Avicenna Journal of Neuro Psycho Physiology
doi: 10.32592/ajnpp.2023.10.4.102 2023 November;10(4): 145-157 https://ajnpp.umsha.ac.ir

Original Article

Modeling Psychosocial Factors Affecting the Severity of Coronary Heart Disease: The Mediating Role of Physiological Responses and Health-related Behaviors

Bahman Lotfikhah[1], Ahmad Alipour[2*], Majid Saffarinia[2], Mehrnoosh Broumandpour[3]

[1] PhD candidate in health psychology, Payame Noor University, Tehran, Iran
[2] Professor, Psychology Department, Payame Noor University, Tehran, Iran
[3] Assistant Professor, Department of Cardiology, Islamic Azad University, Tehran, Iran

*Corresponding author:
Ahmad Alipour, Psychology Department, Payame Noor University, Tehran, Iran.
Tel: +989125391394
Email: alipor@pnu.ac.ir

Received: 02 Nov 2023
Accepted: 04 Jan 2024
ePublished: 13 May 2024

Abstract

Background and Objective: Today, cardiovascular diseases (CVDs) remain a leading cause of mortality. They rank first across the globe and are recognized as a multifactorial disorder, one of which is psychosocial factors. The current study aimed to determine the role of the aforementioned factors in coronary heart disease mediated by physiological responses and health-related behaviors.

Materials and Methods: This descriptive-correlational study was performed on 270 men and women who were referred to the Department of Cardiology of Taleghani, Modares, and Farhikhtegan hospitals in Tehran and were selected via the Convenience sampling method. The research instruments were Depression, Anxiety and Stress Scale -21 Items (DASS-21), perceived social support, hostility, type D personality, and adherence to treatment. To assess social variables, the questionnaire of social support, socioeconomic status, quality of life, social comparison, and psychological response was used. Finally, to examine health-related behaviors, questionnaires related to diet, smoking, and physical activity were used. The level of physiological responses of blood lipids was based on blood test results, and the severity of coronary heart disease was measured by coronary angiography. The causal relationship between variables and the research questions was examined using structural equation modeling, particularly through path analysis within the Lisrel software for the measurement model.

Results: Based on the findings, it is evident that psychosocial factors have a significant impact on the severity of coronary heart disease. This impact is partly mediated by physiological responses and blood lipids. Therefore, it is important to consider psychosocial factors, unhealthy behaviors, and blood lipids in the treatment of coronary heart disease. The squared multiple correlations in the structural model of psychological factors account for 25.33% of the variance in coronary heart disease. This indicates that psychological factors, physiological responses, and unhealthy behaviors collectively explain 25.33% of the variance. Specifically, psychological factors explain 41.8% of the variance in physiological responses and 18.49% of the variance in unhealthy behaviors. Additionally, physiological responses account for 10.89% of the variance in coronary heart disease, while unhealthy behaviors explain 14.14% of its variance.

Conclusions: ???

Keywords: Blood lipids, Coronary heart disease, Psychological factors, Social factors, Unhealthy behaviors

Background

Today, cardiovascular diseases (CVDs) are major causes of death, ranking first across the globe [1]. Social, cultural, and industrial changes have modified the pattern of diseases, and as a result, chronic diseases are recognized as the most important health and psychological problems among people [2]. Therefore, chronic diseases are considered a major source of stress, imposing huge financial costs on societies. One of the main public issues identified by the World Health Organization (WHO)[1] is the rising trend of chronic diseases at the global level, with an estimated one-third of all deaths from cardiovascular diseases.

The burden of these heart diseases is very high, especially in middle and high-income countries [1]. This high prevalence of heart disease typically has significant financial consequences, such as the cost of disease management and loss of income [3]. Coronary heart disease (CHD)[1] is one of the rapidly growing diseases [4] and is known as a multifactorial disorder for which more than 250 risk factors have been identified so far [5]. In fact, since cardiovascular diseases are considered a group of psychosomatic diseases, a combination of social,

biological, and psychological should be considered when investigating the effective factors in the occurrence of these diseases.

Since psychosocial factors and cardiovascular health are closely related to each other, the identification of psychosocial factors and this relationship is of utmost importance. In addition, cardiovascular disease is one of the most common heart complications and is known as a chronic, progressive, and debilitating disorder [6]. This disease, which may be caused by the narrowing and blockage of the coronary arteries, is the most important cardiovascular disorder and health problem in developing and developed countries.

As a result of rapid socioeconomic developments in recent years in many Eastern Mediterranean and Middle Eastern countries, including Iran, cardiovascular diseases are regarded as a major health and social problem, the dimensions of which are increasing rapidly [7]. Currently, heart disease is recognized as a major cause of hospitalization and death across the globe [8].

