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Molecular Seeding of Cerebral Amyloid Angiopathy in Novel Transgenic Mice
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Cerebral amyloid angiopathy (CAA) is a condition in which amyloid plaques form inside the blood vessels of the brain instead of, or in addition to, within the brain matter itself. CAA is common in people with Alzheimer’s disease, but certain genetic mutations are associated with early onset, severe CAA. These mutations occur in the gene for beta-amyloid, the protein fragment that forms the major constituent of amyloid plaques.

Most people with a genetic mutation causing CAA have one copy of the normal beta-amyloid gene and one copy of the mutated gene. In such cases, amyloid plaques arise from interactions between normal and mutant beta-amyloid. Because these interactions are not well understood, William E. Van Nostrand, Ph.D. and colleagues are studying this issue in mice that have been genetically altered to express both normal and mutant human beta-amyloid. The researchers will study how the two forms of beta-amyloid interact to produce amyloid deposits in the blood vessels of the brain, and how such deposits lead to cognitive impairments and brain hemorrhage. These studies will advance our understanding of CAA, and help to identify strategies for slowing or preventing progression of the disease.