Letter by Zuo and Xu Regarding Article, “Delayed Administration of Tat-HA-NR2B9c Promotes Recovery After Stroke in Rats”

To the Editor:

We read with interest the recent study by Zhou et al.1 titled “Delayed Administration of Tat-HA-NR2B9c Promotes Recovery After Stroke in Rats.” The authors performed a study to investigate a potential role and mechanism of Tat-HA-NR2B9c in the subacute phase after stroke, which could provide treatment option to protect neurons against N-methyl-D-aspartate receptor-mediated excitotoxicity after stroke. Based on the authors’ results, we wish to communicate to the authors.

Cyclin-dependent kinase 5 (Cdk5) is a proline-directed serine/threonine kinase involved in the processes of neuronal migration, neurotransmitter release, neuronal plasticity, memory, learning, addiction, and apoptosis.2 Cdk5 activity requires association with neuro-specific activator molecules, called p35, p39 and p67, the most effective activator of cdk5 is p25, which is converted from p35 and has a higher activator activity compared with p35.3

In their study, Cdk5 activity was measured with histone H1 as a substrate, the level of p35, a protein regulating Cdk5 activity was also measured. Integrating existing related research, the elevated Cdk5 activity was consistent with the formation of p25 protein or could be caused by increased levels of p35. But the authors’ results showed that p35 protein level did not change significantly, although Cdk5 activity was significantly increased after stroke. One reason is that the number of animals in each group is too small to detect a significant effect? The other reason is that the increased levels of p35 conversion to p25, and may counteract each other?

Disclosures

None.

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The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/46/8/e192