

# The lowest is the safest value of plasma triglycerides

**Mehmet Rami Helvaci (1)**

**Abdulrazak Abyad (2)**

**Lesley Pocock (3)**

(1) Specialist of Internal Medicine, MD

(2) Middle-East Academy for Medicine of Aging, MD, MPH, MBA, AGSF

(3) medi+WORLD International

## Corresponding Author

Dr Mehmet Rami Helvaci,

07400, Alanya,

Turkey

Phone: 00-90-506-4708759

Email: mramihelvaci@hotmail.com

Received: August 2019; Accepted: September 2019; Published: October 1, 2019.

Citation: Mehmet Rami Helvaci, Abdulrazak Abyad, Lesley Pocock. The lowest is the safest value of plasma triglycerides.

World Family Medicine. 2019; 17(10): 10-15 DOI: 10.5742MEWFM.2019.93684

## Abstract

**Background:** We tried to understand the safest value of triglycerides in the plasma.

**Methods:** Patients with plasma triglycerides values lower than 60 mg/dL were collected into the first, lower than 100 mg/dL into the second, lower than 150 mg/dL into the third, lower than 200 mg/dL into the fourth, and 200 mg/dL and greater into the fifth groups.

**Results:** The study included 669 cases (393 females), totally. Mean age increased up to triglycerides value of 200 mg/dL, and there was an increase of triglycerides about 8.1 mg/dL for each year of aging. Male ratio increased parallel to increased triglycerides, gradually (32.3% versus 50.0%,  $p < 0.001$ ). Mean body mass indexes were 24.4, 27.0, 29.3, 29.9, and 30.1 kg/m<sup>2</sup> in the five groups, respectively, and it was normal only in cases with the plasma triglycerides values of lower than 60 mg/dL. Fasting plasma glucose, hypertension, diabetes mellitus, smoking, and chronic obstructive pulmonary disease increased parallel to increased triglycerides, gradually, whereas low density lipoproteins and white coat hypertension increased just up to triglycerides value of 200 mg/dL. The greatest number of deteriorations (seven components, significantly) was observed just during passage from the first into the second groups.

**Conclusions:** Plasma triglycerides may actually be some acute phase reactants indicating disseminated endothelial damage, inflammation, fibrosis, and eventual atherosclerosis all over the body. There may be some significant relationships between plasma triglycerides and aging, excess weight, and smoking. The greatest number of deteriorations of components of metabolic syndrome were observed just above triglycerides value of 60 mg/dL.

**Key words:** Triglycerides, acute phase reactants, smoking, excess weight, aging, chronic endothelial damage, accelerated atherosclerosis

## Introduction

Chronic endothelial damage may be the most common type of vasculitis and the leading cause of aging and death in human beings (1-4). Much higher blood pressure (BP) of the afferent vasculature may be the major underlying cause by inducing recurrent injuries on endothelium, and 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. Secondary to the chronic endothelial damage, inflammation, edema, and fibrosis, vascular walls thicken, their lumens narrow, and they lose their elastic nature which reduces blood supply to terminal organs and increases systolic BP further. Some of the well-known components of the inflammatory process are physical inactivity, animal-rich diet, overweight, smoking, alcohol, hypertriglyceridemia, hyperbetalipoproteinemia, dyslipidemia, impaired fasting glucose, impaired glucose tolerance, white coat hypertension (WCH), and chronic inflammatory processes including rheumatologic disorders, chronic infections, and cancers. Some of the irreversible consequences of the chronic inflammatory process include obesity, hypertension (HT), diabetes mellitus (DM), cirrhosis, peripheral artery disease (PAD), chronic obstructive pulmonary disease (COPD), chronic renal disease (CRD), coronary heart disease (CHD), mesenteric ischemia, osteoporosis, and stroke (5-7). Although early withdrawal of the causative factors may delay terminal consequences, after development of cirrhosis, COPD, CRD, CHD, PAD, or stroke, endothelial changes cannot be reversed completely due to their fibrotic nature. The underlying causes and terminal consequences were researched under the titles of metabolic syndrome, aging syndrome, or accelerated endothelial damage syndrome in the medicine, extensively (8-11). Although its normal limits have not been determined clearly yet, higher plasma triglycerides may be significant indicators of the metabolic syndrome (12). Due to the strong association between higher plasma triglycerides and prevalence of CHD, Adult Treatment Panel (ATP) III adopts lower cutpoints for triglycerides abnormalities than did ATP II (13, 14). Although ATP II determined the normal triglycerides value as lower than 200 mg/dL in 1994, World Health Organisation in 1999 (15) and ATP III in 2001 reduced its normal limit as lower than 150 mg/dL (14). Although these cutpoints are usually used to define limits of the metabolic syndrome, there are suspicions about the safest upper limits of plasma triglycerides in the medical literature. Beside that, smoking may be found among one of the most common causes of vasculitis all over the world. It is a major risk factor for the development of atherosclerotic endpoints including CHD, PAD, COPD, cirrhosis, CRD, and stroke (16, 17). We tried to understand the safest value of plasma triglycerides according to some known components of the metabolic syndrome in the present study.

