Journal Basrah Researches ((Sciences)) Vol. 35, No. 1, 15 February ((2009))

Frequencies of *GSTM1* and *GSTT1* Polymorphisms in Iraqi Population

[1] Salwa Jaber Al-Awadi , [1]Ismail H. Aziz , [2]* Adnan I. Al-Badran

[1] Institute of Genetic Engineering and Biotechnology for Post Graduate Studies University of Baghdad .

[2] Department of Biology, College of Science, University of Basrah
** Correspondent Author : E-mail : aalbadran@yahoo.com*
ISSN –1817 –2695

((Received 7/10/2007 , Accepted 21/4/2008))

ABSTRACT

The glutathione S-transferase (GST) family of enzymes has a vital role in phase II of biotransformation of environmental carcinogens, pollutants, drugs and other xenobiotics. GSTs are polymorphic, with the type and frequency of polymorphism being ethnically dependent. Polymorphisms in *GST*s genes have been shown to be associated with susceptibility to disease and disease outcome.

The frequencies of *GSTM1* and *GSTT1* polymorphisms are determined in 160 Iraqi volunteers who had been residents of different areas of Baghdad and Basrah. Blood was collected and DNA extracted by proteinase K/SDS digestion. GSTM1 and GSTT1 polymorphisms were analyzed by a PCR-Multiplex procedure.The multiplex PCR protocol was used to simultaneously analyze the presence or absence of gstm1and gstt1 genes. The Albumin gene was used as an internal control(350 bp) whereas the presence of the *GSTM1* and *GSTT1* genes was identified by 215 and 480 bp fragments, respectively. Although these assays do not distinguish between heterozygote and homozygote positive genotypes, they conclusively identify the null genotypes (the genes are completely deleted).

The genetic analysis showed that the deletion percentage in the samples was 30% (*GSTM1* 13.1% and null genotype 10% and 6.9 % for *GSTT1*) as compared to 70% that was normal. The detected deletions in our population(30%)appear to be lower than or similar to the frequencies of these polymorphisms in other Western countries. But they are lower than those found in Asia .

Keywords : Multiplex PCR , GSTM1 , GSTT1 .

INTRODUCTION

Glutathione S-transferases (GSTs) are a super gene family of enzymes involved in phase II of biotransformation, which is characterized by the conjugation of endogenous water-soluble compounds to lipophilic substrates. GSTs catalyze the conjugation of glutathione and a tripeptide (consisting of glycine, glutamic acid and cysteine) to electrophilic compounds resulting in less reactive and more easily excreted glutathione conjugates(1). Substrates of GST-catalyzed reactions include pre-carcinogens such as polycyclic aromatic hydrocarbons, pharmacological drugs, including paracetamol, chemotherapeutic agents and free radicals generated during oxidative stress (2). GSTs have been shown to act as inhibitors of the jun kinase pathway which is an important signaling mechanism for the activation of cytoprotective genes (3 , 4). Human cytosolic GSTs have been well characterized, are polymorphic, and have ethnic-dependent polymorphism frequencies. The GSTM1 gene is located on chromosome 1p13.3, and 20 to 50% of individuals do not express the enzyme due to a homozygous gene deletion, known as the GSTM1*0, or null allele (5). The percentage of individuals who do not express the enzyme is higher in Caucasians and Asians than in Africans (6,7). GSTM1 is involved in the detoxification of polycyclic aromatic hydrocarbons and other

*** Correspondent Author : E-mail : aalbadran@yahoo.com**

mutagens, and cells from GSTM1 null individuals are more susceptible to DNA damage caused by these agents (8). The *GSTT1* gene is located on chromosome 22 . 20% to 60% of individuals do not express the enzyme also this is due to a gene deletion known as the GSTT1*0 allele (9). About 60% of Asians, 40% of Africans and 20% of Caucasians do not express this enzyme (10). This polymorphism accounts for the variation in GST-catalyzed metabolism of halomethanes by human erythrocytes (9). Polymorphisms of *GSTM1* and *GSTT1* have been associated with differences not only in susceptibility to various forms of cancer, particular those caused by cigarette smoking (11) but also in resistance to chemotherapy treatment and in drug response (12), and in disease susceptibility and outcome as well (13,14).

