CHAPTER VI
CONCLUSION

1. Curcumin prevents oxidative and nitrative stress induced by praziquantel treatment in *O. viverrini*-infected hamsters.

2. Curcumin decreased accumulation of eosinophils infiltration, and increased mononuclear cell infiltration in inflamed areas surrounding the worm in the liver after short-term praziquantel treatment.

3. Curcumin enhanced the expression of genes nuclear factor-erythroid 2-related factor 2 (Nrf2) and heme oxygenase-1 (HO-1) at the transcriptional and protein levels. Curcumin also enhanced the expression of genes involved in Nrf2-regulated stress pathway (Kelch-like ECH-associated protein 1, NAD(P)H:quinine oxidoreductase 1, glutamate cysteine ligase, and activating transcription factor 3, peroxiredoxin 3, peroxiredoxin 6, manganese superoxide dismutase, and catalase), leading to increased ferric antioxidant capacity (FRAP) in the plasma.

4. Curcumin suppressed of NF-κB and related molecules cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) and proinflammatory cytokines (IL-1β and TNF- α).

5. Curcumin tended to decrease the plasma levels of oxidative/nitrative stress markers [malondialdehyde (MDA) and nitrate/nitrite (NOx)] and liver injury (ALT) levels. Also curcumin decreased the level of urinary 8-oxo-7, 8-dihydro-2'-deoxyguanosine (8-oxodG) after short-term praziquantel treatment.

6. In summary, curcumin may be an effective chemopreventive agent against oxidative and nitrative stress derived from after short-term praziquantel treatment during *O. viverrini* infection.