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Impact of GSTM1 and GSTT1 Genetic Variations on Prostate Cancer Susceptibility in a Jordanian Cohort

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Abstract

Prostate cancer (PCa) is a major global health concern, characterized by high morbidity and mortality. Factors such as advanced age, androgen influence, and ethnic background are recognized as potential risk factors. Specific genetic variations in glutathione S-transferases (GSTs), enzymes critical for detoxifying environmental carcinogens, may increase PCa risk. Given inconsistent findings in previous studies and limited data from Jordan, we conducted a case-control study to examine the association between GSTM1 and GSTT1 polymorphisms and PCa risk in a Jordanian cohort.

Methods: GSTM1 and GSTT1 genotypes were analyzed in 150 patients with histologically confirmed PCa and 140 age-matched healthy controls. DNA extracted from peripheral blood was genotyped using multiplex polymerase chain reaction (PCR).

Results: The GSTM1 null genotype was significantly associated with increased PCa risk (OR = 3.69, 95% CI = 1.30–10.44; P = 0.01). In contrast, the GSTT1 null genotype showed no significant association (OR = 0.92, 95% CI = 0.32–2.62; P = 0.49). Combined GSTM1/GSTT1 null genotypes were also not associated with risk. Stratified analyses by Gleason score and smoking status revealed no significant differences.

Conclusion: The GSTM1 null genotype may increase susceptibility to prostate cancer in the Jordanian population, whereas GSTT1 null and combined null genotypes do not appear to influence risk. These findings support the potential use of GSTM1 as a molecular biomarker for PCa risk and highlight the need for larger, multi-gene, and multi-population studies.

Introduction

Prostate cancer (PCa) is one of the most common malignancies among men worldwide and represents a major public health concern. It is currently among the leading causes of cancer-related morbidity and mortality in aging male populations (Daniyal et al., 2014). According to global cancer statistics, the incidence of prostate cancer continues to rise, particularly in developed and developing countries. In Jordan, PCa represents one of the most frequently diagnosed cancers among men, accounting for approximately 8.3% of all male cancers, with about 335 newly diagnosed cases reported in 2022 according to the Jordan Cancer Registry and GLOBOCAN estimates.

The development of prostate cancer is multifactorial and involves complex interactions between environmental exposures, lifestyle factors, and genetic susceptibility (Giri and Beebe-Dimmer, 2016). Diagnosis typically relies on digital rectal examination, measurement of serum prostate-specific antigen (PSA), and histopathological confirmation following biopsy.

Among genetic susceptibility factors, polymorphisms in xenobiotic-metabolizing enzymes have been widely investigated because of their role in detoxifying carcinogenic compounds. Glutathione S-transferases (GSTs) constitute an important family of phase II detoxification enzymes that catalyze the conjugation of reduced glutathione with electrophilic substrates, thereby facilitating the detoxification

and elimination of numerous endogenous and exogenous toxic compounds (Chatterjee and Gupta, 2018). In addition to detoxification functions, GSTs are involved in regulating cellular signaling pathways, oxidative stress responses, and apoptosis.

Human GSTs include several classes such as alpha (A), mu (M), pi (P), theta (T), omega (O), sigma (S), kappa (K), and zeta (Z), which are distributed in cytosolic, mitochondrial, and microsomal compartments. Among these, the GSTM1 and GSTT1 genes are of particular interest because they frequently exhibit deletion polymorphisms that lead to a complete loss of enzymatic activity. Individuals carrying the null genotype lack functional enzyme activity, which may reduce the detoxification capacity for carcinogens and increase susceptibility to malignancies (Chirilă et al., 2015).

Several epidemiological studies have evaluated the association between GST gene polymorphisms and prostate cancer risk in different populations. For example, investigations in Northern India demonstrated that polymorphisms in GSTM1, GSTT1, and GSTP1 genes may influence individual susceptibility to PCa (Srivastava et al., 2005). Similar associations have been reported in Japanese populations (Komiya et al., 2005; Nakazato et al., 2003) and in European cohorts (Gsur et al., 2001). However, the results remain inconsistent across populations.

