

Exogenous amyloid β protein ($A\beta$) seeds induce $A\beta$ depositions in the blood vessels

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Alzheimer's disease (AD) involves the deposition of a protein called amyloid β protein ($A\beta$) in the brain. This protein can build up in both the functional tissues, or "parenchyma", of the brain, and in the blood

vessels. This can lead to AD, causing dementia, or cerebral amyloid angiopathy (CAA), which can lead to cerebral hemorrhage. The reason for A β forming deposits in either of these places remains unknown. Now, new research by a team of researchers from Kanazawa University is shedding light on the deposition patterns formed when A β is introduced to the brain from external sources.

A β exists in different conformations, or strains. The Kanazawa team investigated whether the strain of A β affected the [deposition](#) pattern. To study this, they used a mouse model of AD. The most effective mouse models are ones where the mouse is injected with brain extracts from Alzheimer's patients, known as "seeding".

The team seeded the mice with A β from autopsied patients with different deposition patterns of A β , including those with AD, those with CAA, those with both, and those with very little A β . After one year, the team compared the patterns of deposition between the mice and the patients from whom the A β was taken.

Surprisingly, they found no correlation between the strain of A β that the mice were seeded with and the deposition pattern. All the mice showed the A β deposition in the [blood vessels](#) and had a higher incidence of CAA than mice not seeded with A β , regardless of the strain of A β .

Seeding of A β is not exclusive to [mice](#). It is known that neurosurgeries using tools contaminated with A β or the administration of compounds that may be contaminated with A β might lead to the development of "iatrogenic" A β pathologies in contaminated patients.

"When we induced A β deposition using extracts from patients with different A β pathologies, we observed deposition in the blood vessels rather than the brain parenchyma, regardless of the strain of A β ," explains study lead author Tsuyoshi Hamaguchi. "This may explain why

CAA is a predominant feature of the A β pathology that we see in iatrogenic transmission cases."

The researchers also observed that the amount of A β deposition induced by [brain](#) extracts from the group with less A β was higher than that induced by extracts from the other three groups of patients. "This is significant because patients with little A β pathology could still cause contamination leading to iatrogenic transmission—indeed, the early phase of A β deposition, when little pathology is evident, may in fact be when the transmission risk is highest," says senior author Masahito Yamada. "Methods to inactivate amyloid β seeding activity are therefore urgently needed."

The research was published in *Acta Neuropathologica Communications*.

More information: Tsuyoshi Hamaguchi et al, Exogenous A β seeds induce A β depositions in the blood vessels rather than the brain parenchyma, independently of A β strain-specific information, *Acta Neuropathologica Communications* (2021). [DOI: 10.1186/s40478-021-01252-0](#)

Provided by Kanazawa University

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