The study analyzed the frequency of the *GSTM1* and *GSTT1* polymorphisms in Iraqi population since polymorphism in these low-penetrance genes may predispose Iraqis to certain adverse drug reactions or disease occurrences.

MATERIAL AND METHODS

The population studied

The population that took part in the present study composed of 160 volunteers. Those with a cancer history or chronic diseases were excluded in this study as they signed an informed consent data was obtained via a standardized questionnaire, including age, gender, marital status as well as health problems. All the volunteers were residents of Baghdad, and Basrah.

DNA Extraction and Genotyping of *GSTM1* and *GSTT1*

Blood was collected in EDTA-containing tubes (3-5 ml)and DNA was extracted from the lymphocytes by proteinase K/SDS digestion as described by (15).

The polymorphisms of *GSTM1* and *GSTT1* were analyzed by a polymerase chain reaction (PCR)-multiplex procedure as previously described (16).

Genomic DNA (100 ng) was used as a DNA template in 50 µl of total volume reaction. The following primers were used :

GSTM1 : F-(5-GAA CTC CCT GAA AAG CTA AAG C-3)
R-(5 –GTT GGG CTC AAA TAT ACG GTG G-3)

GSTT1 : F- (5 – TTC CTT ACT GGT CCT CAC ATC TC -3)
R- (5 – TCA CCG GAT CAT GGC CAG CA -3)

Albumin : F-(5 – GCC CTC TGC TAA CAA GTC CTA C -3)
R-(5 – GCC CTA AAA AGA AAA TCG CCA ATC-3)

The amplification reactions were carried out in a volume of 50 µl containing (25ng) DNA;10 mM Tris–HCl; 50 mM KCl ; 1.5 mM $MgCl_2$;200 µM (each) dATP, dCTP,dGTP and dTTP (Promega); each primer was at 20 pM and 2.5 unit of Taq polymerase (Promega) .

The amplification was carried out as follows :

The initial denaturation is at 95 ^oC for 3 min, 30 cycles in (Techne,Cambridge Ltd., England) thermocycler which is as follows : 94 ^oC for1 min; 59 ^oC for1 min ; 72 ^oC for1 min and 5 min final extension for the last cycle. The PCR products were analyzed on 2%Agarose gel electrophoresis to detect the absence or presences of these genes. Albumin gene iis used as an internal control. The internal control amplified *Albumin* fragment was 350 bp in length whereas presence of the genes*GSTM1* and *GSTT1* was identified by 215 and 480 bp fragments, respectively.

Results and Discussion

1- Genotyping :

Genomic DNA was extracted from blood samples(of both patients and control group) using the proteinase K/ SDS protocol. The DNA was checked on agarose gel electrophoresis before doing PCR to see the purity and integrity. Fifty to 100 ng of genomic DNA were used for each PCR reaction. Some samples were repeated twice to confirm the PCR result and examine the protocol. A multiplex PCR protocol was used to simultaneously analyze the presence or absence of *GSTM1*and *GSTT1* genes. The *Albumin* gene was used as an internal control. The internal control amplified *Albumin* fragment was 350 bp in length whereas the presence of the *GSTM1* and*GSTT1* genes was identified by 215 and 480 bp fragments, respectively. Although these assays did not distinguish between heterozygote and homozygote positive genotypes, they conclusively identify the null genotypes (Fig 1).

