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A Study of Polycyclic Aromatic Hydrocarbons (Pah) and. Tetra-Ethyl Lead Effect on Polymorphisms Cyp1a1 Gene on Thi-Qar Heat Power Stationary

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Abstract

The influence of polycyclic aromatic hydrocarbon and tetra-ethyl lead (TEL) on the gene *CYP1A1* is highly widespread, as workers can be exspoused with this material. which is effected through exposure or dealing with it. Therefore, the present study aimed to determining the influence of PAH and the role of *CYP1A1* gene according to some criteria (age, smoking, and family history). The current study included 50 workers with highest *CYP1A1* lacking of workers 48%compare with control 40%at the level of significant (P≤0.05). DNA was Extracted from the two sets and amplified *CYP1A1* gene with of technology of Polymerase Chain Reaction (PCR) so as Albumin gene (internal control). The highest percentage of lacking in heavy smoking51.4% compare with light ones 48.57%. The highest gene *CYP1A1* dele tion rate was aged (more than 50) years was (60%).The rate of lacking gene by the family history was 23%and in nob family history was 9% with has significant differences. *at the level of significant (P≤0.05).*

Key words: PCR, CYP1A1 gene, Detoxification, Polycyclic aromatic hydrocarbon

INTRODUCTION

According to researchers' view most of cancer output is made from human and natural ecological factors such as chemical pollutants in the ear, food, water and smoke tobacco. Also cancer risk depended on others factors like age, race within genetics Wild [5]. CYP1A1 is primarily located in your placenta, digestive system, skin and lungs. CYP1A1 detoxifies by linking toxic compounds with cytochrom p (CYP1A1), thus forming a less reactive substance [3]. CYP1A1 genotypes are associated with increased risk of various cancers that is compounded by exposure to cigarette smoke. [2] CYP1A1 are a family of isoenzymes that play an important role in protecting cells from cytotoxic and carcinogenic agents as well as (TEL) [1]. Organic lead (tetraethyl lead; TEL) is used as an antiknock agent in gasoline and jet fuels. TEL is absorbed rapidly from the skin as well as the lungs and gastrointestinal tract and is converted to triethyl lead in the body. This form of lead may be responsible for its toxic effects [7]. Oxidative stress defines as imbalance between reactive oxygen species and anti-oxidant defense systems and it is associated with pathogenesis of diseases. In human, genes involved in antioxidant defenses are highly polymorphic and show association with several multifactorial traits [14,23,34,13,6]. Cancer is a primary human health risk of exposure to PAHs. Exposure to PAHs has also been linked with cardiovascular disease and poor fetal development. PAHs have been linked to skin, lung, bladder, liver, and stomach cancers in well-established animal model studies [5,1]. The structure of a PAH influences whether and how the individual compound is carcinogenic some carcinogenic PAHs are genotoxic and induce mutations that initiate cancer; others are not genotoxic and

