Smoking as an Environmental Xenobiotic and Its Effects on Certain Health Parameters

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Abstract

Data were drawn in the study from patients seen and/or admitted to Merjan teaching hospital in the city of Hilla, Babylon governorate and they include:
100 healthy controlled non-smokers and 100 smokers' non-diabetic and 240 patients with diabetes mellitus also 100 patients with acute myocardial infarction, 52 patients with bronchogenic carcinoma.
It had been shown that there was statically significant increase in leukocyte count in smokers than in non-smokers. There is statically significant increase in platelets count between smoker diabetic and healthy control non-smoker. There are statically significant changes in the level of fibrinogen in diabetic smoker from non-smoker healthy control and also smoker subject from non-smoker subject.
In patient with acute myocardial infarction we noticed that 89% of them had smoking index above 600. Also we noticed that in patients with bronchogenic carcinoma 87% of them were heavy smokers.
Most of smokers showed significant reduction in peak expiratory flow rate (PEFR) than healthy non-smokers, also they showed higher Hb level than non-smoker to extent that venesection in 10 of them was done to relieve symptoms of secondary polycythemia.
In conclusion tobacco usage through cigarette and other means is the most avoidable risk factors for several human diseases including cancer.

الخلاصة

تم اخذ المعلومات الخاصة بهذه الدراسة من المرضى الذين راجعوا او دخلوا في مستشفى مرجان التعليمي في مدينة الحلة (محافظة بابل) وكالاتي:
مجموعة مقارنة ضمت (100) من المرضى الأصحاء وغير المدخنين, و (100) مريض مدخن وغير مصاب بمرض السكري, (52) مريض مصابين بجلطة قلبية حادة, و (52) مصابين بسرطان الرئة.
أثبتت الدراسة أن هنالك زيادة ذات قيمة حسابية في كريات الدم البيض عند المرضى المدخنين بالمقارنة مع غير المدخنين, ووجود زيادة في الأقراص الدموية في المرضى المدخنين والمصابين بداء السكري بالمقارنة مع مجموعة المقارنة غير المدخنين وكذلك وجود زيادة في الفايبرينوجين عند المدخنين المصابين بالسكري بالمقارنة مع مجموعة المقارنة الأصحاء وكذلك بين المدخنين وغير المدخنين.وقد وجد أيضا أن 89% من المرضى المصابين بالجلطة القلبية الحادة (cigarette index >600) وكذلك أن 87% من المصابين بسرطان الرئة هم من المدخنين بإفراط.
هنالك خلل في وظائف الرئة وزيادة في خضاب الدم عند معظم المدخنين بالمقارنة مع الأصحاء غير المدخنين (10 مرضى منهم احتاجوا إلى فصد لمعالجة أعراض زيادة خضاب الدم الثانوي).
الخلاصة: القطران الناتج عن التدخين والوسائل الأخرى يعتبر أهم عامل خطورة (ممكن تجنبه) لأمراض عديدة تصيب الإنسان ومن ضمنها السرطان.

Introduction

Xenobiotic is a compound that is considered as foreign substance in the body. The principle classes of xenobiotics of medical relevance are drugs, chemical carcinogens, and various compounds that the environment by one route or another, including smoking as an environmental pollutant. Xenobiotics can produce a variety of biologic effects including pharmacological responses, toxicity, immunological reactions & cancer. [1]

Medical evidence of harm, which was done by smoking has been accumulated for 200 years at first in relation to cancer of the lip & mouth and then in relation to cardiovascular diseases and respiratory diseases. [2, 3, 4] An estimated 430,000 deaths each year are directly caused by cigarette smoking. Current estimates indicate about one third of all adults smoke and women & both sexes still smoking more. [5]

All cigarettes can damage human body & and any amount of smoke is dangerous, a low tar can be just as harmful as a high tar cigarettes. All cigarettes smokers have a lower level of lung function than non-smokers, the late stage of lung diseases produced by smoking is one of the most miserable of all medical conditions, and it creates a feeling of gasping for breath all the time, similar to the feeling of drowning.

