CHAPTER VI

CONCLUSION

The objectives of the present study were (1) to determine the roles of the 5-HT_{2A} receptor in the chronic pain model and the development of the chronic pain state. (2) To investigate the role of the 5-HT_{2A} receptor in 5-HT depleted state on the changes of pain sensation.

The CFA-induced chronic inflammation resulted in the increase in nociceptive behaviors; shorten the paw withdrawal latency as well as the activation of Fos protein in the somatosensory cortex. It was found that nociceptive behaviors started to decrease in Day 3. On the other hand, Fos protein expressed in the highest level in Day 3 then drop in the later day. While the paw withdrawal latency recovered in 7 days after CFA-introduction. All findings indicate the modulation of pain processing.

These phenomena were reversed by the administration of 5-HT_{2A} receptor, ketanserin. However, in the normal condition, ketanserin failed to alter those of measurements. It may implicate that 5-HT_{2A} receptor does not play a primary role in facilitating inflammatory pain.

In addition, it was found that injection of 100 mg/kg-body-weight of PCPA did not alter the spontaneous behavior of the rats, but it affected the thermal-hyperalgesia. While the number of Fos-IR neurons did not change during the experiment. Moreover, the effect of ketanserin in altering all of the pain measurements was not found. It may indicate that 5-HT_{2A} receptor does not have a role in modulating the sensitivity of thermal-nociceptor.

Theses findings indicate that 5-HT_{2A} receptor facilitates pain in chronic chemical nociception and this receptor does not play a primary role in modulating the thermal-hyperalgesia in normal condition. However, 5-HT_{2A} receptor does not have a role in modulating the sensitivity of thermal-nociceptors in both normal condition and low serotonin state.