CHAPER V

CONCLUSION

The overall results of this thesis demonstrate that for the rat mucosal RGM1 and its corresponding cancer RGK1 cells, very low Mn-SOD activity were measured. The lack of Mn-SOD might cause an increase in the mitochondrial $O_2^{-\bullet}$ concentration thus affected the mitochondrial function and cell growth. Contrary, RGM1 should contain the mitochondrial antioxidant system that have more efficient than RGK1.

Both DMSO and quercetin should be considered as chemical stressors, their pro-oxidant action caused cellular lipid peroxidation resulted in the 4-HNE protein adducts, which were digested in vacuoles of the RGK1 and more extensively in MnSOD transfected cells but not in normal cells. This is the characteristic of autophagy. The density and sizes of autolysosomes affected the cellular physiology that caused later growth arrest and death. The autophagic RGK1 cells were slightly observed in the presence of 200 mM quercetin but extensively found in Mn-SOD transfected cells. This signifies that Mn-SOD regulates the autophagy in rat mucosal gastric RGK1 cells.

These findings clearly point out the window difference of the rat normal mucosal gastric and its corresponding cancer cells provide a new and important approach for cancer diagnosis and anticancer drug research.