Meta-analysis studies have further evaluated the role of GST polymorphisms in prostate cancer susceptibility. For instance, Ntais et al. (2005) reported a modest association between GSTM1 deletion and prostate cancer risk, while Gong et al. (2012), analyzing 57 independent studies, concluded that GSTM1 and GSTT1 polymorphisms may contribute to PCa susceptibility in certain ethnic groups. Similarly, a systematic review by Cai et al. (2014) indicated that the GSTM1 null genotype may increase prostate cancer risk, whereas the GSTT1 null genotype showed less consistent association.

Population-specific studies also suggest that the influence of GST polymorphisms varies geographically. Safarinejad et al. (2011) demonstrated a significant association between GST gene polymorphisms and prostate cancer susceptibility in Iranian patients. Studies conducted in Middle Eastern populations, including Jordanian cohorts, have also investigated the contribution of GSTM1 and GSTT1 polymorphisms to prostate cancer development (Benabdelkrim et al., 2018; Medjani et al., 2020).

Given these inconsistent findings and the limited genetic epidemiological data available for the Jordanian population, further studies are necessary to clarify the potential contribution of GST polymorphisms to prostate cancer susceptibility. Therefore, the present study aims to investigate the association between GSTM1 and GSTT1 gene polymorphisms and prostate cancer risk in a Jordanian population.

Materials and Methods

Study Population

A total of 290 unrelated Jordanian men were enrolled, including 150 histologically confirmed prostate cancer (PCa) patients and 140 age-matched healthy controls, all residing in North Jordan. Patient ages

ranged from 45 to 82 years. PSA levels were measured prior to treatment using an ELISA mini-VIDAS TPSA kit (BioMérieux, France). Clinical data—including age at diagnosis, residence, smoking status, Gleason score, and family history—were collected prospectively. Controls had normal PSA levels (<4 ng/mL) and no evidence of prostate disease or malignancy. The study was approved by the Irbid National University Ethical Committee (IRB) and conducted according to the Declaration of Helsinki, with informed consent obtained from all participants.

DNA Extraction and Genotyping

Peripheral blood (5 mL) was collected in EDTA tubes, and genomic DNA was extracted using the FlexiGene® DNA Kit (Qiagen, Hilden, Germany). DNA concentration was measured with a NanoPhotometer™ (Implen, Germany) and stored at 4°C until analysis. GSTM1 and GSTT1 polymorphisms were analyzed using a multiplex PCR method adapted from Abdel-Rahman et al. (1996), with CYP1A1 as an internal control. PCR products were resolved on 5% polyacrylamide gels and visualized using EuroSafe Nucleic Acid Staining Solution (EuroClone, Italy).

Primer sequences included:

- **GSTM1**: Forward 5’-GTTGGGCTCAAATATACGGTGG-3’, Reverse 5’-GAACTCCCTGAAAAGCTAAAGC-3’
- **GSTT1**: Forward 5’-TCACCGGATCATGGCCAGCA-3’, Reverse 5’-TTCCTTACTGGTCCTCACATCTC-3’
- **CYP1A1 (internal control)**: Forward 5’-GAACTGCCACTTCAGCTGTCT-3’, Reverse 5’-CAGCTGCATTTGGAAGTGCTC-3’

All primers were sourced from Sigma-Aldrich (Germany). The PCR reaction was conducted in a 25 µL volume, comprising 2.5 µL 10X PCR buffer, 200 µM dNTPs, 2 µL 25 mM MgCl2, 1 µL of each primer, and 0.3 µL AmpliTaq Gold® DNA Polymerase (Applied Biosystems, Germany). Amplification was performed using an Applied Biosystems Veriti Thermal Cycler (ThermoFisher Scientific, Germany) with the following conditions: initial denaturation at 95°C for 10 minutes, followed by 40 cycles of denaturation (95°C, 1 min), annealing (68°C, 2 min), and extension (72°C, 1.5 min), concluding with a final extension at 72°C for 7 minutes. PCR products (GSTM1: 215 bp, GSTT1: 480 bp, CYP1A1: 312 bp) were separated on a 5% polyacrylamide gel electrophoresis and visualized using EuroSafe Nucleic Acid Staining Solution (EuroClone, Italy).

