CHAPTER IV

DISCUSSION & CONCLUSION

The exposure chamber used in this study was satisfied and fulfilled our purpose for the study of the toxic carbon monoxide gas. The chamber was made of simple material, cheap and could prevent carbon monoxide leak from the chamber. However, the carbon monoxide produced from the in-house carbon monoxide generator might not be mixed well in the first part of the chamber sometimes and caused the non-homogeneous gas in the second part of the chamber. So, experimental rats might not be exposed to the carbon monoxide gas at the same concentration. But the concentration of the carbon monoxide gas was monitored all the time of our exposure experiments.

It was shown that most of hematology and blood chemistry determinants of the carbon monoxide exposure rats in both sex were not significantly different from the control rats. It seems like female rats were more sensitive to the carbon monoxide toxicity than male rats. It might be the hormonal effect on carbon monoxide poisoning even though there has not been any evidence to support this suggestion.

There was also no reasonable explanation for some of the rat 's organs weight which were not increased after the three months of carbon monoxide exposure. It might be due to the idiosyncratic effects or a technical error while doing animal surgury.

Hematological values in the carbon monoxide exposure rats were increased compared to the control rats. The results show an increase of hemoglobin concentration as well as the hematocrit ratio which might be a response to hypoxia that served to increase the oxygen carrying capacity of the blood (IPCS, 1999). Guyton & Richardson (1961) and Smith & Growell (1967) suggested that changes in hematocrit ratio not only affected the oxygen carrying capacity of the blood, but also affected blood flow as well. Therefore, when hematocrit ratio increased much above normal, oxygen delivery to the tissues would be reduced.

In this study we did not measure urine creatinine of the carbon monoxide exposure rats, then the increase of blood creatinine in the female rats after 3 months carbon monoxide exposure could not be interpreted as renal toxicity.

Cellular toxicity of carbon monoxide exposure was determined by measuring of serum malondialdehyde level. Thom (1990) showed evidence for the occurrence of lipid peroxidation in the brain of carbon monoxide-poisoned rats that serum malondialdehyde level in exposure rats in both sex were not different from the control which was correlated to this study. Actually, the results showed that malondialdehyde level in the carbon monoxide exposure female rats appeared to decrease compared to the control female rats. It was related to high blood carboxyhemoglobin in the female rats after carbon monoxide exposure.

Glutathione is one of the cell oxidative defense mechanism, which protect the cell from oxidative stress. When cell exposed to toxicants that could cause oxidative stress, the cells would adapt with an increased production of glutathione. In this study, we found an increase of glutathione level, even though it was not significantly different from the control rats. The same result had been noticed with cytochrome *c* reduction determination in the rat 's brain which was not significantly different but appeared to be decreased.

This study can be concluded that experimental animal chamber which was designed and constructed in this research was suitable for a study of toxicity of carbon monoxide and other toxic gases in ambient air. Low level exposure to subchronic carbon monoxide in the rats caused an increase of carboxyhemoglobin and a change in hematology parameters. However, the exposure to carbon monoxide at low concentration did not show significant toxicity in the exposed rats. Even though it was appeared that the rats might be harmful if there was a chronic carbon monoxide exposure.