

CHAPTER VI

CONCLUSION AND FUTURE DIRECTION

VI.1 Conclusion

1. GSTM1 null gene tend to be risk factor of ASD and there is no association between GSTM1 null with ASD although the frequency of GSTM1 null in ASD higher compared with control group.
2. GSTT1 null gene tend to be risk factor of ASD and there is no association between GSTT1 null with ASD, while GSTT1 null genotype's frequency is higher compared with control group.
3. Low erythrocyte GST activity tend to be risk factor of ASD.
4. The mean erythrocyte GST activity in ASD is lower compared with controls.
5. GSTM1 null and GSTT1 null is not associated with phenotype expression of ASD.
6. GSTM1 null and GSTT1 null is not associated with erythrocyte GST activity.
7. There is no association between erythrocyte GST activity with phenotype expression of ASD although mean erythrocyte GST activity in severely autistic is lower than mild to moderately autistic.

VI.2 Future Direction

Study for determining polymorphism of detoxification gene in autism is important. GST genes are one of the detoxification genes. Glutathione S-transferases (GST) are antioxidant enzymes that play important role in cellular detoxification and the excretion of environmental pollutants including many carcinogens, environmental toxins and drugs. Some genetic polymorphisms of GST are known to affect enzyme function. This polymorphism potentiated by reduced levels of GSH (one of the substrates of GST) could contribute to the pathogenesis of autism.

Genetic and environmental risk factors could lead to failure of individuals with autism to detoxify important compounds, including agents or products of oxidative stress. This is consistent with the hypothesis of a gene-environment interaction that alters the expression of autism because GSTs are detoxification enzymes that conjugate absorbed xenobiotics. These findings could lead to the mechanism of action of select environmental chemicals and identification of an exogenous or endogenous moiety interacting with GSTs to contribute to the phenotypic presentation of autism (Steven Buyske et al.,2006).

Therefore, this preliminary study needs further investigations with increased sample size,multiple genes and GST activity determination, to find out gene susceptibility of ASD and factors that contribute to the phenotype expression of ASD.