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Smoking Decreases High Density Lipoproteins in the Plasma

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Abstract

Background: We tried to understand whether or not there is a relationship between smoking and high-density lipoproteins (HDL) in the plasma.

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.

Results: The study included 150 smokers (51 females) and 162 non-smokers. The mean age of smokers was 45.9 years, and just 34.0% of them were female. Although the body weight, body mass index, systolic and diastolic blood pressures, and hematocrit values were similar in both groups, HDL (41.1 versus 44.0mg/dL, $p<0.05$) were lower in the smokers, significantly. Similarly, fasting plasma glucose (FPG) was lower in the smokers, again (101.9 versus 111.9mg/dL, $p<0.01$). On the other hand, triglycerides (163.3 versus 151.8mg/dL, $p<0.05$) and low-density lipoproteins (LDL) (126.1 versus 117.4mg/dL, $p<0.05$) were higher in the smokers, significantly. Parallel to the triglycerides and LDL, erythrocyte sedimentation rate (ESR) (10.8 versus 9.4mm/h, $p<0.05$) and C-reactive protein (CRP) (2.5 versus 2.1mg/L, $p<0.05$) values were also higher in the smokers, significantly.

Conclusion: Smoking-induced low-grade inflammation on vascular endothelium all over the body may terminate with an endothelial dysfunction, an accelerated atherosclerosis, end-organ insufficiencies, early aging, and premature death. As some significant indicators of systemic inflammation, smoking decreases HDL and FPG whereas increases triglycerides and LDL, parallel to the ESR and CRP, in the plasma.

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

Abbreviations: CRP: C-Reactive Protein; 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; HDL: High-Density Lipoproteins; ERC: Erythrocyte Sedimentation Rate; CRP: C-Reactive Protein; FPG: Fasting Plasma Glucose; LDL: Low Density Lipoproteins; BMI: Body Mass Index; NT: Normotension; WCH: White Coat Hypertension; NAD: Nicotinamide Adenine Dinucleotide; APR: Acute Phase Reactants; 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 body. Probably, the whole endothelium all over the body acts as a separate organ, and it may be the largest organ of the body. It may contract the peripheral organs' vasculature while relaxing the internal organs during cold, anxiety, depression, and other stressful conditions of the body. Since we measure the systolic and diastolic blood pressures (BP) in the arms and legs, they may not show the real BP of the brain, heart, lung, liver, and kidney-like internal organs. The endothelium may be the major player in the control of blood fluidity, platelets aggregation, and vascular tone. It may also be the main actor in immunology, inflammation, angiogenesis, and endocrinology. The endothelium controls vascular tone and blood flow by synthesizing and releasing nitric oxide, arachidonic acid metabolites, and reactive oxygen species. It may also be important for the generation of vasoactive hormones such as angiotensin II. An endothelial dysfunction-induced accelerated atherosclerosis all over the 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, high blood glucose levels, chronic inflammations, prolonged infections, cancers, smoking, and alcohol are 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 be the major causes of aging in human beings [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. Thus, the term 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]. Although early withdrawal of the accelerating factors may delay terminal consequences, 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 well-known components of the syndrome, there may be a significant relationship between smoking and high-density lipoproteins (HDL) in the plasma.

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 taken into the study. 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), fasting plasma glucose (FPG), triglycerides, low density lipoproteins (LDL), HDL, albumin, creatinine, thyroid function tests, hepatic function tests, markers of hepatitis A, B, C, and human immunodeficiency viruses, urinalysis, a posterior-anterior chest x-ray graphy, and an electrocardiogram was performed. An additional Doppler echocardiogram and/or an abdominal ultrasonography were performed just in cases with requirement. Body mass index (BMI) of each case was calculated by measurements of the Same Physician instead of verbal expressions. Weight in kilograms is divided by height in meters squared [6]. Office BP 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 150 smokers (51 females and 99 males) and 162 non-smokers (55 females and 107 males). The mean age of smokers was 45.9years, and just 34.0% of them were female. Although the mean body weight, BMI, systolic and diastolic BP, and hematocrit values were similar in both groups, HDL (41.1 versus 44.0mg/dL, $p<0.05$) were lower in the smokers, significantly. Similarly, FPG was lower in the smokers, again (101.9 versus 111.9mg/dL, $p<0.01$). On the other hand, triglycerides (163.3 versus 151.8mg/dL, $p<0.05$) and LDL (126.1 versus 117.4mg/dL, $p<0.05$) were higher in the smokers, significantly. Parallel to the triglycerides and LDL, ESR (10.8 versus 9.4mm/h, $p<0.05$) and CRP (2.5 versus 2.1mg/L, $p<0.05$) values were also higher in the smokers, significantly (Table 1).

Table 1: Comparison of cases with smoking and without.