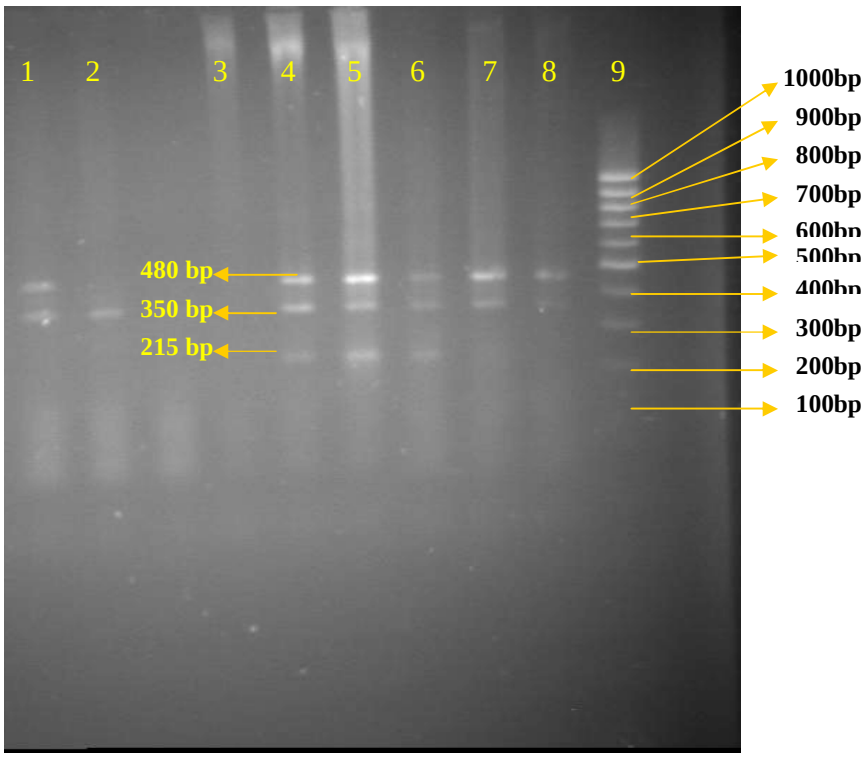

Fig (1): Electrophoresis of PCR products on agarose gel 2% (70 volt / 1 hr).
480 bp = *GSTT1* band, 215 bp = *GSTM1* band, 350 bp = *Albumin* band (Internal Control)
Lane 1, 7, 8: *GSTM1* Deletion, Lane2 : Null Genotype, Lane3 : Negative Control,
Lane 4, 5, 6: Normal Genotype and Lane9 : DNA Marker (100 -1000bp).

The Baghdad and Basrah resident volunteers who took part in this study were categorized diverse parameters , only apparently healthy individual were included in this study. This is because GST polymorphisms can predispose some diseases and patients who have been treated for a particular disease may have a specific GST genotype which would otherwise lead to spurious results (Table 1).

Table (1): Descriptive Parameters of Samples According to Genotype

Parameters	Samples Number	Deletion	Normal
Age			
< 50	110	29(26.3%)	81(73.6%)
≥50	50	19(38%)	31(62%)
Marital Status			
Married	109	35(32%)	74(67.8%)
Single	51	13(25.4%)	38(74.5%)
Gender			
Male	67	30(44.7%)	37(55.2%)
Female	93	13(13.9%)	80(86%)

Marital status

For Marital status, 109 were married and 51 single. Thirty five cases from the married had deletion while 74 were of normal genotype. Thirteen samples from the single had deletion and the remaining (38)were of normal genotype. Most of the case were from the married as compared with the single samples, analysis showed no significant differences. (17) observed a significant multiplicative correlation between GSTM1 and GSTT1 null genotypes and high-risk status of parity factor in all women and in premenopausal women but not in postmenopausal women .The investigators found that single women have more chance to have breast cancer risk than the married women who have no child (18). The available data are not conclusive to statistically compare between the single and married. The outcome indicated that the married women were within high incidence .This is explained by fact that the married women in this population were under general stress as the society has gone through long wars and sanctions, on the one hand estrogen levels are substantially low in Asian women; it may be speculated that environmental carcinogens including those metabolized specifically by GSTs make a larger contribution to breast cancer in populations where estrogen exposure is low (19). Additional studies on the Iraqi population may help to clarify these observations .