## Material and Methods

Chronic endothelial damage may be the most common type of vasculitis and the leading cause of aging and death in human beings (1-4). Much higher blood pressure (BP) of the afferent vasculature may be the major underlying cause by inducing recurrent injuries on endothelium, and 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. Secondary to the chronic endothelial damage, inflammation, edema, and fibrosis, vascular walls thicken, their lumens narrow, and they lose their elastic nature which reduces blood supply to terminal organs and increases systolic BP further. Some of the well-known components of the inflammatory process are physical inactivity, animal-rich diet, overweight, smoking, alcohol, hypertriglyceridemia, hyperbetalipoproteinemia, dyslipidemia, impaired fasting glucose, impaired glucose tolerance, white coat hypertension (WCH), and chronic inflammatory processes including rheumatologic disorders, chronic infections, and cancers. Some of the irreversible consequences of the chronic inflammatory process include obesity, hypertension (HT), diabetes mellitus (DM), cirrhosis, peripheral artery disease (PAD), chronic obstructive pulmonary disease (COPD), chronic renal disease (CRD), coronary heart disease (CHD), mesenteric ischemia, osteoporosis, and stroke (5-7). Although early withdrawal of the causative factors may delay terminal consequences, after development of cirrhosis, COPD, CRD, CHD, PAD, or stroke, endothelial changes cannot be reversed completely due to their fibrotic nature. The underlying causes and terminal consequences were researched under the titles of metabolic syndrome, aging syndrome, or accelerated endothelial damage syndrome in the medicine, extensively (8-11). Although its normal limits have not been determined clearly yet, higher plasma triglycerides may be significant indicators of the metabolic syndrome (12). Due to the strong association between higher plasma triglycerides and prevalence of CHD, Adult Treatment Panel (ATP) III adopts lower cutpoints for triglycerides abnormalities than did ATP II (13, 14). Although ATP II determined the normal triglycerides value as lower than 200 mg/dL in 1994, World Health Organisation in 1999 (15) and ATP III in 2001 reduced its normal limit as lower than 150 mg/dL (14). Although these cutpoints are usually used to define limits of the metabolic syndrome, there are suspicions about the safest upper limits of plasma triglycerides in the medical literature. Beside that, smoking may be found among one of the most common causes of vasculitis all over the world. It is a major risk factor for the development of atherosclerotic endpoints including CHD, PAD, COPD, cirrhosis, CRD, and stroke (16, 17). We tried to understand the safest value of plasma triglycerides according to some known components of the metabolic syndrome in the present study.