Variables	Smokers	p-Value	Non-Smokers
Number	150		162
Female ratio	34.00%	Ns*	33.90%
Mean age (year)	45.9±13.4 (19-76)	Ns	45.2±5.7 (13-77)
Weight (kg)	75.6±14.5 (44-118)	Ns	74.6±13.0 (45-122)
BMI† (kg/m^2)	26.7±4.5 (16.7-39.4)	Ns	26.5±4.5 (18.1-41.1)
Systolic BP‡ (mmHg)	128.0±25.0 (90-200)	Ns	130.2±22.7 (80-200)
Diastolic BP (mmHg)	88.1±12.7 (60-120)	Ns	88.4±12.0 (60-130)
Hematocrit (%)	41.6±5.1 (28-60)	Ns	41.0±3.7 (31-49)

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HDL§ (mg/dL)	41.1±9.5 (26-70)	<0.05	44.0±9.5 (24-70)
FPG‖ (mg/dL)	101.9±25.8 (70-309)	<0.01	111.9±38.1 (74-327)
Triglycerides (mg/dL)	163.3±83.1 (45-385)	<0.05	151.8±86.9 (20-410)
LDL** (mg/dL)	126.1±35.4 (10-282)	<0.05	117.4±28.8 (43-185)
ESR*** (mm/h)	10.8±9.7 (1-51)	<0.05	9.4±8.0 (1-35)
CRP**** (mg/L)	2.5±2.7 (0-13)	<0.05	2.1±2.6 (0-12)

*Nonsignificant (p>0.05) †Body mass index ‡Blood pressures §High density lipoproteins ‖Fasting plasma glucose **Low density lipoproteins ***Erythrocyte sedimentation rate ****C-reactive protein

Discussion

Obesity may be one of the terminal endpoints of the metabolic syndrome. Although some transient successes can be achieved, nonpharmaceutical approaches provide limited benefit to reverse obesity, permanently. Due to the excess weight-induced chronic low-grade inflammation and dysfunction 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 on the endothelial cells, and the systemic atherosclerosis may prevent clearance of malignant cells by means of the natural killer cells, effectively. Similarly, the effects of excess weight on the BP were shown in the literature, extensively [8]. For instance, 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 triggering cause of the metabolic syndrome appears to be weight gain that may be the main cause of insulin resistance, hyperlipoproteinemias, impaired fasting glucose, impaired glucose tolerance, and white coat hypertension (WCH) [10]. Interestingly, weight gain even before development of an obvious overweight or obesity may cause development of several components of the metabolic syndrome. For example, WCH alone may be a strong indicator of weight gain even before development of excess weight [8,9]. On the other hand, prevention of 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 observations, excess weight may actually be a consequence of physical inactivity instead of an excessive eating habit. In another words, there is a problem with burning 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 title of abdominal obesity may be meaningless instead it should be defined as overweight or obesity by means of the BMI. Because adipocytes function as an endocrine organ, and they produce leptin, tumour necrosis factor (TNF)-alpha, plasminogen activator inhibitor-1, and adiponectin-like cytokines in 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 irreversible endpoints of the syndrome [6].

Smoking may be the second common cause of disseminated vasculitis in the body. It is one of the major risk factors for 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 small and medium-sized arteries and veins, and it has never been reported in the absence of smoking in the literature. Beside the well-known atherosclerotic effects of smoking, some studies reported that smoking in human beings and nicotine administration in animals are associated with relatively lower BMI values [15]. Some evidence revealed an increased energy expenditure during smoking both on 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 intermeal time, and simultaneously 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 evidence suggests that risk of weight gain is the highest during the first year after quitting and decreases with the following years [20]. Whereas 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, prevalences 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 prevalences in both genders, prevalences 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 in women about the atherosclerotic endpoints probably due to the higher BMI and its consequences in them. As also observed in the present study, smoking is consistently higher in men in the literature [14]. Several toxic substances

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found in cigarette smoke get into the circulation and cause a vascular endothelial inflammation all over the body. Similarly, smoking is usually associated with depression, irritable bowel syndrome (IBS), chronic gastritis, hemorrhoids, and urolithiasis [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 IBS including loose stool, diarrhea, and constipation. Thirdly, diarrheal losses-induced urinary changes may also 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. Finally, immunosuppression secondary to smoking-induced vascular endothelial inflammation may even terminate with 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 those have the enzyme, urease. Parallel to the results above, urolithiasis was detected in 17.9% of cases with the IBS and in 11.6% of cases without IBS in the other study ($p<0.01$) [25].

