
Response:

We thank Drs Hiltunen and Jolkkonen for their interest in our article regarding secondary neurodegeneration after focal cerebral infarction and their thoughtful comments.1 Focal cerebral infarction after middle cerebral artery occlusion not only causes neuronal damage in the area of ischemia, but also leads to secondary neuronal death and glial reaction in remote regions, including the ipsilateral thalamus, substantia nigra, and distal pyramidal tract.1 Retrograde degeneration of the thalamocortical fibers is believed to contribute to secondary neurodegeneration in the thalamus after stroke, but the underlying mechanisms have not been fully elucidated. Drs Hiltunen and Jolkkonen point out that the secondary pathology in the thalamus may have some difference in ischemic rodents and patients with stroke. For example, β-amyloid was shown to abnormally accumulate in the ipsilateral thalamus of rats subjected to middle cerebral artery occlusion and contribute to secondary thalamic damage,2–4 but it did not display a significant association with cerebrovascular lesions in their large postmortem study.5 We agree with Drs Hiltunen and Jolkkonen that species difference should be taken into consideration when translating the results of animal experiments into human studies. However, the location and size of infarction should also be analyzed when investigating secondary damage in the thalamus, because thalamic degeneration is secondary to the primary infarction and may not occur in the patients without any injury to the thalamocortical fibers.1 In the postmortem study, the patients with different sites and sizes of cerebrovascular lesions were not separately analyzed, which may lead to not detecting a significant association between the load of β-amyloid aggregates and cerebrovascular lesions.5 Further postmortem and imaging studies are needed to investigate the association between thalamic β-amyloid deposition and neuronal death in patients with middle cerebral artery infarction. Additionally, we concur that functional significance of neuroprotection against remote damage should also be addressed in future studies.

Disclosures

None.

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Response to Letter Regarding Article, "Secondary Neurodegeneration in Remote Regions After Focal Cerebral Infarction: A New Target for Stroke Management?"
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