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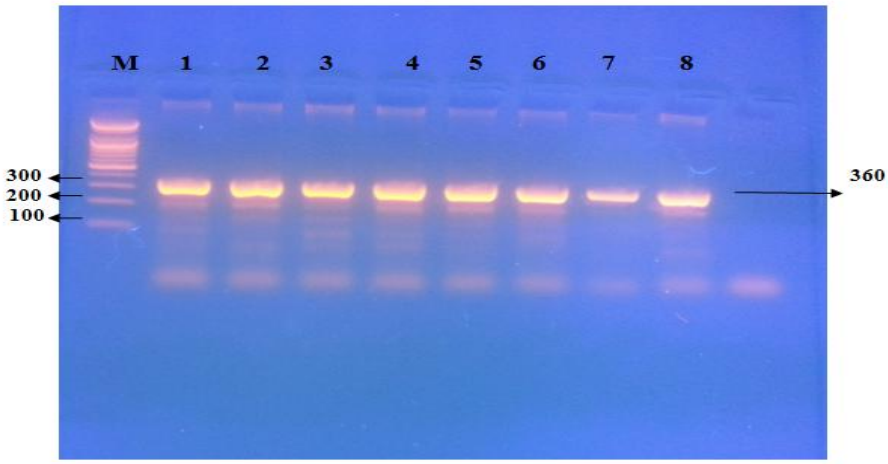
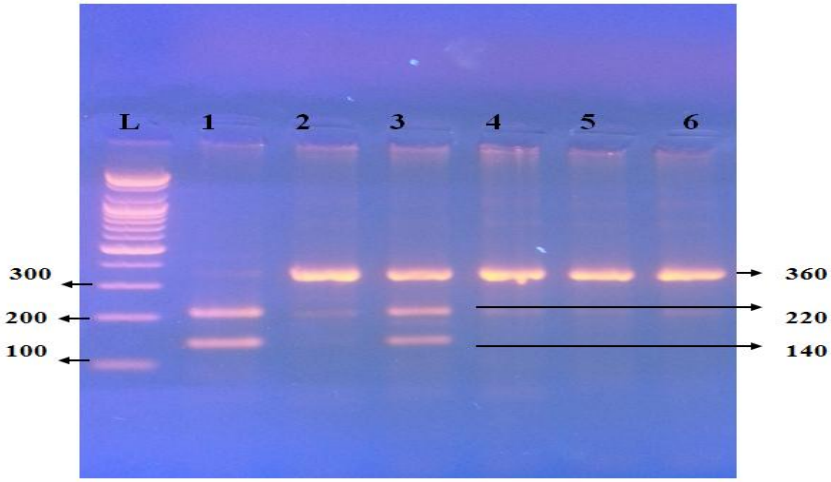
instead affect cancer promotion or progression [9,10]. PAHs that affect cancer initiation are typically first chemically modified by enzymes into metabolites that react with DNA, leading to mutations. When the DNA sequence is altered in genes that regulate cell replication, cancer can result. Mutagenic PAHs, such as benzo[*a*] pyrene, usually have four or more aromatic rings as well as a "bay region", a structural pocket that increases reactivity of the molecule to the metabolizing enzymes [23].

MATERIALS AND METHODS

Data and information on workers with PAH exposure were obtained from Thiqar refinery oil for year 2019.The number of workers is 50workes was collected information on samples collection, age, smoking, work duration, location and family history Samples blood from healthy men and workers were extracted based on the leaflet attached to kit extraction in manufactured by Gene aid (Korean origin). PCR technique was used to amplify CYP1A1 gene according to the method of [7]. DNA samples were detected by electrophoresis [19]. Statically analysis: using SPSS software



Picture 1: Electrophoresis of PCR –products of CYP1A1 gene on agarose gel at concentration of 2%.



Picture 2 : Electrophoresis of PCR –products of CYP1A1 gene on Agarose gel at concentration of 2%.

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RESULTS

The results of the current study showed The highly percentage of gene deletion in refinery workers 80% compare with control groups 24% with significant different at (P≤0.05) as show in Taleb 1. The results of the current gene absent distributed to both smokers by smoking status was the highest the lowest percentage (26%), rate of gene absent in Heavy smoking (30%), Light smoking the percentage of gene absent was calculated from the total number of exposed nonsmoking (20%), workers and the percentage of Heavy smoking and Light smoking of both groups, respectively, as shown in (Table2

Table 1: Comparison between heat stationary workers and control gene deletion

Genotypes	Control	Refinery workers	OR	95%CI*
CYP1A1 (+)	26 (52%)	10 (20%)	1.0	_____
CYP1A!(-)	24 (48%)	40 (80%)	4.33*	10.52-1.78

(+) present gene, (-) Absent gene, OR =Odds Ratio and 95%CI =Confidence Interval. *=significant.

