Chapter 4

Results

4.1. Socio-demographic profile of cases and controls

This case-control study consisted of 115 cases of oral cancer and 116 controls. All the analysis was done based on these unmatched case-control study subjects. The selected socio demographic profiles of cases and controls were summarized in Table 4.1. Of all the selected socio demographic profiles, significant differences between cases and control group were detected in five variables: age, gender, ethnicity, alcohol drinking and betel quid chewing status. In average, cases were noted as significantly older than the controls in which the mean age of the cases and controls were 59.6 $\pm$ 12.0 and 40.8 $\pm$ 11.7 years, respectively. As the gender distributions was reported to be statistically significant among cases and controls ($p = 0.039$), it was also observed that females were overrepresented in the group of cases (63.6%) as compared to the males (36.5%). On the other hand, the gender distributions between males and females were identical within the control group with each gender represented 50%.

With respect to ethnicity, high significant difference between the case and the control groups was seen ($p = 0.000$). Assessing across the groups, Indians dominated among the cases (42.6%), while there were more Indigenous ethnicity (56.9%) compared to others in the controls. The proportion of Malays was marginally different between the two groups (cases 19.1% and controls 22.4%).

Similar pattern of distribution can also be detected in the other two significant variables, alcohol drinking and betel nut chewing status. For both habits, the proportion in the better end which was non-drinkers and non-betel quid chewers was significantly higher in controls than those in the cases. In fact, the non-betel quid chewers among the cases were substantially lower (44.3%) than among the controls (93.1%). On the contrary, there were larger proportions of cases as compared to control that drink alcohol and chew betel quid. Betel-quid chewers constituted 55.7% of the cases and 6.9% of the controls.

No significant difference however was observed among cases and controls in terms of family history of cancer and smoking status. Approximately 81% and 85% of cases and controls appeared do not have prior family history of cancer. As for the smoking habit, the distributions of non-smokers in the cases (67.8%) were comparable to that in the controls (64.7%). Fewer smokers were represented in the case group as there were more smokers among the controls (35.3%) than the cases (32.2%) but the difference was not significant.

As the aim and design of the study was not to re-evaluate well established lifestyle risk factors for oral cancer, there was no attempt made to approximate the total amount of smoking, alcohol consumption or chewing, in terms of amount per day or duration in years.

Table 4.1: Socio-demographic profile of 115 cases and 116 control subjects

Socio demographic profile	Oral cancer status: Control Frequency (%)	Oral cancer status: Cases Frequency (%)	χ^2 statistics (df)	p-value
Age in years	40.8 (11.7)[a]	59.6 (12.0)[a]	12.085 (229)[b]	0.000
Gender				
Male	58 (50.0)	42 (36.5)	4.273 (1)	0.039
Female	58 (50.0)	73 (63.6)		
Ethnic				
Chinese	5 (4.3)	16 (13.9)	34.689 (3)	0.000
Malay	26 (22.4)	22 (19.1)		
Indian	19 (16.4)	49 (42.6)		
Indigenous	66 (56.9)	28 (24.3)		
Family history of cancer				
No	86* (85.1)	92* (80.7)	0.743 (1)	0.389
Yes	15 (14.9)	22 (19.3)		
Smoking status				
No	75 (64.7)	78 (67.8)	0.260 (1)	0.610
Yes	41 (35.3)	37 (32.2)		
Alcohol drinking status				
No	108 (93.1)	76 (66.1)	26.008 (1)	0.000
Yes	8 (6.9)	39 (33.9)		
Betel-quid chewing status				
No	108 (93.1)	51 (44.3)	63.986 (1)	0.000
Yes	8 (6.9)	64 (55.7)		

[a] mean (SD)
[b] t-statistic (df)
* n not tally due to missing data

4.2 Dietary ITC intake and GSTs polymorphisms

Table 4.2 shows the distribution of dietary ITC intake and *GSTs* polymorphisms in the case and control groups. In the beginning, the dietary ITC intake was treated as continuous variable. However, for it to be meaningful, it was then grouped into low and high ITC intake based on the median dietary ITC intake value. Because there is no established cut off point to indicate low and high intake of ITC, median was chosen as cut off point especially in this study where the distribution was skewed. Among all the 231 study subjects, the distribution of estimated dietary intake of ITC per 1000kcal was noted as markedly skewed to the right, with a range of 0.00 – 92.81μmol and a median of 2.31μmol (IQR 0.60 – 4.27). This skewed distribution was almost similar between cases and controls in which the median energy-adjusted dietary intake of ITC among cases was 2.380μmol/1000kcal (IQR 3.86) and among controls 2.255μmol/1000kcal (IQR 3.62). However, the slight difference in these median ITC intakes between cases and controls was not statistically significant (p = 0.671). When grouped into low and high ITC levels, high consumption of dietary ITC was seen as slightly greater in cases (51.3%) than in controls (48.3%) and low consumption of dietary ITC on the contrary was more in controls (51.7%) as compared to cases (48.7%), although these was also not statistically significant (p = 0.645).

