CHAPTER VI

CONCLUSION

In our experiments, VPU obviated the pilocarpine-induced seizures and significantly diminished the enhancement of excitatory amino acid neurotransmitters (glutamate and aspartate). Furthermore VPU was found to abolish the elevation of lipid peroxidation and mitochondria dysfunction induced by pilocarpine. Not only being able to protect the rats from behavioral features of seizures but pretreatment of VPU also could accordingly protect neuronal damage induced by pilocarpine.

The results obtained suggest that protection of VPU against pilocarpine-induced SE was accounted by its ability to reduce the levels of glutamate and aspartate which were significantly increased by pilocarpine. Furthermore it could be considered that VPU protected prolonged seizure-induced by pilocarpine resulting in a reduction of the enhancement of lipid peroxidation and restoring mitochondria dysfunction. These might explain the ability of VPU in the attenuation of the neuronal damage induced by pilocarpine.

Furthermore in addition to those mechanisms being demonstrated in the present study, some other mechanisms should also be explored.