based habitats was at first fortuitous, but gradually led to more intentional and organised coastal occupation and exploration (Crawford, 2006). As long as these shorelines were habitable and provided some drinking water, this exploitation involved both freshwater and saltwater bodies of water.

One of the hallmarks of the mammalian brain is its developmental vulnerability to inadequate nutrition. Indeed, this vulnerability seems to increase in direct proportion to the degree of encephalization. The demographics and scale of iodine and iron deficiency demonstrate that despite encephalized brains, humans only fully escape this developmental vulnerability if they consume shore-based nutrients, particularly iodine, iron and DHA. This is the crucial point - from the start of hominin brain evolution, shore-based diets continue to provide the only reliable source of brain-selective nutrients that mask the mammalian brain's developmental vulnerability.

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Table

Dietary supplementation studies reporting conversion of α-linolenic acid (ALA) to docosahexaenoic acid (DHA) in healthy adult humans[1]

Reference	ALA ($g \cdot d^{-1}$)	Δ % DHA
Nordstrom et al., 1995	9.6	+ 0.5%
Harper et al., 2006	3	+ 4%
Szapary et al., 2007	40	TR
Cunnane et al., 1995	9	TR
Li et al., 1999	3.7	TR
Li et al., 1999	15.4	TR
James et al., 2003	1.5	TR
Finnegan et al., 2003	4.5	TR
Wallace et al., 2003	3.5	+ 2%
de Groot et al., 2004	2.8	TR
Goyens et al., 2006	[1.1% of energy]	+ 0.03%

[1] A more complete assessment of ALA conversion to DHA is available (Plourde & Cunnane, 2007) but is not provided here due to space limitations. The studies shown here each had 8-31 subjects and had durations of 4-26 weeks. In most cases, the ALA was provided as flaxseed oil.

TR Trace (<0.01% change in DHA).

Δ % Refers to the % change in plasma DHA during the ALA supplementation period.