Alcohol may be the third common cause of disseminated vasculitis in 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 damages 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 endpoints 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 in the body [27]. It may even cause unconsciousness and death in high amounts. Hepatic alcohol dehydrogenase is the main enzyme to metabolize alcohol which 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 via passive diffusion, easily. Alcohol works particularly by increasing effects of the gamma aminobutyric acid in the brain. This 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 are sensitive acute phase reactants (APR) in plasma [10]. Although the cases with regular alcohol consumption were excluded, plasma triglycerides were higher in the smokers in the present study (163.3 versus 151.8mg/dL, $p<0.05$), indicating the inflammatory properties of smoking in the human 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 in 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 in the clinic. 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 may actually be secondary to the protection of some amino acids

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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 40 mg/dL, significantly [33]. So, HDL may actually behave as negative and 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 prevalences of HT and DM parallel to the elevated values of LDL and HDL, and the highest prevalences 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]. Interestingly, the most desired values were between 80 and 100 mg/dL for LDL, between 40 and 46 mg/dL for HDL, and lower than 60 mg/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. Cholesterol is an essential component of the animal cell membrane, bile acids, adrenal and gonadal steroid hormones, and vitamin D. It is synthesized by the liver, adrenal glands, reproductive organs, and intestines. Cholesterol is oxidized by the liver into a variety of bile acids. These, in turn, are conjugated. A mixture of conjugated and nonconjugated bile acids, along with cholesterol itself, is excreted into the bile. Cholesterol forms the major constituent of most gallstones. Approximately, 95% of the bile acids are reabsorbed from the intestines. By this way 50% of the excreted cholesterol is reabsorbed by the small bowel, again. The enterohepatic circulation of bile acids is essential for digestion and absorption of dietary fats. Cholesterol is found only in animal-source foods but not in fruits, vegetables, cereals, nuts, and other plants. In fact, most of the dietary cholesterol is esterified, and the esterified cholesterol is poorly absorbed. For these reasons, dietary cholesterol has little effect on plasma cholesterol levels which may also support the hypothesis that plasma lipoproteins mainly behave as positive and negative APR in the body. On the other hand, triglycerides are the major fat found in our foods. Most of the fat in the human body is stored in the form of triglycerides, again. Calories not burned by the body are automatically converted into triglycerides, which explains why eating too much of anything can lead to excess weight. Beside that stored triglycerides help to protect and insulate internal organs. Actually, the number of fat cells does not fluctuate along with changes in the weight instead the fat cells themselves get bigger or smaller. Additionally, triglycerides are the major lipids transported in the blood, again. In another words, triglycerides provide energy for muscles, they are stored as fat in the body, and they are used to produce LDL in the liver. Triglycerides are composed of smaller units, called fatty acids. Fatty acids are described as saturated, polyunsaturated, and monounsaturated depending on how much hydrogen they contain. Saturated fatty acids contain the most hydrogen, and they are the most dangerous ones for health. The saturated fats can raise the blood cholesterol levels more than anything else in the foods. Saturated fats may increase blood cholesterol levels by slowing down the removal of LDL. Therefore, blood cholesterol levels may increase even if the diet is rich for saturated fats but poor for cholesterol. Foods containing saturated fats mainly come from animals, too. These foods also contain excess cholesterol actually, so they can raise blood cholesterol levels in two ways at the same time. Phospholipids are the triglycerides that are covalently bound to a phosphate group, and they regulate membrane permeability, remove cholesterol from the body, provide signal transmission across the membranes, act as detergents, and help in solubilization of cholesterol. Cholesterol, triglycerides, and phospholipids 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 thorasic 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 is 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 the 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 removes fats and cholesterol from cells including the arterial wall atheroma and carry the cholesterol back to the liver and steroidogenic organs such as adrenals, ovaries, and testes for excretion, re-utilization, and 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. Thus, the metabolic syndrome may even cause abnormal lipoproteins levels in the plasma. For instance, 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

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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 human health. Similarly, BMI, DM, and CHD were the lowest between the HDL values of 40 and 46 mg/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 due to the systemic inflammatory effects on the vascular endothelium. On the other hand, triglycerides may be one of the most sensitive indicators of the metabolic syndrome [43,44]. Although ATP II determined the normal plasma triglycerides as lower than 200mg/dL in 1994 [45], World Health Organisation in 1999 [46] and ATP III in 2001 reduced the normal limits as lower than 150mg/dL [6]. Although these cutpoints, there are still suspicions about the safest values of triglycerides in the plasma [44]. On the other hand, triglycerides are the only lipids that were not suppressed with pathological weight losses [47]. For example, plasma triglycerides were increased in contrast to the suppressed body weight and BMI in the SCD [47]. Similarly, prevalences of excess weight, DM, HT, and smoking were all higher in the hypertriglyceridemia group (200mg/dL and higher) [48]. Interestingly, the greatest number of deteriorations in the metabolic parameters was observed with the triglyceride's values of 60mg/dL and higher [44].

Conclusion

As a conclusion, smoking-induced low-grade inflammation on the vascular endothelium all over the body may terminate with an endothelial dysfunction, an accelerated atherosclerosis, end-organ insufficiencies, early aging, and premature death. As some significant indicators of the systemic inflammation, smoking decreases HDL and FPG whereas increases triglycerides and LDL, parallel to the ESR and CRP, in the plasma.

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