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What a Lower Fasting Plasma Glucose in Smokers

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Abstract

Background: We tried to understand whether or not there is a significant relationship between smoking and fasting plasma glucose (FPG).

Methods: Current daily smokers at least for the last six months and age and sex-matched non-smokers were included into the study. Patients with current alcohol consumption (one drink a day) and patients with malignancies or inflammatory, infectious, or devastating disorders were excluded from the study.

Results: The study included 247 smokers (74 females) and 167 non-smokers. The mean age of smokers was 46.2 years, and just 29.9% of them were female. Although the mean body weight, body mass index, systolic and diastolic blood pressures, and hematocrit values were similar in both groups, FPG was lower in the smokers, significantly (102.3 versus 111.6mg/dL, p=0.007). Similarly, high density lipoproteins (HDL) were lower in the smokers, again (40.9 versus 44.0mg/dL, p<0.05). On the other hand, triglycerides (163.1 versus 151.3mg/dL, p<0.05) and low-density lipoproteins (LDL) (123.8 versus 117.5mg/dL, p<0.05) were higher in the smokers, significantly. Parallel to the triglycerides and LDL, erythrocyte sedimentation rate (ESR) (10.6 versus 9.3mm/h, p<0.05) and C-reactive protein (CRP) (2.3 versus 2.0mg/L, p<0.05) values were also higher in the smokers, significantly.

Conclusion: Smoking-induced low-grade inflammation on the vascular endothelium all over the body may terminate with the endothelial dysfunction, accelerated atherosclerosis, end-organ insufficiencies, early aging, and premature death. FPG and HDL may be negative whereas triglycerides, LDL, ESR, and CRP may be positive acute phase reactants indicating the systemic inflammatory effects of smoking.

Keywords: Smoking; Fasting plasma glucose; High density lipoproteins; Triglycerides; Low density lipoproteins; Erythrocyte sedimentation rate; C-reactive protein

Abbreviations: BP: Blood Pressures; HT: Hypertension; DM: Diabetes Mellitus; PAD: Peripheric Artery Disease; COPD: Chronic Obstructive Pulmonary Disease; CHD: Coronary Heart Disease; CRD: Chronic Renal Disease; FPG: Fasting Plasma Glucose; ESR: Erythrocyte Sedimentation Rate; CRP: C-reactive Protein; LDL: Low Density Lipoproteins; HDL: High Density Lipoproteins; BMI: Body Mass Index; NT: Normotension; TNF: Tumor Necrosis Factor; ATP: Adult Treatment Panel; AUD: Alcohol Use Disorder; NAD: Nicotinamide Adenine Dinucleotide; APR: Acute Phase Reactants; VLDL: Very low-density lipoproteins; IDL: Intermediate Density Lipoproteins

Introduction

The monolayer of endothelial cells that forms the inner cellular lining of arteries, veins, capillaries, and lymphatics is called as the endothelium in the whole body. Probably, the endothelium all over the body may act as a separate organ that may be the largest organ of the body. It may contract vasculature of the peripheral organs while relaxing vasculature of the internal organs during cold, anxiety, depression, cancer, infection, or inflammation-like stresses of the body. Because we measure the systolic and diastolic blood pressures (BP) of the arms and legs, they may not show the actual BP of the brain, heart, lung, liver, and kidney-like internal organs. The whole endothelium may be the major player in the control of blood fluidity, platelets aggregation, and vascular tone all over the body. It may also be the main actor in the immunology, inflammation, angiogenesis, and endocrinology. The endothelium may control vascular tone and blood flow by releasing nitric oxide, arachidonic acid metabolites, and reactive oxygen species into the circulation. It may also be important for synthesizing of the vasoactive hormones such as angiotensin II. An endothelial dysfunction-induced accelerated

