Amyloid plaques, consisting of deposited beta-amyloid (Ab) are a neuropathological hallmark of Alzheimer’s Disease (AD). Cerebral vessels play a major role in AD, as Ab is cleared from the brain by pathways involving the cerebrovasculature, most AD patients have cerebrovascular amyloid (cerebral amyloid angiopathy (CAA), and cardiovascular risk factors increase dementia risk. We present a notable advance in vascular tissue engineering by generating the first functional 3-dimensional model of CAA in bioengineered human vessels. We show that lipoproteins including brain (apoE) and circulating (high-density lipoprotein) synergize to facilitate Ab transport across bioengineered human cerebral vessels. These lipoproteins facilitate Ab42 transport more efficiently than Ab40, consistent with Ab40 being the primary species that accumulates in CAA. Moreover, apoE4 is less effective than apoE2 in promoting Ab transport, also consistent with the well-established role of apoE4 in Ab deposition in AD.

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11:30 am
Strathcona Anatomy Building
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