As for *GSTs* polymorphisms, assessing across case and control groups, it was demonstrated that even though less proportion of cases had *GSTM1* null and *GSTT1* null than that of controls, the difference was small thus it became not statistically significant (p = 0.746, 0.831). Polymorphism of *GSTP1* gene also was found lower among cases (47.8%) than that among controls (58.6%). Individuals with this low activity genotype however,

were also observed to be not significantly differed among the cases and controls. Nevertheless, this is actually a slightly different scenario in which although the difference in the proportion of cases and controls having polymorphism in their *GSTP1* was not significant (p = 0.100), it differed by 11%. No significant difference was also found in all non-null genotypes of *GSTM1* and *GSTT1* and wild type genotype of *GSTP* between cases and controls.

When the genotypes were combined, the percentage of polymorphism for the *GSTM1* and *GSTT1* combined was noted as 72.2% among cases and 73.3% among controls, while the presence of the wild-type genotype was only slightly higher among the cases (27.8%) than the controls (26.7%), thus no significant difference in the combined *GSTM1* and *GSTT1* gene between cases and controls was detected (p = 0.851). The proportion of cases and controls with the *GSTM1, GSTT1* and *GSTP1* combined polymorphism genotypes also was almost similar. The combined *GSTM1, GSTT1* and *GSTP1* genotype was polymorphic in 83.5% of the cases and 88.8% of the controls. Meanwhile, a total of 16.5% of the cases was presented with wild-type genotype of the combined *GSTM1, GSTT1* and *GSTP1* gene, and only 11.2% was detected among healthy controls in which it appeared as not statistically significant (p = 0.242).

Table 4.2: Distribution of dietary ITC intake and GSTs polymorphism in 115 cases and 116 control subjects

	Oral cancer status			
	Control Frequency (%)	Cases Frequency (%)	χ^2 statistics (df)	p-value
Dietary ITC intake per 1000kcal	2.255 (3.62)[a]	2.380 (3.86)[a]	-0.424[b]	0.671
Dietary ITC intake level[c]				
Low	60 (51.7)	56 (48.7)	0.212 (1)	0.645
High	56 (48.3)	59 (51.3)		
GSTM1				
Non-null	51 (44.0)	53 (46.1)	0.105 (1)	0.746
Null	65 (56.0)	62 (53.9)		
GSTT1				
Non-null	68 (58.6)	69 (60.0)	0.046 (1)	0.831
Null	48 (41.4)	46 (40.0)		
GSTP1				
Wild-type	48 (41.4)	60 (52.2)	2.703 (1)	0.100
Polymorphism	68 (58.6)	55 (47.8)		
GSTM1/GSTT1				
Wild-type	31 (26.7)	32 (27.8)	0.035 (1)	0.851
Polymorphism	85 (73.3)	83 (72.2)		
GSTM1/GSTT1/GSTP1				
Wild-type	13 (11.2)	19 (16.5)	1.367 (1)	0.242
Polymorphism	103 (88.8)	96 (83.5)		

[a] median (IQR) [b] z-statistic

[c] low (or high) ITC levels were defined as lower (or higher) than the median dietary ITC intake (2.31μmol/1000kcal) among all case-control subjects