atherosclerosis in the whole body may be the main cause of end-organ insufficiencies, aging, and death. Such a dysfunction may also be important in the development of cancers by preventing clearance of malignant cells by the natural killer cells in terminal points of the circulation. Similarly, physical inactivity, animal-rich diet, excess weight, high BP and glucose levels, chronic inflammations, prolonged infections, cancers, smoking, and alcohol may be the accelerating factors of chronic endothelial inflammation and dysfunction terminating with an accelerated atherosclerosis-induced end-organ insufficiencies in the whole body [1]. The chronic endothelial damage, inflammation, and dysfunction may also be the main cause of aging in the human being [2]. The much higher BP of the afferent vasculature may be the major accelerating factor by inducing recurrent injuries on the vascular endothelium. Probably, whole afferent vasculature including capillaries are mainly involved in the process. Therefore, the term of venosclerosis is not as famous as atherosclerosis in the literature. Due to the chronic endothelial damage, inflammation, edema, fibrosis, and dysfunction, vascular walls thicken, their lumens narrow, and they lose their elastic natures, those eventually reduce blood flow to the terminal organs, and increase systolic and decrease diastolic BP further. Some of the terminal endpoints of the systemic inflammatory process are obesity, hypertension (HT), diabetes mellitus (DM), cirrhosis, peripheric artery disease (PAD), chronic obstructive pulmonary disease (COPD), coronary heart disease (CHD), chronic renal disease (CRD), mesenteric ischemia, osteoporosis, stroke, dementia, other end-organ insufficiencies, aging, and death [3]. Early withdrawal of the accelerating factors may delay terminal consequences. But after development of the terminal endpoints, endothelial changes cannot be reversed due to their fibrotic natures, completely. The accelerating factors and terminal endpoints are researched under the titles of the metabolic syndrome, aging syndrome, and accelerated endothelial damage syndrome in the literature, extensively [4,5]. As some of the well-known components of the syndrome, there may be a relationship between smoking and fasting plasma glucose (FPG) in the human body.

Material and Methods

The study was performed in the Internal Medicine Polyclinic of the Dumlupinar University between August 2005 and March 2007. Current daily smokers at least for the last six months were studied. Patients with current alcohol consumption (one drink a day) and patients with inflammatory, infectious, or devastating disorders including eating disorders, malignancies, acute or chronic renal failure, cirrhosis, COPD, hyper- or hypothyroidism, or heart failure were excluded from the study. A routine checkup procedure including hemogram, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), FPG, triglycerides, low density lipoproteins (LDL), high density lipoproteins (HDL), albumin, creatinine, thyroid function tests, hepatic function tests, markers of hepatitis A, B, C, and human immunodeficiency viruses, a urinalysis, a posterior-anterior chest x-ray graph, and an electrocardiogram was performed. An additional Doppler echocardiogram and/or abdominal ultrasonography were performed just in case of a need. Body mass index (BMI) of each individual was calculated by measurements of the Same Physician instead of verbal expressions. Weight in kilograms is divided by height in meters squared [6]. Office BPs were checked after a 5-minute of rest in seated position with mercury sphygmomanometer. Eventually, all smokers were collected into the first, and age and sex-matched non-smokers were collected into the second groups. The mean body weight, BMI, systolic and diastolic BP, triglycerides, LDL, HDL, FPG, ESR, CRP, and hematocrit values were detected in each group, and compared in between. Mann-Whitney U test, Independent-Samples T test, and comparison of proportions were used as the methods of statistical analyses.

Results

The study included 247 smokers (74 females and 173 males) and 167 (55 females and 112 males) non-smokers. The mean age of smokers was 46.2 years, and just 29.9% of them were female. Although the mean body weight, BMI, systolic and diastolic BP, and hematocrit values were similar in both groups, FPG was lower in the smokers, significantly (102.3 versus 111.6mg/dL, p=0.007). Similarly, HDL were lower in the smokers, again (40.9 versus 44.0mg/dL, p<0.05). On the other hand, triglycerides (163.1 versus 151.3mg/dL, p<0.05) and LDL (123.8 versus 117.5mg/dL, p<0.05) were higher in the smokers, significantly. Parallel to the triglycerides and LDL, ESR (10.6 versus 9.3mm/h, p<0.05) and CRP (2.3 versus 2.0 mg/L, p<0.05) values were higher in the smokers, again (Table 1).

