# **Coronary Heart Disease**

Twana Muhammed

Department of Biomedical Engineering, University of Bridgeport Bridgeport, CT 06604, USA

Copyright © 2014 ISSR Journals. This is an open access article distributed under the *Creative Commons Attribution License*, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

**ABSTRACT:** Coronary heart disease is dominant and very dangerous disease in the world. Coronary heart disease causes mortality in the world. CHD is very complicated because of many factors that affect this disease. The main factors effects on coronary heart disease are Cholesterol, Diabetes, Depression, and Hypertension. I believe four factors above have tremendous and direct effects on this disease. The rate of death is different according to country, age, and gender. The lack of physical activity is the same in men and women so the rate of hypertension, diabetes, and obesity more common. The main and dangerous point in coronary heart disease occurs when the coronary artery get atherosclerosis. As a result, the fat particles constrict the coronary artery so the blood, oxygen, and sufficient material don't flow to the heart completely. The main purpose of this model is to minimize the risk factors of coronary heart disease. As a result, I believe we can minimize and control this disease by doing more exercise, getting enough sleep, staying away from smoking and drinking, eating low animal products, and more eating fruit and vegetables. This paper goals to review the coronary heart disease and inquire its connections among different factors that must be taken into consideration throughout the process and how to prevent ourselves from risk factors.

**Keywords:** Cholesterol, diabetes, depression, hypertension, mortality.

# **1** INTRODUCTION

Coronary heart disease is the prevalent and dangerous disease in the world wide [1]. This disease is very broad and complicated because many factors cause the risk of coronary heart disease. CHD has caused the rate of mortality and morbidity in the world. However this rate is different according place, age, and gender [2]. Coronary heart disease in many countries is the number one of death [3]. The main and dangerous point in coronary heart disease the coronary artery gets atherosclerosis [4]. Atherosclerosis means the coronary artery because constricted or made up plagues by accumulating of fat particles in the artery [5]. Thus sometimes sudden death, stroke, and myocardial infraction happen [6]. The most common risk factors cause coronary heart diseases are Cholesterol, Diabetes, Depression, and Hypertension.

Cholesterol is the quantity of fat in food or diet[7]. Every person naturally has cholesterol in body so cholesterol divides into parts HDL and LDL[8]. In general the amount of cholesterol is in animal products such as red meat and dairy products[9]. Indeed, the cholesterol has tremendous effect on coronary heart disease because the atherosclerosis is built up by fat particles and high level cholesterol in the body[10];[11];[12].

Diabetes is the predominant diseases in the world particularly Type 2 mellitus, and it kills disease many people suffering from it[13]. The normal range of sugar in body is 60/120 mg[14]. Diabetes has huge effect on coronary heart disease, and the rate of death is higher because the diabetes Type 2 has a strong impact on coronary artery and build up sever atherosclerosis[15]. In addition, the risk factor on the heart is higher in patients who have diabetes than in patient have not diabetes[16]. The main sign of getting diabetes is obesity[17].

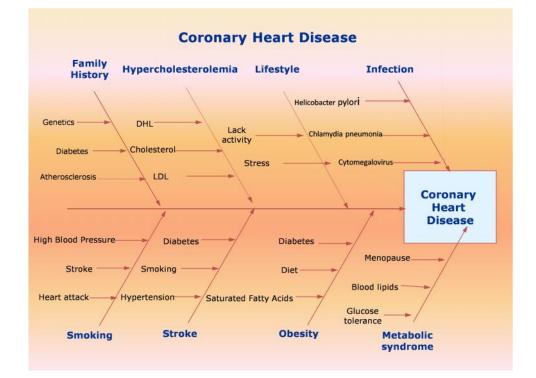
Depression is predicted a risk of heart disease especially for coronary heart disease[18]. Mostly 20 % of patients who have heart problem definitely have great part of depression[19]. Depression causes mortality when the patient has acute

myocardial infraction[20]. It has been proven one out five patients with coronary heart disease have depression[21]. Stress has negative effect on whole body because during stress the physiology of body will change[22]. Depression totally increase the risk of death in patients who have heart problem [23].

Hypertension is definitely linked to the rate of death from coronary heart disease[24]. The normal blood pressure in body is 80/120 mm Hg[25]. The blood pressure has two types Systolic and Diastolic. When the blood pressure increases more than the normal range it is called hypertension[26]. For management the hypertension we should reduce the load of coronary heart disease[27]. The dangerous effect of hypertension can produce strokes[28].

## **Research Method**

Recently combining research methods [5] p. 223 has tried to minimize the rate of death by "coronary heart disease [30] p. 826." One form for "Rate of coronary heart disease" [2]p. 5. Is the review for coronary heart disease according age? In this research method [3] attempted to treat and detect risk factor of "coronary heart disease according gender" [6]p. 199. And [1] p. 159. The model shown here describes reduce the death rate by CHD, realizing that those patients had previous coronary heart disease and during study for diagnosis used cardiac catheterization for 1000 patients. The goal for this study used catheterization for myocardial revascularization [5] p. 226. This study focuses on atherosclerosis and how to control it. This study found many factors caused atherosclerosis like serum lipoprotein. As a result, I could not control it because many factors impact this disease [30]p. 826. I think the first study is more useful and successful for saving human life. In this study, I recognized the symptom called angina for both men and women. Therefore, the women had angina like men and controlled by anti-angina medication. As a result, risk factor of coronary heart disease is more common in women than men because women have menopause during this process the risk of CHD increase. On the other hand, after menopause cycle the high blood pressure and obesity may occur these have main effect on CHD [1]p. 159 and [3]p. 227. In this research I recognized coronary heart disease between sexes and treatment in different way for both men and women. In addition, I recognized the risk factor of obstructive and angiographic CHD in women of all ages are less than men [6] p.205. the comparison between above researches I believe the second study was successful and accomplished I agree with it because the menopause cycle have been only in women so the probability of getting risk factor of more disease particularly CHD and rate of death in women high.



# 2 CORONARY HEART DISEASE

Figure 2. Coronary heart disease (CHD)critical disease factors.

**3** FINDINGS AND DISCUSSION

## **3.1** CORONARY HEART DISEASE (CHD)

The research that has been done about coronary heart disease is definitely detailed and useful. This disease should be taken seriously because of many dangerous factors. I chose this topic because I am interested and its very common disease in the world. It has proven coronary heart disease is the worldwide disease especially more prevalent in women than in men but traditionally same for both[1]. The finding that men in age 40-44 years every 8th man could get coronary heart disease but this rate is lower in younger women[2]. Also, it has been described coronary circulation comprise of coronary artery and vein the function of these provide oxygen and nutrient for myocardium and also remove harm or waste product. When this function has problem or is not working well until cause death [3]. We need to protect our health by doing exercise and eating more fruit and vegetables. It has been proven men and women have different structure or way and life cycle for being sick such as metabolically, therapy [6]. The mortality by coronary heart disease will increase in the world.

## 3.2 CHOLESTEROL

The research has been done involving cholesterol, and its main effect on coronary heart disease. This is completely true and has positive effect on cardio vascular system especially coronary heart disease because when the person eats more red meat and food containing high level of lipid. As a result, the amount of lipid accumulates in body, especially in coronary artery and constricts the artery. It has proven that high rate of cholesterol in the body has highly risk for coronary heart disease and leading cause of death [8]; [9]. The finding that men for prevention and reducing cholesterol in the body by medication using stain led. He believe statin led cause reduce rate of cholesterol thus cut down the risk of coronary heart disease; therefore, reduce death rates by coronary heart disease [10];[12]. We know the body of human is very sensitive if we have a little bit problems we are feeling uncomfortable and pain. I totally agree with research because when the cholesterol increases in the body more than the normal rate the fat particle block oxygen and sufficient nutrient flow to the heart completely. As a result, the person feels fatigue and headache. It has been proven the remain amount of cholesterol before eating it has normal risk of coronary heart disease so independent decrease the HDL- cholesterol in the body [11].we know before eating the rate of cholesterol usually decreases but when we eat the rate of cholesterol will change and increases.