## Results

The study included 669 cases (393 females and 276 males), totally. The mean values of plasma triglycerides were 51.0, 78.2, 121.8, 174.7, and 301.7 mg/dL in the five groups, respectively. The mean age increased just up to the plasma triglycerides value of 200 mg/dL, and there was an increase of triglycerides, about 8.1 mg/dL for each year of aging up to the plasma triglycerides value of 200 mg/dL. Male ratio increased parallel to the increased plasma triglycerides values, gradually (32.3% versus 50.0%,  $p < 0.001$ ). BMI increased just up to the plasma triglycerides value of 150 mg/dL, significantly. Beside that the mean BMI values were 24.4, 27.0, 29.3, 29.9, and 30.1 kg/m<sup>2</sup> in the five study groups, respectively. In other words, only the cases with the plasma triglycerides values of lower than 60 mg/dL had a normal mean BMI value. FPG, HT, DM, and COPD increased parallel to the increased plasma triglycerides values, gradually whereas LDL and WCH increased just up to the plasma triglycerides value of 200 mg/dL. Prevalence of smoking increased parallel to the increased plasma triglycerides values, gradually (16.9% versus 39.1%,  $p < 0.001$ ). Interestingly, the most significant increase of smoking was seen just after the plasma triglycerides value of 200 mg/dL, and there was not any significant effect of aging or excess weight on these patients. On the other hand, the greatest number of deteriorations (seven components, significantly) was observed during passage from the first into the second groups of the study cases. Whereas just six components deteriorated during passage from the second into the third groups, significantly. Three components deteriorated during passage from the third into the fourth, and four components deteriorated during passage from the fourth into the last groups, significantly (Table 1).

## Discussion

Excess weight may lead to both structural and functional abnormalities of many organ systems in the body. Adipose tissue produces leptin, tumor necrosis factor- $\alpha$ , plasminogen activator inhibitor-1, and adiponectin-like cytokines which act as acute phase reactants in the plasma (22, 23). Excess weight-induced chronic low-grade vascular endothelial inflammation may play a significant role in the pathogenesis of accelerated atherosclerotic process all over the body (1, 2). Additionally, excess weight may cause an increased blood volume as well as an increased cardiac output thought to be the result of increased oxygen need of the excessive fat tissue. The prolonged increase in the blood volume may lead to myocardial hypertrophy terminating with a decreased cardiac compliance. Beside that, the prevalence of high FPG and total cholesterol and low high density lipoproteins (HDL) increased parallel to the higher values of BMI (24). Combination of these cardiovascular risk factors will eventually terminate with an increase in left ventricular stroke work, higher risks of arrhythmias, cardiac failure, and sudden cardiac death. Similarly, the prevalence of CHD and stroke increased parallel to the higher BMI values in another study (25), and risk of death from all causes including cancers increased

throughout the range of moderate to severe weight excess in all age groups (26). The relationships between excess weight and increased BP and plasma triglycerides values were described in the metabolic syndrome (12), and clinical manifestations of the syndrome included obesity, dyslipidemia, HT, insulin resistance, and proinflammatory and prothrombotic states (10). Similarly, prevalence of smoking (42.2% versus 28.4%,  $p < 0.01$ ), excess weight (83.6% versus 70.6%,  $p < 0.01$ ), DM (16.3% versus 10.3%,  $p < 0.05$ ), and HT (23.2% versus 11.2%,  $p < 0.001$ ) were all higher in the hypertriglyceridemia group in another study (27). On the other hand, the prevalence of hyperbetalipoproteinemia were similar both in the hypertriglyceridemia (200 mg/dL or higher) and control groups (18.9% versus 16.3%,  $p > 0.05$ , respectively) in the above study (27). Similarly, plasma LDL values increased, just up to the plasma triglycerides value of 200 mg/dL in the present study. Beside that, the mean BMI values increased just up to the plasma triglycerides value of 150 mg/dL, significantly ( $p < 0.05$  for each step).

Smoking causes a chronic inflammatory process on the vascular endothelium, particularly on the respiratory tract and lungs, terminating with an accelerated atherosclerosis, end-organ insufficiencies, early aging, and premature death. Therefore smoking should be accepted as one of the major components of the metabolic syndrome. Strong and irreversible atherosclerotic effects of smoking are the most obviously observed in Buerger's disease. It 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 medicine. Beside the strong and irreversible atherosclerotic effects of smoking, smoking in humans and nicotine administration in animals may be associated with a decreased BMI (28). Evidence revealed an increased energy expenditure during smoking both on rest and light physical activity (29), and nicotine supplied by patch after smoking cessation decreased caloric intake in a dose-related manner (30). According to an animal study, nicotine may lengthen intermeal time and simultaneously decrease amount of meal eaten (31). Additionally, BMI seems to be the highest in former and the lowest in current smokers (32). Smoking may be associated with a postcessation weight gain (33). Similarly, although CHD was detected with similar prevalence in both genders in a previous study (34), prevalence of smoking and COPD were higher in males against the higher mean values or prevalence of the BMI, LDL, triglycerides, WCH, HT, and DM in females with CHD. This result may indicate both the strong atherosclerotic and weight decreasing roles of smoking (35). Similarly, the incidence of myocardial infarction is increased six-fold in women and three-fold in men who smoke 20 cigarettes per day (36). In another definition, smoking is more dangerous for women probably due to the associated higher BMI and its consequences in them. Parallel to the above results, the proportion of smokers is consistently higher in men in the literature (19). So smoking is probably a powerful atherosclerotic risk factor with some suppressor effects on appetite. Smoking-induced weight loss may be related with the smoking-induced vascular endothelial inflammation all over the body, since loss of appetite is one of the major

