An-Najah National University
Faculty of Graduate Studies

# The effect of Smoking on increasing the risk of Type II Diabetes Mellitus and Hypertension 

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This Thesis was defended successfully on 25/11/2006 and approved.

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## Dedication

To My dear Husband, Father, Mother, Brothers and My children Sujude, "Mohammad kathem", and Mo'men for their Patience and Encouragement, with Love and respect.

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## List of Abbreviations:

| Centers for Disease Control and Prevention | $(\mathrm{CDC})$ |
| :--- | :--- |
| World Health Organization | $(\mathrm{WHO})$ |
| Body mass index | (BMI) |
| American Heart Association | $(\mathrm{AHA})$ |
| Nicotinic acetylcholine receptors | (nAchRs) |
| Joint National Committee | $($ (NNC $)$ |
| World Health Organization/International Society of <br> Hypertension | (WHO/ISH) |
| Blood pressure | (BP) |
| Diabetes mellitus | (DM) |
| Systolic blood pressure | (SBP) |
| Diastolic blood pressure | (DBP) |
| Random blood sugar | (RBS) |
| Fasting blood sugar | (FBS) |
| Family history | (F.H) |
| Hypertension | (HTN) |
| D2 dopamine receptor gene | (DRD2). |
| Hepatic glucose output | (HGO) |
| Insulin Resistance Atherosclerosis Study | (IRAS) |

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The effect of Smoking on increasing the risk of Type II<br>Diabetes Mellitus and Hypertension<br>By<br>Buthaina Farah Khalil Salawdeh<br>Supervisors<br>Dr. Suleiman Khalil<br>Dr. Ali Alsha'ar


#### Abstract

The current study aims at exploring the effect of cigarette smoking on the susceptibility to develop Type2 DM and hypertension among adult males in the refugee population in the northern area of West Bank, in period from the $1^{\text {st }}$ of December to $30^{\text {th }}$ of March 2005-2006, for cases not known to have Type2 diabetes or hypertension. The objectives of the study were set to assist public health practitioners in developing health education program aiming at the prevention of smoking and reduce the risks for developing hypertension

We notice from this screening test that $36.2 \%$ of the cases have high BP. 13.1\% have high RBS. This indicates that there are a high percentage of people who have hypertension \& Type 2 DM, this study provides baseline data on these diseases in refugee people in Palestine. Information that is essential for the implementation of national planning and service provision.


It was found that cigarette smoking can increase the risk of Type 2 DM \& there were no statistical effect of smoking on hypertension but smoking can increases cardiovascular disease which lead to HTN disease. Increasing BMI can increase the risk of the HTN \& Type 2 diabetes mellitus.

FH especially in first degree relatives can increase the risk of HTN, \& genetic predisposition was found to be low in Type 2 DM in this study, also practicing sport play a role in decreasing the risk of Type 2 DM .

## Chapter one

## Introduction

### 1.1 Introduction:

Smoking is the leading preventable cause of death in the USA. Regular smoker probably losing about five and half minutes of life expectancy for each cigarette he/she smokes. Up to the age of sixty five, people who smoke twenty or more cigarettes per day die at almost twice the rate for nonsmokers in the same age group. Consider the average smoker, a person who smokes up to twenty cigarettes per day compared with nonsmokers is about fourteen times more likely to die from cancer of the lung, throat, or mouth; four times more likely to die from cancer of the esophagus; twice as likely to die from cancer of the bladder; and twice as likely to die from a heart attack. (Charles B. Clayman 1994)

Cigarettes are a principal cause of chronic bronchitis, emphysema, asthma, and having a chronic lung disease increases the risks of pneumonia and heart failure. Smoking also increases the risk of high blood pressure. Some brands of cigarettes contain less tar and nicotine than others, but there is no such thing as a safe cigarette, switching to mild cigarettes does not usually help; habitually heavy smokers usually adapt their smoking habits to the switch by inhaling longer and more deeply and by smoking more cigarettes. (Charles B. Clayman. 1994)

### 1.2The prevalence of smoking, diabetes \&, hypertension:

### 1.2.1.1 Prevalence of smoking:

Prevalence of smoking in Palestine is: $18.6 \%$ in GS and $23.9 \%$ in WB. This is relatively a high prevalence. (Health Status in Palestine 2003, July 2004).

Cigarette Smoking among Adults in United States in 2004 was 20.9 $\%, 44.5$ million of adults in the United States are current smokers, down from $22.5 \%$ in 2002 and $24.1 \%$ in 1998. In 2004, an estimated 45.6 million adults were former smokers, representing $50.6 \%$ of those who had ever smoked. In developing countries new survey shows that about $20 \%$ of school children are already regular smokers, according to a survey coordinated by WHO in collaboration with the CDC found that nearly $25 \%$ of the children smokers started the habit before the age of 10 and more than two-thirds wanted to quit. The CDC's note in the Bulletin article that worldwide some 250 million children and teenagers alive today will eventually die as a result of their tobacco habit and that $70 \%$ of them will be in developing countries. The high prevalence of smoking in such a young age group portends a lifetime of addiction for a large number of people, half of whom will die prematurely of tobacco-related diseases.

### 1.2.1.2 Smoking-attributable mortality, years of potential life lost, and economic costs in United States, 1995-1999

From 1995-1999, smoking killed over 440,000 people in the United States each year. Each pack Cigarette smoking continues to be a leading cause of death in the unites of cigarettes sold in the United States costs the nation an estimated $\$ 7.18$ in medical care costs and lost productivity. Estimates show that smoking caused over $\$ 150$ billion in annual healthrelated economic losses from 1995 to 1999 including $\$ 81.9$ billion in mortality-related productivity losses (average for 1995-1999) and \$75.5 billion in excess medical expenditures in 1998. The economic costs of smoking are estimated to be about $\$ 3,391$ per smoker per year. Smoking caused an estimated 264,087 male and 178,311 female deaths in the

United States each year from 1995 to 1999.Among adults, the study estimates that most deaths were from lung cancer 124,813 , ischemic heart disease $(81,976)$ and chronic airway obstruction 64,735 .Excluding adult deaths from exposure to secondhand smoke, adult males and females lost an average of 13.2 and 14.5 years of life respectively, because they smoked. Smoking during pregnancy resulted in an estimated 599 male infant and 408 female infant deaths annually. For men, the average number of annual smoking-attributable cancer and cardiovascular disease deaths in 1995-1999 fell while the number of respiratory disease deaths remained stable. For women, the average number of annual smoking-attributable cancer and respiratory disease deaths in 1995-1999 rose while the number of cardiovascular deaths fell. Smoking-attributable neonatal expenditures were estimated at $\$ 366$ million in 1996 or $\$ 704$ per maternal smoker.

### 1.2.2.1 Prevalence of Diabetes mellitus in Palestine:

The prevalence of DM and associated factors in a cross-sectional survey of an urban Palestinian population of 492 men and women aged 3065 years was investigated. The oral glucose tolerance test was used to diagnose diabetes and impaired glucose tolerance. WHO recommended survey protocols were followed. Diabetes was found in $12.0 \%$ of the Palestinian population (including $9.4 \%$ previously diagnosed), and impaired glucose tolerance in $5.9 \%$.(Institute of Community and Public Health 2000 Sep-Nov; 6 (5-6): 1039-45.)

The prevalence of DM and impaired glucose tolerance was investigated in a cross-sectional population-based study in a rural Palestinian population of 500 females and males aged $30-65$ years. The
prevalence of diabetes was $9.6 \%$ and $10.0 \%$ in females and males respectively. The prevalence of impaired glucose tolerance was $8.6 \%$; $10.3 \%$ in females, $6.2 \%$ in males. The prevalence of total glucose intolerance (diabetes mellitus+impaired glucose tolerance) was $18.4 \%$. These studies indicate a high prevalence of glucose intolerance in Palestine. (Institute of Community and Public Health, 2000 Sep-Nov; 6(5-6): 103945.)