Smoking have been shown to have harmful effects on all parts of digestive system and it had been reported that peptic ulcer is more likely to occur, less likely to heal & more likely to cause death in smokers than non-smokers. [6]

It had been shown that nicotine like other addictive drugs attaches to the core neurons of the brain's reward system, and in the ventral tegmental area, these reward neurons called dopaminergic neurons and release dopamine which trigger pleasure response & disposition, drive the process of addiction and nicotine also leads to imbalance between the two neurotransmitters glutamate and gamma-amino butyric acid (GABA) in the ventral tegmental area, this explains how a brief exposure to nicotine results in long-term excitation of the brain's reward area. [7, 8, 9]

Another important aspect in the problem of smoking is the metabolism of nicotine. There are certain isoforms of Cytochrome P450 which are particularly involved in the metabolism of Polycyclic Aromatic Hydrocarbons (PAHs) and related molecules and in the lung it may be involved in the conversion of inactive (PAHs) procarcin-ogens inhaled by smoking into active carcinogens by hydroxylation reaction and smokers have higher levels of this enzyme in some of their cells and tissues than do non-smokers, however, an interesting type of polymorphism is that of Cytochrome enzyme (CYP2 A6) which is involved in the metabolism of nicotine to contain, three alleles have been identified for (CYP2 A6) , one of them is a wild type and the other two are inactive alleles, it had been postulated that individuals with inactive alleles have impaired metabolism of nicotine and they are apparently protected against becoming tobacco-dependent smokers and these individuals smoke less , presumably because their blood & brain concentration of nicotine remains elevated longer than those with wild-type allele, though, it had been speculated that inhibiting (CYP2 A6) may be a novel way to help prevent and treat smoking . [10,11]

It had been recently shown that smoking raises the risk of metabolic syndrome in teens & in one study on 2273 subjects their age between (12 to 19 years) metabolic syndrome which defined:

("clusters of condition in which there is a high BP and/or high blood sugar and high triglyceride , low HDL – cholestrol level, in addition to the presence of abdominal-obesity ") was present in 1.2 % of non-exposed teens , 5.4% of those exposed to passive smoking and 8.7% of those who were active smokers [12].

In this paper we relate the effects of smoking to several health problems, including its effects on certain hemopoitic elements including leucocytes, platelets, haemoglobin level and fibrinogen. Smoking leads to important changes in these elements. Also the effects of smoking on, patients with diabetes mellitus, ischemic heart diseases, pulmonary diseases, & lung cancer, in addition we should not underestimate the problem of smoking in paediatric age groups as there is increasing numbers of smokers

passive and active smoking behaviors. It had been shown that even passive (environmental) tobacco smoking had been shown to increase the risk of endothelial damage, platelets aggregation and urinary excretion of tobacco-specific carcinogens. [13]

Subjects and Methods

Data were drawn in this study from patients seen and /or admitted to Merjan Teaching Hospital in the city of Hilla, Babylon Governorate and they include:
100 healthy controlled non-smokers and 100 non-diabetic smokers and 240 patients with diabetes mellitus (as shown in table 1) also 100 patients with acute myocardial infarction, 52 patients with bronchogenic carcinoma and 270 patients with peptic ulcer disease.
The following changes had been studied blood picture (including Hb %, white cell count and differential count, platelet count). Fibrinogen level was estimated using Grannis et al method and Evotte-method [14].
Neutrophil function was tested using Fikaring and Smith wick method with nitro blue – Tetrazolium dye test [15].
In the control group we had study PEFR (peak Expiratory Flow Rate) using Wright Spiro metter.
Statistical method was done by using t-test and x^2 and p value.

Results

It had been shown that there was a statically significant change in the leukocyte count in smokers than in non-smokers (7753±73) / cu mm versus (6354±119), while higher neutrophil count in smokers than non-smokers (5461±80) versus (4060±81) and this indicate that there is statistically significant ($P<0.05$) increase leukocyte count in diabetic smoker than diabetic non smoker (see table 2 and table 3).
There is statistically significant increase in platelets count between diabetic smoker and healthy control non smoker (table 4).

Table-1: The number of diabetic smokers

sex	Total no.	No. of smokers	Percentage of smokers
Male	91	44	48.35%
Female	149	21	14.1%
Total	240	65	37.14%

Table- 2: The differences in leukocytes count in diabetic smokers and non- smokers