Statistical analysis

Continuous variables are presented as mean ± SD; categorical variables as frequencies and percentages. Odds ratios (OR) with 95% confidence intervals (CI) were calculated. P-values <0.05 were considered statistically significant. Analyses were conducted using SPSS version 20.0 (Chicago, IL, USA).

Table 1: Statistical analysis of Variables

Variable	Category / Statistic	Cases(n=150)	Control(n=140)	Test Used	P value
Age	Mean ± SD	66.25 ± 10.13	66.23 ± 8.38	Independent t-test	0.88
Residence	Urban	135(90.0%)	140 (100%)	Chi-square	0.001*
	Rural	15 (10.0%)	0 (0%)		
Family history of PSA	Yes	9(6.0%)	4 (2.9%)	Chi-square	0.21
	No	141(94.0%)	136 (97.1%		
Smoking status	Nonsmokers	31(20.7%)	31 (22.1%)		0.78
	Smokers	119(79.3%)	109 (77.9%)		
Clinical stage(cases only)	Localized	55 (36.7%)	----		
	Advanced	95 (63.3%)	----		
Gleason score(cases only)	< 7(low)	61 (40.7%)	----		
	7-10(High)	89 (59.3%)	----		
PSA(ng/ml)	Mean ± SD	80.72 ± 24.34	4.26 ± 3.48	Independent t-test	<0.001*
	< 4	6 (4.0%)	109 (77.9%)	Chi-square	<0.001*
	4-10	18 (12.0%)	24 (17.1%)		
	>10	126 (84.0%)	7 (5.0%)		

* Significant at P < 0.05 *** Very highly significant (P < 0.001)

Figure 1. Genotype Analyses of Selected Subjects. Lane M, MspI-digested Bluscript plasmid as molecular weight marker; lane 1, negative control (master mix + molecular water) ; lane 2, subject with null alleles for both *GSTM1* and *GSTT1* (*GSTM1* 0/0 and *GSTT1* 0/0) showing only one band at 312 bp corresponding to the internal control (*CYP1A1* gene fragment); lane 3, subject harboring *GSTT1* +/+ and *GSTM1* +/+ alleles; lane 4, subject harboring *GSTT1* +/+ and *GSTM1* 0/0
alleles.was quite equal in cases and controls.

The frequencies of *GSTM1* null genotype (*GSTM1* 0/0) were 38.77 % in patients and 14.63% in controls. However, for the *GSTM1* active genotype frequencies (*GSTM1* +/+) were 61.22% in patients' group and 85.36% in controls' group. Statistically, significant differences were observed (OR= 3.69, 95% CI= 1.30-10.44; P = 0.01)

(Table 2).

The distribution of *GSTT1* variants in the patient and control groups showed a high similarity. In fact, the frequencies of *GSTT1*0/0 and *GSTT1*+/+ genotypes were
19.51% and 80.48%, respectively, in the controls' group and 18.36% and 81.63% respectively in the patients' group. The statistical analysis showed no association

Table 2. *GSTM1* and *GSTT1* Genotypes Distribution among Individuals with and without Prostate Cancer

Genotype	Cases (n = 150)	Controls (n = 140)	OR (95% CI)	P value
	N (%)	N (%)		
Single genotypes				
GSTM1 non-null	92 (61.33)	120 (85.71)	—	—
GSTM1 null	58 (38.67)	20 (14.29)	3.69 (1.30–10.44)	0.01
GSTT1 non-null	122 (81.33)	113 (80.71)	—	—
GSTT1 null	28 (18.67)	27 (19.29)	0.92 (0.32–2.62)	0.49
Double genotype				
GSTM1 null /GSTT1 null	12 (8.00)	10 (7.14)	0.88 (0.18–4.21)	NS

NS, No Significant

Association Between GSTM1 and GSTT1 Genotypes and Prostate Cancer Risk

Table 3. Distribution of the Genotypic Frequencies According to Gleason Score of Prostate Cancer