### 1.2.2.2 The prevalence of diabetes in developing countries

According to WHO the prevalence of diabetes has reached epidemic proportions. WHO predicts that developing countries will bear the brunt of this epidemic in the $21^{\text {st }}$ century, with approximately $80 \%$ of all new cases of diabetes expected to appear in the developing countries.

The number of people with diabetes in the developing countries is expected to rise to 228 million in 2025 . This represents $76 \%$ of the total number of people with diabetes worldwide. The greatest increase of all is expected to be in India (195\%), if present trends persist, by 2025 the majority of people with diabetes in the developing countries will be in the 45-64 age group.

According to the International Diabetes Federation out of the top ten countries with diabetes sufferers, seven are developing countries. The Caribbean and the Middle East have regions where the percentage of adults with diabetes has reached $20 \%$. In certain parts of Africa developing diabetes can mean a short route to death. While patients in developed countries, with access to proper treatment, can expect to live for several
decades, in countries such as Mali and Mozambique developing diabetes often means a life expectancy of one or two years.

Top five countries with the most diabetes sufferers in 2003 were (India 35.5 million, China 23.8 million, USA 16 million, Russia 9.7 million and Japan 6.7 million). Top five countries with the highest percentage of adults with diabetes in 2003 were: Nauru 30.2 \%, United Arab Emirates $20.1 \%$, Qatar $16 \%$, Bahrain $14.9 \%$, and Kuwait $12.8 \%$. The number of diabetes sufferers by 2025 is expected to Double in Africa, the Eastern Mediterranean and Middle East, and South-East Asia, and rise by 20\% in Europe, $50 \%$ in North America, $85 \%$ in South and Central America and $75 \%$ in the Western Pacific. There are 6 million new diabetes sufferers in the world each year. Every ten seconds someone in the world dies as a result of having diabetes.

### 1.2.2.3 Diabetes costs a burden for families and society

WHO says, $80 \%$ of people in developing countries pay directly for some or all of their own medicine. In many instances, the choice is between health care and food or clothing, and such financial constraints inevitably result in under-consumption of health care services. Studies in India estimate that, for a low-income Indian family with an adult with diabetes, as much as $25 \%$ of family income may be devoted to diabetes care. In developing countries, the prevailing poverty, ignorance, illiteracy and poor health consciousness further adds to the problem. Those who cannot afford or do not have access to even bare minimum healthcare facilities are likely to be diagnosed late and suffer from diabetes related complications because of delay in diagnosis and/or improper treatment.

### 1.2.3. Hypertension prevalence:

The prevalence of hypertension \&abnormal glucose metabolism, in a rural and an urban Palestinian West Bank community was calculated. A total of 500 rural and 492 urban men and women aged $30-65$ years participated in a community-based cross-sectional survey. Blood pressure was taken from each subject. Prevalence of hypertension in the rural and urban populations was (25.4 and $21.5 \%$, respectively). (Journal Diabetes Care 2001; 24,2:275-279).

Also the number of adults in the United States with high blood pressure increased 30 percent over the last decade (from 1988-2000), according to a study published in Journal of the American Heart Association.

The study found that at least 65 million Americans have hypertension; according to this almost a third of U.S. adults have hypertension.

The number of adults with high blood pressure has increased: 59.2 million people had hypertension on the basis of blood pressure measurements or prescriptions for blood pressure medication. More than 6 million people had high blood pressure based on their medical history, resulting in an estimated total of 65.2 million hypertensive adults. The 1999-2000 survey shows that $28.7 \%$ of women and $28.3 \%$ of men have high blood pressure. When prevalence was divided along racial/ethnic categories, non-Hispanic black Americans have the highest prevalence at 38.8 percent. High blood pressure is prevalent in 28.7 percent $\%$ of the

Mexican American population, and in $27.2 \%$ of the non-Hispanic white population.

### 1.3 What is tobacco?

Botanical Tobacco name:
Nicotiana tabacum. Family:
Solanaceae
1.3.1 Part Used: Leaves cured and dried.
1.3.2 Habitat: Virginia, America; and cultivated with other species in China, Turkey, Greece, Holland, France,
 Germany and most subtropical countries.

### 1.3.3 Description:

Tobacco is an annual, with a long fibrous root, stem erect, round, hairy, and viscid; it branches near the top. Leaves are large, numerous, alternate, pointed, hairy, pale-green color, brittle, narcotic odor, with a nauseous, bitter acrid taste.

### 1.3.4 Constituents:

Tobacco smoke contains 4,000 different chemicals at least 200 are known to be poisonous to people, three principal dangerous chemicals are: tar, nicotine, and carbon monoxide. Tar is a mixture of several substances
(hydrocarbons) that condense into a sticky substance in the lungs. Nicotine is an addictive drug that is absorbed from the lungs and acts mainly on the nervous and cardiovascular systems. Carbon monoxide decreases the amount of oxygen that red blood cells can carry throughout the body. (Charles B. Clayman. 1994)

### 1.4 Nicotine:

Nicotine is a pyridine alkaloid obtained from the dried leaves of the tobacco. The leaves contain from 0.6 to 9.0 percent nicotine. Nicotine is colorless to pale yellow, very hygroscopic, oily, volatile liquid with an unpleasant, pungent odor and a sharp, burning, persistent taste. (. James E. Robbers, Ph.D. 1996) Nicotine is a ganglionic (nicotinic) cholinergicreceptor agonist with complex pharmacologic actions that include effects mediated by binding to receptors in autonomic ganglia, the adrenal medulla, the neuromuscular junction, and the brain. Chronic use of nicotine may result in psychological and physical dependence. As a temporary aid for the cessation of cigarette smoking, the drug is available in transdermal systems, and it is also available bound to an ion exchange resin in a chewing gum base. These alternative sources of nicotine help reduce the withdrawal symptoms associated with nicotine addiction. (James E. Robbers.1996).

### 1.4.1 Nicotine absorption metabolism \& elimination:

### 1.4.1.1 Absorption:

Nicotine is distilled from the tobacco burning on tar droplets that are inhaled and deposited in small airways and alveoli. This allows even more
rapid absorption through the lungs than occurs with intravenous administration. Cigarettes are excellently designed methods of rapidly administering and adjusting plasma nicotine concentrations. Smokers appear to be able to regulate the plasma concentration of nicotine on a puff-by-puff basis depending on the type of tobacco and rate and depth of puffing.

### 1.4.1.2. Metabolism:

Metabolism of nicotine occurs in human liver. It is extensively metabolized to a number of metabolites. In humans, about 70 to $80 \%$ of nicotine is converted to cotinine. Another primary metabolite of nicotine extent Nicotine ${ }^{\mathrm{N}}$-oxide ( NNO ).

### 1.4.1.3. Elimination

Nicotine and its metabolites (cotinine and nicotine 1-N-oxide) are excreted into urine, saliva.Passage of saliva containing nicotine into the stomach, combined with the trapping of nicotine in the acidic gastric fluid and reabsorption from the small bowel, provides a potential route for enteric nicotine recirculation. Nicotine freely crosses the placenta and has been found in amniotic fluid and the umbilical cord blood of neonates. Nicotine is found in breast milk and the breast fluid of non lactating women and in cervical mucous secretions.

