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*Original Research Article*

# Docosahexaenoic Acid, the Aquatic Diet, and Hominin Encephalization: Difficulties in Establishing Evolutionary Links

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*ABSTRACT* Distinctive characteristics of modern humans, including language, tool manufacture and use, culture, and behavioral plasticity, are linked to changes in the organization and size of the brain during hominin evolution. As brain tissue is metabolically and nutritionally costly to develop and maintain, early hominin encephalization has been linked to a release of energetic and nutritional constraints. One such nutrient-based approach has focused on the *n*-3 long-chained polyunsaturated fatty acid docosahexaenoic acid (DHA), which is a primary constituent of membrane phospholipids within the synaptic networks of the brain essential for optimal cognitive functioning. As biosynthesis of DHA from *n*-3 dietary precursors (alpha-linolenic acid, LNA) is relatively inefficient, it has been suggested that preformed DHA must have been an integral dietary constituent during evolution of the genus *Homo* to facilitate the growth and development of an encephalizing brain. Furthermore, preformed DHA has only been identified to an appreciable extent within aquatic resources (marine and freshwater), leading to speculation that hominin encephalization is linked specifically to access and consumption of aquatic resources. The key premise of this perspective is that biosynthesis of DHA from LNA is not only *inefficient* but also *insufficient* for the growth and maturation demands of an encephalized brain. However, this assumption is not well-supported, and much evidence instead suggests that consumption of LNA, available in a wider variety of sources within a number of terrestrial ecosystems, is *sufficient* for normal brain development and maintenance in modern humans and presumably our ancestors. Am. J. Hum. Biol. 19:132–141, 2007. 

Studies of both human brain evolution and public health recently converged on an interest in dietary omega-3 (*n*-3) fatty acids (Crawford et al., 1999; Crawford, 1992; Broadhurst et al., 1998, 2002; Institute of Medicine, Food and Nutrition Board, 2002; Heird, 2001; Muskiet et al., 2004). Docosahexaenoic acid (DHA, 22:6*n*-3), eicosapentaenoic acid (EPA, 20:5*n*-3), and α-linolenic acid (LNA 18:3*n*-3) all belong to the *n*-3 family of fatty acids, named for the position of the first double bond along their carbon chain from the methyl end. All mammals, including humans, are unable to synthesize this class of fatty acids de novo, but can synthesize EPA and DHA from the shorter-chained precursor LNA through desaturation and elongation (Fig. 1). Thus LNA is considered dietarily essential, while EPA and DHA are not.

Approximately 50–60% (dry weight) of the adult human brain is made up of lipids (Huang and Brenna, 2001), of which nearly one-third are long-chained polyunsaturated fatty acids (LC-PUFAs) (Clandinin and Jumpsen, 1997). Arachidonic acid (AA, 20:4*n*-6) and DHA are the two most highly concentrated PUFAs in the brain (Sinclair, 1975) (Table 1). In a sample that included five species of mammals and eight species of birds, DHA showed tremendous variability between peripheral tissues and different species (Turner et al., 2003). However, in the brain, concentrations of DHA across species were the least variable among fatty acids tested. The uniformly high concentrations of DHA within the brain relative to peripheral tissue, and compositional immutability across species despite highly variable dietary intakes of *n*-3 fatty acids, suggest a very important role.

DHA is responsible for the maintenance of structural properties as well as intra- and extracellular signaling at the level of the synapse, the cell, and the organism. Molecular

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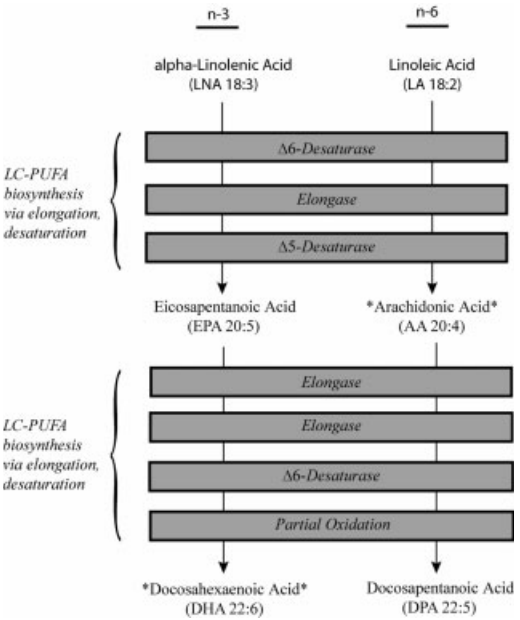


