## Nutrition Discussion Form

## Is docosahexaenoic acid a red herring for the aquatic diet? – Comments by Milligan and Bazinet

Recently, Dr Langdon attempted to address the question of whether or not aquatic-based diets were necessary for hominid brain evolution (Langdon, 2006). In order to do this he assessed (1) if brain functions were sensitive to variations in DHA (22:6*n*-3) supply, (2) if 22:6*n*-3 supply to the brain is sensitive to variations in dietary intake, (3) if an aquatic food chain is the only effective dietary source for 22:6n-3 and (4) if the dietary supply of 22:6n-3 has been a limiting resource for brain evolution. Dr Langdon notes the difficulties in making firm conclusions on several of these points but argues that the body is capable of dealing with fluctuations in 22:6n-3 intake. We agree that the body has developed mechanisms to store (Lefkowitz et al. 2005), synthesise (Burdge & Wootton, 2002) and conserve brain 22:6n-3 (DeMar et al. 2004; Rao et al. 2006), and these processes probably help maintain brain 22:6n-3 concentrations when the dietary supply is sub-chronically limited. However, we feel that the evaluation of 22:6n-3 by the four defined criteria is an inadequate test of the hypothesis that the aquatic diet was important for hominid brain evolution.

There are many facets of the aquatic diet hypothesis that are elegantly discussed in more detail elsewhere (Cunnane, 2005b), and in light of the focus of Dr Langdon's paper published in the Journal, herein we focus on the micronutrient argument. While 22:6n-3 is abundant in aquatic foods and is important in the development (Clandinin et al. 1980; Innis, 2003) and normal functioning (Chen & Bazan, 2005) of the brain, it is only one nutritional component of the aquatic diet. Other nutritional components of the aquatic diet that Dr Langdon has overlooked are I, Fe, Cu, Zn and Se (Cunnane, 2005a, 2006). I deficiency is the leading cause of preventable mental retardation (World Health Organization, 1999) and for over 80 years countries have been using iodised salts to eradicate this deficiency (Delange et al. 2002; Hetzel, 2005). Fe-deficiency anaemia is a leading cause of infant morbidity and mortality worldwide (World Health Organization, 2000). Cunnane has calculated that in order to meet current minimum daily requirements of these five nutrients, one would have to consume 900 g shellfish, or 2500 g eggs or 3500 g fish or 3700 g pulses or 4800 g cereals or 5000 g meats or 5500 g nuts or 9000 g vegetables per d (Cunnane, 2005b). It is important to note that human nutrient requirements have safety factors and overestimate the mean requirement of individuals, but using similar methods we estimate that one would have to consume 1000 g brain or 1200 g liver per d to meet the minimum requirements for these five nutrients. To support the thesis that an aquatic diet would not be necessary for hominid brain evolution and functional development, Dr Langdon would have to apply the four criteria (see above) used to refute the role of dietary 22:6*n*-3 in brain evolution to dietary I, Fe, Cu, Zn, Se and 22:6*n*-3 en masse.

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## References

Burdge GC & Wootton SA (2002) Conversion of  $\alpha$ -linolenic acid to eicosapentaenoic, docosapentaenoic and docosahexaenoic acids in young women. *Br J Nutr* **88**, 411–420.

Chen C & Bazan NG (2005) Lipid signaling: sleep, synaptic plasticity, and neuroprotection. *Prostaglandins Other Lipid Mediat* 77, 65–76.

Clandinin MT, Chappell JE, Leong S, Heim T, Swyer PR & Chance GW (1980) Extrauterine fatty acid accretion in infant brain: implications for fatty acid requirements. *Early Hum Dev* **4**, 131–138.

Cunnane SC (2005a) Origins and evolution of the Western diet: implications of iodine and seafood intakes for the human brain. *Am J Clin Nutr* **82**, 483, author reply 483–484.

Cunnane SC (2005b) Survival of the Fattest: The Key to Human Brain Evolution. Singapore: World Scientific.

Cunnane SC (2006) Survival of the fattest: the key to human brain evolution (article in French). *Med Sci (Paris)* **22**, 659–663.

Delange F, Burgi H, Chen ZP & Dunn JT (2002) World status of monitoring iodine deficiency disorders control programs. *Thyroid* 12, 915–924

DeMar JC Jr, Ma K, Bell JM & Rapoport SI (2004) Half-lives of docosahexaenoic acid in rat brain phospholipids are prolonged by 15 weeks of nutritional deprivation of *n*-3 polyunsaturated fatty acids. *J Neurochem* **91**, 1125–1137.

- Hetzel BS (2005) Towards the global elimination of brain damage due to iodine deficiency the role of the International Council for Control of Iodine Deficiency Disorders. *Int J Epidemiol* **34**, 762–764
- Innis SM (2003) Perinatal biochemistry and physiology of long-chain polyunsaturated fatty acids. *J Pediatr* **143**, S1–S8.
- Langdon JH (2006) Has an aquatic diet been necessary for hominin brain evolution and functional development? *Br J Nutr* **96**, 7–17.
- Lefkowitz W, Lim SY, Lin Y & Salem N Jr (2005) Where does the developing brain obtain its docosahexaenoic acid? Relative contributions of dietary α-linolenic acid, docosahexaenoic
- acid, and body stores in the developing rat. *Pediatr Res* **57**, 157–165.
- Rao JS, Ertley RN, Demar JC Jr, Rapoport SI, Bazinet RP & Lee HJ (2006) Dietary *n*-3 PUFA deprivation alters expression of enzymes of the arachidonic and docosahexaenoic acid cascades in rat frontal cortex. *Mol Psychiatry* Epub ahead of print, doi: 10.1038/sj.mp.4001887.
- World Health Organization (1999) Progress Towards the Elimination of Iodine Deficiency Disorders (IDD). Geneva: WHO.
- World Health Organization (2000) *Malnutrition: the Global Picture*. Geneva: WHO.