### 1.4.2 Physiological Effects of Nicotine:

### 1.4.2.1. Negative Physiological Effects of Nicotine:

Nicotine is the active ingredient in tobacco that acts as a stimulant on the heart and nervous system. In its pure form only one drop of
approximately 50 mg can kill a person within minutes. Nicotine has widespread actions on the cardiovascular system. The typical cardiovascular response to smoking a cigarette is similar to the response to sympathetic stimulation to exercise. There is an increase in heart rate, cardiac output, and coronary blood flow, a rise in blood pressure, peripheral vasoconstriction with a drop in skin temperature in the extremities, and an increase in muscle blood flow, at the same time there is a rise in circulating levels of adrenaline and noradrenaline, a rise in blood sugar and fatty acids, and an increase in the adhesiveness and aggregation of blood platelets. Nicotine produces a further rise in blood pressure and this may be a factor in the increased death rate from rupture of aortic and cerebral aneurysms in smokers. In pregnancy, uterine vasoconstriction may contribute to fetal hypoxia, also smoking reduces fetal breathing movements and makes congenital abnormalities in the infant.

Due to the addictive properties of the nicotine in tobacco, the body builds up a tolerance to the drug. The toxic effects develop rapidly \& the damage to the body is cumulative. Long term effects increase the chances of lung cancer and other lung diseases such as emphysema, asthma, bronchiectasis, and lung abscesses.

### 1.4.2.2 Physiological effect of Nicotine that encourages people to smoke:

Absorption of cigarette smoke from the lung is rapid and complete, producing with each inhalation a high concentration arterial bolus of nicotine that reaches the brain within 10-16 seconds, faster than by intravenous injection. Nicotine has a distributional half life of 15-20
minutes and a terminal half life in blood of two hours. Smokers therefore experience a pattern of repetitive and transient high blood nicotine concentrations from each cigarette, with regular hourly cigarettes needed to maintain raised concentrations, and overnight blood levels dropping to close to those of non-smokers. Nicotine has pervasive effects on brain neurochemistry. It activates nicotinic acetylcholine receptors (nAchRs), which are widely distributed in the brain, and induces the release of dopamine. This effect is the same as that produced by other drugs of misuse (such as amphetamines and cocaine) and is thought to be a critical feature of brain addiction mechanisms. Nicotine is a psychomotor stimulant, and in new users it speeds simple reaction time and improves performance on tasks of sustained attention. However, tolerance to many of these effects soon develops, and chronic users probably do not continue to obtain absolute improvements in performance, cognitive processing, or mood. Smokers typically report that cigarettes calm them down when they are stressed and help them to concentrate and work more effectively, but little evidence exists that nicotine provides effective self medication for adverse mood states or for coping with stress.

### 1.4.2.3 Behavioral aspect toward smoking:

Experimenting with smoking usually occurs in the early teenage years and is driven predominantly by psychosocial motives. For a beginner, smoking a cigarette is a symbolic act conveying messages such as "I am no longer my mother's child," and "I am tough" \& they tend to come from backgrounds that favor smoking (for example, with high levels of smoking in parents and peers; schools where smoking is common). They also tend not to be succeeding according to their own or society's terms (for example,
they have low self esteem, have impaired psychological wellbeing, are overweight, or are poor achievers at school). The desired image is sufficient for the new smoker, but after that pharmacological factors assume much important. In adults also the link with nicotine addiction does not imply that pharmacological factors drive smoking behavior in a simple way and to the exclusion of other influences. Social, economic, personal, and political influences all play an important part in determining patterns of smoking, prevalence and cessation. Although drug effects courage the behavior, family and wider social influences are often critical in determining who starts smoking, who gives up, and who continues.

### 1.4.2.4 NICOTINE GENE \& SMOKING BEHAVIOR

Many of the forbidden pleasures of the modern day-nicotine, alcohol and over-eating- appear to be linked by common genetic factors, according to recent studies. Genetic variables appear to play a key role in every aspect of nicotine addiction, from the tendency to begin smoking, to the chances of quitting.

Evidence is now converging from behavioral studies, twin studies and molecular genetic research that provide a clearer understanding of the biobehavioral basis for nicotine dependence. Ultimately this should lead to the development of improved methods for assessment and treatment of dependence.

Behavioral scientists have made great progress in defining the genotype and carefully pointing out the variables that have to be taken into account in describing individual differences in smoking behaviors. Molecular biologists have made great progress in identifying an array of
nicotinic receptors, the genes involved and their locations, and other neurochemicals (particularly dopamine) that may be involved in regulation and activation of nicotine related behavior. They analyzed more than 20 studies of smoking behaviors in monozygotic and dizygotic twins. They found consistent evidence of genetic influences governing the developmental stages of smoking (initiation, maintenance, cessation), smoking intensity (light to heavy). Five years ago they reported the discovery of a gene that appeared to be associated with alcoholism, the D2 dopamine receptor gene (DRD2). Since that time they have conducted further studies implicating this gene in behaviors associated with tobacco, cocaine and obesity.

There are two main dopaminergic pathways in brain. The first begins in the area called the substantia nigra and is involved with movement. Defects in this part of the brain are associated with movement disorders such as Parkinson's disease. The second pathway, the mesolimbic dopamine system, is associated with emotion activation.
"When alcohol, nicotine, cocaine, or food is ingested, dopamine levels increase in this area. Therefore, we think these areas are associated with reward and pleasure.

The DRD2 gene is found on chromosome 11, in the q22-23 region. There are two alleles of interest, A1 found in $25 \%$ of the population and A2 found in $75 \%$. Studies comparing alcoholics to controls showed a significantly higher incidence of A1 allele. The A1 allele is associated with significantly reduced levels of D 2 dopamine receptors in the brain.
"This led us to hypothesize that individuals with the A1 allele may have an inherent deficit of the dopaminergic system. To compensate for that deficiency, they are high risk for using alcohol, and other substances which by releasing dopamine activate these areas."

These findings could point the way to useful therapies for those attempting to quit smoking or drinking. A recent study with bromocriptine, a drug that increases levels of DRD2, showed significant improvements in craving and anxiety among alcoholics trying to quit.
( Nature Medicine 1995)

### 1.5 What is Diabetes Mellitus?

Diabetes mellitus is a chronic and progressive disease which can affect people in all age groups and can cause ill health, disability and premature death. It is a heterogeneous disorder characterized by varying degrees of insulin resistance and insulin deficiency, which lead to a disturbance in glucose homeostasis. In the short term, uncontrolled diabetes is characterized by symptoms of high blood glucose levels (hyperglycemia). (Anjana Patel 2003)

### 1.5.1 Type2 diabetes mellitus:

Type2 diabetes is classified as non-insulin-dependent diabetes mellitus (NIDDM). (Francis S. Greenspan 2004)

This is generally characterized by peripheral insulin resistance and relative insulin deficiency which may range from predominant insulin resistance with relative insulin deficiency to predominant insulin secretory
defect with insulin resistance. Some patients develop severe insulin deficiency. Peripheral plasma insulin levels are usually high, with relative insulin deficiency being characterized by a delayed initial first-phase insulin response with the second-phase insulin response being weakened over several years. Resistance to the action of insulin takes the form of a decrease in the ability of skeletal muscle both to store glucose (due to a reduction in activity of the enzyme glycogen synthase ) and to oxidize glucose (due to a reduction in pyruvate dehydrogenase activity). There is also an increase in hepatic glucose output (HGO) due to inhibition of glycolysis and an increase in glucogenesis leading to chronic hyperglycaemia. Development of hyperglycaemia is a gradual process which frequently goes undiagnosed for many years due to an absence of any classic symptoms of diabetes during the early stages of the disease. (Anjana Patel 2003)

The risk of developing Type 2 diabetes increases with age, obesity (particularly central obesity), family history of diabetes or cardiovascular disease (particularly hypertension or dyslipidaemia), and lack of physical activity.(Anjana Patel 2003)

### 1.5.2 The Effect of Smoking on Type 2 Diabetes Mellitus:

Smoking may increase the risk of developing diabetes, according to new research by investigators at Wake Forest University School of Medicine and colleagues. They examined the relationship between smoking and diabetes among participants in a major national study, the Insulin Resistance Atherosclerosis Study (IRAS). They compared the incidence of diabetes after five years among smokers and those who had never smoked.