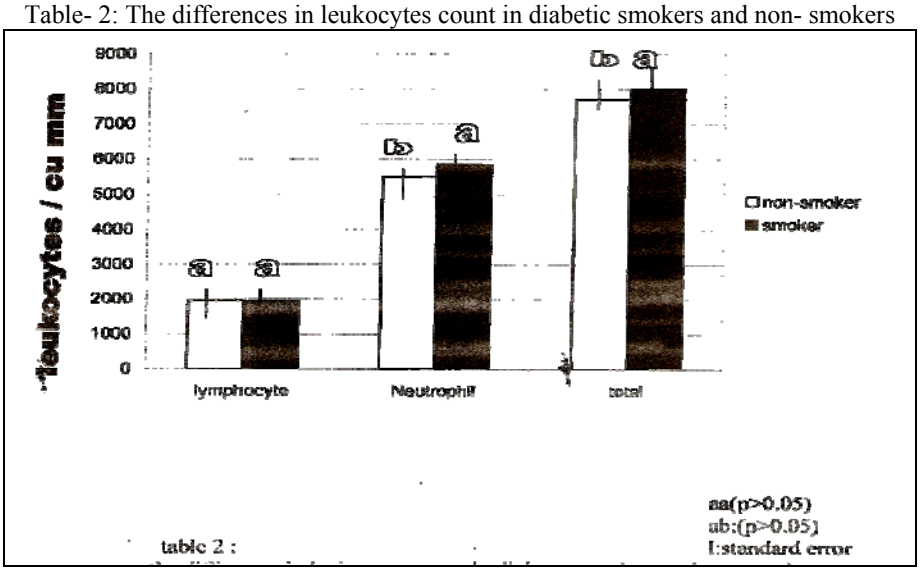


Table 3
The differences in leukocytes count between diabetic non- smoker s & in healthy controlled human

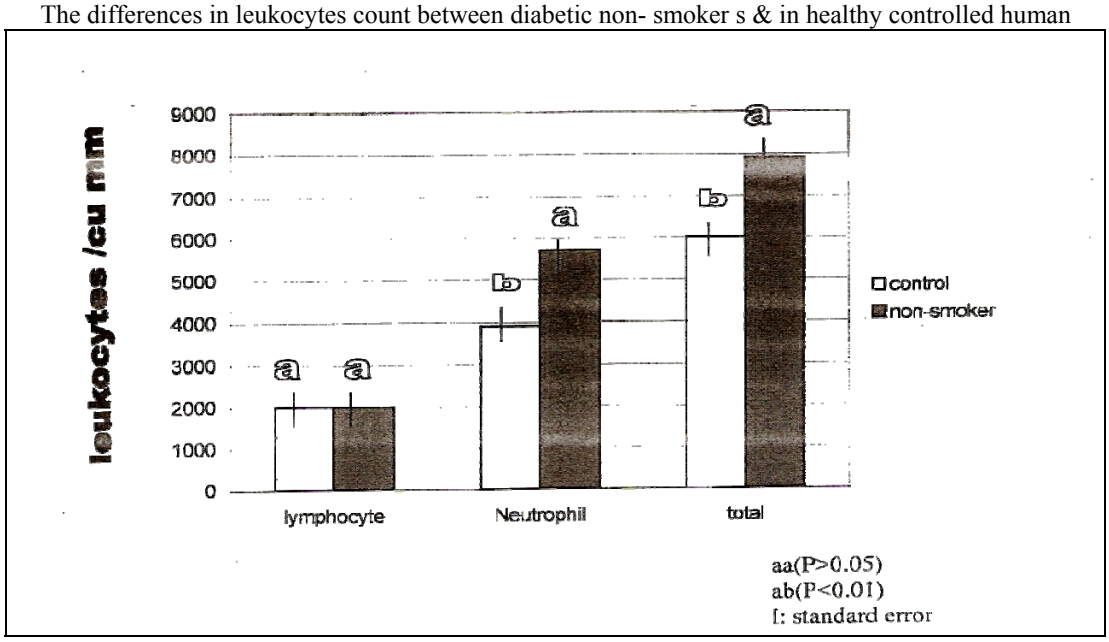
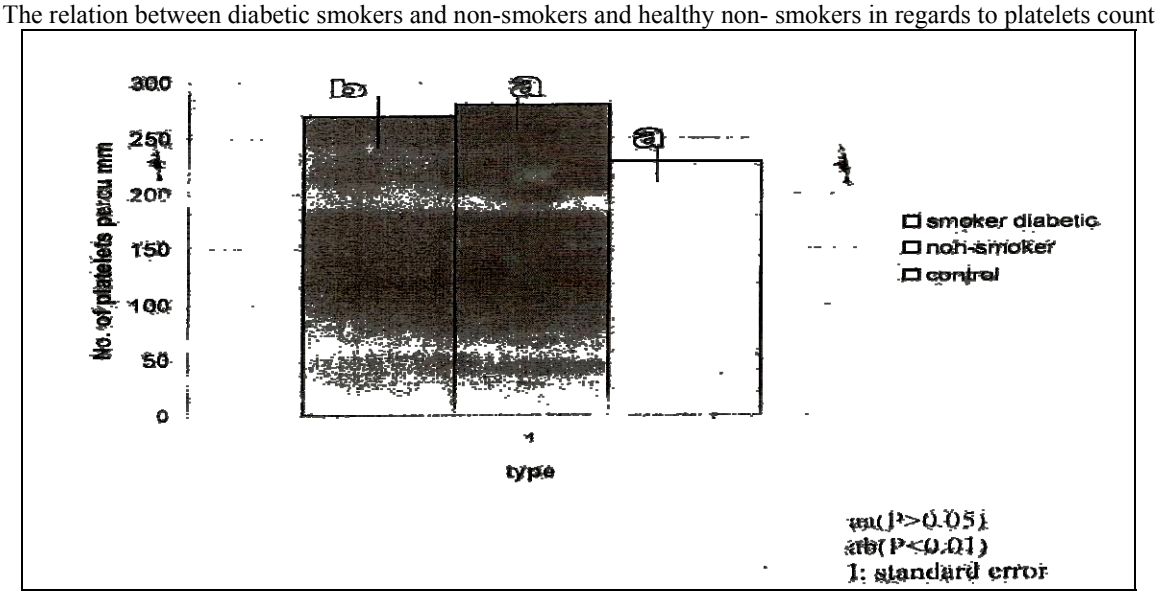