Table 1: Comparison of smokers and non-smokers.

Variables	Smokers	P-Value	Non-Smokers
Number	247		167
Female ratio	29.90%	Ns*	32.90%
Mean age (year)	46.2±13.4 (19-76)	Ns	44.8±15.7 (13-77)
Weight (kg)	76.1±13.8 (44-118)	Ns	74.7±13.0 (45-122)
BMI† (kg/m^2)	26.6±4.4 (16.2-39.4)	Ns	26.5±4.5 (16.6-41.1)
Systolic BP‡ (mmHg)	127.5±23.7 (80-200)	Ns	130.0±22.6 (80-200)
Diastolic BP (mmHg)	88.0±12.4 (60-130)	Ns	88.5±11.9 (60-130)
Hematocrit (%)	41.9±4.6 (28-60)	Ns	41.0±3.7 (31-49)

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FPG§ (mg/dL)	102.3±25.5 (70-309)	0.007	111.6±37.6 (74-327)
HDL‖ (mg/dL)	40.9±9.6 (26-70)	<0.05	44.0±9.4 (24-70)
Triglycerides (mg/dL)	163.1±101.4 (40-585)	<0.05	151.3±86.2 (20-410)
LDL** (mg/dL)	123.8±34.3 (10-282)	<0.05	117.5±29.0 (43-185)
ESR*** (mm/h)	10.6±10.2 (1-51)	<0.05	9.3±8.0 (1-35)
CRP**** (mg/L)	2.3±2.6 (0-13)	<0.05	2.0±2.5 (0-12)

*Nonsignificant (p>0.05) †Body Mass Index ‡Blood Pressures §Fasting Plasma Glucose ‖High Density Lipoproteins **Low Density Lipoproteins ***Erythrocyte Sedimentation Rate ****C-Reactive Protein.

Discussion

Obesity may be one of the terminal consequences of the metabolic syndrome. Although some transient successes can be achieved, nonpharmaceutical approaches provide limited benefit to reverse the obesity, permanently. Due to the excess weight-induced chronic low-grade inflammation of the vascular endothelium all over the body, the risk of death from all causes including cardiovascular diseases and cancers increases parallel to the range of excess weight in all age groups [7]. The chronic low-grade inflammation may even cause genetic changes of the endothelial cells, and the systemic atherosclerosis may prevent clearance of malignant cells via the natural killer cells, effectively. Similarly, the effects of excess weight on the BP were shown in the literature, extensively [8]. For example, prevalence of sustained normotension (NT) was higher in the underweight than the normal weight (80.3% versus 64.0%, p<0.05) and overweight groups (80.3% versus 31.5%, p<0.001) [8], and 52.8% of patients with HT had obesity against 14.5% of patients with the sustained NT (p<0.001) [9]. So, the major underlying cause of the metabolic syndrome appears as weight gain that may be the main cause of insulin resistance, hyperlipoproteinemia's, impaired fasting glucose, impaired glucose tolerance, and white coat hypertension (WCH) [10]. Interestingly, weight gain before the development of an obvious overweight or obesity may even cause development of several components of the metabolic syndrome. For instance, WCH alone may be a strong indicator of weight gain even before development of excess weight [8,9]. On the other hand, prevention of the weight gain with physical activity even in the absence of a prominent weight loss usually results with resolution of many parameters of the syndrome [11]. According to our experiences, excess weight may actually be a result of physical inactivity instead of an excessive eating habit. In another words, there is a problem with burning of calories instead of getting them. Thus, prevention of weight gain cannot be achieved by diet, alone [12]. On the other hand, limitation of excess weight as an excessive fat tissue around abdomen under the heading of abdominal obesity may be meaningless, instead it should be defined as overweight or obesity via the BMI. Because adipocytes function as an endocrine organ, and they produce leptin, tumor necrosis factor (TNF)-alpha, plasminogen activator inhibitor-1, and adiponectin-like cytokines into the plasma [13]. Eventual hyperactivities of sympathetic nervous system and renin-angiotensin-aldosterone system are probably associated with insulin resistance, elevated BP, and chronic endothelial inflammation and dysfunction. Similarly, the Adult Treatment Panel (ATP) III reported that although some people classified actually as overweight with larger muscular masses, most of them also have excessive fat tissue predisposing to the terminal consequences of the syndrome [6].