Table 1: Characteristics features of the study cases according to plasma values of triglycerides

Variable	Lower than 60 mg/dL	p-value	Lower than 100 mg/dL	p-value	Lower than 150 mg/dL	p-value	Lower than 200 mg/dL	p-value	200 mg/dL or higher
Number of cases	65		158		188		110		148
Age (year)	<u>36.1 ± 16.6</u> (17-79)	<u>0.011</u>	<u>41.9 ± 17.0</u> (16-83)	<u>0.001</u>	<u>47.1 ± 15.0</u> (16-82)	<u>0.005</u>	<u>51.3 ± 12.0</u> (19-73)	Ns*	<u>49.5 ± 11.6</u> (21-86)
Male ratio	<u>32.3%</u>	Ns	<u>36.0%</u>	Ns	<u>40.4%</u>	Ns	<u>43.6%</u>	Ns	<u>50.0%</u>
Smoking	<u>16.9%</u>	Ns	<u>18.3%</u>	<u>0.05</u> >	<u>25.0%</u>	Ns	<u>24.5%</u>	<u>0.001</u> >	<u>39.1%</u>
BMI† (kg/m <sup>2</sup> )	<u>24.4 ± 4.5</u> (16.7-38.1)	<u>0.003</u>	<u>27.0 ± 5.9</u> (16.7-49.3)	<u>0.000</u>	<u>29.3 ± 6.0</u> (18.4-50.5)	Ns	<u>29.9 ± 4.8</u> (19.2-49.0)	Ns	<u>30.1 ± 5.1</u> (21.0-51.1)
FPG‡ (mg/dL)	<u>98.5 ± 39.6</u> (77-377)	Ns	<u>107.1 ± 52.0</u> (59-400)	Ns	<u>104.7 ± 31.9</u> (71-327)	<u>0.029</u>	<u>116.2 ± 48.5</u> (68-386)	Ns	<u>122.0 ± 49.1</u> (74-338)
Triglycerides (mg/dL)	<u>51.0 ± 7.7</u> (27-59)	<u>0.000</u>	<u>78.2 ± 11.1</u> (60-99)	<u>0.000</u>	<u>121.8 ± 14.9</u> (100-149)	<u>0.000</u>	<u>174.7 ± 14.8</u> (150-199)	<u>0.000</u>	<u>301.7 ± 108.7</u> (200-1.144)
LDL§ (mg/dL)	<u>99.5 ± 23.4</u> (56-161)	<u>0.02</u>	<u>114.4 ± 34.1</u> (43-269)	<u>0.021</u>	<u>132.5 ± 31.2</u> (64-228)	Ns	<u>138.8 ± 29.9</u> (50-210)	Ns	<u>129.9 ± 38.7</u> (10-239)
WCH	<u>16.9%</u>	<u>0.01</u> >	<u>25.3%</u>	Ns	<u>30.8%</u>	Ns	<u>34.5%</u>	Ns	<u>33.7%</u>
HT**	<u>6.1%</u>	<u>0.001</u> >	<u>12.6%</u>	<u>0.01</u> >	<u>20.7%</u>	Ns	<u>23.6%</u>	Ns	<u>24.3%</u>
DM***	<u>3.0%</u>	<u>0.001</u> >	<u>10.7%</u>	Ns	<u>13.2%</u>	Ns	<u>16.3%</u>	<u>0.01</u> >	<u>25.6%</u>
COPD****	<u>6.1%</u>	Ns	<u>9.4%</u>	Ns	<u>12.7%</u>	Ns	<u>13.6%</u>	<u>0.01</u> >	<u>22.9%</u>
Number of deteriorations		<u>7</u>		<u>6</u>		<u>3</u>		<u>4</u>	