## 3.2.1 DIABETES

Research has been done on the risk factors of coronary heart disease. In addition, diabetes has a common risk factor on coronary heart disease especially diabetes Type 2. This is true because we know when people have diabetes automatically they decrease physical activity; then oxidative stress in body happens and affects endothelial dysfunction particularly coronary artery. It has proven the person who has diabetes has greater risk of having CHD. [13]. I think this is true because we know if the patient who has coronary heart disease so they should reduce eating fat foods, doing exercise, taking medication, and surgery treat or reduce the risk of coronary heart disease. However, in those patients who have diabetes we cannot treat by surgery and prevention. The patient must be using medication for the rest his life. This author said people with diabetes have 2 to 4 time greater risk to coronary heart disease than patient has no diabetes and the rate of death in person has diabetes with coronary heart disease is 65-75% [4];[14]. This is true because we know one problem better than two problems. It mean better to who has diabetes and coronary heart disease. It has been proven between two groups of patients. One has diabetes but not myocardial infraction before. The other group has myocardial infraction but they have not diabetes. As a result, in the first group, 43% have lower risk for growing coronary heart disease events compared with the second group [15]; [17]. As we know only one disease has little risk, but if we have two diseases at the same time, it will be more complicated. It has been proven the effect diabetes on coronary heart disease rate of death in female is higher than in males, statically women are 2.58, in men are 1.85 [16]. We know many factors cause the rate of death higher than men. For instance, women become pregnant, care more about her children and family and she has not enough time on herself, and stays at home thus increasing her weight.

#### 3.3 DEPRESSION

This research has been done involving depression and that their effect on coronary heart disease. We know depression has direct effect on the whole body. Depression disturbs the physiology our bodies and secret many hormones. It has been proven depression increases the probabilities the patients will have coronary heart disease and death rates[18]. This completely makes sense because depression is associated with high level of cortisol, so this cortisol increases the blood pressure and heart beats. These are two types of depression, major and minor depression. More than 20% of coronary heart diseases have major depression. Major depression starts after one month of acute myocardial infraction [19]. We know

depression affects physiological, social and work. As we know many people when they have stress they try to decrease their stress less smoke, insomnia, drinking more, and loss of appetite by using those increases risk to coronary heart disease. It has been proven one of the most consequence of depression is an increased death rate in person already had coronary heart disease is beat to beat fluctuations in heart rate [20]. If we have depression, the physiology of our bodies will changes one of the changes in heart beat and heart rhythm, so it has negative effect on patients who have heart problem and might cause sudden death. It has been proven, that depression has more negative effects on those patient who have myocardial infraction than patients have not myocardial infraction [22]; [23]. This is true because we know when the person gets heart disease, the first advice is "Don't be stressed". The person should be relax and happy because if he is depressed the physiology all body release hormones increase heart beats and increase pressure on coronary artery, so the person will be get myocardial infraction again or sudden death.

#### 3.4 HYPERTENSION

This research has been conducted about hypertension of coronary heart disease. Hypertension is measured by two things, which are systolic and diastolic. Hypertension has negative effect on many parts of body especially on the heart. It has been proven for control and treatment hypertension the main step is reducing the heart disease events (Perreault et al., 1999; Van Den Hoogen, Seidell, Menotti, & Kromhout, 2000; Wolf–Maier et al., 2003). I agree because we know hypertension means increase pressure so it overloads the heart artery which might rupture, causes heart failure. The author found the rate of death by hypertension is different from population to populations. For instance, the rate of death in USA and Northern Europe of 10000 people were more than 70 death per year, but the rate of death in Japan and Mediterranean Southern Europe of 10000 person was more than 20 death each year (P. C. van den Hoogen et al., 2000). This is totally make sense because we know every country has own culture. If people use more salt and lipid, the rate of hypertension is high because the people get over weight so the coronary heart disease increases and vice versa in other country. It has been proven the effect of hypertension changes according to age. The effect of systolic in young person of coronary heart disease is more powerful than diastolic in middle age (Stanley S. Franklin & Wong, 2013). We know in young age, the enough relax less, sleep less and work more, so it has a negative effect on heart. The author the found the rate of hypertension with coronary heart disease is different between men and women. During 14 years of survey of 323 men and 169 women first time got coronary heart disease. It shows in the severity and killing is more common in men than in women (Kannel, Schwartz, & McNamara, 1969). We know the hypertension appear earlier in 45 years old men but in women later. The second reason the rate of smoking and drinking more common in men.

#### 3.5 INFECTION

The last factors appeared in organization is infection which are Chlamydia pneumonia, Helicobacter pylori, Cytomegalovirus.

#### 3.6 SMOKING AND SMOKING

Stroke and smoking these are the most important factors effect on coronary heart disease because these are consists of High blood pressure, Heart attack, Diabetes. These have dangerous interact with coronary heart disease.

#### 3.7 METABOLIC SYNDROME

These have dangerous interact with coronary heart disease. The role of metabolic syndrome shown in classified 10 factors which are recognized by menopause, blood lipids, and glucose tolerance.