4.3 Factors associated with oral cancer

4.3.1 Socio-demographic profiles and oral cancer

Table 4.3 shows the results of simple logistic regression (SLR) analysis for the association between socio demographic profiles and oral cancer risk. It reveals that age was significantly associated with oral cancer risk. Older patients have 13% increased in risk of having oral cancer with an OR of 1.13 (95%CI 1.097 – 1.169). Significant associations were also found between gender, ethnicity, alcohol drinking, betel quid chewing status and oral cancer risk. The females posed to be 1.7 times higher risk of having oral cancer than males (OR 1.74, 95% CI: 1.027 – 2.941). As compared to Chinese, Malays was observed to have 74% less oral cancer risk (OR 0.26, 95% CI: 0.083 – 0.838) while the Indigenous also had reduction in risk by 87% (OR 0.13, 95% CI: 0.044 – 0.397). Habitual alcohol drinkers had a significantly 6.9 times higher risk of having oral cancer than non-drinkers, showing an OR of 6.93 (95% CI: 3.065 – 15.656) and a very significant increased in oral cancer risk was also detected among subjects who chewed betel-quid compared with those who did not, giving an OR of 16.94 (95% CI: 7.560 – 37.965). On the other hand, there was no association discovered between family history of cancer, smoking status and oral cancer risk. Although those with family history of cancer showed 1.3 times higher oral cancer risk as compared to those without family history of cancer (OR 1.37, 95% CI 0.668 – 2.814) and being cigarette smokers showed lower risk of getting oral cancer (OR of 0.87, 95% CI: 0.503 – 1.498), the findings appeared to be not statistically significant. Therefore, of all the socio demographic profiles of interest, only five factors namely age, gender, ethnicity, alcohol drinking and betel quid chewing status were found to be significantly associated with oral cancer risk at univariate level.

Table 4.3: Association between socio-demographic profiles and oral cancer by simple logistic regression analysis

Factor	Oral cancer		Crude OR	95% CI	Wald χ^2 (df)	p-value[b]
	Control Frequency (%)	Cases Frequency (%)				
Age in years	40.8 (11.7)[a]	59.6 (12.0)[a]	1.13	1.097 – 1.169	60.05 (1)	0.000
Gender						
Male	58 (50.0)	42 (36.5)	1			
Female	58 (50.0)	73 (63.5)	1.74	1.027 – 2.941	4.245 (1)	0.039
Ethnic						
Chinese	5 (4.3)	16 (13.9)	1			
Malay	26 (22.4)	22 (19.1)	0.26	0.083 – 0.838	5.108 (1)	0.024
Indian	19 (16.4)	49 (42.6)	0.81	0.259 – 2.508	0.139 (1)	0.710
Indigenous	66 (56.9)	28 (24.3)	0.13	0.044 – 0.397	13.029 (1)	0.000
Family history of cancer						
No	86 (85.1)	92 (80.7)	1			
Yes	15 (14.9)	22 (19.3)	1.37	0.668 – 2.814	0.740 (1)	0.390
Smoking status						
No	75 (64.7)	78 (67.8)	1			
Yes	41 (35.3)	37 (32.2)	0.87	0.503 – 1.498	0.279 (1)	0.610
Alcohol drinking status						
No	108 (93.1)	76 (66.1)	1			
Yes	8 (6.9)	39 (33.9)	6.93	3.065 – 15.656	21.647 (1)	0.000
Betel-quid chewing status						
No	108 (93.1)	51 (44.3)	1			
Yes	8 (6.9)	64 (55.7)	16.94	7.560 – 37.965	47.244 (1)	0.000

[a] mean (SD) [b] Wald test

4.3.2 Dietary ITC intake and oral cancer

Table 4.4 summarizes the effect of dietary ITC intake on oral cancer risk at univariate level which was analyzed using simple logistic regression. From the table, it shows that the median dietary intake of ITC per 1000 kcal was found to be not significantly associated with oral cancer risk (OR = 1.00, 95% CI = 0.965 – 1.034). Interestingly, it was noticed that when dietary ITC intake was grouped into high (greater than/equal to median) and low (less than median) intake, high ITC intake was observed to be associated with increased risk of having oral cancer by 13% (OR = 1.13, 95% CI = 0.674 – 1.891), although this also was not statistically significant (P = 0.645).

Table 4.4: Association between dietary ITC intake and oral cancer by simple logistic regression analysis

	Oral cancer status					
	Control Freq (%)	Cases Freq (%)	Crude OR	95% CI	Wald χ^2 (df)	p-value[b]
Dietary ITC intake per 1000kcal	2.255 (3.62)[a]	2.380 (3.86)[a]	1.00	0.965 – 1.034	0.004 (1)	0.950
Dietary ITC intake level[c]						
Low	60 (51.7)	56 (48.7)	1	0.674 - 1.891	0.212 (1)	0.645
High	56 (48.3)	59 (51.3)	1.13			

[a] median (IQR)
[b] Wald test
[c] low (or high) ITC levels were defined as lower (or higher) than the median dietary ITC intake (2.31μmol/1000kcal) among all case-control subject

