CHAPTER VI

Conclusion

In summary, the present results indicate a compensatory up-regulation of NMDA receptor expression reflecting reduced glutamatergic function. The alterations in NMDAR1 receptor found in the striatum and frontal cortex suggest that other components of glutamate synapse may be abnormal in these regions in methamphetamine abuse. Furthermore, the results of EAAT3 expression provide evidence to support the hypothesis of glutamatergic system involvement in the drug dependence. These findings suggest deficits of cortico-striatal innervation and may contribute to the cognitive dysfunction related to hippocampus in drug dependence. Administration of methamphetamine produces the differential changes in glutamategic transmission and a regional specific of glutamatergic dysfunction, and the brain regions are differentially vulnerable to the neurotoxic effects of methamphetamine However, further studies of other glutamatergic markers would be valuable to determine if these changes of EAAT3 expression in striatum and frontal cortex reflect deficits in glutamatergic cortico-striatal neurons. Moreover, other components of glutamatergic neurotransmission are still needed to study in order to investigate abnormalities of glutamatergic system in methamphetamine dependence.