#### 3.8 ADDITIONAL FACTORS FOR CORONARY HEART DISEASE

coverweight and obesity[33], [34], [35],smoking[33], [34], [35],smoking[35], [37], [38],Family history(Nasir et al, 2004), [39], [00].Metabolic syndrome[41], [42], [43].High density lipporotein[45],High density lipporotein[45],diastolic blood pressure (DBP) with stroke[46], [47], [32].staturated faity acids in the ditet[32]lifestyle changes reverse coronary heart disease[48](low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise)[48]Psychosocial factors[49]Menopause[50]Sex and age[51]Absence of an effect of liposuction on insulin action[53]Adiponectin[55]Adiponectin[55]Adiponectin[56]Estrogen reglacement therapy[57]Hypertrugivecridemia[56]Prognostic[56]UV-HOL-changes in subclinical hypothyroidism[61]Infection with Helicobacter pylori[62]Prognostic[66]UV-HOL-changes in subclinical phosphate common and independent reversible[71]Hyper-homocysteinemia[73]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[73]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible </th <th>Additional Factors for Coronary Heart Disease</th> <th>References</th>	Additional Factors for Coronary Heart Disease	References
smoking[31, [34, [35].Lack of physical activity[36].[37]. [38].Family history(Nair et al., 2004, [39], [40].Metabolic syndrome[41].High density lipoprotein[42].high density lipoprotein[43].diastolic blood pressure (DBP) with stroke[46].diastolic blood pressure (DBP) with stroke[46].diastolic blood pressure (DBP) with stroke[48].[16xylc changes reverse coronary heart disease[48][16xylc changes reverse coronary heart disease[49].Menopause[50]Sex and age[51].Absence of an effect of liposuction on insulin action[53].Multivariate analysis[54].Tumor necrosis factor-alpha G-308 A polymorphism[56].Adiponectin[56].Estrogen replacement therapy[57].Hypertriglyceridemia[58].Prognostic[61].UD/LHOL-changes in subclinical hypothyroidism[63].UD/LHOL-changes in subclinical hypothyroidism[63].UD/LHOL-changes in subclinical hypothyroidism[63].UD/LHOL-changes in subclinical hypothyroidism[63].UD/LHOL-changes in subclinical hypothyroidism[64].UD/LHOL-changes in subclinical hypothyroidism[63].UD/LHOL-changes in subclinical hypothyroidism[64].UD/LHOL-changes in subclinical hypothyroidism[64].UD/LHOL-changes in subclinical hypothyroidism[65].Prognostic[64].UD/LHOL-changes in subclinical hypothyroidism[65].	Overweight and obesity	[29], [30], [31], [32]
Lack of physical activity[36], [37], [38].Family history[Naisre tal., 2004.] [39], [40].Metabolic syndrome[41], [42], [43].Metabolic syndrome[41].[14] phoets hitty inportein[43]high density inty inportein[46].[14] hypercholesterolemia[43]diasolic blood pressure (DBP) with stroke[48](16w-fat vegetarian diet, stoping smoking, stress management training, and moderate exercise][48]Psychosocial factors[53]Menopause[50]Sex and age[53]Absence of an effect of liposuction on insulin action[53]Absence of an effect of liposuction on insulin action[53]Tumor necrosis factor-alpha G-308 A polymorphism[55]Estrogen replacement therapy[57]Hypertrigivectidema[53]Prognotic[56]Estrogen replacement therapy[57]Hypertrigivectidema[53]Prognotic[56]Evitage neplacement science[56]Estrogen replacement science[56]Evitage neplacement science[56]Evitage neplacement science[56]Prognotic[56]U/HOL-thangs in subclinical hypothyroidism[56]Evitage protein[56]Prognotic[56]U/HOL-thangs in subclinical hypothyroidism[56]Evitage protein[56]Prognotic[56]Evitage protein[56]Prognotic[56]Evitage protein[56]Progno	smoking	
Netabolic syndrome[41, [42], [43],High density lipoprotein[44],hypercholesterolemia[45]diastoli blood pressure (DBP) with stroke[46],sturated fatty adds in the diet[32]lifestyle changes reverse coronary heard disease[48](low-fat vegetarian diet, stopping snoking, stress management training, and moderate exercise)[49]Psychosocial factors[49]Menopause[50]Sex and age[51]Absence of an effect of liposuction on insulin action[52]Chronic Chlamydia pneumonia infection[53]Tumor encercisa factor-alpha G-308 A polymorphism[56]Catrogen replacement therapy[56]Prognostic[59]Prognostic[59]Prognostic[61]LDL/HDL-changes in subclinical hypothyroidism[61]Uifection with Helicobacter pylori[62]Vortage protein[63]Cercactive protein[63]Haportein[63]Homerysteine[66]Haporteiphoury pyriox of paraoxonase gene[67]Vest Hormones and Adrogen[71]Hyper rurenia[73]Folze and vitamin B form diet and supplements[74]Homerysteine[73]Polate and vitamine Strom diat and supplements[74]Homerysteine[73]Hoursteine and low pyridoxal phosphate common and independent reversible[73]Hoursteine and diver pyridoxal phosphate common and independent reversible[74]Homerysteine[75] <tr< td=""><td>Lack of physical activity</td><td></td></tr<>	Lack of physical activity	
Netabolic syndrome[41, [42], [43],High density lipoprotein[44],hypercholesterolemia[45]diastoli blood pressure (DBP) with stroke[46],sturated fatty adds in the diet[32]lifestyle changes reverse coronary heard disease[48](low-fat vegetarian diet, stopping snoking, stress management training, and moderate exercise)[49]Psychosocial factors[49]Menopause[50]Sex and age[51]Absence of an effect of liposuction on insulin action[52]Chronic Chlamydia pneumonia infection[53]Tumor encercisa factor-alpha G-308 A polymorphism[56]Catrogen replacement therapy[56]Prognostic[59]Prognostic[59]Prognostic[61]LDL/HDL-changes in subclinical hypothyroidism[61]Uifection with Helicobacter pylori[62]Vortage protein[63]Cercactive protein[63]Haportein[63]Homerysteine[66]Haporteiphoury pyriox of paraoxonase gene[67]Vest Hormones and Adrogen[71]Hyper rurenia[73]Folze and vitamin B form diet and supplements[74]Homerysteine[73]Polate and vitamine Strom diat and supplements[74]Homerysteine[73]Hoursteine and low pyridoxal phosphate common and independent reversible[73]Hoursteine and diver pyridoxal phosphate common and independent reversible[74]Homerysteine[75] <tr< td=""><td>Family history</td><td>(Nasir et al., 2004), [39], [40].</td></tr<>	Family history	(Nasir et al., 2004), [39], [40].
High density lipoprotein[44],hypercholestrenolemia[45]distolic blood pressure (DBP) with stroke[46], [47], [32]aturated fatty acids in the diet[32]lifestyle changes reverse coronary heart disease[48](low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise)[48]Menopause[50]Sex and age[51]Absence of an effect of liposuction on insulin action[52]Chronic Changwidg ane unonia infection[53]Multivariate analysis[54]Tumor necrosis factor-alpha G-308 A polymorphism[55]Adjonectin[56]Estrogen replacement therapy[57]Hypertridy/eridemia[58]Plasma trafyceride[59]Prognostic[60]LDU/HOL-changes in subclinical hypothyroidism[61]Turde context protein[63]C-reactive protein[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Homocysteine[74]Omega-3[74]Orega-3[75]Folder and vitamin 6 forn diet and supplements[74]Changet and yitaming endothila progenitor cells inversely correlate[75]Polare and vitaming 6 forn diet and supplements[74]Omega-3[75]Folder and yitaming endothila progenitor cells inversely correlate[76]C-reactive protein[76]Changet and vitaming endothila progenitor cells i	Metabolic syndrome	
diastolic blood pressure (DBP) with stroke[43]saturated fatty acids in the diet[32]ifestyle changes reverse coronary heart disease[48](low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise)[49]Psychosoical factors[49]Menopause[50]Exe and age[51]Absence of an effect of liposuction on insulin action[52]Chronic Chlamydia pneumonia infection[53]Multivariate analysis[54]Tumor necrosis factor-alpha G-308 A polymorphism[55]Estrogen replacement therapy[57]Pyperrigiveride[59]Prognostic[60]LU/HD/ changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]For genetic polymorphisms of paraoxonase gene[66]Four genetic polymorphisms of paraoxonase gene[66]Happer cylicopycentien[67]Eave Hormones and Androgen[68]Homocysteine[73]Foldensing and low pyridoxal phosphate common and independent reversible[71]Hyper-Inderustine and low pyridoxal phosphate common and independent reversible[73]Foldensingen gene T235 variant[76]Foldensingen gene tic 235 variant[76]Hotherald dyfunction, xidukte stress[78]Hyper-Inderustine differendie in progenitor cells inversely correlate[78]Hotherald dyfunction, xidukte stress[78]Hotherald dyfunction, xidukte stress[78]Hotherald dyfunction, xidukte stress <t< td=""><td></td><td></td></t<>		
sturated fatty acids in the diet[32]lifestyle changes reverse coronary heart disease[48](bw-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise)[48]Psychosocial factors[50]Sex and age[50]Absence of an effect of liposuction on insulin action[52]Chronic Chlamydia pneumonia infection[53]Multivariate analysis[54]Tumor necrosis factor-alpha G-308 A polymorphism[55]Adiponectin[58]Estrogen replacement therapy[57]Prognostic[60]Dup the licobacter pylori[56]Prognostic[61]Dup the licobacter pylori[62]Creactive protein[63]Creactive protein[64]Use of calcium supplements[66]Four genetic polymorphisms of paraoxonase gene[66]Four genetic polymorphisms of paraoxonase gene[66]Working hours, sleep duration[71]Hypert-indocysteinemia and low pyridoxal phosphate common and independent reversible[72]Working hours, sleep duration[73]Colate and vitamin B6 from diet and supplements[74]Orga-3[75]angiotensingene pent endities and low pyridoxal phosphate common and independent reversible[73]Polate and vitamin B6 from diet and supplements[74]Orga-3[75]angiotensingene pent 235 variant[76]Endothelial dysfunction, oxidative stress[78]Number and mingratory activity of circulating endothelial progen	hypercholesterolemia	[45]
iffestive changes reverse coronary heart disease[48](low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise)[48]Psychosocial factors[50]Menopause[51]Absence of an effect of liposuction on insulin action[53]Absence of an effect of liposuction on insulin action[53]Multivariate analysis[54]Tumor necrosis factor-alpha G-308 A polymorphism[55]Adjonectin[56]Estrogen replacement therapy[57]Hyperriglyceride[59]Pregnostic[60]LDL/HDL-changes in subclinical hypothyroidism[61]Infection with Helicobacter pylori[62]Vorsing energic enotype[63]Four genetic polymorphisms of paraoxonase gene[66]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Hormocysteinemi and long-term[71]Hyper-Inducysteinemian and long-term[73]Folate and vitami B from diet and supplements[73]Folate and vitami B from diet and supplements[73]Folate and vitami B from diet and supplements[73]Folate and vitami B from diet and supplements[74]Omega-3[75]Hyper-Inducysteinemi and low pyridoxal phosphate common and independent reversible[73]Folate and vitami B from diet and supplements[73]Folate and vitami B from diet and supplements[73]Hyper-Inducysteinemi and low pyridoxal phosphate common an	diastolic blood pressure (DBP) with stroke	[46], [47], [32]
(low-fat vegeTarian diet, stopping smoking, stress management training, and moderate exercise)[48]Psychosocial factors[49]Menopause[50]Sex and age[51]Absence of an effect of liposuction on insulin action[52]Chronic Chlamydia pneumonia infection[53]Multivariate analysis[54]Tumor necrosis factor-alpha G-308 A polymorphism[56]Adigonectin[56]Estrogen replacement therapy[57]Plasma triglyceridemia[59]Prognostic[60]LDU-HDL-changes in subclinical hypothyroidism[61]Infection with Helicobacter pylori[62]Vergenetic polymorphisms of paraoxonase gene[65]Four genetic polymorphisms of paraoxonase gene[66]Four genetic polymorphisms of paraoxonase gene[66]Vergening huining horu divola phosphate common and independent reversible[71]Hyper-Imonocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper-Imonocysteinemia and low pyridoxal phosphate common and independent reversible[73]Polate and vitamin BG from diet and supplements[76]Folate and vitamin BG from diet and supplements[76]Puttra-son graphically assessed carotid morphology[80]Ultra-son graphically assessed carotid morphology[81]Ultra-son graphically assessed carotid morphology[82]Ultra-son graphically assessed carotid morphology[83]Ultra-son graphically assessed carotid morphology[83]Ultra-son graphically assessed ca	saturated fatty acids in the diet	[32]