Smoking may be the second common cause of disseminated vasculitis all over the body. It is one of the major risk factors for the atherosclerotic end-organ insufficiencies [14]. Its atherosclerotic effect is the most obviously seen in Buerger's disease. Buerger's disease is an obliterative vasculitis characterized by inflammatory changes in the small and medium-sized arteries and veins, and it has never been reported in the absence of smoking in the literature. Besides the obvious atherosclerotic effects of smoking, some studies reported that smoking in human being and nicotine administration in animals are associated with the lower BMI values [15]. Some evidence revealed an increased energy expenditure during smoking both on the rest and light physical activity [16]. Nicotine supplied by patch after smoking cessation decreased caloric intake in a dose-related manner [17]. According to an animal study, nicotine may lengthen inter mealtime, and decrease amount of meal eaten [18]. Similarly, the BMI seems to be the highest in the former, the lowest in the current, and medium in never smokers [19]. Smoking may be associated with a post cessation weight gain, but the risk is the highest during the first year after quitting and decreases with the following years [20]. As the opposite findings to the above studies, the mean body weight and BMI were similar both in the smokers and non-smokers in the present study (p>0.05 for both). Similarly, prevalence's of smoking were similar in the normal weight (35.9%), overweight (32.9%), and obesity groups (33.7%, p>0.05 between all) in the other study [21]. On the other hand, although the CHD was detected with similar prevalence's in both genders, prevalence's of smoking and COPD were higher in males against the higher BMI, LDL, triglycerides, WCH, HT, and DM in females [22]. Beside that the incidence of myocardial infarctions is increased six-fold in women and three-fold in men who smoked at least 20 cigarettes per day [23]. In another words, smoking may be more dangerous for women about the atherosclerotic consequences probably due to the higher BMI and its consequences in them. Similar to the present study, smoking is consistently higher in men in the literature [14]. Several toxic substances found in the cigarette smoke get into

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the circulation and cause a vascular endothelial inflammation in all organ systems of the body. For example, smoking is usually associated with depression, irritable bowel syndrome (IBS), chronic gastritis, hemorrhoids, and urolithiasis in the literature [24-26]. There may be several underlying mechanisms to explain these associations in the smokers [24]. First of all, smoking may have some antidepressant properties with several side effects. Secondly, smoking-induced vascular endothelial inflammation may disturb epithelial functions for absorption and excretion in the gastrointestinal and genitourinary tracts. These functional problems may terminate with urolithiasis and components of the IBS including loose stool, diarrhea, and constipation. Thirdly, diarrheal losses-induced urinary changes may even cause urolithiasis [25,26]. Fourthly, smoking-induced sympathetic nervous system activation may cause motility problems in the gastrointestinal and genitourinary tracts terminating with the IBS and urolithiasis. Eventually, immunosuppression secondary to smoking-induced vascular endothelial inflammation may even terminate with the gastrointestinal and genitourinary tract infections causing loose stool, diarrhea, and urolithiasis, because some types of bacteria can provoke urinary supersaturation, and modify the environment to form crystal deposits in the urine. Actually, 10% of urinary stones are struvite stones which are built by magnesium ammonium phosphate produced during infections with bacteria producing urease. Parallel to the results above, urolithiasis was detected in 17.9% of cases with the IBS and 11.6% of cases without in the other study ($p<0.01$) [25].