Fig. 1. Metabolic pathway for biosynthesis of LC-PUFA. This pathway is highly regulated by competitive inhibition between families of fatty acids and negative feedback from downstream products.

shape, fluidity, lipid packing and elasticity, permeability, and bilayer instability are unique properties resultant from the lipid composition of the neural membrane (Stillwell et al., 2005). LC-PUFAs also regulate the eruption and expression of receptors, and if released from the membrane, may contribute to prostaglandin synthesis or inhibition, participate as second messengers, or move into the nucleus and interact with various transcription factors to influence gene expression.

As a primary component of membrane lipids within the brain, the period for greatest demand for neural DHA occurs while the brain is most actively growing and neurons are increasing connectivity via cell differentiation and synaptogenesis. This corresponds to a period extending from the third trimester of pregnancy through approximately 2 years of age as the fetus and infant brain cells grow, replicate, and differentiate (Clandinin et al., 2001; Lauritzen et al., 2001). The brain continues to accrue DHA while decreasing its concentration of AA throughout the first 18–20 years of life (Carver et al., 2001). While the neural demand for DHA after 20 years of age need not account for growth, dietary *n*-3 fatty acids must make up for any turnover of tissue lipids. Turnover with adults, however, is very low (Strokin et al., 2004), and thus demand for dietary *n*-3 fatty acid in the nonpregnant adult is comparatively low. Several studies in rats indicate that the brain tenaciously retains neural DHA in the face of a dietary deficiency thereof. Those studies testing cognitive impairment following *n*-3 deficiency often keep their subjects on *n*-3-deficient diets for 2–3 generations before observing significant impairment (Catalan et al., 2002; Moriguchi et al., 2000; Moriguchi and Salem, 2003).

*TABLE 1. Abbreviations*

| | |
|---|---|
| LA | Linoleic acid (18:2*n*-6) |
| LNA | Alpha-linolenic acid (18:3*n*-3) |
| AA | Arachidonic acid (20:4*n*-6) |
| EPA | Eicosapentanoic acid (20:5*n*-3) |
| DHA | Docosahexaenoic acid (22:6*n*-3) |
| DPA | Docosapentanoic acid (22:5*n*-6) |
| PUFA | Polyunsaturated fatty acid |
| LC-PUFA | Long-chain polyunsaturated fatty acid |
| *n*-3 | Omega-3 |
| *n*-6 | Omega-6 |

AA and DHA available for structural deposition may originate either preformed in the diet or via synthesis from the shorter-chained linoleic acid (LA, 18:2*n*-6) or LNA, respectively. AA belongs to the *n*-6 class of fatty acids, which relative to the *n*-3 fatty acids are considered widely available both ecologically and dietarily. Thus, their acquisition is not considered a limiting factor for brain growth and development. The relative efficacy by which LNA is elongated and desaturated to form DHA was reviewed extensively (Huang and Brenna, 2001; Gerster, 1998; Heird, 2001; Innis, 2000; Lauritzen et al., 2001; Muskiet e al., 2004; Su et al., 1999; Woods et al., 1996; Burdge, 2004), and the accumulation as such in neural tissue is reported as highly variable, but generally considered low.

Early examination of *n*-3 fatty acid metabolism and the essentiality of *n*-3 fatty acids for brain development and function have led to speculation on the implications of DHA availability for human brain evolution and the diet of our earliest hominin ancestors (Crawford, 1992; Chamberlain, 1996). Several characteristics of dietary DHA make it a potentially key nutritional component for supporting or rejecting evolutionary hypotheses of encephalization and hominin dietary strategies: 1) no mammalian species (including humans) can synthesize *n*-3 fatty acids de novo, and thus must be consumed in the diet; 2) synthesis of DHA from dietary precursors appears to occur relatively inefficiently; 3) dietary inclusion of preformed DHA is limited to foods of aquatic origin; and

4) in humans, its absence from the diet appears to be of relatively little consequence outside the neural system. It then follows that if DHA is required for brain development, and its synthesis from dietary LNA is considered unreliable and insufficient for the demands of a developing brain, lineages with an evolutionary trajectory of encephalization would have required a dietary niche abundant in preformed DHA. With aquatic dietary resources providing the only concentrated source of preformed DHA, this hypothesis proposes that encephalized hominin species must have lived in proximity to and made deliberate and consistent use of aquatic resources (Crawford, 1992; Broadhurst et al., 1998; Crawford et al., 1999).