Twenty-five percent of the participants who smoked and did not have diabetes when the study began had developed diabetes by the fiveyear follow-up, compared to 14 percent of the participants who had never smoked. (Jjournal Diabetes Care 2005)

### 1.6 Hypertension:

Hypertension is not itself a disease but is a condition of consistently raised blood pressure above 'normal' that, if left untreated, carries a risk of increased morbidity and mortality from various cardiovascular diseases, including stroke and coronary heart disease and renal impairment (Susan Skankie 2001)

### 1.6.1 Definition:

The term 'blood pressure' refers to the pressure of blood against the blood vessel walls. It generally means arterial blood pressure as it is usually measured indirectly in the brachial artery just above the elbow using a mercury sphygmomanometer and is expressed in mmHg. Two measurements are made: systolic or maximum blood pressure: the pressure measured during ventricular contraction of the heart, and diastolic or minimum blood pressure: the pressure measured during ventricular dilatation. Blood pressure is therefore usually quoted as two figures, for example, $140 / 80 \mathrm{mmHg}, 140 \mathrm{mmHg}$ being the systolic blood pressure and 80 mmHg being the diastolic blood pressure. (Susan Skankie 2001)

### 1.6.2 Causes

In the majority of cases of hypertension (over 98\%) there is no immediately obvious underlying cause; such cases are referred to as
primary or essential hypertension. It is suspected that such primary or essential hypertension is multifactorial in origin, with various factors such as environmental influences, diet and body weight playing a role. Genetic factors are also thought to contribute since hypertension clusters in families.

In a small minority of patients (2-5\% of hypertensive), hypertension is due to an underlying disease, usually involving the kidneys or endocrine system, or may be due to the adverse effects of drugs. Such hypertension is referred to as secondary hypertension. Secondary hypertension may be suspected particularly in resistant or malignant hypertension. Effective treatments of the underlying condition can sometimes, but not necessarily, abolish the hypertension. (Susan Skankie 2001)

### 1.6.3 The effect of smoking on HTN:

Smoking can cause peripheral vascular disease as well as hardening of the arteries. This leads to an increase in heart rate, cardiac output, coronary blood flow \& a rise in blood pressure.

The most effective lifestyle measure to reduce overall cardiovascular risk is smoking cessation. Although stopping smoking has no effect on blood pressure, smoking multiplies the cardiovascular risk as much as twoto five fold.. Smoking was one factor related to the persistent excess coronary mortality in men with treated hypertension. Cardiovascular mortality and morbidity fall within a few months of stopping smoking. In particular there are large reductions in risk among those who quit before 35 years of age or middle age; life expectancy in these patients is typically equal to that of lifelong non-smokers. (Susan Skankie 2001)

## Chapter two

## Methodology

### 2.1 Methodology:

The current study aims at exploring the effect of cigarette smoking on the susceptibility to develop type 2 diabetes mellitus and hypertension among adult males in the refugee population in the northern area of West Bank. The objectives of the study were set to assist public health practitioners in developing health education program aiming at the prevention of smoking and reduce the risks for developing hypertension and diabetes.

### 2.1.1Subject of the study:

A total number of 221 male adults visiting the UNRWA clinics in the North West Bank were included in the study. Females were excluded as prevalence of smoking among females is very low and this would have affected the sample with a huge number of non-smoking individuals.

The cases were selected from patients visiting the clinic for routine examination or treatment and those who agreed to be included in the study.

### 2.1.2 Case definition:

A male adult in the age 40-50 years old attending UNRWA clinic in the period over four months from the $1^{\text {st }}$ of December 2005 to $30^{\text {th }}$ of march 2006, not known to have type 2 diabetes or hypertension.

### 2.1.3 Exclusion criteria:

- Younger male adults were excluded for the reason that type2 diabetes and hypertension occurrence would be low and would therefore negatively affect findings.
- Individuals older that 50 were also excluded as the natural prevalence of type 2 diabetes and hypertension might affect the findings and the exclusion of smoking as a risk factor.
- Individuals with already established diagnosis of diabetes and hypertension were excluded


### 2.1.4 Collection of cases:

Cases were collected from United Nations Relief and Works Agency for Palestine refugees in the North West Bank (UNRWA), Health division in the following clinics:

- Balata camp clinic, Al-Ayn camp clinic, Askar camp clinic, Tolkarm camp clinic, Jenin camp clinic, Qalqilya clinic, Al-faraa camp clinic, Nur-shams camp

There were coordination with UNRWA and permission obtained to conduct the study. There was some difficulties occurring during collecting data, that sample collection was for specific age group of male attendants \& number of the volunteers was small, so it took a relatively long time to collect the data. Also there were difficulties in communication with nurses in remote clinics due to difficulties in transport and military barriers.

### 2.2 Approaches to collect data:

The cases that made the screening tests visiting UNRWA clinics for routine examination or treatment volunteer to answer the questionnaire $\&$ make the tests needed for the research.

### 2.2.1. Random blood sugar test:

Random blood sugar tests for the cases were taken; cases didn't have to fast in this test, blood samples were taken regardless of eating or drinking. These blood test samples were drawn intravenously by professional lab technicians who work at UNRWA clinics \& blood glucose test was made for each sample, so if there was a high result of random blood sugar,
$140 \mathrm{mg} / \mathrm{dl}$ or higher fasting blood sugar was ordered and performed thereafter. And in this study pre diabetes stage is included.

Table (1) Classification of blood glucose levels

| Diagnosis | Fasting blood glucose | Random blood glucose |
| :--- | :--- | :--- |
| Normal | $100 \mathrm{mg} / \mathrm{dl}$ or less | $140 \mathrm{mg} / \mathrm{dl}$ or less |
| Pre-Diabetes | $100-125 \mathrm{mg} / \mathrm{dl}$ | $140-199 \mathrm{mg} / \mathrm{dl}$ |
| Diabetes | $126 \mathrm{mg} / \mathrm{dl}$ or higher | $200 \mathrm{mg} / \mathrm{dl}$ or higher |

To make fasting blood sugar test, samples had to fast at least eight hours, then a blood sample was drawn intravenously and examined for fasting blood glucose level. If the fasting blood glucose level result was $126 \mathrm{mg} / \mathrm{dL}$ or higher, the person was considered as diabetic.

Then the results were analyzed according to SPSS.

### 2.2.2 Blood pressure:

Blood pressure was examined with sphygmomanometer by UNRWA nurses trained up to the competency level and supervised by doctors to detect the blood pressure levels for each sample. Levels of BP is categorized according to both WHO/ISH guidelines \& cases classified as
hypertensive or non-hypertensive \& in this study pre hypertension is included. Then the results were analyzed according to SPSS.

Table (2) Classification of hypertension according to WHO

| Normal | Pre <br> hypertension | Grade 1/stage <br> I | Grade 2/ stage <br> 2 | Grade 3/ stage <br> 3 |
| :---: | :---: | :---: | :---: | :---: |
|  |  | •hypertension | hypertension | hypertension |
|  |  | (mild) | (moderate) | (severe) |
| $120 /$ | $120-139 /$ | $140-159 /$ | $160-179 /$ | $\uparrow 180 /$ |
| 80 | $80-89$ | $90-99$ | $100-109$ | $\uparrow 110$ |

## Chapter Three

## Results and discussion

### 3.1 Hypothesis examined in the study

In addition to providing a frequency and descriptive analysis of the main background and independent variables, the study will provide crosstabulation analysis to verify the level of statistical significance among different dependent and independent variables.

While it was possible to elaborate on high number of possible statistical relations due to richness of data, the present study focused on examining the effect of smoking and other lifestyle factors on the development of type 2 diabetes mellitus and hypertension.

