

Towards understanding the role of neuroprotective effect of adenosine deaminase

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Adenosine deaminase (ADA) is a widely expressed enzyme that catabolizes adenosine and deoxyadenosine into inosine and deoxyinosine, respectively. Adenosine is known to play a protective role by interacting with adenosine receptors when its extracellular concentration is increased. Therefore, ADA has been considered toxic to the central nervous system under ischemia, hypoxia and tissue damage. However, ADA-deficiency results in neurological disorders. We were interested in determining whether ADA is protective or harmful in the striatum, which is especially vulnerable during cerebral ischemia. In order to determine the effects of ADA on transient ischemic stress of the striatum, we used acute rat corticostriatal slices. We found that ADA has substantial neuroprotective effects in the striatum. In addition, we examined whether the neuroprotective effect of ADA is due to the removal of deoxyadenosine. The deoxyadenosine administration does not show acute cytotoxicity, suggesting that the neuroprotective effect of ADA may be due to other mechanisms, such as the recently reported direct effect of ADA on the adenosine A_{2A} receptors.