Psychosocial factors[49]Menopause[50]Sex and age[51]Absence of an effect of liposuction on insulin action[52]Chronic Chlamydia pneumonia infection[53]Multivariate analysis[54]Tumor nerrosis factors alpha G-308 A polymorphism[55]Adjonentin[56]Estrogen replacement therapy[57]Hypertrighveridemia[59]Prognostic[59]UD/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[63]C-reactive protein[64]UJ & Bord Calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[73]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[73]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[73]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[73]Hyper and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dictar y antioxiant flavonoid[79]Hiersen and hypothypology[81]Periad disease[82]Arterial stiffness[83]Periad disease[82]Arterial stiffness[83]Periad disease[83]Arterial stiffness[84]Calcium and poly photic disculating endothelial progenitor cells inversely correlat	lifestyle changes reverse coronary heart disease	[48]
Nenopause[50]Sex and age[51]Absence of an effect of liposuction on insulin action[52]Chronic Chlamydia pneumonia infection[53]Multivariate analysis[54]Tumor necrosis factor-alpha G-308 A polymorphism[55]Adiponectin[56]Estrogen replacement therapy[57]Hypertriglyceridemia[56]Prognostic[60]LDL/HDL-changes in subclinical hypothyroidism[61]Infection with Helicobacter pylori[62]C-reactive protein[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Four genetic polymorphisms of paraoxonase gene[66]Vorking hours, sleep duration[70]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[71]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[76]Folate and vitamin B6 from diet and supplements[76]Folate and vitamin B6 from diet and supplements[76]Folate and vitamin B6 from diet and supplements[76]Polate and vitamin B6 from diet and supplements[76]Folate and vitamin B6 from diet and supplements[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxitaft favonoidis[79]Heterogeneity[80]Ultra-soor graphically assesed carot	(low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise)	[48]
sex and age[51]Absence of an effect of liposuction on insulination[52]Absence of an effect of liposuction on insulination[53]Multivariate analysis[54]Tumor necrosis factor-alpha G-308 A polymorphism[55]Adiponectin[56]Estrogen replacement therapy[57]Hypertrig/veridemia[58]Plasma trig/veride[59]Prognostic[60]LD/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haytoglobin Genotype[67]Sex Hormones and Androgen[70]Haytoglobin Genotype[71]Working hours, sleep duration[73]Folate and vitamin B6 from diet and supplements[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Hubmer and Ingratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[83]Periodontitis[83]Periodontitis[84]Auterial stiffness[83]Periodontitis[84]Other and supplements[76]Endothelial diposity[84]Other and supplements <t< td=""><td>Psychosocial factors</td><td>[49]</td></t<>	Psychosocial factors	[49]
Absence of an effect of liposuction on insulin action[52]Chronic Chlamydia pneumonia infection[53]Multivariate analysis[54]Turnor necrosis factor-alpha G-308 A polymorphism[55]Adiponectin[56]Estrogen replacement therapy[57]Hypertriglyceridemia[58]Plasma triglyceride[59]Prognostic[60]LDU-HD-changes in subclinical hypothyroidism[61]Infection with Helicobacter pylori[62]Creactive protein[63]Creactive protein[63]Use of calcium supplements[63]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[70]Flavondi intake and long-term[71]Hyper urenia[72]Problemia for midiet and supplements[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensingen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Heterogeneity[83]Dietary antioxidant flavonoids[83]Pietard atlikease[83]Pietard atlikease[83]Pietard atlikease[83]Pietard atlikease[83]Pietard atlikease[84]Omega-3[85]Dietard atlikease[83]Pietard atlikease[83]Pietard atlikease[84]Dietard atlikease[83]	Menopause	[50]
Chronic Chlamydia pneumonia infection[53]Multivariate analysis[54]Multivariate analysis[55]Adiponettin[56]Estrogen recrosis factor-alpha 6-308 A polymorphism[57]Hypertriglyceridemia[58]Plasma triglyceride[59]Prognostic[60]LDL/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]Vort proBN[63]C-reactive protein[64]Use of calciun supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[70]ProPhonocysteinemia and low pyridoxal phosphate common and independent reversible[71]Hyper-Inomocysteinemia and low pyridoxal phosphate common and independent reversible[72]Polate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[82]Periodonitifie[83]Periodonitifie[83]Periodonitifie[83]Periodonitis[84]Calcification of the aortic arch[85]Addoninal alioposity[86]Periodonitis[86]Periodonitis[86]Periodonitis[86]	Sex and age	[51]
Multivariate analysis[54]Tumor necrosis factor-alpha G-308 A polymorphism[55]Adiponectin[56]Estrogen replacement therapy[57]Hypertriglyceridemia[58]Plasma triglyceride[59]Prognostic[60]LU/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[63]C-reactive protein[63]C-reactive protein[66]Haptoglobin Genotype[66]Homocysteine[66]Homocysteine[66]Homocysteine[67]Sex Hormones and Androgen[68]Homocysteine[71]Hyper-Iomocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antixidant flavonoids[81]Lettra disease[82]Arterial stiffness[83]Periodontitis[83]Periodontitis[83]Periodontitis[83]Periodontitis[84]Caldification of the aortic arch[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitami te Consumption[88]	Absence of an effect of liposuction on insulin action	[52]
Tumor necrosis factor-alpha G-308 A polymorphism[55]Adiponectin[56]Estrogen replacement therapy[57]Hypertriglyceridemia[58]Plasma triglyceride[59]Prognostic[60]LDL/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[70]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vispfuncturing dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antixidiant flavonoids[79]Heterogeneity[80]Uttraven graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[83]Periodontitis[84]Calcification of the aortic arch[83]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[83]Periodontitis[84]Calcification of the aortic arch[83]Arterial stiffness[83]Periodontitis[84]	Chronic Chlamydia pneumonia infection	[53]
Adiponectin[56]Estrogen replacement therapy[57]Hypertrig/veridemia[58]Plasma triglyceride[59]Prognostic[60]LD/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-thomocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental diffess[83]Periodonttis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[87]Frequent nut consumption[87]Frequent nut consumption[88] </td <td>Multivariate analysis</td> <td>[54]</td>	Multivariate analysis	[54]
Estrogen replacement therapy[57]Hypertriglyceridemia[58]Prognostic[60]LDL/HD-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[66]Use of calcium supplements[66]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocrysteine[69]Working hours, sleep duration[70]Flavonoil intake and long-term[71]Hyper-homocrysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[81]Dental disease[82]Arterial stiffness[83]Periodontitis[83]Periodontitis[84]Calcification of the arcit arch[85]Very low levels of micro albuminuria[86]Very low levels of micro albuminuria[86]Ver	Tumor necrosis factor-alpha G-308 A polymorphism	[55]
Hypertriglyceride[58]Plasma triglyceride[59]Prognostic[60]LDL/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[70]Flaxonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper-uremia[73]Foltae and vitamin B6 from diet and supplements[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antixidant flavonoids[79]Heterogeneity[81]Detradisease[82]Arterial stiffness[83]Periodonttis[83]Periodonttis[83]Periodonttis[84]Calcification of the archic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[88]	Adiponectin	[56]
Plasma triglyceride[59]Prognostic[60]LDL/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[70]Havonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[83]Periodontitis[83]Periodontitis[83]Periodontitis[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[88]	Estrogen replacement therapy	[57]
Prognostic[60]LDL/HDL-changes in subclinical hypothyroidism[61]Infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Hattoglobin Genotype[67]Sex Hormones and Androgen[68]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Foltae and vitamin B6 from diet and supplements[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Detrat diffess[82]Arterial stiffness[83]Periodontitis[83]Periodontitis[83]Periodontitis[83]Periodontitis[83]Periodontitis[85]Abdominal adiposity[86]Vitamin E consumption[86]Vitamin E consumption[86]	Hypertriglyceridemia	[58]
LDL/HDL-changes in subclinical hypothyroidism[61]infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homcocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[83]Periodontitis[83]Periodontitis[85]Abdominal adiposity[86]Very low levels of micro albuminuria[86]Very low levels of micro albuminuria[86]Vitamin E consumption[88]Vitamin E consumption[88]	Plasma triglyceride	[59]
infection with Helicobacter pylori[62]NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Habtoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[76]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[86]Vitamin E consumption[88]Vitamin E consumption[89]	Prognostic	[60]
NT-proBNP[63]C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodonttiis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Vitamin E consumption[88]Vitamin E consumption[89]	LDL/HDL-changes in subclinical hypothyroidism	
C-reactive protein[64]Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Hetterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Vitamin E consumption[88]	infection with Helicobacter pylori	
Use of calcium supplements[65]Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Ultra-son graphically assessed carotid morphology[81]Periodontitis[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[88]		[63]
Four genetic polymorphisms of paraoxonase gene[66]Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endethelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Vitamin E consumption[88]		[64]
Haptoglobin Genotype[67]Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]	Use of calcium supplements	[65]
Sex Hormones and Androgen[68]Homocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[88]		
Homocysteine[69]Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Working hours, sleep duration[70]Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]	Sex Hormones and Androgen	
Flavonoid intake and long-term[71]Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Hyper-homocysteinemia and low pyridoxal phosphate common and independent reversible[72]Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Hyper uremia[73]Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]	•	
Folate and vitamin B6 from diet and supplements[74]Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Omega-3[75]angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
angiotensinogen gene T235 variant[76]Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Endothelial dysfunction, oxidative stress[77]Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Number and migratory activity of circulating endothelial progenitor cells inversely correlate[78]Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Dietary antioxidant flavonoids[79]Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Heterogeneity[80]Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Ultra-son graphically assessed carotid morphology[81]Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Dental disease[82]Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Arterial stiffness[83]Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Periodontitis[84]Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Calcification of the aortic arch[85]Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Abdominal adiposity[86]Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Very low levels of micro albuminuria[87]Frequent nut consumption[88]Vitamin E consumption[89]		
Frequent nut consumption[88]Vitamin E consumption[89]		
Vitamin E consumption [89]		
	Fibrinogen, viscosity, and white blood cell count	[90]
Erectile dysfunction [91]		[91]

