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EPA and Huntington's chorea: treatment and associated cerebral changes

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Huntington's disease (Huntington's chorea) is an autosomal dominant disease of the human brain caused by an unstable expansion of CAG trinucleotide triplet repeats in the huntingtin gene at 4p16.3; the CAG repeats are transcribed and translated into polyglutamine expansion (polyQ) stretches, and the length of the repeats correlates inversely with age of onset. Huntington's disease is characterized by motor dysfunction, with chorea and incoordination occurring relatively early and dystonia, rigidity and bradykinesia becoming more prominent with time; death usually occurs within 15-25 years of onset of motor symptomatology. The key neuropathological change is neuronal degeneration, particularly in the striatum. The scientific background is given for why fatty acids may play an important role in Huntington's disease. Evidence is then presented from a randomized double-blind placebo-controlled to show that ultra-pure ethyl-eicosapentaenoic acid (ethyl-EPA), a semi-synthetic, ethyl ester of eicosapentaenoic acid, is associated with clinical improvement in motor functioning in Huntington's disease. The likely mechanisms of this beneficial action are then described. Finally, the results are detailed of a further recent study to determine the extent to which ethyl-EPA might reduce the rate of progress of cerebral atrophy. High-resolution cerebral magnetic resonance imaging scans were acquired at baseline, six months and one year in up to 34 patients with stage I or II Huntington's disease who took part in the trial of ethyl-EPA. For each subject and each pair of structural images, the two-timepoint brain volume change was calculated in a double-blind manner.

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