\*Nonsignificant (p>0.05) †Body mass index ‡Fasting plasma glucose §Low density lipoproteins || White coat hypertension \*\*Hypertension \*\*\*Diabetes mellitus \*\*\*\*Chronic obstructive pulmonary disease



symptoms of disseminated inflammations in the body. Physicians can even understand healing of patients via their normalizing appetite. Several toxic substances found in cigarette smoke get into the circulation by means of the respiratory tract, and cause a vascular endothelial inflammation until their clearance from the circulation. But due to the repeated smoking habit of the individuals, the clearance process never terminates. So the patients become ill with loss of appetite, permanently. In another explanation, smoking-induced weight loss is an indicator of being ill instead of being healthy (30-32). After smoking cessation, normal appetite comes back with a prominent weight gain in the patients but the returned weight is their physiological weight, actually.

Although the obvious consequences of excess weight on health, nearly three-quarters of cases above the age of 30 years have excess weight (37). The prevalence of excess weight increases by decades, particularly after the third decade, up to the eighth decade of life (37). So 30th and 70th years of age may be the breaking points of life for weight, and aging may be the major determiner factor of excess weight. Probably, partially decreased physical and mental stresses after the age of 30 years and debility and comorbid disorders-induced restrictions after the age of 70 years may be the major causes for the changes of BMI at these ages. Interestingly, the mean age and BMI increased just up to the plasma triglycerides values of 200 mg/dL and 150 mg/dL, respectively, in the present study. So smoking remained the major causative factor for the hypertriglyceridemia after the plasma triglycerides values of 200 mg/dL. Beside that the mean BMI values were 24.4, 27.0, 29.3, 29.9, and 30.1 kg/m<sup>2</sup> in the five study groups, respectively. In other words, only the cases with the plasma triglycerides values of lower than 60 mg/dL had a normal mean BMI value. On the other hand, the mean age and triglycerides value of the first group were 36.1 years and 51.0 mg/dL, respectively. They were 41.9 years and 78.2 mg/dL in the second, 47.1 years and 121.8 mg/dL in the third, and 51.3 years and 174.7 mg/dL in the fourth groups, respectively. In another definition, the triglycerides values increased about 8.1 mg/dL for each year of aging up to 200 mg/dL in the plasma. So aging alone may be another risk factor for chronic low-grade inflammation on vascular endothelium all over the body. In this way, we may estimate the approximate age of patients by using their plasma triglycerides values below 200 mg/dL in the absence of any comorbid disorder or smoking.

Although ATP III reduced the normal border of plasma triglycerides as lower than 150 mg/dL in 2001 (14), whether or not much lower limits provide additional benefits for health is unclear. In the present study, prevalence of smoking was the highest in the highest triglycerides having group which may also indicate inflammatory roles of smoking in the metabolic syndrome, since triglycerides may actually be some acute phase reactants in the plasma. The mean FPG and BMI and prevalence of HT, DM, and COPD increased parallel to the plasma triglycerides values from the first up to the last groups, gradually. As one of our opinions, significantly elevated mean age by the increased

plasma triglycerides values may be secondary to aging-induced decreased physical and mental stresses, which eventually terminates with onset of excess weight and its consequences. Interestingly, although the mean age increased from the lowest triglycerides having group up to the triglycerides value of 200 mg/dL, it then decreased. The similar trend was also seen with the mean LDL value. These trends may be due to the fact that although the borderline high triglycerides values (150-199 mg/dL) is seen together with physical inactivity and overweight, the high triglycerides (200-499 mg/dL) and very high triglycerides values (500 mg/dL and higher) may be secondary to both genetic factors, smoking, and terminal consequences of the metabolic syndrome including obesity, DM, HT, COPD, cirrhosis, CRD, PAD, CHD, and stroke (14). But although the underlying causes of the high and very high plasma triglycerides values may be a little bit different, probably risks of the terminal endpoints of the metabolic syndrome do not change in them. For example, prevalence of HT, DM, and COPD were the highest in the highest triglycerides having group in the present study. Eventually, although some authors reported that lipid assessment can be simplified by measurements of total cholesterol and HDL (38), the present study and most of the others indicated a causal relationship between higher triglycerides and terminal consequences of the metabolic syndrome (39).

