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Smoking Increases Triglycerides and Low Density Lipoproteins in the Plasma

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Abstract

Background: There may be some significant relationships between smoking and triglycerides and low density lipoproteins (LDL) in the plasma.

Methods: Consecutive daily smokers at least for a period of six months and age and sex-matched non-smokers were included into the study. Cases with regular alcohol consumption (one drink a day) and patients with inflammatory, infectious, or devastating disorders including eating disorders, malignancies, acute or chronic renal failure, cirrhosis, chronic obstructive pulmonary disease, hyper- or hypothyroidism, or heart failure were excluded from the study.

Results: The study included 150 smokers (99 males) and 162 non-smokers. The mean age of smokers was 45.9 years, and 66.0% of them were male. Although the mean body weight, body mass index, systolic and diastolic blood pressures, and hematocrit values were similar in both groups, triglycerides (163.3 versus 151.8 mg/dL, $p<0.05$) and LDL (126.1 versus 117.4 mg/dL, $p<0.05$) were higher in the smokers, significantly. Similarly, erythrocyte sedimentation rate (ESR) (10.8 versus 9.4 mm/h, $p<0.05$) and C-reactive protein (CRP) (2.5 versus 2.1 mg/L, $p<0.05$) values were also higher in the smokers. On the other hand, high density lipoproteins (HDL) (41.1 versus 44.0 mg/dL, $p<0.05$) and fasting plasma glucose (FPG) (101.9 versus 111.9 mg/dL, $p<0.01$) values were lower in the smokers, significantly.

Conclusion: Smoking causes a low-grade systemic inflammation on vascular endothelium terminating with an accelerated atherosclerosis-induced end-organ insufficiencies all over the body. As significant indicators of the systemic inflammation, smoking increases triglycerides and LDL, parallel to ESR and CRP, whereas decreases HDL and FPG in the body.

Keywords: Smoking, triglycerides, low density lipoproteins, high density lipoproteins, fasting plasma glucose, erythrocyte sedimentation rate, C-reactive protein

Introduction

The endothelium is a monolayer of endothelial cells that forms the inner cellular lining of arteries, veins, capillaries, and lymphatics. It may also be the major player in the control of blood fluidity, platelets aggregation, and vascular tone. It may also be the main actor in the 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. They may also be important for generation of vasoactive hormones such as angiotensin II. On the other hand, overweight and obesity, smoking, and alcohol are obvious causes of chronic endothelial inflammation terminating with an accelerated atherosclerosis-induced end-organ insufficiencies all over the body [1]. Chronic endothelial damage may be the main cause of end-organ insufficiencies, aging, and death [2]. Much higher blood pressures (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, and fibrosis, 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 obvious accelerating factors of the inflammatory process are physical inactivity, animal-rich diet, excess weight, smoking, alcohol, chronic inflammations, prolonged infections, and cancers for the development of terminal end-points including 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, early aging, and premature death [3]. Although early withdrawal of the accelerating factors may delay terminal consequences, after development of HT, DM, cirrhosis, COPD, CRD, CHD, PAD, mesenteric ischemia, osteoporosis, stroke, dementia, other end-organ insufficiencies, and aging, endothelial changes can not be reversed due to their fibrotic natures, completely. The accelerating factors and terminal end-points are researched under the titles of the metabolic syndrome, aging syndrome, and accelerated endothelial damage syndrome, extensively [4, 5]. We tried to understand whether or not there are some significant relationships between smoking and triglycerides and low density lipoproteins (LDL) 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. Consecutive daily smokers at least for a period of six months were taken into the study. Cases with regular 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. A routine check up procedure including hemogram, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), fasting plasma glucose (FPG), triglycerides, LDL, high density lipoproteins (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, 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 (99 males and 51 females) and 162 non-smokers (107 males and 55 females). The mean age of smokers was 45.9 years, and 66.0% of them were male. Although the mean body weight, body mass index, systolic and diastolic BP, and hematocrit values were similar in both groups, the mean triglycerides (163.3 versus 151.8 mg/dL, $p<0.05$) and LDL (126.1 versus 117.4 mg/dL, $p<0.05$) were higher in the smokers, significantly. Similarly, ESR (10.8 versus 9.4 mm/h, $p<0.05$) and CRP (2.5 versus

2.1 mg/L, $p<0.05$) values were also higher in the smokers. On the other hand, HDL (41.1 versus 44.0 mg/dL, $p<0.05$) and FPG (101.9 versus 111.9 mg/dL, $p<0.01$) values were lower in the smokers, significantly (Table 1).

Variables	***Smokers***	***p-value***	***Non-smokers***
Number	150		162
Male ratio	*66.0%*	Ns*	66.0%
Mean age (year)	45.9 ± 13.4 (19-76)	Ns	45.2 ± 15.7 (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)
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)
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)*
*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 §Low density lipoproteins ‖High density lipoproteins
**Fasting plasma glucose ***Erythrocyte sedimentation rate ****C-reactive protein

Table 1: Comparison of cases with smoking and without.