# Table 1. Additional factors for coronary heart disease

# 3.9 DISCUSSION

There is much information about roles and effects these specific factors impact on coronary heart disease. The coronary heart disease is very complicated and common disease in the world. With mutuality of risk factors, we can reduce the mortality and morbidity of coronary heart disease. The aim or important of this model could be united with our planned model. Figure two shows the relation or conjunction of the above 10 factors categorized of considered level of effects – Hypercholesterolemia, lifestyle, infection, metabolic syndrome, obesity, high density lipoprotein, lack physical activity, family history, and stroke. This compartmentalization of factors is very useful for discussion. The main purpose of this topic is to find new ways to minimize the rate of death by coronary heart disease. Actually, this disease more complicated by having many factors effects on our bodies. Only two of the top 10 factors are connected with high level of cholesterol of coronary heart disease which are hypercholesterolemia and obesity. The hypercholesterolemia focuses on HDL, LDL, and Cholesterol. In addition, obesity focused on diet, diabetes, and saturated fatty acid. Other factors in additional Table One such as Dental disease, Arterial stiffness, Periodontitis, Calcification of the aortic arch, Abdominal adiposity, Very low levels of micro albuminuria, Frequent nut consumption, Vitamin E consumption, Fibrinogen, viscosity, and white blood cell count, Erectile dysfunction, Tumor necrosis factor-alpha G-308 A polymorphism, Adiponectin Estrogen replacement therapy, Hypertriglyceridemia, Plasma triglyceride, Prognostic, LDL/HDL-changes in subclinical hypothyroidism, NT-proBNP, C-reactive protein Use of calcium supplements, Four genetic polymorphisms of paraoxonase gene, Haptoglobin Genotype Sex Hormones and Androgen, Homocysteine, Working hours, sleep duration, Flavonoid intake and long term, Hyperhomocysteinemia and low pyridoxal phosphate common and independent reversible, Hyperuricemia, Folate and vitamin B6 from diet and supplements, Omega-3, angiotensinogen gene T235 variant, Endothelial dysfunction, oxidative stress, Number and migratory activity of circulating endothelial progenitor cells inversely correlate, Dietary antioxidant flavonoids, Heterogeneity, Ultrasonographically assessed carotid morphology, (low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise), Psychosocial factors, menopause, sex , and age ranked low or have not main effects on coronary heart disease so I don't mentioned in Table Two. Although these factors are important, that research focuses on the main factors and organized factors because they have greater or direct effect on coronary heart disease. Four out 10 factors have more important effects on coronary heart disease: - lifestyle, lack physical activity, obesity, and high density lipoprotein. These have direct effect on coronary heart disease. Another factor two of the top 10 factors are stroke and smoking. These are the most important factors that affect coronary heart disease because these are consistent with High blood pressure, Heart attack, Diabetes. These have dangerous interaction with coronary heart disease. The role of metabolic syndrome shown in 10 factors which recognized by menopause, blood lipids, and glucose tolerance. The last factors appeared in organization is infection which are Chlamydia pneumonia, Helicobacter pylori, Cytomegalovirus.

