EFFECT OF SMOKING ON CARBOHYDRATE METABOLISM

THESIS
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BY

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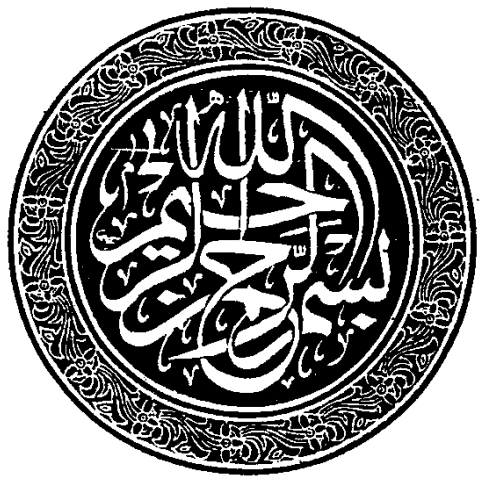

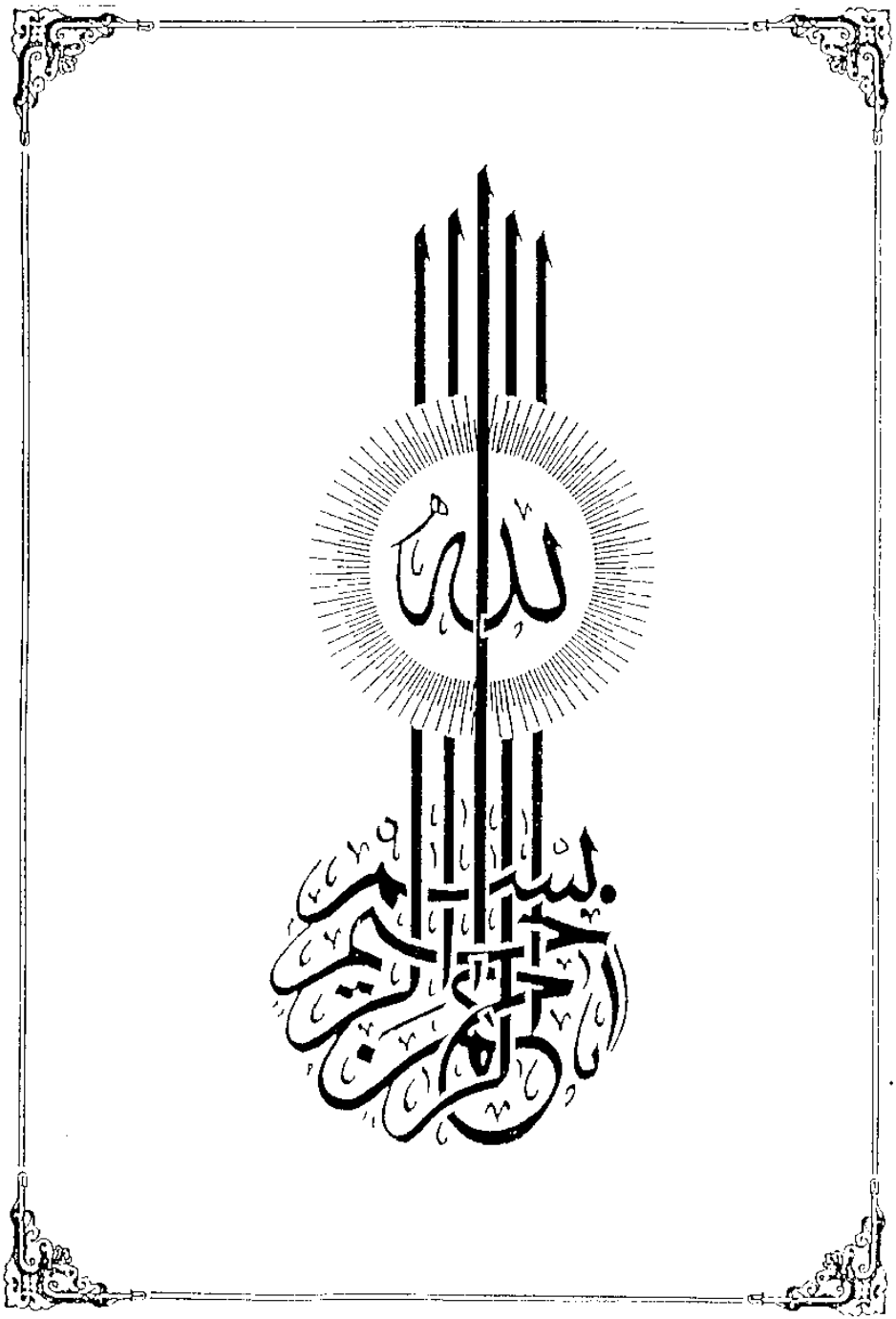

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INTRODUCTION & AIM OF THE WORK

Introduction and aim of work

Nicotine exerts a sympathomimetic effect by facilitating transmission across sympathetic ganglia, adrenal medullary stimulation and release of norepinephrine from chromaffin tissues (Meyers et al., 1970).

Epinephrine stimulates the breakdown of glycogen into glucose with the result of an increase in blood glucose level (glycogenolysis) through cyclic adenosine monophosphate. (CAMP). (Haugard and Hess, 1965).

Again catecholamines has a direct inhibitory effect on insulin secrection through alpha receptor predominance (Porte, 1967).

The aim of the present work is to study the effect of smoking on carbohydrate metabolism. 20 insulin dependant patients, 20 non insulin dependant patients and 20 non diebetic healthy persons, half number of each group are smokers and the others are not smokers. In all groups blood glucose levels will be estimated at fasting, 5, 10, 30 and 60 minutes also C peptide levels will be estimated at fasting, 30 and 60 minutes these procedures will be done for smoker and non smoker candidates.