4.3.3 GSTs polymorphisms and oral cancer

A summary of the results from simple logistic regression analysis for the association between the *GSTM1, GSTT1* and *GSTP1* genotypes and oral cancer risk a univariate level is shown in Table 4.5. It reports that the OR for *GSTM1* null genotype was 0.92 (95% CI: 0.546 – 1.542), relative to *GSTM1* non – null, and that for the *GSTT1* null genotype was 0.94 (95% CI: 0.559 – 1.597) versus *GSTT1* non-null genotype. The OR for *GSTP1* polymorphism genotype, as compared to the wild-type genotype, was 0.65 (95% CI 0.385 – 1.088). Although all OR values of *GSTs* polymorphisms indicated reduced risk against oral cancer risk, neither *GSTM1* or *GSTT1* null nor the *GSTP1* polymorphism genotype was revealed as significantly associated with oral cancer risk. Overall, there was no significant association observed between the *GSTM1, GSTT1,* and *GSTP1* genotypes and oral cancer risk.

For the combined *GSTM1* and *GSTT1* gene, as compared to the wild type genotype, the polymorphism of *GSTM1* and *GSTT1* combined has demonstrated an OR as 0.95 (95% CI 0.530 – 1.688) which indicate that individuals with polymorphism for both *GSTM1* and *GSTT1* genotypes was associated with reduced and not with increased oral cancer risk. Unfortunately, this finding was not statistically significant. For the combined polymorphism of *GSTM1, GSTT1* and *GSTP1* in relative to the wild-type genotype, the OR was reported to be 0.64 (95% CI 0.299 – 1.361). Subjects carrying the polymorphism genotype of all the three analyzed GSTs however, also were not significantly associated with the risk of having oral cancer. Therefore, there was no significant association between the polymorphisms in *GSTM1, GSTT1* and *GSTP1* and oral cancer risk was discovered. The effect of the GST-susceptible genotypes on oral

cancer risk is not increased with the combined polymorphism of either combination of *GSTM1* and *GSTT1* or *GSTM1*, *GSTT1* and *GSTP1*.

Table 4.5: Association between GSTs polymorphisms and oral cancer by simple logistic regression analysis

	Oral cancer status					
	Control Frequency (%)	Cases Frequency (%)	Crude OR	95% CI	Wald χ^2 (df)	p-value[a]
GSTM1						
Non-null	51 (44.0)	53 (46.1)	1			
Null	65 (56.0)	62 (53.9)	0.92	0.546 - 1.542	0.105 (1)	0.746
GSTT1						
Non-null	68 (58.6)	69 (60.0)	1			
Null	48 (41.4)	46 (40.0)	0.94	0.559 - 1.597	0.046 (1)	0.831
GSTP1						
Wild-type	48 (41.4)	60 (52.2)	1			
Polymorphism	68 (58.6)	55 (47.8)	0.65	0.385 - 1.088	2.692 (1)	0.100
GSTM1/GSTT1						
Wild-type	31 (26.7)	32 (27.8)	1			
Polymorphism	85 (73.3)	83 (72.2)	0.95	0.530 - 1.688	0.035 (1)	0.851
GSTM1/GSTT1/GSTP1						
Wild-type	13 (11.2)	19 (16.5)	1			
Polymorphism	103 (88.8)	96 (83.5)	0.64	0.299 - 1.361	1.352 (1)	0.245

[a] Wald test

4.3.4 Dietary ITC intake, GSTs polymorphisms and oral cancer

Table 4.6 summarizes the results of multiple logistic regression for the association between dietary ITC, *GSTs* polymorphisms and oral cancer risk adjusted for other associated factors such as socio-demographic profiles. At multivariate level, neither dietary ITC nor *GSTs* polymorphisms have significant association with oral cancer risk. Among other factors investigated, four factors namely age, ethnic, alcohol intake and betel quid chewing were significantly associated with oral cancer risk. Older people had higher risk against oral cancer with an OR of 1.13 (95% CI: 1.084 – 1.174). Among ethnic groups, Indigenous was found to have lower risk compared to reference group Chinese with an OR of 0.05 (95% CI: 0.033 – 1.050). Consumption of alcohol and betel quid chewing also conferred higher risk against oral cancer with OR of 15.04 (95% CI: 3.652 – 61.966) and 21.25 (95% CI: 6.366 – 70.955) respectively. However, further steps to check for interaction between the significant factors was not pursued since all significant factors were not the factors of interest in this study. Therefore, we conclude that there was no significant association found between the dietary ITC & *GSTs* polymorphisms and oral cancer risk.