Table -4
The relation between diabetic smokers and non-smokers and healthy non- smokers in regards to platelets count



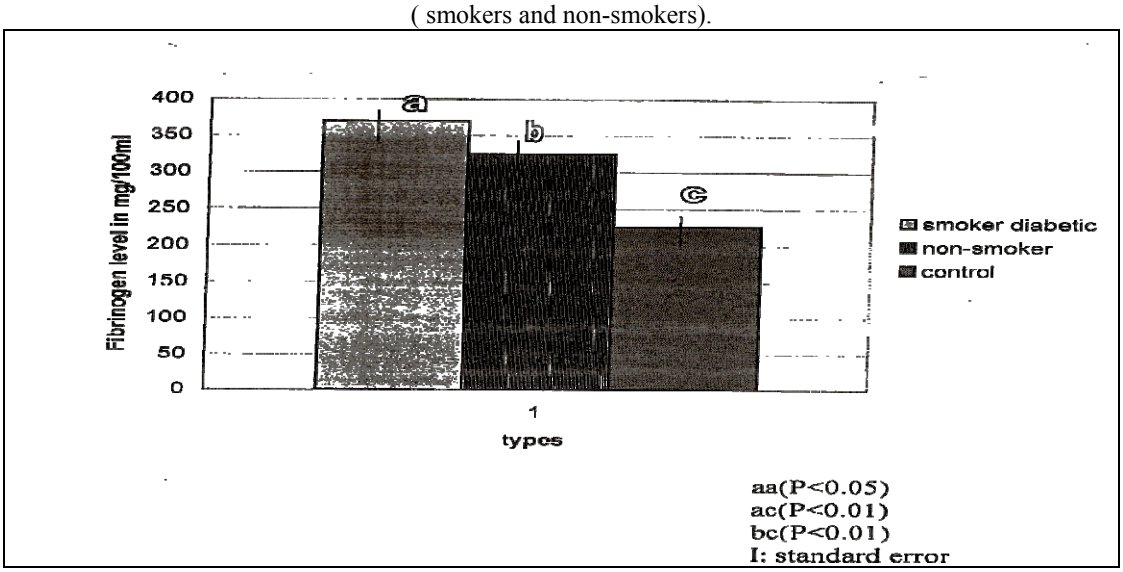
There are statistically significant changes in the level of fibrinogen in diabetic smoker from healthy controlled non-smoker and also smoker subject from non-smoker subject (table 5)

Platelet count in diabetic smokers (10^3 X 4.07 ± 10^3X276.587) Platelet count in healthy non smokers (10^3X3.344± 10^3X221.817)

The study shows that marked reduction in the phaqgocytic activity in smoker persons and diabetic smokers than healthy non-smoker: Fibrinogen level in diabetic smokers is (375 mg/dl ±5) Fibrinogen level in healthy non-smokers is (275mg/dl±4) Fibrinogen level in smoker is (293mg/dl ± 8)

In healthy fibrinogen level is (275 mg/dl±4)

Table-5
The level of fibrinogen in the control group non-smokers and the diabetic patients (smokers and non-smokers).