Genotype	Cases (n = 150)	Controls (n = 140)	OR (95% CI)	P value
	N (%)	N (%)		
Single genotypes				
GSTM1 non-null	92 (61.33)	120 (85.71)	—	—
GSTM1 null	58 (38.67)	20 (14.29)	3.78 (2.13–6.74)	<0.0001
GSTT1 non-null	122 (81.33)	113 (80.71)	—	—
GSTT1 null	28 (18.67)	27 (19.29)	0.96 (0.53–1.73)	0.89
Double genotype				
GSTM1 null / GSTT1 null	12 (8.00)	10 (7.14)	1.13 (0.47–2.70)	0.78

Association Between GST Genotypes and Gleason Score

Table 4. Distribution of the Genotypic Frequencies According to Smoking Statue

Genotype / Variable	GS <7 (n = 60)	GS ≥7 (n = 90)	OR (95% CI)	P value
	N (%)	N (%)		
GSTM1 non-null	33 (55.0)	57 (63.3)	0.71 (0.36–1.40)	0.32
GSTM1 null	27 (45.0)	33 (36.7)	—	—
GSTT1 non-null	51 (85.0)	73 (81.1)	1.32 (0.52–3.37)	0.55
GSTT1 null	9 (15.0)	17 (18.9)	—	—

GS, Gleason Score; OD, Odds Ratio; NS, No Significant

Table 5.Association Between Smoking Status and GST Null Genotypes Among Cases

Cases / Variable	Non-smokers (n = 30)	Smokers (n = 120)	OR (95% CI)	P value
	N (%)	N (%)		
GSTM1 (0/0)	12 (40.0)	46 (38.3)	1.07 (0.47–2.43)	0.86
GSTT1 (0/0)	6 (20.0)	22 (18.3)	1.11 (0.40–3.09)	0.83

NS*: No Significant

between *GSTT1* null genotype and the risk of PCa (OR= 0.92, 95% IC= 0.32-2.62; P = 0.49). Individuals

with combined genotypes (*GSTM1* 0/0 and *GSTT1* 0/0), exhibited no change in the risk for PCa compared to controls (OR= 0.88, 95 % CI= 0.18-4.21) (Table 2).

Table 3 presents the results of association between the studied polymorphisms and the Gleason score at diagnosis of PCa and differences were not significantly important with either the low or high grade cancer.The smoking status was not associated with *GSTM1*and *GSTT1*polymorphisms (Table 4).

Discussion

This study explored the relationship between GSTM1 and GSTT1 gene polymorphisms and the risk of prostate cancer within a Jordanian cohort. Our results indicate that individuals carrying the GSTM1 null genotype have a higher susceptibility to prostate cancer. In contrast, no significant correlation was observed for the GSTT1 null genotype or for the combined deletion of both GSTM1 and GSTT1.

The frequency of GSTM1 and GSTT1 deletions varies considerably among populations. Earlier research reported GSTM1 null genotype prevalence of 47–58% and GSTT1 null genotype prevalence of 13–25% in European populations (Drozdz-Afelt et al., 2020). In the present Jordanian cohort, the rates were 38.77% for GSTM1 and 18.36% for GSTT1 null genotypes, aligning with the range reported in other ethnic groups.

Our observation linking the GSTM1 null genotype to increased prostate cancer risk aligns with several prior studies. For example, Srivastava et al. (2005) reported a significant elevation in prostate cancer risk among GSTM1-deleted individuals in an Indian population. Meta-analyses have similarly indicated that the GSTM1 null genotype could serve as a notable genetic risk factor for prostate cancer development (Ntais et al., 2005; Gong et al., 2012; Cai et al., 2014).

However, some investigations have not found a significant association between GSTM1 deletion and prostate cancer risk. Studies in Japanese populations (Komiya et al., 2005) and European case-control studies (Sivonova et al., 2009) reported no meaningful relationship. Such discrepancies may be attributable to variations in genetic background, environmental exposures, lifestyle factors, or sample size limitations.