REVIEW OF LITERATURE

Tobacco smoking

Cigarette smoke is aheterogenous aerosol produced by incomplete combustion of the tobacco leaf. It is composed of gases and vapour in which droplets are dispersed. Main stream smoke emerges from the mouth piece during puffing while side-stream smoke is emitted between puffs at the burning cone as well as from mouth piece. the composition of smoke is influenced by several factors including type of tobacco, temperature of combustion, length of cegarette, porosity of paper, additives and filters. the major constituents of tobacco are carbohydrate, non organic fatty acid, nitrogen containing compounds and resin.

Cigarette temperature vary greaty from $30^{o}c$ at mouth piece to $900^{o}c$ at the burning cone (Holbrook- 1980).

Volatile susbances are distilled directly into the smoke. In the presence of intense heat some tobacco constituents undergo thermic decomposition (pyrolysis). Unstable molecules recombine again to generate new compounds (pyrosynthesis). Some substances found in tobacco pass unchanged into cigarette smoke.each cigarette generates approximately 500 mg mainstream smoke of which

92% is present in the gas phase and 8% in a particulate phase. Main stream smoke contain 2-5 bilion particles per milliliter with particle size ranging from 0.1 to 1.0 um, Nitrogen, Oxygen and Carbon dioxide account for 85% of the smoke weight. The remaining gases, vapours and particulate matter are the substances of medical importance. some smoke constituents are absorbed directly through the mucosa of the mouth, nose, pharynx and upper air ways while others are inhaled into the lungs and undergo absorption (Schmeltz and Hoffmann. 1976).

Selected cigarette smoke constituents (Holbrook. 1980)

Particulate phase:

. Tss	carcinogenic
. Polynucleararomatic hydro carbon	,,
. N nitroso- nor- nicotine	,,
. B- naphthylamine	,,
. Cresol	,, & irritant
. Phenol	,, ,,
. Nicotine	ganglion stimulant& depressor
. Indol	tumour accelerator
. Carbazol	,, ,,

Gas phase:

. Carbon monoxide	impair O_2 transport& utilization
. Hydrocyanic acid	cilio toxin & irritant
. A cetaldehyde & Acrolein.	,, ,,
. Ammonia & formaldehyde.	,, ,,
. Oxides & nitrogen	,, ,,
. Nitrosamine	carcinogenic

Nicotine

Nicotine is an alkaloid isolated from tobacco. The free base is a clear liquid that become brown upon exposure to air (Meyer et al, 1978).

Nicotine is well absorbed from all body surfaces. It exerts a sympathomimetic effect by facilitating transmission across sympathetic ganglia. a drenal medullary stimulation and release of norepinephrine from chromasffin tissues in the cardic atria and arterioles para sympathetic effect or muscarinic action due to stimulation of para sympathetic ganglia occur espicially on the gastro intestinal tract and eye. Acute central nervous system effect are mild central stimulation followed by depression and may be convulsions (Meyer et al, 1970).

Nicotine in large dose causes paralysis of ganglionic transmission and of voluntary muscles.

Nicotine is able to stimulate the release of anti diuretic hormone from posterior pitutary gland., also nicotine is very active in stimulating the carotid sinus chemoreceptor and the initial actions on blood pressure and respiration are due to this effect (Cryer et al,1976).

Smoking and catecholamine

Cigarette smoking is associated with the release of sympathetic neurotransmittor norepinephrine as well as the adrenal medullary hormone epinephrine.

So the smoking associated sympathetic dischange is physiologically reflected and there is increase in the mean plasma norepinephrine and epinephrine. (Cryer et al, 1976).

It has been found that injection of nicotine into laboratory animals increase urinary catecholamine excretion (Westfall et al, 1971).

On the other hand it has been reported by Auge (1974) that there is increase in urinary excretion of epinephrine during smoking of nicotine free cigarette.

Smoking is associated with significant inrement in the mean pulse rate, systolic and diastolic blood pressure, glycerol and lactate pyruvate ratio. This effect is due to catecholamine release induced by smoking.

On contrary smoking is also associated with significant increment in the plasma growth hormone and cortisal level which is not catecholamine mediation because not prevented by infusion formed of mixed alpha and Beta blockade (Cryer et al, 1976).

Cigarette smoking has often been found to increase the corticosteroid level which is considered persumptive evidence that there is an increase in adreno cortico trophic hormone (ACTH) production. This elevated ACTH level is associated with elevated level of B endorphin which may be responsible for the continuation of smoking habit. So that endrophin system if involved may also act as an intermediary in the amount of nicotine consumed. (Karras et al, 1980).(Cherek et al, 1982).

The high contisal level give an idea about diseases associated with smoking espicially C.V.S due to its sensitising action on the heart to catecholamine (Pomer leau et al 1981), (Hill et al, 1974).,elevated plasma catecholamine levels exacerbate platlet thrombus with reduction of blood flow. (Folts et al, 1980).

Smoking and canboxy haemoglobin

Cigarette smoke contains 2-6% of carbon monoxide which is toxic gas that interferes with oxygen transport and utilisation.

Smokers inhale concentration as high as 400 parts per million (ppm) and develop elevated carboxyhaemoglobin. (COHB).

The range of COHb for smoker is 2-15% and for non smoker is near 1% but in moderate smoker is 5%. Carbon monoxide produces its effect by reducing the amount of available oxyhemoglobin and myoglobin and displacing oxygen dissociation curve to the left.

Chronic mild elevation of COHb due to smoking is a common cause of polycythemia and may produce impairment of the CNS. (Holbrook 1980).