As a conclusion, plasma triglycerides may actually be some acute phase reactants indicating disseminated endothelial damage, inflammation, fibrosis, and eventual atherosclerosis all over the body. There may be some significant relationships between plasma triglycerides values and aging, excess weight, and smoking. Interestingly, the greatest number of deteriorations of components of the metabolic syndrome including mean age, BMI, triglycerides, LDL, WCH, HT, DM was observed just above the plasma triglycerides value of 60 mg/dL.

## References

1. Widlansky ME, Gokce N, Keaney JF Jr, Vita JA. The clinical implications of endothelial dysfunction. *J Am Coll Cardiol* 2003; 42(7): 1149–1160.
2. Ridker PM. High-sensitivity C-reactive protein: potential adjunct for global risk assessment in the primary prevention of cardiovascular disease. *Circulation* 2001; 103(13): 1813–1818.
3. Helvacı MR, Seyhanlı M. What a high prevalence of white coat hypertension in society! *Intern Med* 2006; 45(10): 671-674.
4. Helvacı MR, Kaya H, Seyhanlı M, Cosar E. White coat hypertension is associated with a greater all-cause mortality. *J Health Sci* 2007; 53(2): 156-160.
5. Helvacı MR, Kaya H, Yalcin A, Kuvandik G. Prevalence of white coat hypertension in underweight and overweight subjects. *Int Heart J* 2007; 48(5): 605-613.
6. Helvacı MR, Kaya H, Duru M, Yalcin A. What is the relationship between white coat hypertension and dyslipidemia? *Int Heart J* 2008; 49(1): 87-93.