Regarding GSTT1, our data did not reveal a significant link between the GSTT1 null genotype and prostate cancer susceptibility. Similar findings were reported by Agalliu et al. (2006), who found no association between GSTT1 polymorphism and prostate cancer risk or prognosis. Other studies across different populations have supported these results (Lavender et al., 2009; Sivonova et al., 2009). Nevertheless, contrasting evidence exists; for instance, Safarinejad et al. (2011) observed a significant association between GSTT1 deletion and elevated prostate cancer risk in an Iranian cohort. These inconsistencies underscore the need to account for ethnic and environmental differences when assessing genetic susceptibility.

The combined effect of GSTM1 and GSTT1 deletions has also been investigated. Some researchers suggested that the absence of both enzymes could impair detoxification processes and increase carcinogen accumulation (Gsur et al., 2001). Yet, consistent with our findings, several case-control studies reported no significant association between the double-null genotype and prostate cancer risk (Agalliu et al., 2006).

The potential biological mechanism linking GSTM1 deletion to prostate cancer may involve the detoxification role of GST enzymes. A lack of GSTM1 activity could reduce the capacity to neutralize reactive carcinogens and oxidative stress products, leading to DNA damage and higher risk of malignancy (Chatterjee and Gupta, 2018). Additionally, environmental factors like tobacco smoking could interact with GST polymorphisms, as GST enzymes help detoxify carcinogens in smoke. While prior studies suggested GST variants might modulate the effect of smoking on prostate cancer risk (Lavender et al., 2009), our study did not detect a significant effect in smokers, possibly due to limited sample size.

Molecular approaches, such as multiplex PCR, are widely employed to detect GST gene deletions and polymorphisms. For example, Buchard et al. (2007) developed a multiplex PCR method capable of identifying GSTM1, GSTT1, and GSTP1 variants simultaneously, facilitating large-scale genetic studies.

Despite the insights provided, this study has limitations. The modest sample size may reduce the ability to detect subtle genetic effects. Additionally, prostate cancer is a complex disease influenced by

multiple genetic and environmental factors, so the impact of a single gene may be relatively limited.

In summary, our findings suggest that the GSTM1 null genotype may increase prostate cancer susceptibility in the Jordanian population, whereas GSTT1 deletion does not appear to significantly affect risk. These results add to the evidence linking GST polymorphisms to prostate cancer, though larger multicenter studies incorporating multiple genetic and environmental factors are necessary to fully elucidate their role.

Conclusion

Prostate cancer is a multifactorial disease with a complex etiology influenced by both genetic and environmental factors. Among genetic contributors, glutathione S-transferases (GSTs) have been recognized as potential modulators of prostate cancer susceptibility. In the present study, which represents the first investigation of GSTM1 and GSTT1 polymorphisms in Jordanian prostate cancer patients, we found that the GSTM1 null genotype is associated with an increased risk of prostate cancer, whereas GSTT1 null and the combined GSTM1/GSTT1 null genotypes are not significantly associated with disease susceptibility. These findings are largely consistent with previous meta-analyses and population-based studies.

Additionally, our stratified analysis of GST variants relative to Gleason scores and smoking status revealed no significant association with disease aggressiveness or environmental risk factors, underscoring the complex interplay between genetic and non-genetic determinants of prostate cancer. While GSTM1 deficiency may impair detoxification of electrophilic carcinogens, contributing to tumor initiation, the effect of GSTT1 appears less pronounced in the studied population.

Overall, this study highlights the potential of GSTM1 as a molecular biomarker for prostate cancer risk, emphasizing the need for larger, multi-gene, and multi-population studies to validate these findings and explore the combined effects of genetic variants. Understanding the role of GST polymorphisms may ultimately contribute to improved risk assessment, early detection, and personalized management strategies for prostate cancer.

Declarations

Acknowledgments

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Conflict of Interest

The authors declare no conflicts of interest.

