A discussion of the metabolic pathway by which dietary ALA can be converted by a series of sequential desaturation (D) and elongation (E) reactions into EPA and then DHA in the mammalian liver was provided in the metabolism of omega-3 and omega-3 section. This multi-step sequence is briefly outlined below:

The pioneering rodent studies, reported by Lamptey and Walker in 1976 and subsequently supported in numerous subsequent experimental studies, showed that omega-3 deprivation impaired the learning ability of young animals and that rats could convert ALA into DHA for assimilation into brain tissue (membrane phospholipid components). These rodent-based studies had a significant influence on official recommendations from Health and Welfare Canada (1990) and the Food and Nutrition Board (2002) wherein dietary ALA and not DHA, the brain/retina omega-3 fatty acid for structure-functioning, was recognized as the only essential dietary fatty acid. Official recommended minimal intakes were established for ALA for all members of the population (young and old alike). However, extensive controlled studies in human subjects have now all consistently confirmed the very limited metabolic conversion of dietary ALA into DHA, the final end product of the sequential pathway.
A common approach for assessing the apparent conversion efficiency of dietary ALA to the longer-chain products (EPA plus DHA) in controlled human trials is to determine the net mass rise in circulating (blood) levels of EPA and DHA after increasing the dietary intake of ALA from food sources. The early studies in 1993 conducted at the University of Manitoba in collaboration with the University of Guelph in Canada involved 42-day phases of feeding controlled diets with markedly different levels of omega-3 fatty acid as ALA (alpha-linolenic acid) along with varying amounts of omega-6 fatty acid as LA (linoleic acid) and varying omega-6:omega-3 ratios. While some moderate net rise in the level of EPA (eicosapentaenoic acid) was found with higher levels of ALA in conjunction with omega-6:omega-3 ratios lowered to 3:1; no net rise in the level of circulating DHA was found across the various fatty acid mixtures and ratios. This early evidence for a very limited conversion of dietary ALA into DHA has been supported subsequently by other controlled studies in humans using mass measurements. For example, the feeding of 10.7 grams (10,700 mg) of ALA from flaxseed oil over a four week period failed to provide any significant net rise in the low levels of DHA present in the breast milk of lactating women. It is noteworthy that the levels of ALA supplementation used in the latter study from the Oregon Health and Science University (by Francois et al.) was approximately seven-fold that recommended for pregnant recommended by Health and Welfare Canada (1990) and by the Food and Nutrition Board (2002) during pregnancy.

The availability of ALA labeled with stable isotopes, which avoid potential biological hazards associated with the use of radio-isotopes in human studies, has allowed detailed and sophisticated investigations of the metabolic conversion efficiency of ALA to EPA and DHA. This technology has been utilized and reported upon in a number of recent studies in medical and nutrition journals. The original study using this technology was reported from the U.S. Department of Agriculture in 1994 wherein the conversion efficiency of ALA to DHA in young adult male subjects was reported to be at the level of a 4% efficiency, which would predict that 25 parts of dietary ALA would be needed to provide the equivalent rise in circulating levels of DHA which could be delivered by the direct consumption of one part of DHA. The overall conversion efficiency from ALA to EPA plus DHA combined was estimated to be 12%. It is noteworthy that the very limited conversion of ALA to DHA was also highly variable between the individual subjects thereby indicating difficulty in predicting those in the population who may have extremely compromised capacities for the conversion of ALA to DHA. Subsequent studies by Pawlosky et al. (2001) using similar technology and that more recently by Hussein et al. (2005) showed estimated conversions from ALA to DHA of less than 0.1% and a conversion to EPA plus DHA combined of less than 0.4% efficiency overall. The latter study was conducted over a fairly lengthy time period of 12-weeks in duration. Burgee et al. from the U.K. has compared the apparent conversion efficiency of ALA to DHA in young adult men and women. Interestingly, no detectable formation of DHA was found in the men whereas an approximate conversion efficiency from ALA to DHA of 9% was found in women. These authors suggest that the greater fractional conversion in women may be due in part to a significantly lower rate of utilization of dietary ALA for beta-oxidation and/or the influence of estrogen or other hormonal factors on the conversion efficiency. In summary, the conversion efficiency from ALA to DHA is very limited in healthy individuals; furthermore, the apparent inefficiency of the conversion from ALA to DHA is markedly variable between individuals within different sectors of the populations.
such that the lack of sufficient dietary DHA could compromise optimal health in those with very minimal conversion capacities. The very low conversion efficiencies and wide variation in capacities lend support to serious consideration being given to dietary DHA as an 'essential' fatty acid and/or a 'conditionally essential' fatty acid depending upon the conversion capacity of individuals within the population.


Conversion Efficiency of ALA to DHA in Humans