Age

Regarding to age samples , 110 where under 50 years of age . The multiplex PCR results showed that 29 samples had genetic deletion and the 81 were of normal genotype whereas 50 persons above 50 years of age, 19 had deletion and the 31 were of normal *GSTs* genotype.(Table 2)

Table 2: Association between Age and *GSTs* Genotype

Age	Total	*GSTM1* Deletion	*GSTT1* Deletion	Null Genotype	Normal
≥50	50	8 (16%)	3 (6%)	8 (16%)	31 (62%)
<50	110	13 (11.8%)	8 (7.28%)	8 (7.28%)	81 (73.63%)
Total	160	21	11	16	112

The percentage of the genetic deletion four samples over 50 age was 38 % distributed as follows : null genotype and *GSTM1* were 16 % for each, 6 % *GSTT1* deletion and 62 % were normal . For under 50 age samples , the percentage was : 73.63 % normal , 7.28 % *GSTT1* deletion, 11.8 % *GSTM1* deletion and 7.28 % null genotype. The *GSTM1* had a percentage of deletion higher than others. This study did not find a difference between *GSTs* polymorphisms and age. Similar to this finding, (20) observed no correlation between null GSTM1 and age at cancer patients diagnosis. As described above it is possible that other factors such as exposure to certain carcinogens or lifestyle may influence this result. The results fall with those by (21), who reported no association for GSTM1 null genotype in pre or postmenopausal women .(6) and (20) also reported that GSTM1 null genotypes were not associated with postmenopausal women neither in African-American nor Caucasian women.

Gender :

Table 3: Association Between Gender and *GSTs* Genotype

Gender	Samples No.	Genetic Deletion	Normal Genotype
Male	67	30 (44.77%)	37 (55.23%)
Female	93	18 (19.35%)	75 (70.65%)

We found that (44.77%) of men had genetic deletion while (19.35%) of women had deletion (Table 3). To our knowledge, there is no study focusing on gender regarding to *GSTs* polymorphisms but our explanation is that men are more exposed to chemicals or pollutants in our society . Women are less exposed to either if the social situation in our country taken into consideration. This might be explained as an influence of *GSTs* and polymorphisms that depends on the genetic background or possibly environmental cofactors (11,19). Or it may be due to the fact that men are numbered higher as cigarette smokers than women and there is an association between deletion of these genes and smoking (10).

Geographical Distribution

The most of samples were from Baghdad (115) followed by Basrah Province (45). However, samples were collected from the outskirts of Baghdad to ensure more random distribution and be close to reality because most of residents in these areas are known of central and Southern origins (Table 4).

Table 4 : Geographical Distribution of Samples According to Residence .

Residence of control Samples	Total No. of Sample	Normal Genotype	Genetic deletion
Baghdad and Outskirts	115	83 (72%)	32 (27.8%)
Basrah and Near Areas	45	29 (64.4%)	16 (35.5%)

This distribution is not representative indeed because we had no access to reach other provinces due to security situations The most of samples were from Baghdad and adjacent areas . This can be explained by the exposure to air pollution and industrial factories surrounded Baghdad. This might explain an influence of *GSTs* and polymorphisms that depends on the genetic background or possibly environmental cofactors

(11,19*)* The differential distribution of variant polymorphic genes in different human populations around the world may influence the environmental diseases which they acquire (22). We found(Table 5) that 21 samples (13.12%) were homozygote for the GSTM1 deletion. The frequency of GSTM1 null was not similar to that found in studies made on some other populations. The *GSTM1* homozygote deletion is present in 46% of Americans (6), in 49% of Polish (23), and in 51% of Swedish people (24). However, the frequency of Iraqis possess the *GSTM1* deletion is higher than that of Chileans (21.4%) (25). This difference can probably be explained by the ethnic mixture that makes up populations.We observed that 11 of samples (6.9 %) were homozygote for the gene deletion *GSTT1*. This frequency is less than that observed in some other populations, found in 14% of Americans (6) and 20% of Swedish (23). Although the frequencies of *GSTM1*(13.1%) and GSTT1 (6.9%) gene and null genotype(10 %) deletions in our population (30%) appear to be lower than or similar to the frequency of these polymorphisms in other Western countries, they are lower than those found in Asia (Table 5).

Table (5) : Frequency of *GSTs* Gene in the sample Studied .