References

1. Srivastava DS, Mandhani A, Mittal B, Mittal RD (2005). Genetic polymorphism of glutathione S-transferase genes (GSTM1, GSTT1 and GSTP1) and susceptibility to prostate cancer in Northern India. BJU Int, 95, 170-3. PubMed
2. Ntais C, Papadopoulou E, Papadopoulou C, et al (2005). Association of GSTM1, GSTT1, and GSTP1 gene polymorphisms with prostate cancer risk: a meta-analysis. Cancer Epidemiol Biomarkers Prev, 14, 176-81. PubMed
3. Safarinejad MR, Shafiei N, Safarinejad SH (2011). Glutathione S-transferase gene polymorphisms (GSTM1, GSTT1, GSTP1) and prostate cancer: a case-control study in Tehran, Iran. Prostate Cancer Prostatic Dis, 14, 105-13. Nature
4. Gong M, Dong W, Shi Z, Xu Y, Ni W, An R (2012). Genetic polymorphisms of GSTM1, GSTT1, and GSTP1 with prostate cancer risk: a meta-analysis of 57 studies. PLoS One, 7, e50587. PLOS One
5. Cai Q, Wang Z, Zhang W, et al (2014). Association between glutathione S-transferases M1 and T1 gene polymorphisms and prostate cancer risk: a systematic review and meta-analysis. Tumour Biol, 35, 247-56. SpringerLink
6. Drozdz-Afelt JM, Koim-Puchowska B, Kaminski P (2020). Polymorphism of glutathione S-transferase in the population of Polish patients with carcinoma of the prostate. Environ Sci Pollut Res Int, 27, 28996-9003. SpringerLink
7. Medjani S, Chellat-Rezgoune D, Satta D (2020). Association of CYP1A1, GSTM1 and GSTT1 gene polymorphisms with risk of prostate cancer in Jordanian population. Afr J Urol, 26, 49. SpringerOpen
8. Gsur A, Haidinger G, Hinteregger S, et al (2001). Polymorphisms of glutathione-S-transferase genes (GSTP1, GSTM1 and GSTT1) and prostate-cancer risk. Int J Cancer, 95, 152-5. PubMed
9. Lavender NA, Benford ML, VanCleave TT, et al (2009). Examination of polymorphic glutathione S-transferase (GST) genes, tobacco smoking and prostate cancer risk among men of African descent: a case-control study. BMC Cancer, 9, 397. PubMed
10. Agalliu I, Lin DW, Salinas CA, et al (2006). Polymorphisms in the glutathione S-transferase M1, T1, and P1 genes and prostate cancer prognosis. Prostate, 66, 1535-41. PubMed
11. Agalliu I, Langeberg WJ, Lampe JW, et al (2006). Glutathione S-transferase M1, T1, and P1 polymorphisms and prostate cancer risk in middle-aged men. Prostate, 66, 146-56. PubMed
12. Komiya Y, Tsukino H, Nakao H, et al (2005). Human glutathione S-transferase A1, T1, M1, and P1 polymorphisms and susceptibility to prostate cancer in the Japanese population. J Cancer Res Clin Oncol, 131, 238-42. PubMed
13. Nakazato H, Suzuki K, Matsui H, et al (2003). Association of genetic polymorphisms of glutathione-S-transferase genes (GSTM1, GSTT1 and GSTP1) with familial prostate cancer risk in a Japanese population. Anticancer Res, 23, 2897-902. PubMed
14. Sivonov M, Waczul kov I, Dobrota D, et al (2009). Polymorphisms of glutathione-S-transferase M1, T1, P1 and the risk of prostate cancer: a case-control study. J Exp Clin Cancer Res, 28, 32. JECCR