Discussion

Obesity may be one of the terminal end-points of the metabolic syndrome since after development of the obesity, nonpharmaceutical approaches provide limited benefit either to heal obesity or to prevent its complications. Overweight and obesity may cause a chronic low-grade inflammation on the vascular endothelium all over the body, and risk of death from all causes including cardiovascular diseases and cancers increases parallel to the range of excess weight in all age groups [7], since the chronic low-grade inflammation may cause genetic changes on the endothelial cells, and the systemic atherosclerosis may prevent clearance of malignant cells, effectively. The effects of overweight and obesity on 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%, $p<0.001$), 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, hyperlipoproteinemias, impaired fasting glucose, impaired glucose tolerance, and white coat hypertension (WCH) by means of the prolonged low-grade inflammation on the vascular endothelium all over the body [10]. Prevention of the weight gain with physical activity, even in the absence of a prominent weight loss, will probably result with resolution of many parameters of the metabolic syndrome [11]. According to our experiences, overweight and obesity may actually be some consequences of physical inactivity instead of an excessive eating habit. Therefore prevention of weight gain can not be achieved by diet, alone [12]. Additionally, 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 and renin-angiotensin-aldosterone systems are probably associated with insulin resistance, elevated BP, and chronic endothelial inflammation. 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 irrevers-

ible end-points of the metabolic syndrome [6].

Smoking may be the second common cause of vasculitis all over the body. It is one of the main risk factors for the atherosclerotic end-organ insufficiencies [1, 14]. Its atherosclerotic effect is the most obvious 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 obvious atherosclerotic effects of smoking, some studies reported that smoking in human being and nicotine administration in animals are associated with lower BMI values [15]. Some evidences revealed an increased energy expenditure during smoking both on rest and light physical activity [16], and 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]. Additionally, 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 postcessation weight gain, but evidences suggest that risk of weight gain is the highest during the first year after quitting, and decreases with the following years [20]. Interestingly, the mean body weight and BMI were similar both in the smokers and non-smokers in the present study (p>0.05 for both). 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 [21]. 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 [22]. In another word, smoking may be more dangerous for women about the atherosclerotic end-points 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 found in the cigarette smoke get into the circulation mainly via the respiratory tract, and cause a vascular endothelial inflammation all over the body. On the other hand, smoking is usually associated with depression, irritable bowel syndrome (IBS), chronic gastritis, hemorrhoids, and urolithiasis [23-25]. There may be several underlying mechanisms to explain these associations in the smokers [23]. 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 [24, 25]. Fourthly, smoking-induced sympathetic nervous system activation may cause motility disorders 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, since 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 in the other study (p<0.01) [24].

Alcohol may be the third common cause of 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 [26]. For example, 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 [27]. People with AUD have three-fold higher mortality in men and four-fold in women [28]. Similar to the smoking, alcohol may be more dangerous for women about the atherosclerotic end-points probably due to the higher BMI and its consequences in them, again. 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 [7]. Women are generally more sensitive to the harmful effects of alcohol, primarily due to their smaller body weight, lower capacity to metabolize alcohol, and higher proportion of fat in their body. Alcohol is one of the main causes of cancers all over the body [26]. Alcohol can 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. Acetaldehyde is subsequently metabolized by the aldehyde dehydrogenase into acetate that in turn is broken down into carbon dioxide and water. 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 cause fetal disorders in pregnancy because ethanol is classified as a teratogen. Regular consumption leads to cell death in the liver, scarring, cirrhosis, and hepatocellular carcinoma. Heavy consumption may even terminate with permanent brain damage. Similarly, alcohol is a major contributing factor of elevated triglycerides. It is obvious that triglycerides are sensitive acute phase reactants (APR) in the plasma [8]. Although the cases with regular alcohol consumption were excluded, plasma triglycerides were higher in the smokers in the present study (163.3 versus 151.8 mg/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 stresses of the body. Certain mediators known as APR are increased or decreased during the response [29, 30]. These markers are commonly measured in the clinical practice as the indicators of acute inflammation 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 response, respectively. The acute phase response is predominantly mediated by the pro-inflammatory cytokines including TNF, interleukin (IL)-1, and IL-6 secreted by immune cells. In case of inflammation, infection, or tissue damage, neutrophil and macrophages release such cytokines into the circulation. The liver and some other organs respond to the cytokines by producing many positive APR. Some of the obvious positive APR are ESR, CRP, fibrinogen, ferritin, procalcitonin, hepcidin, haptoglobin, ceruloplasmin, complement proteins, and serum amyloid A. CRP is a useful indicator of inflammation, infection, and tissue damage, clinically. It is responsible for activation of the complement pathway. CRP increases rapidly to the maximum concentration within two days, and decreases quickly with the resolution of the inflammation. It correlates with ESR, but not always simultaneously, because ESR is largely dependent upon elevation of fibrinogen with a half-life of one week, approximately. Thus ESR remains higher for a longer period of time despite the removal of the inflammatory stimulus. Whereas CRP rises with a half-life of 6-8 hours rapidly, and then it returns to normal with a successful treatment, rapidly. On the other hand, productions of the negative APR are suppressed at the same time. Some of the obvious negative APR are albumin, transferrin, retinol-binding protein, antithrombin, transcortin, alpha-fetoprotein, and hemoglobin. The suppressions of such negative APR are also used as the indicators of inflammation, infection, and tissue damage in the body. Suppression of such negative APR may actually be secondary to the protection of amino acids for the production of positive APR, sufficiently. As also observed together with smoking in the present study, productions of HDL may also be suppressed in the liver during the acute phase responses [31]. Similarly, triglycerides, DM, and CHD were all higher in patients with plasma HDL values of lower than 40 mg/dL, significantly [31]. 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 40 mg/dL can also be explained by the same hypothesis in the other study [8]. Additionally, plasma triglycerides increased whereas HDL decreased during infections [32]. On the other hand, a 10 mg/dL increase of plasma LDL values was associated with a 3% lower risk of hemorrhagic stroke [33]. Similarly, the highest prevalences of HT and DM parallel to the increased 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 [34]. 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 [8]. Parallel to ESR and CRP, plasma triglycerides and LDL may behave as positive whereas FPG and HDL behave as negative APR in smokers in the present study. In another definition, low HDL values should alert clinicians for researching of any inflammation, infection, or tissue damage in the body [35, 36].