To date, reports of low "efficiency" in conversion from dietary LNA to neural DHA were sufficient to promote assumptions about the dietary essentiality of preformed DHA, while the sufficiency of LNA to also meet human neural requirements was downplayed and, to date, not critically assessed. Uncriticized, these hypotheses are increasingly being considered by paleoanthropologists (Session 8 AAPA Meetings, 2002), and therefore it is important to critically explore the evolutionary relevance of DHA within nutritional hypotheses of constraint. Conversion of dietary LNA to DHA may not occur efficiently, but this pathway may still provide sufficient DHA for growing and maintaining an encephalized human brain. Given a relative abundance of LNA in the environment within plant lipids (Tinoco, 1982; Simopoulos et al., 1992; Simopoulos, 2004; Neuringer et al., 1988; Guil et al., 1996), if dietary LNA can sufficiently provide DHA for the developing brain, encephalization need not be tethered to an aquatic dietary niche.

## DIETARY ACQUISITION OF *n*-3 FATTY ACIDS

DHA is synthesized by phytoplankton (Ratledge, 2001) and accumulated up the aquatic food chain in both fresh- and saltwater ecosystems (Drevon, 1993). Fish appear to lack the enzymes required for synthesis of DHA de novo or via conversion from LNA (Ratledge, 2001), and thus bioaccumulation within the aquatic food web must be responsible for the high percentages of DHA within fish lipids. While aquatic algae represent the primary source of ecological preformed DHA, both fresh- and saltwater species of fish, shellfish, and crustaceans represent key dietary sources of preformed DHA (US Department of Agriculture, Agriculture Research Service, 2004) (Table 2).

*TABLE 2. DHA content from aquatic dietary resources (g DHA / kg meat)*[a]

| Fish | Average | SD |
|---|---|---|
| Freshwater (n = 14) | 2.51 | 2.22 |
| Anadromous (n = 10) | 7.98 | 3.62 |
| Saltwater (n = 33) | 4.11 | 3.38 |
| Mollusks (n = 8) | 1.83 | 1.12 |
| Crustaceans (n = 4) | 1.30 | 0.77 |

[a]USDA nutrient database (last accessed July 2005). n = number of species/varieties in sample.

Eggs also contain a small but potentially significant amount of preformed DHA (18 mg DHA per large egg) (US Department of Agriculture, Agriculture Research Service, 2004). However, by weight, fish contain on average 13.5 times more DHA than eggs. Alternatively, to reach the DHA equivalent of a single fillet of salmon (6 ounces), one would need to consume approximately 105 large eggs. Although some terrestrial mammalian tissues (brain) concentrate DHA, such tissues would be extremely limited across the landscape, and could hardly have constituted a regular or significant component of the diet. Given the previous exceptions, preformed DHA has not been significantly identified in any other potential dietary resources outside an aquatic context.

Unlike DHA, LNA is found in a wide variety of potential dietary resources. Most plant tissues contain the enzymes required for LNA synthesis (Murphy and Piffanelli, 1998), and many plants are relatively enriched in *n*-3 PUFAs (Guil et al., 1996). Leaf lipids of plants are particularly concentrated in LNA as an important component of chloroplast membranes. Dark green leafy plants (Neuringer et al., 1988) (including, but not limited to, liverworts, purslane, ferns, mosses, and algae), muscle tissue from fauna consuming the above flora, along with flaxseed, hempseed, rapeseed, and walnuts, were all identified as significant sources of LNA (Simopoulos, 2004). Several authors reported on the compositional analysis of various wild plants across modern equatorial Africa (Achinewhu et al., 1995; Guil et al., 1996; Harris and Mohammed, 2003; Sena et al., 1998). These few examples, however, are not representative of the broad spectrum of foodstuffs potentially available over a million years ago to an encephalizing hominin lineage. Sampling and analysis of the environmental and dietary availability of *n*-3 fatty acids across regions identified as potential "environments of human adaptedness" are critical. If *n*-3 fatty acids (LNA in particular) appear to be fairly widespread throughout a