Alcohol may be the third common cause of disseminated vasculitis all over the body. Alcohol is addictive to humans, and can result in alcohol use disorder (AUD), dependence, and withdrawal. Alcohol is the only drug that mostly damage the other individuals. It is causally associated with more than 200 different pathologies [27]. Eventually, people hospitalized with AUD have an average life expectancy of 47-53 years in men and 50-58 years in women and die 24-28 years earlier than the others [28]. People with AUD have three-fold higher mortality in men and four-fold higher mortality in women [29]. Similar to the smoking, alcohol may be more dangerous for women about the atherosclerotic consequences probably due to their smaller body weight, lower capacity to metabolize alcohol, and higher proportion of fat in their body. A very substantial part of the Danish excess mortality and lower life expectancy compared to Sweden can be attributed to higher mortality related to alcohol and smoking [28]. Alcohol is one of the main causes of cancers all over the body [27]. It may even cause unconsciousness and sudden death if taken in high amounts. Hepatic alcohol dehydrogenase is the main enzyme to metabolize alcohol that requires the cofactor, nicotinamide adenine dinucleotide (NAD). The products are acetaldehyde and reduced NAD. Normally, NAD is used to metabolize fats in the liver, but alcohol competes with these fats for the use of NAD. Eventually, prolonged exposure of alcohol causes fatty liver. Ethanol is the only alcohol that is found in alcoholic beverages. Ethanol crosses biological membranes and blood-brain barrier by means of passive diffusion, easily. Alcohol works particularly by increasing effects of the gamma aminobutyric acid that is the major inhibitory neurotransmitter of the brain. Alcohol induces happiness and euphoria, decreased anxiety, increased sociability, sedation, generalized depression of central nervous system, and impairment of cognitive, memory, motor, and sensory functions. It may even cause fetal disorders in pregnancy because ethanol is classified as a teratogen. Regular alcohol consumption leads to cell death in the liver, scarring, cirrhosis, and hepatocellular carcinoma. Heavy alcohol consumption may even terminate with permanent brain damage. Alcohol is a major contributing factor of elevated triglycerides. It is well-known that triglycerides behave as sensitive acute phase reactants (APR) in the plasma [10]. Although the regular alcohol consumers were excluded, plasma triglycerides were higher in the smokers in the present study (163.1 versus 151.3mg/dL, $p<0.05$), indicating the inflammatory effects of smoking in the body.

The acute phase response occurs in case of infection, infarction, foreign body, autoimmune disorder, allergy, neoplasm, trauma, and burn-like inflammatory conditions of the body. Certain mediators known as APR are increased or decreased during the response [30,31]. These markers are commonly measured in the clinical practice as the indicators of acute and chronic inflammations of the body. The terms of acute phase proteins and APR are usually used synonymously, although some APR are polypeptides rather than proteins. Positive and negative APR are those whose concentrations increase or decrease during the acute phase response, respectively. The response is predominantly mediated by the pro-inflammatory cytokines including TNF, interleukin-1, and interleukin-6 secreted by neutrophils and macrophages into the circulation. The liver and other organs respond to the cytokines by producing many positive APR. ESR, CRP, fibrinogen, ferritin, procalcitonin, hepcidin, haptoglobin, ceruloplasmin, complement proteins, and serum amyloid A are some of the well-known positive APR. CRP is a useful indicator of the acute phase response, clinically. It is responsible for activation of the complement pathway. CRP reaches up to the maximum concentration within two days and decreases with the resolution of the inflammation with a half-life of 6-8 hours, rapidly. It correlates with ESR, but not simultaneously. Because ESR is largely dependent upon elevation of fibrinogen with a half-life of one week, approximately. Therefore, ESR remains higher for a longer period of time despite the removal of the inflammatory stimulus. Similarly, white blood cells and platelet counts may also behave as some other positive APR in the body [32]. On the other hand, productions of the negative APR are suppressed at the same time. Albumin, transferrin, retinol-binding protein, antithrombin, transcortin, alpha-fetoprotein, and hemoglobin are some of the well-known negative APR in the body. Suppressions of such negative APR are also used as the indicators of the acute phase response in the body. Suppressions of such negative APR

