Immunotherapy for Stroke and Cognition in a Cerebral Amyloid Angiopathy Animal Model (Ramot)

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Cerebrovascular dysfunction is a fundamental part of the pathology of several neurodegenerative diseases and rated as one of the most prominent cause for dementia. Cerebral amyloid angiopathy (CAA) results in intra-parenchymal and subarachnoid bleeding, which is caused by cerebrovascular amyloid deposits and multiple infarcts, and can lead to hemorrhagic stroke and cognitive impairment (Greenberg et al. Stroke. 2004; 35:2616-2619; Frangione et al. Amyloid. 2001; 8 Suppl 1:36-42). The prevalence of CAA, estimated from autopsy series, is approximately 10% to 40% of the general elderly population (Greenberg et al. Stroke. 2004; 35:2616-2619). Although the most common form of cerebrovascular amyloid is Aβ-CAA, there are other proteins that have been linked to familial forms of CAA such as: APP, cystatin C, BRI, prion protein, gelsolin, and transthyretin (Burgermeister et al. Lessons from mouse models. Ann N Y Acad Sci. 2000; 903:307-316). Clinical interventions usually consist of blood pressure control (i.e., hypertension treatment) as well as avoidance of medications that increase systemic hemorrhage risk (e.g., aspirin). However, there are no current treatments to reduce amyloid pathology in CAA (Maia et al. J Neurol Sci. 2007; 257:23-30).

US Patent.

Additional information can be provided upon request.

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