

CHAPTER V

CONCLUSION

In summary, the findings of the present study suggest that the effects of METH cause change of behavioral profiles in both behavioral observation and locomotor activity. The results indicate that ED-METH binge; ED-METH and AB-METH have effects on behavioral responses of rats induced by METH dependence. These results suggested that escalating and binge doses-METH can increase behavior responses and cause hyperlocomotion of rats. Moreover, an impairment of cognitive performance in short-term and long-term memory may be due to METH induce excitotoxicity leading to neuronal apoptosis and neuronal cell death. This result indicates a decrease of PCNA and MAP2 immunoreactive cells may be METH has neurotoxic effects to diminish or induce neuronal cell death. Moreover, a decrease of MBP immunoreactive following METH administration may be because of METH-induced neurotoxicity or neuronal cells death and lead to diminish of myelin sheaths that envelop around the axon of the neurons. However, an increase of GFAP-IR cells may be METH administration can induce an elevation of astrocyte immediately for maintaining brain homeostasis. Therefore, further studies are needed to study with other makers of neuronal stem cells and specific mature neurons such as cholinergic and glutamate neurons which play important role in learning and memory. Moreover, it is interesting to investigate the potential of herb or drug that can elevate neurogenesis and gliogenesis for treatment METH abuser after METH dependence.