Chapter 6

Conclusion

For the first objective which was to determine the dietary ITC intake and its association with oral cancer risk, this study has concluded that dietary ITC intake was not associated with oral cancer risk. However, analyses based solely on FFQ data may be limited by reporting errors, constrained food lists, and natural variability in glucosinolate profiles.

As for the second objective which was to determine the *GSTM1, GSTT1* and *GSTP1* polymorphism and its association with oral cancer risk, it was observed that there was no significant association between *GSTM1, GSTT1* and *GSTP1* polymorphism and oral cancer risk. However, this study suggests that the *GSTP1* codon 105 polymorphism may play an important role in risk for oral cancer.

Lastly, for the third objective which was to determine the association between dietary ITC-*GSTs* polymorphisms interaction and oral cancer risk, the present study found there was no significant association between dietary ITCs -*GSTs* polymorphisms interaction and oral cancer risk. Nevertheless, there were some indications of interactions between intake of ITCs and the *GSTP1* polymorphism, which suggest that susceptible persons might lower their risk of oral cancer by increasing their intake of cruciferous vegetable, thus need further investigation.

Overall, this study was not able to totally reject all the three null hypotheses as there were no significant association observed between dietary ITC intake and oral cancer risk, *GSTs* polymorphisms and oral cancer risk and dietary ITC-*GSTs* polymorphisms interaction and oral cancer risk.

6.1 Recommendations

Again, this study has three objectives which in brief were to determine if any associations occur between dietary ITC intake, *GSTs* polymorphisms and ITC-*GSTs* polymorphisms interaction with oral cancer risk. Based on the conducting, findings and limitation of this study in achieving these objectives, it is recommended that future studies involve a larger sample size in order to be able to detect even a smaller but meaningful and clinically important difference in dietary ITC intake between cases and controls when the difference truly exists. Sample size estimation also needs to take into account the objective of *GST* polymorphism and oral cancer risk including getting the prevalence of the respective genes from different population. Besides increasing the sample size, increased attention should also be given to methodological considerations such as the appropriate selection of controls, establishing a meaningful and standardized cut-off point of high and low ITC intake and method of ITC assessment.

The mechanisms by which ITCs exert their effect deserve additional study and may provide useful clues to the etiology of oral cancer in these populations. Further investigation should also be directed to ITC-*GST* interaction roles in developing oral cancer especially for *GSTs* that have shown indication of influencing oral cancer risk. More genetic factors should be assessed together in future studies since attribution of *GSTM1*, *GSTT1* and *GSTP1* gene deficiency as a risk for oral cancer was found to be of possible significance but of limited magnitude. Eg CYP1A1 inter related in activation of carcinogen before excretion by phase II enzyme.

Studying populations with varying dietary or smoking behaviors also may provide insight into how both absolute and relative exposure levels of tobacco smoke

and ITCs may alter the ability of ITCs to inhibit carcinogenesis. In addition, studies focused on other components in cruciferous vegetables and additional metabolic polymorphisms will help us understand potential interactions between these multiple factors. In fact, besides investigating only into ITCs-*GSTs* polymorphisms roles in modifying oral cancer risk, it is also very much recommended to study in depth about risk habits such as tobacco smoking, alcohol drinking and betel-quid chewing as in this study; these established risk factors showed statistical significance association with oral cancer risk. Looking into if interaction exists between smoking, alcohol drinking and betel quid chewing with ethnicity or with *GST* polymorphism would be very interesting. Furthermore, these *GSTM1*, *GSTT1* and *GSTP1* genes are related in detoxifying the tobacco PAHs compounds. Measurement of lifetime exposures to tobacco (measured as both dose and duration) will help to minimize heterogeneity in the assessment of gene-environment interaction. Indeed or perhaps ethnicity actually does not play a role in this genetic susceptibility study but rather their habits.

Overall, it is strongly recommended that this study should be given further attention in future, thus to confirm certain speculations or uncertainties and also interesting findings observed in this study.