7. Helvacı MR, Kaya H, Sevinc A, Camci C. Body weight and white coat hypertension. *Pak J Med Sci* 2009; 25(6): 916-921.
8. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. *Lancet* 2005; 365(9468): 1415-1428.
9. Grundy SM, Brewer HB Jr, Cleeman JI, Smith SC Jr, Lenfant C. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. *Circulation* 2004; 109(3): 433-438.
10. Tonkin AM. The metabolic syndrome(s)? *Curr Atheroscler Rep* 2004; 6(3): 165-166.
11. Franklin SS, Barboza MG, Pio JR, Wong ND. Blood pressure categories, hypertensive subtypes, and the metabolic syndrome. *J Hypertens* 2006; 24(10): 2009-2016.
12. Helvacı MR, Kaya H, Gundogdu M. Association of increased triglyceride levels in metabolic syndrome with coronary artery disease. *Pak J Med Sci* 2010; 26(3): 667-672.
13. National Cholesterol Education Program. Second Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). *Circulation* 1994; 89(3): 1333-1445.
14. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation* 2002; 106(25): 3143-3421.
15. World Health Organization. Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications. Report of a WHO consultation 1999.
16. Helvacı MR, Aydin Y, Gundogdu M. Smoking induced atherosclerosis in cancers. *HealthMED* 2012; 6(11): 3744-3749.
17. Fodor JG, Tzerovska R, Dorner T, Rieder A. Do we diagnose and treat coronary heart disease differently in men and women? *Wien Med Wochenschr* 2004; 154(17-18): 423-425.
18. Helvacı MR, Kaya H, Borazan A, Ozer C, Seyhanli M, Yalcin A. Metformin and parameters of physical health. *Intern Med* 2008; 47(8): 697-703.
19. Helvacı MR, Aydin Y, Varan G, Abyad A, Pocock L. Acarbose versus metformin in the treatment of metabolic syndrome. *World Family Med* 2018; 16(5): 10-15.
20. O'Brien E, Asmar R, Beilin L, Imai Y, Mallion JM, Mancia G, et al. European Society of Hypertension recommendations for conventional, ambulatory and home blood pressure measurement. *J Hypertens* 2003; 21(5): 821-848.
21. Vestbo J, Hurd SS, Agustí AG, Jones PW, Vogelmeier C, Anzueto A, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *Am J Respir Crit Care Med* 2013; 187(4): 347-65.
22. Funahashi T, Nakamura T, Shimomura I, Maeda K, Kuriyama H, Takahashi M, et al. Role of adipocytokines on the pathogenesis of atherosclerosis in visceral obesity. *Intern Med* 1999; 38(2): 202-206.
23. Yudkin JS, Stehouwer CD, Emeis JJ, Coppack SW. C-reactive protein in healthy subjects: associations with obesity, insulin resistance, and endothelial dysfunction: a potential role for cytokines originating from adipose tissue? *Arterioscler Thromb Vasc Biol* 1999; 19(4): 972-978.
24. Zhou B, Wu Y, Yang J, Li Y, Zhang H, Zhao L. Overweight is an independent risk factor for cardiovascular disease in Chinese populations. *Obes Rev* 2002; 3(3): 147-156.
25. Zhou BF. Effect of body mass index on all-cause mortality and incidence of cardiovascular diseases--report for meta-analysis of prospective studies open optimal cut-off points of body mass index in Chinese adults. *Biomed Environ Sci* 2002; 15(3): 245-252.
26. Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW Jr. Body-mass index and mortality in a prospective cohort of U.S. adults. *N Engl J Med* 1999; 341(15): 1097-1105.
27. Helvacı MR, Aydin LY, Maden E, Aydin Y. What is the relationship between hypertriglyceridemia and smoking? *Middle East J Age and Ageing* 2011; 8(6).
28. Grunberg NE, Greenwood MR, Collins F, Epstein LH, Hatsukami D, Niaura R, et al. National working conference on smoking and body weight. Task Force 1: Mechanisms relevant to the relations between cigarette smoking and body weight. *Health Psychol* 1992; 11: 4-9.
29. Walker JF, Collins LC, Rowell PP, Goldsmith LJ, Moffatt RJ, Stamford BA. The effect of smoking on energy expenditure and plasma catecholamine and nicotine levels during light physical activity. *Nicotine Tob Res* 1999; 1(4): 365-370.
30. Hughes JR, Hatsukami DK. Effects of three doses of transdermal nicotine on post-cessation eating, hunger and weight. *J Subst Abuse* 1997; 9: 151-159.
31. Miyata G, Meguid MM, Varma M, Fetisov SO, Kim HJ. Nicotine alters the usual reciprocity between meal size and meal number in female rat. *Physiol Behav* 2001; 74(1-2): 169-176.
32. Laaksonen M, Rahkonen O, Prattala R. Smoking status and relative weight by educational level in Finland, 1978-1995. *Prev Med* 1998; 27(3): 431-437.
33. Froom P, Melamed S, Benbassat J. Smoking cessation and weight gain. *J Fam Pract* 1998; 46(6): 460-464.
34. Helvacı MR, Kaya H, Gundogdu M. Gender differences in coronary heart disease in Turkey. *Pak J Med Sci* 2012; 28(1): 40-44.
35. Helvacı MR, Aydin Y, Gundogdu M. Atherosclerotic effects of smoking and excess weight. *J Obes Wt Loss Ther* 2012; 2: 145.
36. Prescott E, Hippe M, Schnohr P, Hein HO, Vestbo J. Smoking and risk of myocardial infarction in women and men: longitudinal population study. *BMJ* 1998; 316(7137): 1043-1047.
37. Helvacı MR, Kaya H, Ozer C. Aging may be the major determiner factor of excess weight. *Middle East J Age and Ageing* 2008; 5(2).
38. Di Angelantonio E, Sarwar N, Perry P, Kaptoge S, Ray KK, Thompson A, et al. Major lipids, apolipoproteins, and risk of vascular disease. *JAMA* 2009; 302(18): 1993-2000.
39. Sarwar N, Sandhu MS, Ricketts SL, Butterworth AS, Di Angelantonio E, Boekholdt SM, et al. Triglyceride-mediated pathways and coronary disease: collaborative analysis of 101 studies. *Lancet* 2010; 375(9726): 1634-1639.