Diabetic smoker (9.28%) reduction.
Smoker (5.83%) reduction.
Non-smoker (4.93%) reduction.

In Patients with acute myocardial infarction, we noticed that 89 % of them had smoking index above 600. Also we noticed that in patients with bronchogenic carcinoma 87% of them were heavy smokers.

Most of the smokers show significant reduction in PEFR than healthy non-smoker

Also they showed higher Hb level than non-smoker to extent that we did vene section in 10 of them to relive symptoms of secondary polycythemia.

During the study on 270 patients with peptic ulcer disease it was noticed that a significant number of patients showing failure to respond to therapy and developed relapses and impaired healing of ulcer.

Discussion and Conclusion:

The health consequences of smoking are large and many of the health problem caused by the used of tobacco are well-known even to smoker for that reason we have to tackle the problem from earlier age as 13% middle school student used cigarette and 28% of higher school students are smoker [16]. we expect the rate the figure among our students higher than this .

The real problem related to smoking is its effect on hemopoetic system which had been shown in our study where secondary polycythemias contribute to increase blood viscosity and the complication related to this.

Smoking also affect on the neutrophil count and function in smoker patients and diabetic smokers and this may lead to vascular complications that can be seen in those patients, also the change in the neutrophil function leads to some immunological complication associated with diabetes mellitus. In previous epidemiological study it had been found that there was association between elevated neutrophil counts and risk and mortality from coronary heart disease [17], [18].

In smokers and in diabetic smokers both show higher fibrinogen level and higher platelet count and this adds further risk to the development of cardiovascular disease and atherosclerosis and this confirms the study that suggests cigarette smoking approximately doubles a person's risk of stroke and causes

reduced circulation by causing narrowing of the arteries, thus, smokers are more than 10 times as likely as non-smokers to develop peripheral vascular disease [19 , 20] .

Smoking causes a variety of life threatening respiratory diseases, including lung cancer, emphysema, chronic bronchitis and impairment of lung function, in previous study, 90% of all deaths from chronic obstructive pulmonary diseases are attributable to cigarette smoking [21]. In a study which was done on workers who worked one of the cement factory in the city of Badosh. Mosul governorate in Iraq , and the surrounding families , in which pulmonary function tests including arterial oxygen done on 108 workers who works for a period between (1-30) years , these workers were non-smokers and another 100 smoker workers whom smoked at least daily 20 cigarette were tested and also 74 persons from the region surrounding the factory who had lived in that area for a period between (1-13) years undergone pulmonary function testing . The result of the study showed that non-smoker workers had 1.8% reduction in lung function , whereas there was 14.7% reduction in smoker workers , also pulmonary function showed 17.5% reduction in persons from the families who were exposed to environmental pollution and they were cigarette-smoker ,whereas non-smoker-persons showed only 2.7% reduction . [22]

If we talk about the relation of smoking to lung cancer, we should remember the writing of William Lambe who outlined the understanding of the causes of cancer when he wrote: (we say a person dies of cancer, the truth must be a person dies of the causes of cancer and the cancer is not the cause but the mode of dying). This saying goes with the effect of smoking on the pulmonary system and smoking under suitable condition with air pollution and industrial exposure will lead to occurrence of cancer. [23]

Smoking is harmful to all parts of the digestive system and contributing to common GIT disorders including worsening of peptic ulcer disease, gall stones. Pancreatic disorders and even some kind of lever disease might be related to smoking. [24]. Currently we advice people with GIT disorder not to smoke. Smoking dose not only affect major organ and system in the body but it affects cellular function on a molecular level through its affect on various enzyme levels (e.g. smoking inactivates anti-protease and leads to inflammatory reaction at different sites). [25, 26, 27]

In conclusion

Tobacco use through cigarette and other means is the most avoidable risk factor for several human diseases including cancer. We should consider smoking prevention a paediatric issue as 80% of smokers begin their smoking before the age of 18 years. School health education should begin during the elementary grade when self-concept and self-care attitudes are being formed also we have to enrich teachers of the harmful effects of smoking.

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