The following relations were the main areas of interest in the analysis and will be those to consider in the results and discussion chapter:

1. The relation between smoking and type 2 diabetes mellitus.
2. The relation between smoking and Hypertension.
3. The relation between cigarettes no and HTN
4. The relation between cigarettes no \&type 2 diabetes mellitus
5. The relation between place of residence \& HTN.
6. The relation between BMI and HTN .
7. The relation between BMI and type 2 diabetes mellitus
8. The relation between practicing sport and HTN.
9. The relation between practicing sport and type 2 diabetes mellitus
10. The relation between family history and HTN.
11. The relation, between family history and type 2 diabetes mellitus

### 3.2. Description of the sample:

### 3.2.1 Geographic distribution of the sample

In this study 221 adult males in the age of 40-50 years old, visiting UNRWA clinics were enrolled in the study as shown in the table (1) which describes the geographic distribution of the sample.

Table (3) Geographic distribution of the sample

| Clinic name | Frequency | Percent |
| :--- | :---: | :---: |
| Faraa | 19 | 8.6 |
| Balata | 44 | 19.9 |
| Al ayn | 30 | 13.6 |
| Nur shams | 12 | 5.4 |
| Qalqilia | 25 | 11.3 |
| Jenin | 37 | 16.7 |
| Askar | 30 | 13.6 |
| Tulkarm | 24 | 10.9 |
| Total | 221 | 100.0 |

### 3.2.2 Study sample according to place of residence

The table (2) shows that the highest percent of cases live in refugee camps. Residents of refugee camps comprise $45.7 \%$ of the total sample, residents of cities form $29.4 \%$, and $25.9 \%$ of the study participants come from villages. Even with the distribution of the study participants to different residential locations, the status of being refugees is a common characteristic among those participants, this is because the UNRWA healthcare system is providing services to refugee population in Palestine
and the status of refugee is the precondition for entitlement to these services. As seen in the table (2), refugee population in Palestine is distributed among different residential areas (cities, villages and refugee camps).

Table (4) Distribution of study sample according to Place of residence

| Place of residence | Frequency | Percent |
| :--- | :---: | :---: |
| City | 65 | 29.4 |
| Village | 55 | 25.9 |
| Refugee camp | 101 | 45.7 |
| Total | 221 | 100 |

### 3.3 Description of results:

### 3.3.1 Smoking \& number of cigarettes

As shown in table (3) $55.5 \%$ of cases collected are smokers and $45.5 \%$ of the cases are not smoker.

Table (5) Distribution of study sample according to Number of cigarettes a day

|  | Frequency | Percent |
| :--- | :---: | :---: |
| 10 cigarettes and less per day | 24 | 10.9 |
| more than 10 cigarettes per day | 98 | 44.6 |
| Total | 122 | 54.5 |
| non smoker | 99 | 45.5 |
| Total | 221 | 100.0 |

We notice that the highest percent of cases smoke more than 10 cigarettes per day with 44.6 . $\%$ and $10.9 \%$ smoke 10 cigarettes and less per day, which indicates that most of the smokers are heavy ones.

### 3.3.2 The percent of HTN:

Table (6) Distribution of study sample according to SBP

| HTN | Frequency | Percent |
| :--- | :---: | :---: |
| 120 mmHg and less | 141 | 63.8 |
| more than 120 mmHg | 80 | 36.2 |
| Total | 221 | 100.0 |

We notice from table (4) that $63.8 \%$ of cases have normal systolic blood pressure and $36.2 \%$ have high SBP. This screening test for cases not known to have hypertension indicates that there is a high percentage of people, who have hypertension. This study provides baseline data on HTN in refugees in Palestine, information that is essential for the implementation of national planning and service provision.

Table (7) Distribution of study sample according to DBP

|  | Frequency | Percent |
| :--- | :---: | :---: |
| 80 mmHg and less(normal) | 170 | 76.9 |
| more than 80 mmHg | 51 | 23.1 |
| Total | 221 | 100.0 |

We notice from table above that most of these cases have normal DBP with $76.9 \%$, and the rest of cases have high DBP with $23.1 \%$.

### 3.3.3 The percent of type 2 Diabetes Mellitus:

Table (8) Distribution of study sample according to RBS

| RBS | Frequency | Percent |
| :--- | :---: | :---: |
| $140 \mathrm{mg} / \mathrm{dl}$ and less | 170 | 76.9 |
| more than140mg/dl | 29 | 13.1 |
| Total | 199 | 90.0 |
| Who had FBS test only | 22 | 10.0 |
| Total | 221 | 100.0 |

As shown in the table above, the percent of the normal RBS of (140 $\mathrm{mg} / \mathrm{dl}$ and less) is $76.9 \% \& 13.1 \%$ of the cases have high RBS (more than $140 \mathrm{mg} / \mathrm{dl})$.This screening test for cases not known to have diabetes shows that this study provides baseline data on diabetes mellitus in refugees in Palestine. The results indicate a high percentage of RBS in the study sample.

Table (9) Distribution of study sample according to FBS

| FBS | Frequency | Percent |
| :--- | :---: | :---: |
| $110 \mathrm{mg} / \mathrm{dl}$ and less(normal) | 23 | 10.4 |
| more than $110 \mathrm{mg} / \mathrm{dl}($ high $)$ | 8 | 3.6 |
| Total | 31 | 14.0 |
| RBS | 190 | 86.0 |
| Total | 221 | 100.0 |

From the table above $10.4 \%$ of the cases have normal FBS and $3.6 \%$ have high FBS.RBS was performed in $86 \%$ of the cases.

### 3.3.4 The percent of people practicing sport:

Table (10) Distribution of study sample according to practicing sport

| practicing sport | Frequency | Percent |
| :--- | :---: | :---: |
| No | 171 | 77.4 |
| Yes | 50 | 22.6 |
| Total | 221 | 100.0 |

The table above shows that $77.4 \%$ of the sample is not practicing sport and $22.6 \%$ do.

### 3.3.5 The percent of high BMI:

Obesity is an excess of body fat that frequently results in a significant impairment of health. Obesity is a known risk factor for chronic diseases including heart disease, diabetes, high blood pressure \& stroke. We notice from table (9) that $22.2 \%$ of the cases have normal BMI and $67.4 \%$ have high BMI, so the percent of overweight \& obese cases are very high. This is attributed to bad eating habits, bad life style, and lack of exercise.

Table (11) Distribution of study sample according to BMI

| BMI | Frequency | Percent |
| :--- | :---: | :---: |
| 25 and less(normal) | 49 | 22.2 |
| more than 25 (high) | 149 | 67.4 |
| Total | 198 | 89.6 |
| Missing | 23 | 10.4 |
| Total with missing | 221 | 100.0 |

### 3.4 Discussion of the Results Of The Hypothesis

3.4.1 Discussion of the result of the first Hypothesis: The relation between smoking and type 2 diabetes mellitus.

Table (12) The frequency and percentages of the smoking and RBS

|  |  | RBS |  | Total |
| :---: | :---: | :---: | :---: | :---: |
|  |  | $140 \mathrm{mg} / \mathrm{dl}$ <br> and less | more <br> than140mg/dl |  |
| smoking | No | Count of non smokers | 78 | 8 |

From the table above we notice that $90.7 \%$ of non smokers have normal RBS and $9.3 \%$ of them have high RBS and $81.4 \%$ of smoker have normal RBS and $18.6 \%$ of them, have high RBS. Since the level of significance is $(0.066)$ it is very near to $(0.05)$ which means that smoking is an independent risk factor for diabetes.This finding can be supported by (IRAS) study, as they compared the incidence of diabetes after five years among smokers and those who had never smoked.