Total No.	gstm1 Deletion	gstt1 Deletion	Null Genotype	Normal Genotype
160	21(13.1%)	11 (6. 9%)	16 (10%)	112 (70 %)

The frequency of the GSTM1 null genotype in humans ranges from 30 to 50% depending on the ethnic origin of the individual (28).The frequency of the null genotype has been estimated in some ethnic groups. It is the highest among Asian Populations (46.52%). Among Europeans, it ranges from 11% to 22% (27).This deletion occurs in about 30% of individuals and is linked to an increased DNA damage from carcinogens (27). About 60% of Asians, 40% of Africans and 20% of Caucasians do not express this enzyme (10).The Iraqi population is multi-ethnic, being mainly composed of peoples of Arab, Kurd and Turkman origins, with a smaller number of individuals other origins such as Assyrian. Polymorphisms in *GST* genes can lead either to a lack of expression or to the expression of GST enzymes that possess a different catalytic activity than the wild-type protein. Since GST enzymes play a vital role in cellular defence against environmentally toxic compounds, such as carcinogens polymorphism of *GSTs* genes in Iraqis may predispose them to diseases caused by such xenobiotics.Finally, comprehensive studies are needed to gain a clear picture of the polymorphisms of these genes in our population: these studies should consider interactions with genes encoding Phase I enzymes that produce reactive GST substrates (cytochrome monooxygenases) and, potentially, other detoxifying enzyme systems (i.e., glucuronosyl transferases and sulfotransferases) that may work cooperatively with the *GSTs* to guard the genome during or after chemical damage.

REFERENCES:

1- JG, Li Y, MM,**Ford** O'Sullivan Demopoulos R, Garte S, Taioli E and Rauf PWB Glutathione S-transferase M1 polymorphism and lung cancer risk in African-Americans. Carcinogenesis 21:1971-1975.(2000).

2- R.C., M.A., S. **Strange**, Spiteri, Ramachandran, and A.A. Fryer, Glutathione S-transferase family of enzymes. *Mut. Res. 482*: 21-26. (2001).

3- V., **Adler**, Z., Yin, S.Y., Fuchs, M., Bezerra, L., Rosario, K.D.,Tew, M.R., Pincus, M., Sardana, C.J., Henderson, C.R., Wolf, R.J.Davis, and Z.Ronai, Regulation of JNK signalling pathway by GSTPp. *EMBO J. 18*: 1321-1334. (1999).

4- LE **Johns,** and RS Houlston Glutathione S-transferase L (GSTM1) status and bladder cancer risk: a meta-analysis. Mutagenesis 15:399-404. (2000).

5- J., **Seidgard**, W.R., Vorachek, R.W. Pero, and W.R.Pearson, Hereditary differences in the expression of the human glutathione S-transferase activity on trans-stilbene oxide are

due to a gene deletion. *Proc. Natl. Acad. Sci.USA 85*: 7293-7297. (1988).

6- L.R., **Bailey**, N., Roodi, C.S., Verrier, C.J., Yee, Dupont, W.D. and Parl, F.F.Breast cancer risk and CYP1A1, GSTM1, and GSTT1 polymorphisms: evidence of a lack of association in Caucasians and African Americans. Cancer Res. 58: 65-70. (1998).

7- M.J., **Roth**, S.M., Dawsey, G.Q., Wang, J.A., Tangrea, Zhou, B., Ratnasinghe, D., Woodson, K.G., Olivero, O.A., Poirier, M.C., Frye, B.L., Taylor, P.R. and Weston, A. Association between GSTM1*0 and squamous dysplasia of the esophagus in the high risk region of Linxian, China. *Cancer Lett. 156*: 73-81. (2000).

8- L.W., **Harries**, M.J., Stubbins, D., Forman, G.C.W. Howard, and C.R. Wolf, Identification of genetic polymorphism at the glutathione S-transferase P locus and association with susceptibility to bladder, testicular and prostate cancer. *Carcinogenesis 18*: 641-644. (1997).

