those seen in rat hippocampal neurons.[17] The decrease of kinase activity appears to be the result of excitotoxicity, because both kinases remain at the same level in the cortical neurons. These findings are consistent with those reported by Churn *et al.*,[19] in which they found that excitotoxic activation of the NMDA receptor resulted in inhibition of CaMK II activity in cultured hippocampal neurons. Exposure of cultured cerebellum granule neurons to subtoxic concentrations of NMDA induces a state of excitoprotection when measured by subsequent exposure to toxic concentrations of amphetamine and NMDA. The excitoprotection phenomenon agrees with those observed in cerebral granule neurons.[20]

We conclude that amphetamine-induced PKC and CaMK II translocation and down-regulation are mediated through NMDA-type glutamate receptors. NMDA receptor is involved in the expression of c-fos mRNA after cortical injury.[25] Further investigation is required to delineate whether the immediate early gene expression is secondary to the PKC or CaMK II signaling pathway. It would also be interesting to investigate whether other addictive drugs exhibit the same response with respect to PKC and CaMK II translocation and down-regulation.

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