# 4 CONTRIBUTION AND NEW INSIGHT

This model exemplifies the factors that work to find or minimize the risk factor of coronary heart disease. The significant thing of this model is how to minimize the rate of death by coronary heart disease. CHD is a very dangerous disease of the integration between these factors: Cholesterol, diabetes, depression, hypertension, and other factors I showed in additional tables. Indeed, to control and minimize this disease many ways have been proven. Regarding cholesterol, I prefer to decrease this factor of eating those foods that it has less or low level of for example, red meat, animal products it has high level of fat instead of fatty food intake more vegetables and fruit. Another way to decrease or prevent this factor is not to drink alcohol. However, we are doing body exercise. When viewing the act of the diabetes factor, we can prevent or minimize this risk factor by losing weight, stop smoking, and eating low red meat because cholesterol has main effect for getting diabetes. Regarding depression, depression is joined with decrease exercise capability to perform in CHD patients and connect with people who have few physical activity especially in older and domicile people [19]. Thus the depression will be controlled by more relaxing, staying away from problems or trouble, physical activity, hanging out with relative, and laughter. Actually we cannot forget the effect of hypertension. High blood pressure is well set up risk factor of coronary heart disease, for treatment high blood pressure should reduction the load of coronary heart disease and doing better health life [27]. Thus the hypertension is main risk of coronary heart disease for control. This risk we can prevent our bodies from obesity, low salt intake, exercise, maintaining healthy cholesterol, and eating more sea products such as fish. Regarding other factors as I mentioned, the main things to avoid are overweight, smoking, drinking alcohol. Eating more fruits and vegetables than those foods that have high level fats, relax and getting enough sleep, more laugh, and spend more times at gym daily. Therefore, it could reduce the death rate and risk of this disease on the humanity by finding new technique for coronary heart surgery and make equipment to reduce rate of infection during surgery and publicizing on social media about risk of coronary heart disease till people know about this terrible disease.

#### 5 CONCLUSION

Coronary heart disease is the most dangerous disease in the world wide. With this disease, the coronary artery will be constricted or narrowed thus the oxygen, blood, and other substances cannot flow to the heart completely. The common risk factors of coronary heart disease are cholesterol, diabetes, depression, and hypertension. The rate of coronary heart disease is different between man and women. On the other hand, the rate of mortality by coronary heart disease is different from place to place. In addition, the rate of getting of coronary heart disease changes according ages and in people have another disease or not. Coronary heart disease has been proven number one disease for killing people in the world especially in North Europa and North America. Family history has less effect on coronary heart disease

Cholesterol is major risk factor on coronary heart disease because the constriction in coronary artery can leads to lipid particles. As a result, it sudden death, stroke, and myocardial infraction happen. It has two types LDL and HDL, the accumulate of cholesterol caused by avoiding exercise, more eating red meat, more drink alcohol, more eating dairy products and low eating fruits and vegetables. With cholesterol many time the patient feels headache, heavily around neck, and fatigue. For control cholesterol in the body, we can take medication, exercise, and eat less animal products.

Diabetes is the very prevalent disease in the world particularly Type 2 mellitus has direct relation with coronary heart disease. If the person has diabetes the probability for getting coronary heart disease is more than the person has not diabetes about 5-7 fold higher. The rate of diabetes is higher in women than men; thus the rate of death in women is higher than men. The main reason of diabetes is overweight and other cause of diabetes avoiding exercise, more eating sugar, the problem in pancreas while it cannot produce the insulin.

Depression is the other factor of coronary heart disease. Indeed, depression has direct effect on the whole body. When the person has depression, the physiology of body could change and dysfunction happens. The main part of the body at risk is the heart. In addition, depression is related with another disease. For example, most of people who have anxiety trying drinking, smoking, and getting medication. The good things to prevent depression are relaxing, staying away from problem, and laughing.

Hypertension is highly common factor due to mortality in those patients who have coronary heart disease in the world. Hypertension has two kinds' systolic blood pressure and diastolic blood pressure. The normal range of blood sugar in body is 120/90 mm hg. Hypertension is caused by eating more salt, obesity, eating more fatty food.

Overall, coronary heart disease is prevalent disease in the world so I believe we can prevent our bodies from this disease by doing more exercise, getting good sleep, eating healthy food such as fruits, vegetables, fish and more laugh. I read proverb that said "Happy heart is better than a full purse of money." So just laugh and enjoy your life.

#### REFERENCES

- [1] Tan, Y.Y., G.-C.M. Gast, and Y.T. van der Schouw, *Gender differences in risk factors for coronary heart disease*. Maturitas, 2010. **65**(2): p. 149-160.
- [2] Castelli, W., *Epidemiology of coronary heart disease: the Framingham study.* The American journal of medicine, 1984. **76**(2): p. 4-12.
- [3] Beltrame, J.F., R. Dreyer, and R. Tavella, *Epidemiology of coronary artery disease*. Coronary artery disease-current concepts in epidemiology, pathophysiology, diagnostics and treatment, 2012: p. 1-30.
- [4] Ali, M.K., K.V. Narayan, and N. Tandon, *Diabetes & coronary heart disease: current perspectives.* The Indian journal of medical research, 2010. **132**(5): p. 584.
- [5] Hertzer, N.R., et al., *Coronary artery disease in peripheral vascular patients. A classification of 1000 coronary angiograms and results of surgical management.* Annals of surgery, 1984. **199**(2): p. 223.
- [6] Wenger, N.K., *Coronary heart disease: the female heart is vulnerable.* Progress in Cardiovascular Diseases, 2003. **46**(3): p. 199-229.
- [7] Turpeinen, O., *Effect of cholesterol-lowering diet on mortality from coronary heart disease and other causes.* Circulation, 1979. **59**(1): p. 1-7.
- [8] Troisi, A., *Cholesterol in coronary heart disease and psychiatric disorders: same or opposite effects on morbidity risk?* Neuroscience & Biobehavioral Reviews, 2009. **33**(2): p. 125-132.
- [9] Ebrahim, S., et al., Cholesterol and coronary heart disease: screening and treatment. Quality in health care: QHC, 1998.
  7(4): p. 232.
- [10] Després, J.-P., et al., *HDL-cholesterol as a marker of coronary heart disease risk: the Quebec cardiovascular study.* Atherosclerosis, 2000. **153**(2): p. 263-272.
- [11] Varbo, A., et al., *Remnant cholesterol as a causal risk factor for ischemic heart disease*. Journal of the American College of Cardiology, 2013. **61**(4): p. 427-436.