9- S.,**Pemble**, K.R., Schroeder, S.R., Spencer, , D.J., Meyer, E., Hallier, H.M., Bolt, B. Ketterer, and J.B. Taylor, Human glutathione S-transferase theta (GSTT1): cDNA cloning and the characterization of a genetic polymorphism. *Biochem. J. 300* (Part 1): 271-276. (1994).

10- R.C. **Strange**, and A.A.Fryer, The glutathione S-tranferases: influence of polymorphism on cancer susceptibility. In: *Metabolic Polymorphisms and Susceptibility to Cancer* (Vineis, P., Malats, N., Lang, M., d'Errico, A., Caporaso, N., Cuzick, J. and Boffetta, P., eds.). IARC Scientific Publications, No. 148, Lyon, France, pp. 231-249. (1999).

11- J E.; **Curran** Weinsyein S. R.and Griffith R. Polymorphisms of glutathione S-transferase genes (GSTM1, GSTP1, and GSTT1) and breast cancer susceptibility. Cancer Letters . 153: 113-120.(2000).

12- J.D. **Hayes**, and D.J. Pulford, The glutathione S-transferase supergene family: regulation of GST and the contribution of the isoenzymes to cancer chemoprotection and drug resistance. *Crit. Rev. Biochem. Mol. Biol. 30*: 445-600. (1995).

13- J.Y., **Lear**, A.H.M., Heagerty, A., Smith, B., Bowers, C., Payne, C.A.D., Smith, P.W., Jones, Gilford, J., Yengi, L., Alldersea, J., Fryer, A. and Strange, R.C.Multiple cutaneous basal cell carcinomas: glutathione-S-tranferase (GSTM1, GSTT1) and cytochrome P450 (CYP2D6, CYP1A1) polymorphisms influence tumour numbers and accrual. *Carcinogenesis 12*: 1891-1896. (1996).

14- A.A., **Fryer**, A., Bianco, M., Hepple, P.W., Jones, R.C. Strange, and M.A. Spiteri, Polymorphism at the glutathione S-transferase, GSTP1 locus: a new marker for bronchial hyperresponsiveness and asthma. *Am. J. Respir. Crit. Care Med. 161*: 1437-1442. (2000).

15- S.A., **Miller**, D.D. Dykes, and H.F. A Polesky, simple salting out procedure for extracting DNA from human nucleated cells. *Nucleic Acids Res. 16*: 1215. (1988).

16- L.M.F., **Amorim**, A., Rossini, G.A.S., Mendonça, P.F., Lotsch, T.A., Simão, Gallo, C.V.M. and Ribeiro Pinto, L.F. CYP1A1, GSTM1, and GSTT1 polymorphisms and breast cancer risk in Brazilian women. *Cancer Lett. 181*: 179-186. (2002).

17- S K., **Park**, D.,Kang Noh DY., Lee KM., Kim SU, Choi JY., Choi IM., Ahn S H., Choe KJ., Hirvonen A., Stricklan PT and Yoo KY . Reproductive factors, glutathione S-transferase M1 and T1 genetic polymorphisms and breast cancer risk. Breast Cancer Res Treat.78:89-96. (2003).

18- G.; **Colditz**, A.Graham, and Wolftz, R.The use of estrogen and progesterone and the risk of breast cancer in postmenopusal women. The New England Journal of Medicine . 332 : 1589-1593 . (1995).

19- S.; **Garte**, L.; Gaspari A.K; Alexandrie, W.R Pearson, and W.R. Vorachek, Metabolic gene polymorphism frequencies in control populations. Cancer Epidemiol Biomarkers Prev;10:1239–48.(2001).

20- R., **Milikan**, G.; Pittman, C.; Tse, D. A.; Savitz B.Newman, and D.Bell, Glutathione S-transferase M1, T1, and P1 and breast cancer.

Cancer Epidemiol Biomarkers Prev . 9: 567-73 . (2000) .

21- K.; **Garcia-Closas**, Kelesy, S.;Hankinson Spiegel Man, D.;Springer, K.; Willett, W.; Speizer F. and Hunter, D.Glutathione S-transferase class mu and theta polymorphisms and breast cancer susceptibility. J Natl Cancer Inst . 91: 1960-1964 . (1999).