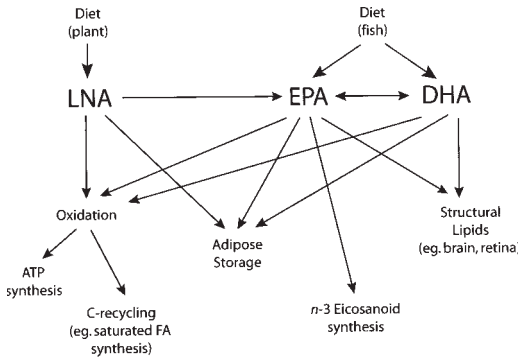


Fig. 2. Potential bodily depots and metabolic fates of *n*-3 fatty acids. Details discussed in text.

number of ecosystems and dietary niches, this suggests that *n*-3 fatty acids currently do not and likely did not serve as a dietary constraint upon primate encephalization. However, the identification of *n*-3-rich and *n*-3-poor dietary niches may yield further support of particular environments critical for the growth and maintenance of an encephalizing lineage.

Dietary *n*-3 fatty acids serve a number of metabolic functions aside from neural structure (Fig. 2). Ingested LNA does not represent a pool of *n*-3 fatty acid exclusively preserved for the inevitable synthesis of LC-PUFA (Menard et al., 1998; Cunnane, 2001), nor ingested DHA as a pool for neural substrate alone. Dietary LNA may be utilized for energy production via $\beta$-oxidation, for the synthesis of *n*-3 LC-PUFA (including DHA) or *n*-3 eicosanoids (via EPA), deposited within adipose tissue, or partially oxidized and recycled for the de novo synthesize of non-essential fatty acids (Cunnane, 2001). Dietary DHA similarly may be oxidized for energy, stored in adipose tissue, and retroconverted to EPA and *n*-3 eicosanoids or as a component of phospholipids within various tissue membranes. The fate of ingested *n*-3 fatty acid (particularly LNA) is highly dependent on a number of factors, including gender (Burdge, 2004), energy status (Cunnane, 2001; Leyton et al., 1987), pregnancy/lactation (Burdge, 2004; Otto et al., 2001), protein intake (Gerster, 1998), preformed DHA (Burdge, 2004; Demar et al., 2005; Pawlosky, 2001), and the dietary ratio of *n*-6 to *n*-3 (Clandinin and Jumpsen, 1997; Holman, 1997; Gerster, 1998; Burdge, 2004) or saturated to polyunsaturated fatty acids (Gerster, 1998). The body has variable intrinsic priorities, and distributes nutrients differentially to meet those demands.

Elongation and desaturation of dietary LNA to form DHA are metabolically costly, and in the face of sufficient tissue DHA, likely represent an energetic waste. The final steps in the conversion pathway are at least partly controlled by the concentration of preformed DHA in the diet. When the concentration of dietary DHA is high, conversion rates decline, but when dietary consumption of DHA is decreased, conversion from LNA increases to compensate for this deficit and to facilitate an adequate supply of DHA for brain function and growth (Pawlosky, 2001).

It is important to keep this list of priorities and options in mind when considering the efficiency or sufficiency of the *n*-3 elongation and desaturation pathway. Many studies reporting low rates of DHA synthesis from LNA were designed to investigate the relative efficiency of this conversion and accretion as DHA within tissue membranes relative to that accretion following the consumption of preformed DHA. As discussed below, such study designs are inappropriate for testing the *sufficiency* of this pathway to meet neural demands in the dietary absence of DHA.

## BUILDING A BRAIN

The periods of highest human brain growth rates, during the final trimester of gestation and immediately following birth, represent the most critical intervals for the supply and deposition of DHA. As the developing fetus and young infant are entirely dependent on maternal nutrition, demands upon the mother to meet the requirements of the growing brain are considerable and critical.