Table 4.6: Association between dietary ITC, GSTs polymorphisms and oral cancer by multiple logistic regression analysis

	Oral cancer status		Adjusted OR	95% CI	Wald χ^2 (df)	p-value[b]
	Control Frequency (%)	Cases Frequency (%)				
Age in years	40.8 (11.7)[a]	59.6 (12.0)[a]	1.13	1.084 – 1.174	35.293 (1)	< 0.001
Ethnic						< 0.001
Chinese	5 (4.3)	16 (13.9)	1			
Malay	26 (22.4)	22 (19.1)	0.53	0.109 – 2.586	0.614 (1)	0.433
Indian	19 (16.4)	49 (42.6)	0.19	0.009 – 0.216	3.626 (1)	0.057
Indigenous	66 (56.9)	28 (24.3)	0.05	0.033 – 1.050	14.937 (1)	< 0.001
Alcohol drinking status						
No	108 (93.1)	76 (66.1)	1			
Yes	8 (6.9)	39 (33.9)	15.04	3.652 – 61.966	14.086 (1)	< 0.001
Betel-quid chewing status						
No	108 (93.1)	51 (44.3)	1			
Yes	8 (6.9)	64 (55.7)	21.25	6.366 – 70.955	24.694 (1)	< 0.001

[a] mean (SD) [b] Wald test

To achieve the third objective which was to investigate the association between dietary *ITC-GSTs* polymorphisms interaction and oral cancer, Mantel Haenszel stratified analysis was employed. Table 4.7 presents the association between dietary ITC intake of cruciferous vegetables and oral cancer risk stratified by *GSTs* wild-type and polymorphism genotypes separately for *GSTM1, GSTT1* and *GSTP1*. Low ITC intake level was used as the reference group.

From Table 4.7, there was no significant association between dietary ITC and oral cancer risk was observed when stratified by *GSTM1* or *GSTT1* genotypes. Fairly similar odd ratios were found across strata when the association between dietary ITC and oral cancer risk was examined among subjects grouped by either the *GSTM1* or *GSTT1*. The OR of high ITC in relative to low ITC intake among *GSTM1* non-null and null individuals was 1.16 (95% CI 0.538 – 2.511) and 1.10 (95% CI 0.545 – 2.196) respectively. It suggests that there was no evidence that effect of high dietary ITC intake on oral cancer was modified by *GSTM1* deletion, however it was not significant. Meanwhile, the OR of high ITC intake was 1.23 (95% CI: 0.627 – 2.399) among *GSTT1* non-null genotype subjects and 1.00 (95% CI: 0.443 – 2.239) among *GSTT1* null genotype. Although it suggests a slight reduction in risk in absence of *GSTT1*, it was not statistically significant. These ORs did not exactly differ from their common OR indicating that neither confounding nor interaction exists between *GSTM1*, *GSTT1* and the dietary ITC. However, this interpretation is valid in case of significant association.

There was also no significant association observed between dietary ITC and oral cancer risk when *GSTP1* was controlled. However, difference in OR of high versus low ITC was observed between *GSTP1* wild type and polymorphism individuals.

Among *GSTP1* polymorphism subjects, the OR for high versus low ITC intake was 0.80 (95% CI 0.387 – 1.635) compared with an OR of 1.48 (95% CI: 0.686 – 3.194) among *GSTP1* wild type subjects. It was noted that high ITC intake conferred a 20% reduction in risk among those with polymorphism genotype for *GSTP1* although it was not statistically significant. The differences between the OR for high versus low ITC intake among total subjects, *GSTP1* non-null and null suggest towards a possibility of interaction occurs between the *GSTP1* and dietary ITC intake, however this also was not significant.

When the association between dietary ITC intake and oral cancer was stratified by *GSTM1* and *GSTT1* genotypes combined, the risk of oral cancer among individuals with wild-type genotypes was discovered as not significantly associated with ITC intake even though the OR indicating protective effect of high ITC (OR 0.93, 95% CI: 0.346 – 2.515). Likewise, there was also no significant association between cruciferous vegetable intake and oral cancer risk among the polymorphism genotype of the combined *GSTM1* and *GSTT1* (OR 1.21, 95% CI: 0.659 – 2.215).

Meanwhile, among the combined *GSTM1, GSTT1* and *GSTP1* genotypes, the protective effect of high dietary ITC intake against oral cancer was observed as not significant among individuals wild-type genotype (OR 0.96, 95% CI 0.210 – 4.421). Among subjects with *GSTM1, GSTT1* and *GSTP1* polymorphism genotypes, high ITC also seemed has no significant effect on the oral cancer risk (OR 1.10, 95% CI 0.628 – 1.914). Perhaps there were too few cases ($n < 10$) that were wild type for the combined *GSTM1, GSTT1* and *GSTP1* genotypes for a meaningful analysis.