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may actually be secondary to the protection of some amino acids and polypeptides required for the production of positive APR, sufficiently. As also observed in the smokers in the present study, production of HDL may also be suppressed in the liver during the acute phase response [33]. Similarly, triglycerides, DM, and CHD were all higher in patients with plasma HDL values of lower than 40mg/dL, significantly [33]. So, HDL may actually behave as negative whereas triglycerides behave as positive APR in the plasma. Similarly, the highest CHD of the group with HDL values of lower than 40mg/dL can also be explained by the same hypothesis in the other study [10]. Additionally, plasma triglycerides increased whereas HDL decreased during infections [34]. On the other hand, a 10 mg/dL increase of plasma LDL values was associated with a 3% lower risk of hemorrhagic stroke [35]. Similarly, the highest prevalence's of HT and DM parallel to the elevated values of LDL and HDL, and the highest prevalence's of COPD, CHD, and CRD in contrast to the lowest values of LDL and HDL may show initially positive but eventually negative behaviors of LDL and HDL as the APR [36]. Probably, HDL turn to the negative direction much earlier than LDL in the plasma. Interestingly, the most desired values were between 80 and 100mg/dL for LDL, between 40 and 46mg/dL for HDL, and lower than 60mg/dL for triglycerides in the plasma [10]. Parallel to ESR and CRP, plasma triglycerides and LDL may behave as positive whereas HDL and FPG behave as negative APR in smokers in the present study. In another words, low HDL values should alert clinicians for researching of any acute phase response in the body [37,38].

Cholesterol, triglycerides, and phospholipids are the major lipids of the body. They do not circulate in the plasma, freely instead they are bound to proteins, and transported as lipoproteins. There are five major classes of lipoproteins in the plasma. Chylomicrons carry exogenous triglycerides to the liver via the thoracic duct. Very low-density lipoproteins (VLDL) are produced in the liver and carry endogenous triglycerides to the organs. In the capillaries of adipocytes and muscle tissue, VLDL are converted into intermediate density lipoproteins (IDL) by removal of 90% of triglycerides by lipases. Then IDL are degraded into LDL by removal of more triglycerides. So VLDL are the main source of LDL in the plasma, and LDL deliver cholesterol from the liver to the other organs. Although the liver removes majority of LDL from the circulation, a small amount is up taken by scavenger receptors of the macrophages migrating into the arterial walls, and become the foam cells of atherosclerotic plaques. HDL remove fats and cholesterol from cells including the arterial wall atheroma, and carry the cholesterol back to the adrenals, ovaries, and testes-like steroidogenic organs and liver for excretion, re-utilization, or disposal. All of the carrier lipoproteins are under dynamic control, and are readily affected by diet, drug, chronic inflammation, prolonged infection, cancer, tissue damage, smoking, alcohol, and excess weight. Thus, lipid analysis should be performed during a steady state. But the metabolic syndrome alone is a low-grade inflammatory process, therefore the metabolic syndrome may even cause abnormal lipoproteins levels in the plasma. For example, HDL may normally show various anti-oxidative, anti-inflammatory, and anti-atherogenic properties including reverse cholesterol transport [39]. However, HDL may become 'dysfunctional' in pathologic conditions which means that relative compositions of lipids and proteins, as well as the enzymatic activities of HDL are altered [39]. For example, properties of HDL are compromised in patients with DM by means of the oxidative modification, glycation, and/or transformation of HDL proteomes into the proinflammatory proteins. Additionally, the drugs increasing HDL values such as niacin, fibrates, and cholesteryl ester transfer protein inhibitors cannot reduce all-cause mortality, CHD mortality, myocardial infarction, and stroke [40]. In another words, HDL may just be some indicators instead of being the main actors of the human health. Similarly, BMI, DM, and CHD were the lowest between the HDL values of 40 and 46mg/dL, and the prevalence of DM was only 3.1% between these values against 22.2% outside these limits [41]. Similar to the present study, HDL and FPG values were also suppressed in the sickle cell diseases (SCD), probably due to the severe inflammatory nature of the diseases [42]. Smoking may reduce HDL and FPG by means of the systemic inflammatory effects on the vascular endothelium all over the body. On the other hand, triglycerides may be one of the most sensitive APR of the metabolic syndrome [43]. Although ATP II determined the normal plasma triglycerides as lower than 200 mg/dL in 1994 [44], World Health Organization in 1999 [45] and ATP III in 2001 reduced the normal limits as lower than 150mg/dL [6]. Although these cut points, there are still suspicions about the safest values of triglycerides in the plasma [43]. Beside that triglycerides are the only lipids which were not suppressed with the pathological weight losses [46]. For example, plasma triglycerides increased in contrast to the suppressed body weight and BMI in the SCD [46]. Similarly, prevalence's of excess weight, DM, HT, and smoking were all higher in the hypertriglyceridemia group (200mg/dL and higher) in the other study [47]. Interestingly, the greatest number of deteriorations in the metabolic parameters was observed with the triglyceride's values of 60 mg/dL and higher [43].