In 2002, the Institute of Medicine convened an expert panel to create and release the most recent dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein, and amino acids (Institute of Medicine and Food and Nutrition Board, 2002). The mission was to draw upon the current literature, and issue official recommendations for the dietary intake of energy, carbohydrate, fiber, and other basic macronutritient requirements. Included in this report is an extensive review of the literature relevant to forming recommendations on the consumption of *n*-3 fatty acids. Interestingly, this panel found insufficient evidence for the essentiality of DHA. All final recommendations for *n*-3 fatty acids for men and women across all age categories are given exclusively in units of LNA. Even during pregnancy/lactation, the period of greatest demand for

neural DHA for the fetus/infant, the panel concluded that "evidence is not available to show that increasing intakes of preformed DHA in pregnant and lactating women consuming diets that already meet requirements for *n*-6 and *n*-3 fatty acids have any physiologically significant benefit to the infant" (Institute of Medicine and Food and Nutrition Board, 2002).

To date, many clinical trials have tested various measures of cognition and visual acuity in infants following maternal DHA supplementation relative to nonsupplemented controls. While improvement of visual acuity and some measures of cognition were repeatedly observed in infants following supplementation with DHA, it remains unclear whether function is improved via increased intake of *n*-3 fatty acids in general or DHA in particular. Also equivocal is whether the supplementation of *n*-3 fatty acids corrects for essential fatty acid deficiency, or whether there is a pharmacological effect above and beyond a baseline requirement for neural DHA.

Future trials testing dietary supplementation of LNA compared to that of DHA will potentially elucidate the most critical aspects of *n*-3 fatty-acid metabolism and function. Valenzuela et al. (2004) conducted such a trial with rats, comparing various tissue concentrations of DHA following maternal supplementation with LNA or DHA in relation to a control diet deficient in *n*-3 fatty acids. The results showed that equivalent quantities of neural DHA were deposited following the LNA- and DHA-supplemented diets, which were both significantly greater than deposition within *n*-3-deficient controls. While this indeed lends support to a hypothesis of the dietary sufficiency of LNA for neural growth, such studies and results must be replicated with human subjects.

### *Transfer of* n-*3 across placenta*

Several mechanisms are in place to ensure a sufficient supply of DHA to the growing fetus, even in the absence of preformed dietary DHA. Concentrations of DHA within maternal plasma were observed to increase throughout pregnancy, and notably without dietary alteration of *n*-3 fatty acid intake (Burdge, 2004; Otto et al., 2001). Given the volumetric expansion of maternal plasma during pregnancy, the increase in plasma concentration of DHA represents a very significant increase in the absolute quantity of circulating DHA. This indicates an increase in either the maternal synthesis of DHA from dietary LNA, or an increase in the release of stored DHA from maternal tissue (i.e., adipose or tissue membrane phospholipids).

As evidence for increasing DHA synthesis during gestation, an increase in the activity of the elongation/desaturation pathway (Fig. 1) was documented in the absence of preformed dietary DHA (Pawlosky, 2001) and in the presence of circulating estrogens (Burdge, 2004; Demar et al., 2004). Specifically, administration of ethinyl estradiol was found to increase DHA production by 42% (Giltay et al., 2004), implicating estrogens in the upregulation of DHA synthesis during periods of conditional demand such as pregnancy.

### *Transfer of* n-*3 via breast milk*

The concentration of DHA within human milk varies widely (Lauritzen et al., 2004; Francois et al., 2003; Heird, 2001). This variability may arise from a number of factors, including differential dietary intake of preformed DHA, baseline storage of LNA for future conversion, or storage of DHA for mobilization. However, given the mechanisms noted above to ensure availability of DHA and a sufficient supply of *n*-3 fatty acids in the maternal diet, the lowest observed concentrations of DHA within human breast milk should prove sufficient for normal brain and retinal development.

Lauritzen et al. (2004) conducted a randomized supplementation trial with fish oil, highly concentrated in DHA, on lactating mothers to test the effect on the visual acuity of their infants. All mothers in the trial had a relatively low baseline intake of fish, and thus the supplementation tested whether the inclusion of dietary DHA above a low baseline level can improve infant visual function by increasing the DHA content of their breast milk. After 4 months, the authors noted no difference between the supplementation group and controls in scores of infant visual acuity (Lauritzen et al., 2004). Since *n*-3 deficiency has been most implicated in functional impairment, this supports the hypothesis that mothers are able to provide for the development of their infants' neural tissue requirements without the consumption of preformed DHA (or at most in small quantities), the addition of which may add no functional advantage. While a number of studies indeed showed positive effects on visual and/or cognitive function in association with diets rich in, or supplemented with, preformed DHA (Agostoni and Giovannini, 2001;

Heird, 2001; Helland et al., 2003; Malcolm et al., 2003; Willats et al., 1998; Williams et al., 2001), the precise relationship between essential fatty acids in the diet and optimal neural function remains to be fully understood.