Interestingly, the same trend was observed among the individuals with wild-type genotype for *GSTM1* and *GSTT1* combined or *GSTM1*, *GSTT1* and *GSTP1* combined where high dietary ITC intake could possibly conferred a 7% or 4% decrease in oral cancer risk respectively although it has yet to prove its significance.

Overall, either using multivariate or stratified analysis, there was no significant association observed between the dietary ITC intake, *GSTs* polymorphisms, *ITC-GSTs* polymorphisms interaction and oral cancer risk. These observations may be the result of a true lack of association and interaction or to the effect of small sample sizes.

Table 4.7: Dietary ITC intake in relation to risk of oral cancer stratified by GST genotypes

	Oral cancer status				
	Control Freq (%)	Cases Freq (%)	OR	95% CI	p-value
Total subjects					
Low ITC[c]	60 (51.7)	56 (48.7)	1		
High ITC	56 (48.3)	59 (51.3)	1.12	0.670 – 1.885	0.658
GSTM1 Non-null					
Low ITC	25 (49.0)	24 (45.3)	1		
High ITC	26 (51.0)	29 (54.7)	1.16	0.538 – 2.511	0.703
GSTM1 Null					
Low ITC	35 (53.8)	32(51.6)	1		
High ITC	30 (46.2)	30(48.8)	1.10	0.545 – 2.196	0.801
Total subjects					
Low ITC	60 (51.7)	56 (48.7)	1		
High ITC	56 (48.3)	59 (51.3)	1.13	0.673 – 1.888	0.650
GSTT1 Non-null					
Low ITC	35 (51.5)	32 (46.4)	1		
High ITC	33 (48.5)	37 (53.6)	1.23	0.627 – 2.399	0.551
GSTT1 Null					
Low ITC	25 (52.1)	24 (52.2)	1		
High ITC	23 (47.9)	22 (47.8)	1.00	0.443 – 2.239	0.993
Total subjects					
Low ITC	60 (51.7)	56 (48.7)	1		
High ITC	56 (48.3)	59 (51.3)	1.06	0.673 – 1.888	0.819
GSTP1 Wild-type					
Low ITC	4 (30.8)	6 (31.6)	1		
High ITC	9 (69.2)	13 (68.4)	1.48	0.686 – 3.194	0.317
GSTP1 Polymorphism					
Low ITC	56 (54.4)	50 (52.1)	1		
High ITC	47 (45.6)	46 (47.9)	0.80	0.387 – 1.635	0.534

[c]Low (or high) ITC levels were defined as lower (or higher) than the median dietary ITC intake (2.31μmol/1000kcal) among all case-control subjects

Table 4.7: Dietary ITC intake in relation to risk of oral cancer stratified by GST genotypes (continue)

	Oral cancer status				
	Control Freq (%)	Cases Freq (%)	OR	95% CI	p-value
Total subjects					
Low ITC[c]	60 (51.7)	56 (48.7)	1		
High ITC	56 (48.3)	59 (51.3)	1.12	0.672 – 1.888	0.652
GSTM1/GSTT1 Wild-type					
Low ITC	14 (45.2)	15 (46.9)	1		
High ITC	17 (54.8)	17 (53.1)	0.93	0.346 – 2.515	0.891
GSTM/GSTT1					
Polymorphism	46 (54.1)	41(49.4)	1		
Low ITC	39 (45.9)	42(50.6)	1.21	0.659 – 2.215	0.540
High ITC					
Total subjects					
Low ITC	60 (51.7)	56 (48.7)	1		
High ITC	56 (48.3)	59 (51.3)	1.08	0.640 – 1.822	0.774
GSTM1/GSTT/GSTP1					
Wild-type					
Low ITC	35 (51.5)	32 (46.4)	1		
High ITC	33 (48.5)	37 (53.6)	0.96	0.210 – 4.421	0.961
GSTM/GSTT1/GSTP1					
Polymorphism					
Low ITC	25 (52.1)	24 (52.2)	1		
High ITC	23 (47.9)	22 (47.8)	1.10	0.628 – 1.914	0.747

[c]Low (or high) ITC levels were defined as lower (or higher) than the median dietary ITC intake (2.31μmol/1000kcal) among all case-control subject