The body's homeostatic mechanism keeps blood glucose levels within a narrow range. There are two groups of mutually antagonistic hormones affecting the blood glucose levels. Glucagon, cortisol, and catecholamines are the catabolic hormones increasing the blood glucose, whereas insulin is the anabolic hormone decreasing the blood glucose levels. Glucagon is secreted from the alpha cells, while insulin is secreted from the beta cells of pancreatic islets which are the bundles of endocrine tissues. They regulate the blood glucose levels together, through a negative feedback mechanism. When the blood glucose level is too high, insulin tells muscles to take up excess glucose for storage. When the blood glucose level is too low, glucagon informs the tissues to produce more glucose. Glucagon and insulin are secreted into the portal vein and extracted from the portal vein

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by the liver. Epinephrine prepares the muscles and respiratory system for a 'fight to fight' response. Cortisol prepares the body for the stresses. Glucose is stored in the skeletal muscles and hepatocytes in the form of glycogen. There are approximately 100 and 400grams of glycogen stored in the skeletal muscles and liver in the human body, respectively [48]. A blood glucose level of four grams, or about a teaspoon, is critical for the normal function of millions of cells in the body [48]. The brain consumes about 60% of the blood glucose during fasting. The four grams of glucose circulates in the blood stream of a person with the body weight of 70kg. This amount is kept constant with a sophisticated control mechanism in the body. The constant blood glucose levels are maintained via the hepatic and muscular glycogen stores during fasting. FPG, which is measured after a fasting period of 8 hours, is the most commonly used indication of overall glucose homeostasis. Infections, inflammations, surgical operations, depressions, alcohol, smoking, and various stresses may affect the blood glucose homeostasis. For example, smoking was negatively associated with FPG and type 2 DM in Chinese men with the normal weight, but no significant association was found in men with excess weight or in women [49]. Similarly, smokers have a lower likelihood of newly diagnosed DM in Chinese males with a lower BMI in the other study [50]. Parallel to the above studies, FPG was also lower in the smokers in the present study, significantly (102.3 versus 111.6mg/dL, p=0.007).

Conclusion

As a conclusion, smoking-induced low-grade inflammation on the vascular endothelium all over the body may terminate with the endothelial dysfunction, accelerated atherosclerosis, end-organ insufficiencies, early aging, and premature death. FPG and HDL may be negative whereas triglycerides, LDL, ESR, and CRP may be positive acute phase reactants indicating the systemic inflammatory effects of smoking.

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