## CONSEQUENCES OF DHA DEFICIENCY

Various tissue concentrations of DHA (e.g., liver, heart, and red blood cells) are highly variable, depending on the fatty-acid composition of the diet (Valenzuela, 2004). However, the brain retains LC-PUFA to a greater extent than other tissue, and this retention is extended during periods of dietary *n*-3 deficiency (Institute of Medicine and Food and Nutrition Board, 2002; Demar et al., 2004).

In the complete absence of dietary *n*-3 fatty acid, the concentration of DHA within neural tissue declines, but is precisely compensated for by reciprocal incorporation of the *n*-6 equivalent fatty acid, docosapentaenoic acid (DPA, 22:5 *n*-6) (Schiefermeier and Yavin, 2002; Innis, 2003). These two fatty acids (DHA and DPA) are very similar in structure. Both are 22 carbons in length and are highly unsaturated, but likely result in significant structural differences (Crawford et al., 1999) within the membrane tissue. Notably, the complete absence of *n*-3 fatty acids (and thus any form of DHA for brain growth and maintenance) was not shown to result in impaired growth of the brain or body (Schiefermeier and Yavin, 2002). Thus early hominin species likely could have survived periods of *n*-3 deficiency while maintaining an encephalized brain. This further argues against the necessary and regular dietary incorporation of DHA-rich foods, namely those of aquatic origin.

## ADDITIONAL INSIGHTS FROM POPULATION OBSERVATION

### *Global consumption*

Globally, there is high variability in population-level consumption of fish products and, by extension, dietary DHA. Table 3 shows global per capita consumption of fish foods per year of the 10 lowest and highest consuming nations in the world (FAOSTAT, 2002). On the low end, many of these countries supply less than 2 kg of fish per year to the average citizen. Given an average concentration of approximately 5 g DHA/kg fish in Table 2, daily consumption would average approximately 28 mg DHA, or less than 200 mg DHA per week. Notably, eggs may also contribute to dietary DHA. On the order of 18 mg DHA per large egg (US Department of Agriculture, Agricultural Research Service, 2004), individuals from populations with low per capita consumption of fish may still obtain significant *n*-3 fatty acids in the diet.

*TABLE 3. Global availability of aquatic dietary resources (kg/capital/yr)*[a]

| | Fish, marine | Fish, freshwater | Total |
|---|---|---|---|
| *Lowest consumers* | | | |
| Ethiopia | 0.2 | 0.2 | 0.4 |
| Mongolia | 0.5 | 0.0 | 0.5 |
| Djibouti | 1.0 | 0.0 | 1.0 |
| Palestine, Occupied Territory | 0.8 | 0.2 | 1.0 |
| Niger | 0.6 | 0.6 | 1.2 |
| Guinea-Bissau | 1.2 | 0.1 | 1.3 |
| Honduras | 1.4 | 0.1 | 1.5 |
| Zimbabwe | 1.4 | 0.9 | 2.3 |
| Serbia and Montenegro | 2.0 | 0.4 | 2.4 |
| Mozambique | 2.1 | 0.4 | 2.5 |
| *Highest consumers* | | | |
| Maldives | 185.9 | 0.1 | 186.0 |
| Kiribati | 75.2 | 58.7 | 133.9 |
| Iceland | 91.0 | 5.3 | 96.3 |
| Samoa | 91.6 | n/v | 91.6 |
| Norway | 54.7 | 19.2 | 73.9 |
| Japan | 66.3 | 5.9 | 72.2 |
| Seychelles | 62.0 | 0.0 | 62.0 |
| Malaysia | 57.0 | 3.3 | 60.3 |
| Portugal | 59.3 | 0.9 | 60.2 |
| Korea, Republic of | 58.7 | 1.0 | 59.7 |
| *Other* | | | |
| UK | 23.2 | 3.1 | 26.3 |
| USA | 21.3 | 3.2 | 24.5 |

[a]According to FAOSTAT (2002) last accessed July 2005 (last update Aug 2004).
n/v, no value given in database.