22- WW, **Au** CHS, Torres NC Salazar and AS Salama Inheritance of polymorphic metabolizing genes and environmental disease and quality of life. Mutat Res 428:131-140.(1999).

23- B.G., **Szklarz**, M., Wojciki, A., Kuprianowicz, K., Kedzierska, M., Kedzierski, W.Gornik, and A. Pawlik, CYP2D6 and GSTM1 genotypes in a Polish population. Eur. J. Clin. Pharmacol. 55: 389-392. (1999). .

24- H., **Zhang**, A., Ahmadi, G., Arbman, J., Zdolesk, J., J., Castensen, B., Nordenskjold, P.Soderkvist, and X.F. Sun, Glutathione S-transferase Ti and Mi genotypes in normal mucosa, transitional mucosa and colorectal adenocarcinoma.Int.J.Cancer 48:135-138.A. (1999).

25- L., **Quinones**, F., Berthou, N., Varela, B., Simon, L.Gil, and D.Lucas, Ethnic susceptibility to lung cancer: differences in CYP2E1, CYP1A1 and GSTM1 genetic polymorphisms between French Caucasian and Chilean populations. Cancer Lett. 141: 167-171. (1999).

26- SJ, **London**, AK, Daly J, Cooper WC,Navidi CL Carpenter and JR Idle Polymorphysm of glutathione S-transferase M1 and lung cancer risk among African-American and Caucasians in Los Angeles County, California. J Natl Cancer Inst 87:1246-1253. (1995).

27- J.K.;**Wiencke**, S.;Pemble, B.Ketterer, and K.T.Kelsey, Gene deletion of glutathione S-transferase theta: correlation with induced genetic damage and potential role in endogenous mutagenesis. Cancer Epidemiol Biomarkers Prev.4:253-259.(1995).

تكرار تعدد الاشكال الوراثية للجينين *GSTM1* و *GSTT1* في المجتمع العراقي

سلوى جابر العوادي[1] و اسماعيل حسين عزيز[1] وعدنان عيسى البدران [2]

[1] معهد الهندسة الوراثية والتقانات الاحيائية للدراسات العليا – جامعة بغداد

[2] قسم علوم الحياة – كلية العلوم – جامعة البصرة

الخلاصة

العائلة الانزيمية Glutathion S-transferase (GSTs) لها دور حيوي في الطور الثاني من تفاعلات التحول الحيوي للمسرطنات البيئية والملوثات والادوية والسموم الاخرى . وتعتبر من الجينات المتعددة الاشكال الوراثية ويعتمد تكرارها على الاصول الاثنية في المجتمعات المختلفة وقد لوحظ ارتباط هذه الجينات مع مجموعة من الامراض المختلفة.

لقد درس تكرار الاشكال الوراثية للجينين *GSTT1* و *GSTM1* في هذه الدراسة ل 160 فردا عراقيا من بغداد والبصرة . جمعت عينات الدم من هؤلاء المتطوعين واستخلص DNA منها بطريقة Proteinase K / SDS ودرس تعدد الاشكال الوراثية في الجينين المذكورين بتقنية Multiplex- PCR .استخدمت هذه التقنية للتعرف على وجود او فقدان الجينين *GSTT1* و*GSTM1* . استخدم الجين البومين *Albumin* كسيطرة داخلية وكان حجمه (340 bp) بينما عرف وجود الجينين *GSTM1* و *GSTT1* بالقطعتين 215 و480 bp على التوالي . لقد اظهر التحليل الوراثي والاحصائي ان 30% من عينات الدراسه كانت فاقده لاحد الجينين او كليهما (13.1% *GSTM1* ، 6.9% *GSTT1* و 10% لكليهما) بينما كان 70% من الاشخاص طبيعين أي يمتلكون الجينين .

ان ماتوصلت اليه نتائج الدراسه تبين ان فقدان هذين الجينين في مجتمعنا العراقي تبدو اقل او مشابهة لتكرار الجينين في المجتمعات الغربية لكنها كانت اقل من تكرارها في المجتمعات الاسيوية .