Twenty-five percent of the participants who smoked and did not have diabetes when the study began had developed diabetes by the fiveyear follow-up, compared to 14 percent of the participants who had never smoked

Another study indicates that insulin-mediated glucose disposal of patients with type 2 diabetes was markedly lower (about 45\%) in smokers,
as compared with nonsmokers, cigarette smoking can acutely impair insulin action, and hyperinsulinemia and/or insulin resistance in non diabetic smokers. This supports the idea that cigarette smoking may adversely affect insulin sensitivity both in healthy subjects and in patients with type 2 diabetes. (The Journal of Clinical Endocrinology \& Metabolism, Vol. 82, No. 11 3619-3624).

And the bar chart below shows the frequency of the two variables: the smokers \& non smokers and RBS.

## Bar chart (1)


3.4.2 Discussion of the result of the second Hypothesis: The relation between smoking and Hypertension

Table (13) The frequency and percentages between smoking and SBP

| SBP | Normal SBP (120 <br> and less) | High SBP <br> (morethan120) | Total |
| :---: | :---: | :---: | :---: |
| Count of non smokers | 61 | 38 | 99 |
| \% of non smokers | $61.6 \%$ | $38.4 \%$ | $100.0 \%$ |
| Count of smokers | 80 | 42 | 122 |
| \% of smokers | $65.6 \%$ | $34.4 \%$ | $100.0 \%$ |
| Total Count | 141 | 80 | 221 |
| Total percent | $63.8 \%$ | $36.2 \%$ | $100.0 \%$ |

From the table above we notice that $61.6 \%$ of non smokers have normal systolic blood pressure \& 38.4\% of them have high SBP. 65.6\% of smokers have normal systolic blood pressure and $34.4 \%$ of them have high SBP. The P value is $(0.543)$ which is bigger than 0.05 . This indicates that in our study there was no effect of smoking found on HTN. But studies showed that smoking a cigarette raises the blood pressure by $5-10 \mathrm{~mm} \mathrm{Hg}$ for about 30 minutes. If this is combined with drinking a cup of coffee, the effects are bigger and last longer.

Despite this, numerous epidemiological studies have found that people with hypertension are not more likely to be smokers than those with normal blood pressure, and conversely, that smokers are not more likely to be hypertensive than non-smokers. One possible explanation for this might be that the blood pressure measurements are usually made when people are not smoking. If person smoke a pack a day, it will raise average daytime pressure by about 5 mm Hg , even though doctor may not detect this during an office visit.

The important thing about smoking is not what it does to blood pressure, but that it greatly increases risk of heart disease as cigarette smoking is a powerful risk factor that predisposes the smoker to CHD. (Tobacco Control 2005; 14:315-320).

Also genes play a role in hypertension according to Erasmus medical center, Rotterdam study, made by Anna Schut showed that smoking has been found to interact with polymorphisms of the angiotensin-converting enzyme (ACE) gene in a way that increases the risk of hypertension. The study showed that in smokers, SBP was higher in those carrying the DD
alleles as opposed to the II alleles. Furthermore, the risk of developing hypertension was significantly increased in smokers who carried a D allele.. (Journal of Medical Genetics. 2004; 22: 313-31).

In this study we don't know the genotype of Palestinian population.

### 3.4.3 Discussion of the result of the third Hypothesis:

The relation between cigarettes no. and HTN
Table (14) The frequency and percentages between cigarettes no. in a day and systolic blood pressure

|  |  |  | systolic bloo categ | od pressure rized |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | $\begin{gathered} 120 \text { and } \\ \text { less } \\ \text { (normal) } \end{gathered}$ | $\begin{gathered} \hline \text { more than } \\ 120 \\ \text { (high) } \\ \hline \end{gathered}$ | Total |
| $\begin{aligned} & \text { 옴. } \\ & 0, \\ & 0 \\ & 0 \\ & 0 \\ & 0 \\ & 0 \\ & 0 \\ & 0 \\ & 0 \\ & 0 \end{aligned}$ | 10 cigarettes and less per day | Count of cases who smoke 10 cigarettes and less per day | 14 | 10 | 24 |
|  |  | $\%$ of cases who smoke 10 cigarettes and less per day | 58.3\% | 41.7\% | 100\% |
|  | more than 10 <br> cigarettes per day | Count of cases who smoke more than 10 cigarettes | 66 | 32 | 98 |
|  |  | $\%$ of cases who smoke more than 10 cigarettes | 67.7\% | 32.3\% | $\begin{gathered} 100.0 \\ \% \end{gathered}$ |
| Total |  | Count | 80 | 42 | 122 |
|  |  | \% within cigarettes no | 65.8\% | 34.2\% | $\begin{gathered} 100.0 \\ \% \end{gathered}$ |

From the table above we notice that $58.3 \%$ of cases who smoke 10 cigarettes and less per day have normal systolic blood pressure, and 41.7\%
of them have high systolic blood pressure; and $67.7 \%$ of cases who smoke more than 10 cigarettes per day have normal SBP and $32.3 \%$ of them have high SBP. In this study it seems that there is no effect of cigarette no. on HTN. But larger studies on smokers have shown that the main health dangers from tobacco diseases are related to how much the person smokes.

The more someone smokes, the greater his or her risk of diseases. (Charles B. Clayman, MD 1994) Studies have also shown that there is no safe level of exposure to tobacco; someone who smokes occasionally is still at greater risk of disease than in a nonsmoker. Tobacco smoke is like nuclear radiation; although increased exposure leads to increased risk, any exposure carries a threat to health and life. (Tobacco Control 2005).
3.4.4 Discussion of the result of the forth hypothesis: The relation between cigarettes no \&type 2 diabetes mellitus

Table (15) the frequency and percentages of cigarettes no. smoked in a day \& RBS


From the table above we notice that $83.4 \%$ of cases who smoke 10 cigarettes and less have normal RBS and $16.6 \%$ of them have high RBS, and $71.4 \%$ of cases who smoke heavy more than 10 per day have normal RBS and $28.6 \%$ of them have high RBS so there is a trend that increasing the number of cigarettes increases the risk of type 2 diabetes mellitus.

### 3.4.5 Discussion of the result of the fifth hypothesis: The relation

 between place of residence $\&$ HTN.Table (16) The frequency and percentages of residence and systolic blood pressure

|  |  |  | $\begin{aligned} & \text { systol } \\ & \text { pre } \end{aligned}$ | c blood <br> sure | Total |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | 120 and less | more than $120$ |  |
| 0000000000000 | city | Count of cases lives in city | 49 | 16 | 65 |
|  |  | \% of cases lives in city | 75.4\% | 24.6\% | 100\% |
|  | village | Count of cases lives in village | 31 | 24 | 55 |
|  |  | \% of cases lives in village | 56.4\% | 43.6\% | 100\% |
|  | camp | Count of cases lives in camp | 61 | 40 | 100 |
|  |  | \% of cases lives in camp | 60.0\% | 40.0\% | 100\% |
|  | otal | Total Count | 141 | 80 | 221 |
|  |  | Total \% within residence | 63.6\% | 36.4\% | 100\% |

From the table above we notice that $75.4 \%$ of cases from city have normal blood pressure and $24.6 \%$ have high blood pressure; and $56.4 \%$ of cases from village have normal blood pressure, $\& 43.6 \%$ of them have high blood pressure; and $60 \%$ of those cases living in a camp have normal blood pressure and $40 \%$ of them have high blood pressure. The level of significance is $(0.058)$ near to 0.05 , So the city residents have the lowest $\%$
of hypertension then camp residents, and finally the village residents have the highest \% of HTN. This can be attributed to better income \& health education in cities than in villages $\&$ camps which lead to a healthier life style.
3.4.6 Discussion of the result of the sixth hypothesis: The relation between BMI and HTN.