During the final trimester of pregnancy, the growing fetus is estimated to assimilate at least 400 mg DHA per week (Burdge, 2004). Therefore, assuming that 100% of the maternal dietary DHA could be absorbed and preferentially shuttled to the growing infant brain, an average weekly consumption of less than 200 mg DHA would not provide for half the neural demand during this period. Admittedly, DHA destined for fetal development may come in part from maternal adipose and not directly from dietary *n*-3 fatty acids. DHA stored during periods of less demand may then be mobilized via lipolysis throughout pregnancy as fetal neural demand is increased. Therefore, 200 mg DHA per week may be sufficient to provide enough neural substrate for the growing fetus. However, birth spacing must then account for the need to rebuild maternal stores of DHA.

Within each population there is certainly a distribution of consumption that will include a large number of individuals consuming no fish, and thus very little or no preformed DHA. Assuming the essentiality of dietary DHA, it

remains questionable how individuals in countries with very low consumption of fish reconcile their dietary deficiency of preformed DHA with normal neural development.

### *Vegetarians/vegans*

Millions of individuals around the modern world, including some 2.5% of Americans and 4% of Canadians (American Dietetic Association and Dieticians of Canada, 2003), consume a diet classified as vegan or vegetarian. There are also a number of religious doctrines that emphasize abstention from animal consumption, including Jainism, Buddhism, Hinduism, and Taoism, involving a significant percentage of modern human populations. In the case of these vegetarians, many have maintained such a restricted diet for generations. Neurological impairment under generational deficiency of DHA should result if dietary DHA is essential for neural function. Given that these populations experience normal brain growth and development in the absence of dietary DHA, it seems reasonable to question the nature of our dietary requirements for *n*-3 fatty acids. If preformed DHA is essential, and only significantly available from aquatic dietary sources, the expected outcome of a vegetarian lifestyle is the failure of neural growth and development. On the other hand, there is no evidence to suggest that the capacity for DHA synthesis in vegetarians is limited (Sanders, 1999). A logical explanation involves the sufficiency of LNA from the dietary intake of plants to provide sufficient DHA for the neural development of these populations.

### *Economic expense*

In further consideration of availability, dietary sources rich in preformed DHA come at a greater economic expense. For the lowest-income individuals, even in areas where fish products are readily available, these items are often unaffordable (Drewnowskiki, 2004; Drewnowski and Specter, 2004). Those with low income were shown to consume a lower-quality diet, including low intake of fish (Shelton, 2005), and thus their consumption along with preformed DHA is negligible. The implications of a hypothesis for DHA essentiality are that children born to parents in the lowest income bracket should suffer impaired brain growth and development. No data found by these authors, however, showed impaired brain growth in these children.

## ENCEPHALIZATION IN HUMAN EVOLUTION

Theories proposed to explain a dramatic increase in encephalization in the human lineage over the past 2–3 Ma have essentially adopted two alternate approaches. One strategy is to isolate and identify specific selective pressures for increased cognitive capacity requiring a larger brain, including social (e.g., Bryne and Whiten, 1988; Dunbar, 2000; Harcourt, 1989) and ecological pressures (e.g., Clutton-Brock and Harvey, 1980; Parker and Gibson, 1977; Gibson, 1986). These selective pressures are linked to notions of increased use of open habitats with attendant predation risks (Foley, 1987), increased dietary quality and wider home range, and higher activity levels in hot, arid environments (Leonard and Robertson, 1997; McHenry and Coffing, 2000; Aiello and Wells, 2002).

The second approach assumes inherent adaptive advantages to increased encephalization, and focuses on the role of biological constraints on increasing brain size. Prominent among these is the expensive tissue hypothesis, which advocates the release of an energetic constraint by a dietary shift to denser, high-nutrient food sources (Aiello and Wheeler, 1995), or in lowering the "expected" muscle mass relative to other primates (Leonard et al., 2003). Metabolic costs are especially high during infancy, when brain metabolism can account for more than 60% of the resting metabolic rate (Leonard et al., 2003). Falk (1990) argued that a physiological constraint to encephalization is developing a means of efficiently cooling the brain in equatorial environments, which requires a distributional shift in cranial blood circulation.

