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Poster Sessions

Transport of docosahexaenoic acid across the blood–brain barrier is decreased in aged mice

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Nutrients are actively taken up by the brain through various transporters at the blood–brain barrier (BBB). In the aged brain, central nervous system (CNS) disorders can result from a lack of nutrients, including decreased levels of docosahexaenoic acid (DHA), which is important for many neurophysiological functions and is associated with memory and cognitive dysfunction. To compensate for decreased brain DHA, orally supplied DHA must be transported from circulating blood to the brain across the BBB via its transporter, the major facilitator superfamily domain-containing protein 2a (Mfsd2a). Although BBB integrity is known to be altered in aging, the influence of aging on DHA transport across the BBB it is not well understood.

In this study, we used young (7–8 weeks old) and aged (35–55 weeks old) female C57BL/6 mice to evaluate brain uptake of [^{14}C]DHA using an *in situ* transcardiac perfusion technique.

In the whole brain of aged mice, DHA uptake was significantly decreased by 52.5%, without any change to the expression level of Mfsd2a protein in brain microvasculature compared with young mice. Interestingly, significant decreases in the expression levels of tight junction-associated proteins (claudin-5 and ZO-1) were observed in aged mice, but brain uptake of FITC-albumin, a marker of BBB integrity, did not differ between young and aged mice. These results indicate that the age-related structural changes to tight junctions did not lead to increased BBB permeability to albumin and DHA.

These findings suggest that the aged brain exhibits decreased DHA brain uptake via the BBB, probably as a result of reduced Mfsd2a transporter activity.