Table (17) The frequency and percentages of BMI and systolic blood pressure

|  |  |  | systolic blood pressure categorized |  | Total |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | 120 mmHg and less | $\begin{array}{\|c} \hline \text { more than } \\ 120 \\ \mathrm{mmHg} \\ \hline \end{array}$ |  |
| $$ | $\begin{gathered} 25 \mathrm{~kg} / \mathrm{m} . \mathrm{m} \\ \text { and } \\ \text { less(normal) } \end{gathered}$ | Count normal BMI cases | 35 | 14 | 49 |
|  |  | \% of normal BMI cases | 71.4\% | 28.6\% | 100\% |
|  | more than 25 $\mathrm{kg} / \mathrm{m} . \mathrm{m}$ (high) | Count high BMI cases | 85 | 64 | 149 |
|  |  | \% of high BMI cases | 57.0\% | 43.0\% | 100\% |
| Total |  | Total Count | 120 | 78 | 198 |
|  |  | Total \% within BMI | 60.6\% | 39.4\% | 100\% |

From the table above we notice that $71.4 \%$ of cases who have normal BMI have normal SBP and $28.6 \%$ of cases who have normal BMI have high SBP; and $57 \%$ of cases who have high BMI have normal SBP and $43 \%$ of cases who have high BMI have high SBP. \& the level of significance ( 0.074 ) is near to 0.05 , so this indicates that increase in BMI increases the risk of HTN.

And the bar chart below shows the frequency of the two variables: the BMI and systolic blood pressure


## Bar chart (2)

BMI
3.4.7 Discussion of the result of the seventh hypothesis: The relation between BMI and type2 DM

Table (18) The frequency and percentages of BMI and RBS

|  |  |  | RBS |  | Total |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | $140 \mathrm{mg} / \mathrm{dl}$ and less | more than $140 \mathrm{mg} / \mathrm{dl}$ |  |
| 荡 | $\begin{gathered} 25 \text { and } \\ \text { less(normal) } \end{gathered}$ | Count normal BMI cases | 43 | 6 | 49 |
|  |  | \% normal BMI cases | 87.8\% | 12.2\% | 100\% |
|  | more than 25 | Count high BMI cases | 119 | 30 | 149 |
|  | high) | $\begin{aligned} & \text { \% high BMI } \\ & \text { cases } \end{aligned}$ | 79.9\% | 20.1\% | 100\% |
|  |  | Total Count | 153 | 26 | 198 |
|  | Total | Total \% within BMI | 85.5\% | 14.5\% | 100\% |

From the table above we notice that cases who have normal BMI $87.8 \%$ of them have normal RBS and $12.2 \%$ of them have high RBS; and
cases who have high BMI with normal RBS constitute $79.9 \%$ and $20.1 \%$ of cases who have high BMI have high RBS. So we can conclude that there is a trend indicating that increasing BMI increases the risk of having type 2 DM.

Obesity is common in people with type 2 diabetes and itself causes insulin resistance. Body fat distribution (around the waist) rather than obesity may have great effect on type2 diabetes. (Anjana Patel 2003)
3.4.8 Discussion of the result of the eighth hypothesis: The relation between practicing sport and HTN

Table (19) The frequency and percentages of practicing sport and SBP


From the table above we notice that $66.7 \%$ of cases who do not practice sport have normal SBP and $33.3 \%$ have high SBP; and from those who practice sport $54 \%$ have normal SBP and $46 \%$ have high SBP. As
shown in this study there is no effect of practicing sport on HTN, but studies do indicate that although acute exercise increases BP by an amount that depends on the degree of fitness $\&$ level of exertion, regular exercise 30-40 minutes aerobic exercise 3 times a week reduce BP , as reduced sympathetic nervous system activity may be responsible. Increasing exercise also contributes to weight loss. (Susan Skankie 2001)

### 3.4.9 Discussion of the result of the ninth hypothesis: The relation

 between practicing sport and type 2 DM .Table (20) The frequency and percentages of practicing sport and RBS


From the table above we notice that $83.9 \%$ of the cases who do not practice sport have normal RBS and $16.1 \%$ of them have high RBS; and $90.9 \%$ of the cases who practice sport have normal RBS and $9.1 \%$ of them have high RBS. So we can conclude that there is a trend indicating that practicing sport has a positive effect by reducing the likelihood of type 2

DM, which is supported by studies that show that exercise have an effect to decrease the risk of diabetes, as exercise generally decreases insulin resistance and reduces blood glucose concentrations. Also moderate, regular exercise reduce cardiovascular risk factors due to an improvement in blood lipids profile, blood pressure \& cardiovascular fitness \& enhancement of body weight loss in obese diabetic patients. (Anjana Patel 2003)

### 3.4.10 Discussion of the result of the tenth hypothesis: The relation

 between family history (FH) of HTN and DBP.Table (21) The frequency and percentages of the family history and DBP

|  |  |  | DBP |  | Total |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | 80 mmHg and less(normal) | more than 80 <br> mmHg (high) |  |
|  | no | Count of cases not having family history | 103 | 23 | 126 |
|  |  | \% of cases not having family history | 81.7\% | 18.3\% | 100\% |
| $\stackrel{\rightharpoonup}{\mathbf{c}}$ 0 0 0 0 0 | yes | Count of cases having family history | 67 | 28 | 95 |
| $\stackrel{\stackrel{\rightharpoonup}{e}}{\substack{0}}$ |  | \% of cases having family history | 70.5\% | 29.5\% | 100\% |
| Tot |  | Total Count | 170 | 51 | 221 |
|  |  | Total \% family history | 76.9\% | 23.1\% | 100\% |

FH plays a role in hypertension. From the table (19) above we notice that $81.7 \%$ of cases who have no family history of HTN have normal DBP
and $18.3 \%$ of them have high DBP; and $70.5 \%$ of cases who have family history have normal DBP and $29.5 \%$ have high DBP \& the level of significance equals 0.05 . So FH increases the risk of HTN, as hypertension tends to cluster within families. A very few rare forms of hypertension can be attributed to a single gene mutation. (Anjana Patel 2003) However, the majority of cases of high blood the pressure appear to be the result of an interaction of several genes with each other and with the environment. (Anjana Patel 2003)
3.4.11 Discussion of the result of the eleventh hypothesis: The relation between family history (FH) of DM and RBS

Table (22) The frequency and percentages of family history and RBS

|  |  |  | RBS |  | Total |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  | $140 \mathrm{mg} / \mathrm{dl}$ and less | $\begin{gathered} \text { more } \\ \text { than } 140 \mathrm{mg} / \mathrm{dl} \end{gathered}$ |  |
|  | no | Count of cases not have family history | 100 | 16 | 116 |
|  |  | $\%$ of cases not have family history | 86.2\% | 13.8\% | 100\% |
|  | yes | Count of cases have family history | 70 | 13 | 83 |
|  |  | \% of cases have family history | 84.3\% | 15.7\% | 100\% |
| Total |  | Count | 170 | 29 | 199 |
|  |  | \% family history. | 85.4\% | 14.6\% | 100\% |

The table above shows that $86.2 \%$ of the cases with negative FH have normal RBS and $13.8 \%$ of them have high RBS; and $84.3 \%$ of cases who have a family history have normal RBS and $15.7 \%$ of them have high RBS. There is no significant result among F.H \& type 2 diabetes mellitus,
although a small trend can be elicited. So FH plays a role in type 2 diabetes mellitus. But FH association with a strong genetic predisposition is higher in type 1 diabetes mellitus than type 2 diabetes mellitus (Charles B. Clayman, MD 1994)

## Chapter Four

## Conclusion \& Recommendations

### 4.1 Conclusion:

Cigarette smoking can increase the risk of type 2 DM \& there is no statistical evidence shows that there is a direct effect of smoking on hypertension. The important thing about smoking is not what it does to blood pressure, but that it greatly increases risk of heart disease as cigarette smoking is a powerful risk factor that predisposes the smoker to CHD. Also high BMI can increase the risk of HTN \& type 2 DM.