Given the neural essentiality and limited dietary availability of DHA and AA in terrestrial sources, it has been hypothesized that a consistent consumption of fish, crustaceans, mollusks, and other marine or aquatic species with lipid profiles (including DHA and AA values) similar to brain phospholipids was essential in initiating and sustaining growth of the cerebral cortex throughout the *Homo* lineage (Broadhurst et al., 1998; Crawford et al., 1999). While brain, bone marrow, and some organ tissues of mammalian herbivores represent concentrated sources of dietary LC-PUFA, their insignificance relative to total carcass mass and the highly perishable nature of these fatty acids preclude their contribution as a consistent and readily available source of AA and DHA for early hominins (Broadhurst et al., 1998). Birds, small mammals, reptiles, and

amphibians consuming aquatic resources may also have provided a link in a food chain rich in DHA (Broadhurst et al., 2002). In addition, the association of early hominin fossils with lacustrine and fluvial sediments, the presence of major lake systems in the East African Rift Valley throughout the course of human evolution, and an apparent association of large shell middens and fish with some of the earliest hominins are invoked to support the possibility of regular and repeated access to an aquatic resource base (Broadhurst et al., 1998, 2002; Crawford et al., 1999).

Within this basic framework, a series of key hominin evolutionary events was linked to consumption of DHA-rich lacustrine and marine resources, including: 1) enlargement of the *Homo* brain from *H. habilis* to *H. sapiens* (Broadhurst et al., 1998); 2) an increase in intellectual capacity and creativity in the emergence of modern humans (Broadhurst et al., 2002); 3) increased adaptability of Upper Paleolithic humans relative to terrestrial resource-based Neanderthals (Broadhurst et al., 2002); 4) more sophisticated tool manufacture and use at near-shore environments relative to inland sites (Broadhurst et al., 1998, 2002); 5) differential trajectory of *Homo* spp. (lacustrine association) and australopithecines (forested association) (Crawford et al., 1999); and 6) an aquatic phase of human evolution (Crawford, 1992). These links remain problematic, as they rely on ambiguous criteria that result in possible but not necessarily probable explanations of the data. While many if not most fossil hominins were recovered from stratigraphic sequences dominated by lacustrine or fluvial litholofacies, the association is overwhelmingly a function of taphonomic processes (Behrensmeyer and Hook, 1992), as these depositional regimes are most likely to preserve vertebrate faunas. Preservation potentials for many habitats currently found in equatorial Africa such as lowland forest, open grassland, or montane forest are poor, and the vertebrate fossil record of faunas, including potential hominins, in these habitats is rare to nonexistent.

While aquatic dietary resources may have represented a component in early hominin diets, there is no archeological evidence for aquatic resource use by early *Homo*. Evidence of intensive use of aquatic resources such as harpoons, hooks, shell debris, or exploited fish bones is not evident until the Middle Stone Age at such sites at Katanda, D.R. Congo, where bone points are found in association with fish at ca. 75 ka (reviewed by McBrearty and Brooks, 2000). Intermittent or even extensive use of aquatic resources, however, may not have left a retrievable or recognizable fossil or archeological record.

## CONCLUSIONS

Docosahexaenoic acid (DHA), an *n*-3 fatty acid, is essential for the development of the human nervous system. DHA concentrations are much higher in the brain and retina, and while connections were drawn to improved visual acuity and cognitive abilities in infants receiving DHA supplements, results remain inconclusive as to the benefits of supplementing *n*-3 fatty acids in general or DHA in particular. Although humans are relatively inefficient synthesizers of DHA, there is currently no evidence to indicate that we are incapable of producing sufficient DHA for optimal neurodevelopment, given sufficient access to dietary *n*-3 in the form of LNA. In fact, it appears that there are several physiological mechanisms to ensure sufficient availability of DHA during the critical periods of human brain growth, despite dietary fluctuations in the availability of *n*-3 fatty acid, namely, the upregulation of DHA biosynthesis from dietary LNA, and mobilization during periods of conditional need. While access to the aquatic food chain would ensure a consistent source of dietary DHA, the fossil and archeological records do not support an exclusive association of early hominins to littoral habitats or an aquatic resource base. Thus while an aquatic dietary niche may certainly have met the metabolic, developmental, and functional demands of our evolving hominin ancestors, the available nutritional, physiological, and archaeological evidence does not support the argument that such a dietary niche was necessary for hominin brain evolution. Rather, an encephalizing hominin brain may have been fueled within a terrestrial, or aquatic, setting, provided sufficient dietary acquisition of LNA, EPA, DHA, or any combination thereof.

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