Cholesterol, triglycerides, and phospholipids are the major lipids of the body. Cholesterol is an essential structural 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 crystallizes in the gall bladder and 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. In fact, most of the dietary cholesterol is esterified, and the esterified cholesterol is poorly absorbed by the body. For these reasons, dietary cholesterol has little effect on plasma cholesterol levels which may also support the hypothesis that plasma lipoproteins may mainly behave as some positive and negative APR in the body. Cholesterol is found only in animal-source foods but not in fruits, vegetables, cereals, nuts, and other plants. 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. On the other hand, triglycerides help to protect and insulate internal organs and cushion the blow of a fall when stored by the body. Actually, the number of fat cells in the body 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, too. In another word, triglycerides provide energy for muscles, they are stored as the body fat, and they are used to produce LDL in the body. Triglycerides are composed of smaller units of fat called as fatty acids. Fatty acids are described as saturated, polyunsaturated, or monounsaturated depending on how much hydrogen they contain. Saturated fatty acids contain the most hydrogen, and they are considered as the most dangerous for the 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. Thus, 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 too much cholesterol actually, so they can raise blood cholesterol levels in two ways at the same time. Phospholipids are 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 freely in the plasma, 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 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 uptaken 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 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, inflammation, infection, tissue damage, smoking, alcohol, overweight, and obesity. Therefore lipid analysis should be performed during a steady state. But the metabolic syndrome alone is a low grade inflammatory process on the vascular endothelium all over the body. Therefore the metabolic syndrome may even cause abnormal lipoproteins levels in the plasma. For example, HDL may normally show various anti-atherogenic properties including reverse cholesterol transport and anti-oxidative and anti-inflammatory properties [37]. 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 [37]. For instance, properties of HDL are compromised in patients with DM via 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 can not reduce all cause mortality, CHD mortality, myocardial infarction, and stroke [38]. In other definition, 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 46 mg/dL, and the prevalence of DM was only 3.1% between these values against 22.2% outside these limits in the other study [39]. 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 [40]. Smoking may reduce HDL and FPG in the plasma due to the systemic inflammatory effects on the vascular endothelium. On the other hand, although their normal limits have not been determined clearly yet, increased plasma triglycerides may be one of the most initial indicators of the metabolic syndrome [41-44]. Due to the growing evidences about the strong association between higher plasma triglycerides and increased prevalences of CHD, ATP III determined lower cutpoints for triglycerides abnormalities than did ATP II [6, 45]. Although ATP II determined the normal plasma triglycerides as lower than 200 mg/dL in 1994 [45], World Health Organisation in 1999 [46] and ATP III in 2001 reduced the normal limits as lower

than 150 mg/dL [6]. Although these cutpoints, there are still suspicions about the safest values of triglycerides in the plasma [42-44]. On the other hand, plasma triglycerides may be the only lipids those were not suppressed in pathological weight losses in the body [47]. For example, plasma triglycerides were not decreased, even 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 (200 mg/dL and higher) in the other study [48]. Additionally, the greatest number of deteriorations in the metabolic parameters was observed just above the plasma triglycerides of 60 mg/dL [44].

As a conclusion, smoking causes a low-grade systemic inflammation on vascular endothelium terminating with an accelerated atherosclerosis-induced end-organ insufficiencies all over the body. As significant indicators of the systemic inflammation, smoking increases triglycerides and LDL, parallel to ESR and CRP, whereas decreases HDL and FPG in the body.

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