In this study practicing sport reduced the risk of type 2 DM , but had no positive effect on HTN, although studies show that moderate, regular exercise reduces cardiovascular risk factors due to an improvement in blood lipids profile, blood pressure \& cardiovascular fitness.

FH especially in first degree relatives can increase the risk of HTN. But genetic predisposition is low in type 2 diabetes mellitus in this study.

### 4.2 Recommendations:

1. Strategic health authorities, community pharmacies, local authorities and local community groups should make programs for smoking cessation policies and practices to reduce smoking phenomena in Palestine.
2. Public health efforts can be effective, mostly by creating the idea that smoking is no longer normal. This concept of denormalization is best instituted by laws and local regulations making smoking inaccessible in public places, raising prices, and putting stricter limitations on cigarette advertising.
3. Everyone who smokes should be advised to quit smoking in primary and secondary health care settings during visit for treatment or routine check up.
4. Nurses in primary and community care should advise everyone who smokes to stop smoking \& all other health professionals, such as hospital clinicians, pharmacists and dentists, community workers, should aware people to the risk of smoking, offer a pharmacotherapy and behavioral support under the supervision of special medical group.
5. Healthy life style must be followed like practicing exercises, eating healthy diet, decreasing overweight \& the health education about diseases $\&$ how to prevent them is necessary.

## References:

Anjana Patel. (2003). Diabetes in focus [2 $2^{\text {nd }}$ edition]. pharmaceutical press 2001 Great Britain by TJ international, Padstow, Cornwall.

Susan Skankie. (2001). Hypertension in focus [2 ${ }^{\text {nd }}$ edition]. pharmaceutical press. Great Britain by TJ international, Padstow, Cornwall.

Braunwald, Isselbacher, Petersdrof, Wilson Martin, Fauci. (1987). $\underline{\text { Harrison's Principles of internal Medicin [11 }{ }^{\text {th }} \text { edition]. London }}$

Charles B. Clayman, MD, (1994). The American Medical Association Family Medical Giude [3 ${ }^{\text {rd }}$ edition]. U.S.

Heather Ashton, Rob Stepney, Tavistock Publications. Smoking psychology pharmacology. London \& New York.

James E. Robbers, Ph.D., LAVlariIyn K. Speedie, Ph.D. L,-Varro E. Tyler, Ph.D., Sc.D. Lilly. (1996). Pharmacocnosy and Pharmacobiotechnology. Williams \& Wilkans. US.

Francis S.Greenspan, MD, FACP, David G. Gardner, MD. (2004).Basic \& Clinical Endocrinology [7 $7^{\text {th }}$ edition]. A Lange medical book Medical publishing Division. McGraw_Haill Companis. New York.

The Journal of Clinical Endocrinology \& Metabolism Vol. 82, No. 11 3619-1997 by The Endocrine Society.

Anna Schut (2004). Angiotensin Converting Enzyme gene polymorphism and smoking the Rotterdam Study. Journal of Medical Genetics. 2004; 22: 313-31.

Tobacco Control (2005); 14: 315-320; doi: 10.1136/ tc. 2005. 011932 2005 BMJ Publishing Group Ltd.

Institute of Community and Public Health, Birzeit University, West Bank, Palestine. Journal East Mediterr Health. (2000) Sep-Nov; 6(5-6): 1039-45).

Institute of Community and Public Health, Birzeit University, Palestine. The Metabolic Syndrome in the West Bank Population: An urbanrural comparison. Journal Diabetes Care (2001); 24, 2:275-279.

Nature Medicine, 4/4/95, Noble.; Science, 4/26/95, Wehner et al.
Primary Prevention of Coronary Heart Disease: November 25, 2005 Guidance From Framingham: 1998 71-0139 Circulation. 1998; 97: 1876-1887.

Dr. Ernest Noble, Gary Swann. (1995). Nicotine Gene \& smoking behavior.http://www.accessexcellence.org/WN/SUA01/nicotine.html

S Capri G. Foy, Ph.D (2006), Smoking may increase the risk of developing diabetes. http://www.hmnews.org/article2618.html

Journal of the American Heart Association (2004) today's Hypertension http://www.americanheart.org/presenter.jhtml?identifier=3024254

Mrs. M. Grieve (1995). A modern herbal tobacco http://www.botanical.com/botanical/mgmh/t/tobcco.html

استبيان عن أثر التذخين على زيادة مخاطر الاصابة بالضغطو السكر من النوع الثاني

$$
\begin{aligned}
& \text { التاريخ: } \\
& \text { اسم العياده: } \\
& \text { رقم الملف الطبي: } \\
& \text { 1. العمر: } \\
& \text { 2. الطول: } \\
& \text { 3. الوزن: } \\
& \text { 4. :المهنة } \\
& \text { 5. مكان السكن : أ. مدينة } \\
& \text { 6. هل أنت مدخن : نعم } \\
& \text { 7. :عدد السجائر التي تدخنها يوميا } \\
& \text { 8. :عدد سنو ات التنخين } \\
& \text { 9. هل تمارس الرياضة : نعم } \\
& \text { نو عها: } \\
& 10 \text { :كم ساعة تمارس الرياضة اسبو عيا } \\
& 11 \text { :هل يعاني احد من افر اد أسرتك (أب, ام, أخ, أخت) من مرض } \\
& \text { 1-الضغط: } \\
& \text { 2-السكري: }
\end{aligned}
$$

12 هل تعاني من اي مرض حاليا: مثال (رشح ، انفلونزا..... إلخ) مـا .................................... هو 13 هل تستعطل اي نوع من العلاج حاليا:..........................

BP result: $\qquad$

RBS: $\qquad$

FBS:

## جامعة النجاح الوطنية

 كلية الاراسات (لعليا
# أثر التدخين على زيـادة قابلية الإصابة بمرض ضغط الام المرتفع و اللسكري من النوع الثاني 

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قدمت هذه الأطروحة اسنكمالاً لمتطلبات درجة الماجستير في الصحة العامة بكليــة الار اســـات العليا في جامعة النجاح الوطنية في نابلس، فلسطين. 2006

أثر التّخين على زيادة قابلية الإصابة بمرض ضنظ الام المرتفع والسكري من النوع الثاني

إعداد
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د. سليمـان خليل
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## الملخص

تهدف الدر اسة الجارية إلى دراسة اثز التدخين على زيادة القابلية لحدوث مرض ارتفاع ضغط الام و السكر من النوع الثاني.

جمعت الحالات من الرجال في الفئة العمرية من 40-50 سنة من اللاجئين الفلسطينيين
في منطقة شمال الضفة الغربية من المر اجعين في عيادات وكالة الغوث الدولية ، و عمل مســـح
لمرض الضغط و السكر للمتطو عين اللذين لم تعرف إصابتهم بالضغط و السكر من قبل.
وفي هذه الدر اسة وجد أن التخخين يزيد من مخاطر الإصـابة بمرض السكر من النــوع ع الثاني، ولم يظهر إحصائيا اثر التـخين على زيادة مخاطر الإصـابة بمرض ارتفاع ضغط الام.

كهـا ووجد أن الزيادة في الوزن تزيد من مخاطر الإصـابة بمرض ارتفاع ضـــغط الــــــم و السكر من النوع الثاني

ولم يظهر تأثنثر ممارسة الرياضة إحصـائيا على النقليل من مخاطر الإصـــابة بمــرض السكر من النوع الثاني. ولكن أثبتت الار اسات الحديثة أن الرياضة نساهم في النقلبل من مخاطر الإصـابة بارنفاع ضغط الدم.

ووجد أن الور اثة تزيد من احتمالية الإصابة بمرض ارتفاع الضغط و السكر من النــوع