15. Benabdelkrim M, Djeffal O, Berredjem H (2018). GSTM1 and GSTT1 polymorphisms and susceptibility to prostate cancer: a case-control study of the Jordanian population. Asian Pac J Cancer Prev, 19, 2853-8. PubMed
16. Buchard A, Sanchez JJ, Dalhoff K, Morling N (2007). Multiplex PCR detection of GSTM1, GSTT1, and GSTP1 gene variants: simultaneously detecting GSTM1 and GSTT1 gene copy number and the allelic status of the GSTP1 Ile105Val genetic variant. J Mol Diagn, 9, 612-7. PubMed
17. Cai Q, Wang Z, Zhang W, et al (2014). Association between glutathione S-transferases M1 and T1 gene polymorphisms and prostate cancer risk: a systematic review and meta-analysis. Tumour Biol, 35, 247-56. SpringerLink
18. Chatterjee A, Gupta S (2018). The multifaceted role of glutathione S-transferases in cancer. Cancer Lett, 433, 33-42. PubMed
19. Chiril DN, Popp R, Vesa Ș, et al (2015). GSTM1, GSTT1 and GSTP1 genetic variants in multiple urologic cancers. Chirurgia (Bucur), 110, 54-61. PubMed
20. Daniyal M, Siddiqui ZA, Akram M, et al (2014). Epidemiology, etiology, diagnosis and treatment of prostate cancer. Asian Pac J Cancer Prev, 15, 9575-8. PubMed
21. Giri VN, Beebe-Dimmer JL (2016). Familial prostate cancer. Semin Oncol, 43, 560-5. PubMed
22. Gong M, Dong W, Shi Z, et al (2012). Genetic polymorphisms of GSTM1, GSTT1, and GSTP1 with prostate cancer risk: a meta-analysis of 57 studies. PLoS One, 7, e50587. PLOS One
23. Gsur A, Haidinger G, Hinteregger S, et al (2001). Polymorphisms of glutathione-S-transferase genes (GSTP1, GSTM1 and GSTT1) and prostate-cancer risk. Int J Cancer, 95, 152-5. PubMed
24. Lavender NA, Benford ML, VanCleave TT, et al (2009). Examination of polymorphic glutathione S-transferase (GST) genes, tobacco smoking and prostate cancer risk among men of African descent: a case-control study. BMC Cancer, 9, 397. PubMed.

Figures

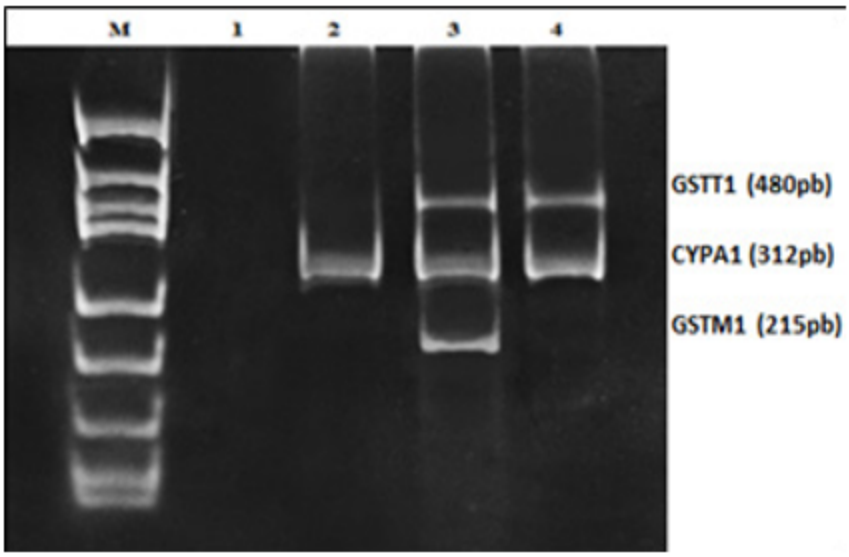


Figure 1

Genotype Analyses of Selected Subjects. Lane M, MSpI-digested Bluscript plasmid as molecular weight marker; lane 1, negative control (master mix + molecular water) ; lane 2, subject with null alleles for both *GSTM1* and *GSTT1* (*GSTM1* 0/0 and *GSTT1* 0/0) showing only one band at 312 bp corresponding to the internal control (*CYP1A1* gene fragment); lane 3, subject harboring *GSTT1* +/+ and *GSTM1* +/+ alleles; lane 4, subject harboring *GSTT1* +/+ and *GSTM1* 0/0 alleles.was quite equal in cases and controls.