- [12] Cromwell, W.C., *High-density lipoprotein associations with coronary heart disease: Does measurement of cholesterol content give the best result?* Journal of clinical lipidology, 2007. **1**(1): p. 57-64.
- [13] Saely, C.H. and H. Drexel, *Is type 2 diabetes really a coronary heart disease risk equivalent?* Vascular pharmacology, 2013. **59**(1): p. 11-18.
- [14] Hu, F.B., et al., *The impact of diabetes mellitus on mortality from all causes and coronary heart disease in women: 20 years of follow-up.* Archives of Internal Medicine, 2001. **161**(14): p. 1717-1723.
- [15] Bulugahapitiya, U., et al., *Is diabetes a coronary risk equivalent? Systematic review and meta-analysis.* Diabetic Medicine, 2009. **26**(2): p. 142-148.
- [16] Lee, W.L., et al., Impact of diabetes on coronary artery disease in women and men: a meta-analysis of prospective studies. Diabetes care, 2000. **23**(7): p. 962-968.
- [17] Haffner, S.M., et al., Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. New England Journal of Medicine, 1998. **339**(4): p. 229-234.
- [18] Carney, R.M. and K.E. Freedland, *Depression in patients with coronary heart disease*. The American journal of medicine, 2008. **121**(11): p. S20-S27.
- [19] Carney, R.M., et al., *Depression as a risk factor for cardiac mortality and morbidity: a review of potential mechanisms.* Journal of psychosomatic research, 2002. **53**(4): p. 897-902.
- [20] Goldston, K. and A.J. Baillie, *Depression and coronary heart disease: a review of the epidemiological evidence, explanatory mechanisms and management approaches.* Clinical psychology review, 2008. **28**(2): p. 288-306.
- [21] Rugulies, R., Depression as a predictor for coronary heart disease: a review and meta-analysis1 1The full text of this article is available via AJPM Online at www. ajpm-online. net. American journal of preventive medicine, 2002. 23(1): p. 51-61.
- [22] Rowan, P.J., et al., *Depressive symptoms have an independent, gradient risk for coronary heart disease incidence in a random, population-based sample.* Annals of epidemiology, 2005. **15**(4): p. 316-320.
- [23] Poole, L., C. Dickens, and A. Steptoe, *The puzzle of depression and acute coronary syndrome: reviewing the role of acute inflammation*. Journal of psychosomatic research, 2011. **71**(2): p. 61-68.
- [24] Perreault, S., et al., *Impact of treating hyperlipidemia or hypertension to reduce the risk of death from coronary artery disease.* Canadian Medical Association Journal, 1999. **160**(10): p. 1449-1455.
- [25] van den Hoogen, P.C., et al., *The relation between blood pressure and mortality due to coronary heart disease among men in different parts of the world.* New England Journal of Medicine, 2000. **342**(1): p. 1-8.
- [26] Franklin, S.S. and N.D. Wong, *Hypertension and Cardiovascular Disease: Contributions of the Framingham Heart Study.* Global Heart 2013. **8**(1): p. 49-57.
- [27] Franklin, S.S., et al., *Does the relation of blood pressure to coronary heart disease risk change with aging? The Framingham Heart Study.* Circulation, 2001. **103**(9): p. 1245-1249.
- [28] Kannel, W.B., M.J. Schwartz, and P.M. McNamara, *Blood pressure and risk of coronary heart disease: the Framingham study.* CHEST Journal, 1969. **56**(1): p. 43-52.
- [29] Manson, J.E., et al., A prospective study of obesity and risk of coronary heart disease in women. New England Journal of Medicine, 1990. **322**(13): p. 882-889.
- [30] Bibbins-Domingo, K., et al., *Adolescent overweight and future adult coronary heart disease*. New England Journal of Medicine, 2007. **357**(23): p. 2371-2379.
- [31] Li, T.Y., et al., *Obesity as compared with physical activity in predicting risk of coronary heart disease in women.* Circulation, 2006. **113**(4): p. 499-506.
- [32] Keys, A., Coronary heart disease in seven countries. Circulation, 1970. 41(1): p. 186-195.
- [33] Critchley, J.A. and S. Capewell, Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. Jama, 2003. **290**(1): p. 86-97.
- [34] He, J., et al., *Passive smoking and the risk of coronary heart disease—a meta-analysis of epidemiologic studies.* New England Journal of Medicine, 1999. **340**(12): p. 920-926.
- [35] Doyle, J.T., et al., *The relationship of cigarette smoking to coronary heart disease: The second report of the combined experience of the Albany, NY, and Framingham, Mass, studies.* Jama, 1964. **190**(10): p. 886-890.
- [36] Taylor, R.S., et al., *Exercise-based rehabilitation for patients with coronary heart disease: systematic review and metaanalysis of randomized controlled trials.* The American journal of medicine, 2004. **116**(10): p. 682-692.
- [37] Powell, K.E., et al., *Physical activity and the incidence of coronary heart disease*. Annual review of public health, 1987.
  8(1): p. 253-287.
- [38] Rodriguez, B.L., et al., *Physical activity and 23-year incidence of coronary heart disease morbidity and mortality among middle-aged men. The Honolulu Heart Program.* Circulation, 1994. **89**(6): p. 2540-2544.
- [39] Silberberg, J.S., et al., Risk Associated with Various Definitions of Family History of Coronary Heart Disease The Newcastle Family History Study II. American journal of epidemiology, 1998. 147(12): p. 1133-1139.

- [40] Marenberg, M.E., et al., *Genetic susceptibility to death from coronary heart disease in a study of twins*. New England Journal of Medicine, 1994. **330**(15): p. 1041-1046.
- [41] Lakka, H.-M., et al., *The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men.* Jama, 2002. **288**(21): p. 2709-2716.
- [42] Wannamethee, S.G., et al., *Metabolic syndrome vs Framingham Risk Score for prediction of coronary heart disease, stroke, and type 2 diabetes mellitus.* Archives of Internal Medicine, 2005. **165**(22): p. 2644-2650.
- [43] Sattar, N., et al., *Metabolic syndrome with and without C-reactive protein as a predictor of coronary heart disease and diabetes in the West of Scotland Coronary Prevention Study*. Circulation, 2003. **108**(4): p. 414-419.
- [44] Gordon, T., et al., *High density lipoprotein as a protective factor against coronary heart disease: the Framingham Study.* The American journal of medicine, 1977. **62**(5): p. 707-714.
- [45] Group, S.S.S.S., Randomised trial of cholesterol lowering in 4444 patients with coronary heart disease: the Scandinavian Simvastatin Survival Study (4S). The Lancet, 1994. **344**(8934): p. 1383-1389.
- [46] MacMahon, S., et al., Blood pressure, stroke, and coronary heart disease: part 1, prolonged differences in blood pressure: prospective observational studies corrected for the regression dilution bias. The Lancet, 1990. 335(8692): p. 765-774.
- [47] Wilson, P.W., et al., *Prediction of coronary heart disease using risk factor categories*. Circulation, 1998. **97**(18): p. 1837-1847.
- [48] Ornish, D., et al., Can lifestyle changes reverse coronary heart disease?: The Lifestyle Heart Trial. The Lancet, 1990.
  336(8708): p. 129-133.
- [49] Haynes, S., M. Feinleib, and W.B. Kannel, *The relationship of psychosocial factors to coronary heart disease in the Framingham Study. III. Eight-year incidence of coronary heart disease.* American journal of epidemiology, 1980. 111(1): p. 37-58.
- [50] Matthews, K.A., et al., *Menopause and risk factors for coronary heart disease*. New England Journal of Medicine, 1989.
  321(10): p. 641-646.
- [51] Jousilahti, P., et al., *Sex, age, cardiovascular risk factors, and coronary heart disease A prospective follow-up study of 14* 786 middle-aged men and women in Finland. Circulation, 1999. **99**(9): p. 1165-1172.
- [52] Klein, S., et al., *Absence of an effect of liposuction on insulin action and risk factors for coronary heart disease*. New England Journal of Medicine, 2004. **350**(25): p. 2549-2557.
- [53] Saikku, P., et al., *Chronic Chlamydia pneumoniae infection as a risk factor for coronary heart disease in the Helsinki Heart Study*. Annals of internal medicine, 1992. **116**(4): p. 273-278.
- [54] Wilhelmsen, L., H. Wedel, and G. TIBBLIN, *Multivariate analysis of risk factors for coronary heart disease*. Circulation, 1973. **48**(5): p. 950-958.
- [55] Chu, H., et al., *Tumor necrosis factor-alpha G-308 A polymorphism and risk of coronary heart disease and myocardial infarction: A case–control study and meta-analysis.* Journal of Cardiovascular Disease Research, 2012. **3**(2): p. 84-90.
- [56] Ai, M., et al., Adiponectin: An independent risk factor for coronary heart disease in men in the Framingham offspring Study. Atherosclerosis, 2011. **217**(2): p. 543-548.
- [57] Stampfer, M.J. and G.A. Colditz, *Estrogen replacement therapy and coronary heart disease: a quantitative assessment of the epidemiologic evidence.* Preventive medicine, 1991. **20**(1): p. 47-63.
- [58] Fontbonne, A., et al., *Hypertriglyceridaemia as a risk factor of coronary heart disease mortality in subjects with impaired glucose tolerance or diabetes.* Diabetologia, 1989. **32**(5): p. 300-304.
- [59] Austin, M.A., *Plasma triglyceride as a risk factor for coronary heart disease. The epidemiologic evidence and beyond.* American journal of epidemiology, 1989. **129**(2): p. 249-259.
- [60] Schächinger, V., M.B. Britten, and A.M. Zeiher, *Prognostic impact of coronary vasodilator dysfunction on adverse longterm outcome of coronary heart disease.* Circulation, 2000. **101**(16): p. 1899-1906.
- [61] Althaus, B., et al., LDL/HDL-CHANGES IN SUBCLINICAL HYPOTHYROIDISM: POSSIBLE RISK FACTORS FOR CORONARY HEART DISEASE. Clinical endocrinology, 1988. 28(2): p. 157-163.
- [62] Danesh, J. and R. Peto, *Risk factors for coronary heart disease and infection with Helicobacter pylori: meta-analysis of 18 studies.* Bmj, 1998. **316**(7138): p. 1130-1132.
- [63] Sattar, N., et al., *NT-proBNP is associated with coronary heart disease risk in healthy older women but fails to enhance prediction beyond established risk factors: Results from the British Women's Heart and Health Study.* Atherosclerosis, 2010. **209**(1): p. 295-299.
- [64] Mojiminiyi, O.A., et al., Association of C-reactive protein with coronary heart disease risk factors in patients with type 2 diabetes mellitus. Diabetes Research and Clinical Practice, 2002. **58**(1): p. 37-44.
- [65] Pentti, K., et al., Use of calcium supplements and the risk of coronary heart disease in 52–62-year-old women: The Kuopio Osteoporosis Risk Factor and Prevention Study. Maturitas, 2009. **63**(1): p. 73-78.