Table 2: Genotypes Distribution of CYP1A1 gene samples of heat stationary workers

CYP1A1(-)	*CYP1A1*(+)	Smoking status	OR	95% CI
10 (20%)	5 (10%)	Non -smoking	1.0	_____
13 (26%)	10 (20%)	Light smoking	1.875	6.559-0. 303
15 (30%)	1 (2%)	Heavy smoking	7. 0	69.49-0.0705

according to Smoking status

The results of the current study show the risk Gene deletion was increased more than three times for men who have age less than 50 years (OR=3. 16) compare with control group shows significant difference (OR=3.16,95% CI=1.209-8.247), while multiplied about ten times for men who have age more than 50 years(OR=10) compare with control group shows significant difference (OR=10,95% CI=0.95-90.99)

Table 3: Genotypes Distribution of CYP1A1 gene of heat stationary workers according to the ages.

Genotype		Refinery workers	Control	OR	95% CI
Older than 50	CYP1A1(+)	0	7(14%)	1.0	_____
	CYP1A1(-)	10(20%)	%)12(6	10*	90.99-0.95
(-Younger less than 50)	CYP1A1(+)	10(20%)	19(12%)	1.0	_____
	CYP1A1(-)	30(60%)	18(36%)	3.16*	8.247–1.209

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The results of the current study of the risk Gene deletion was not increased the risk of gene deletion distributed to both groups and location were highest in rural family history (23%) compare with nonfamily (9%), as shown in Table 4. There were significant differences between workers in nonfamily history and family history

Table 4: Genotypes Distribution of CYP1A1 gene according to family history.

Geno type	Family history	%	Non-family history	%	OR	95% CI
Normal	88	44%	22	11%	1.0	-
CYP1A1 -	36	23%	18	9%	1.638	0.231-1.763
CYP1A1+	20	10%	6	3%	0.833	0.195-3.551

DISCUSSION

[3]. Reported that PAH is one of the most common factor which has been influence on human health since low molecular weight PAHs are prevalent in the environment, thus posing a significant risk to human health at the promotional phases of cancer and these result agree with our results. The statistically analyses of results show that there is significant difference between the mutant and PAH influence. These results indicated that there is correlation between polymorphisms of CYP1A1 and PAH number of people with is increasing due to population growth, PAH especially Benzo (a) pyrene, urbanization, aging, [21,23,24]. has been shown to play a major obesity and mobile phone radiation exposure The results of the present study have showed a table (4) the percentage of workers in ورole nonfamily was 9%, while those living in family history l reached 23%. There was significant difference between the nonfamily urban and family rural areas (OR=1.638). This is may be because family history are more active to exposed to risk factors such as mobile phone radiation, smoking and eating fast food than non family history, and non family history [4,3,2].This result agree with[16].the highest percentage Carbone exhausts in the city within people which have family history due to truck and car exhaust and this car exhausts avoid the linking between CYP1A1 and toxic compounds such as heavy toxic metal, solvents and cheered food and this lead to reactive substances forming. Compare with people which This study differed from a study conducted by [11,1] which showed that there was haven' significant difference between the frequency of CYP1A1 genotype and incidence of PAH when compared refinery oil workers and control groups was also different from the study conducted by [18] Which showed that there was no effect of smoking difference among smoking workers and non smoking (OR=0.3239) and these results disagree with our result which has been shown correlation between smokers and non smokers workers (OR=7.0) so (OR=1.875). The main factors that make older as between heavy and light smokers were long period workers higher than younger are related with several another factors such as

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exposure to PAH molecule which increased influence with ages progressive (OR=10.0) and this result agree with that[11]

CONCLUSION

According to these results, we can conclude that polycyclic aromatic hydrocarbon was played important influence on the gene detoxification deletion. Risk factors such as age smoking and family history have been appeared differentiation relationship with the *CYP1A1* gene deletion, where the highest percentage was found in workers aged more than 50 years and family history

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