- [66] Wang, M., et al., Four genetic polymorphisms of paraoxonase gene and risk of coronary heart disease: A meta-analysis based on 88 case–control studies. Atherosclerosis, 2011. **214**(2): p. 377-385.
- [67] Cahill, L.E., et al., *Haptoglobin Genotype Is a Consistent Marker of Coronary Heart Disease Risk Among Individuals With Elevated Glycosylated Hemoglobin.* Journal of the American College of Cardiology, 2013. **61**(7): p. 728-737.
- [68] Cao, J., et al., Sex Hormones and Androgen Receptor: Risk Factors of Coronary Heart Disease in Elderly Men. Chinese Medical Sciences Journal, 2010. **25**(1): p. 44-49.
- [69] Shai, I., et al., *Homocysteine as a risk factor for coronary heart diseases and its association with inflammatory biomarkers, lipids and dietary factors.* Atherosclerosis, 2004. **177**(2): p. 375-381.
- [70] Cheng, Y., et al., Working hours, sleep duration and the risk of acute coronary heart disease: A case-control study of middle-aged men in Taiwan. International Journal of Cardiology, 2014. 171(3): p. 419-422.
- [71] Hertog, M.G., et al., *Flavonoid intake and long-term risk of coronary heart disease and cancer in the seven countries study.* Archives of Internal Medicine, 1995. **155**(4): p. 381.
- [72] Robinson, K., et al., *Hyperhomocysteinemia and low pyridoxal phosphate common and independent reversible risk factors for coronary artery disease*. Circulation, 1995. **92**(10): p. 2825-2830.
- [73] Brand, F., et al., *Hyperuricemia as a risk factor of coronary heart disease: The Framingham Study.* American journal of epidemiology, 1985. **121**(1): p. 11-18.
- [74] Rimm, E.B., et al., Folate and vitamin B6 from diet and supplements in relation to risk of coronary heart disease among women. Jama, 1998. **279**(5): p. 359-364.
- [75] Harris, W.S. and C. von Schacky, *The Omega-3 Index: a new risk factor for death from coronary heart disease?* Preventive medicine, 2004. **39**(1): p. 212-220.
- [76] Katsuya, T., et al., Association of angiotensinogen gene T235 variant with increased risk of coronary heart disease. The Lancet, 1995. **345**(8965): p. 1600-1603.
- [77] Heitzer, T., et al., *Endothelial dysfunction, oxidative stress, and risk of cardiovascular events in patients with coronary artery disease.* Circulation, 2001. **104**(22): p. 2673-2678.
- [78] Vasa, M., et al., Number and migratory activity of circulating endothelial progenitor cells inversely correlate with risk factors for coronary artery disease. Circulation research, 2001. **89**(1): p. e1-e7.
- [79] Hertog, M.G., et al., Dietary antioxidant flavonoids and risk of coronary heart disease: the Zutphen Elderly Study. The Lancet, 1993. **342**(8878): p. 1007-1011.
- [80] Bhopal, R., et al., *Heterogeneity of coronary heart disease risk factors in Indian, Pakistani, Bangladeshi, and European origin populations: cross sectional study.* Bmj, 1999. **319**(7204): p. 215-220.
- [81] Salonen, J.T. and R. Salonen, *Ultrasonographically assessed carotid morphology and the risk of coronary heart disease.* Arteriosclerosis, Thrombosis, and Vascular Biology, 1991. **11**(5): p. 1245-1249.
- [82] DeStefano, F., et al., Dental disease and risk of coronary heart disease and mortality. BMJ: British Medical Journal, 1993.
  **306**(6879): p. 688.
- [83] Mattace-Raso, F.U., et al., *Arterial stiffness and risk of coronary heart disease and stroke the rotterdam study*. Circulation, 2006. **113**(5): p. 657-663.
- [84] Beck, J.D., et al., *Periodontitis: a risk factor for coronary heart disease?* Annals of periodontology, 1998. **3**(1): p. 127-141.
- [85] Iribarren, C., et al., *Calcification of the aortic arch: risk factors and association with coronary heart disease, stroke, and peripheral vascular disease.* Jama, 2000. **283**(21): p. 2810-2815.
- [86] Rexrode, K.M., et al., Abdominal adiposity and coronary heart disease in women. Jama, 1998. 280(21): p. 1843-1848.
- [87] Klausen, K., et al., Very low levels of microalbuminuria are associated with increased risk of coronary heart disease and death independently of renal function, hypertension, and diabetes. Circulation, 2004. **110**(1): p. 32-35.
- [88] Hu, F.B., et al., *Frequent nut consumption and risk of coronary heart disease in women: prospective cohort study.* Bmj, 1998. **317**(7169): p. 1341-1345.
- [89] Rimm, E.B., et al., Vitamin E consumption and the risk of coronary heart disease in men. New England Journal of Medicine, 1993. 328(20): p. 1450-1456.
- [90] Yarnell, J., et al., *Fibrinogen, viscosity, and white blood cell count are major risk factors for ischemic heart disease. The Caerphilly and Speedwell collaborative heart disease studies.* Circulation, 1991. **83**(3): p. 836-844.
- [91] Feldman, H.A., et al., *Erectile dysfunction and coronary risk factors: prospective results from the Massachusetts male aging study.* Preventive medicine, 2000. **30**(4): p. 328-338.