Understanding the integration of interactions between multiple psychological and biological factors in regulating the cardiovascular system and developing cardiovascular disorders is a daunting challenge posed to future research. Psychological and social factors significantly affect the cardiovascular system and play a major role in the pathogenesis of cardiovascular disorders. Over the past few decades, assiduous attention has been devoted to psychosocial and behavioral factors in cardiovascular diseases. In addition to patients' physical health, cardiovascular diseases affect their social relationships, lifestyle, family atmosphere, job, and income level.

Based on previous studies, psychosocial risk factors (PSRFs) have a role to play in both the risk of the onset of CHD events, cardiac events, and quality of life. the relative risk of cardiac events and mortality is affected by socioeconomic status, social isolation, lack of social support, acute and chronic stress, depression, anxiety, or other mental disorders (1.2-2.0). [9-11].

Apart from physical problems, cardiovascular diseases can cause significant mental disorders, such as anxiety and depression. These complications, along with the chronic, progressive, and irreversible nature of the disease, have numerous negative consequences for patients, affecting all aspects of their personal and social lives. They are considered one of the plausible reasons for a dramatic decline in their quality of life [12,13]. In addition, there is strong evidence that smoking, physical inactivity, obesity, unhealthy diet, and poor medication adherence increase the risk of cardiovascular disease and increase cardiovascular risk factors, such as hypertension, abnormal levels of blood lipids, insulin resistance, and diabetes [14].

Coronary heart disease is the result of the convergence of several risk factors [15]. Age, gender, family history, and race are among the risk factors that cannot be corrected. On the other hand, blood pressure, diabetes, blood lipids, such as cholesterol, low density, high-density cholesterol, triglyceride, and total cholesterol, obesity, social isolation, low social support and limited social networks, low physical activity, nutrition (food and food additives, including salt, sugar, and fatty acids), alcohol abuse, and smoking are among the risk factors that can be modified for coronary heart disease [16- 20].

Theoretical foundations and research background

Stress is one of the fundamental psychological concepts that disrupt a person's performance in social, psychological, physical, or physiological domains. This word, which entered the field of psychology from physics, has taken on a psychological meaning. Stress is commonly used to describe negative responses and emotions that uncertain changes that affect a biological system." Selye, in his latest conceptualization, considered stress an uncertain response to any environmental demand [21].

This disorder (depression) affects a person's mood with a constant feeling of sadness, restlessness, social isolation, and suicidal thoughts. In addition, it can have physical consequences in terms of disturbed sleep, energy, and appetite [22]. The American Psychological Association defines anxiety as "feelings characterized by tension, anxious thoughts, and physical changes, such as increased blood pressure" [23]. It is defined as a stable trait that is characterized by emotional, cognitive, and behavioral sub-components. Cognitive factors include pessimism, mistrust, and frequent malicious interpretations of others' behavior. The emotional components include anger, and the behavioral component encompasses aggressive verbal and non-verbal behaviors [24]. It is a set of relatively stable and unique characteristics that may change in response to different situations [25].

Background and research literature

Past research has identified several demographic predictors of unhealthy behaviors. For instance, several studies have pinpointed that men follow medical advice to a lesser extent than women [26; 27], while married patients follow the advice to a greater extent than single ones [28]. In the study by Jahangirpour et al. (2013) and in another research

by Vella and Friedman (2009), hostility and were recognize as the risk factors of coronary heart disease [29,30]. Nonetheless, some studies did not link these characteristics and coronary heart disease [31]. The results of several studies actually demonstrated that psychological reaction can be defined as a state consisting of affective, cognitive, and motivational components[32-35], such as anger, which is both motivational and emotional [36] and negative cognitions

Research findings emphasize that different behavioral styles, such as unhealthy diet, lack of physical activity, and smoking, are related to the onset and spread of symptoms of coronary heart disease [37]. A peer-reviewed meta-analysis illustrated that smoking leads to increased mortality while reducing smoking after cardiac surgery increases life expectancy [38]. Smoking alone is a major risk factor for CVD [39, 40]; moreover, its combination with other risk factors aggravates cardiovascular diseases. In the study by Libin Tan et al., it has been reported that the risk of cardiovascular disease was higher in hypertensive smokers than in non-smokers [41].

Several other studies have also demonstrated that smoking is a potential risk factor for CVD, and its combined effect with hypertension increases the risk of stroke [41-43]. In addition to hypertension, smoking increases the risk of vascular diseases [44]. Considerable biological evidence suggests that cigarette smoke leads to endothelial damage, cellular dysfunction, arteriosclerosis, acute thrombosis, and a decrease in the oxygen-carrying ability of the blood.

Base on a recent study, a major part (59.0%) of the relationship between coronary heart disease and physical activity can be explained by differences in known risk factors. The findings of the study by Thomas et al. (2020) demonstrated that inactivity, along with low consumption of fruits and vegetables, is the most common behavioral combination for cardiovascular disease. In addition, most smokers (88%) reported three or four high-risk behaviors [45].

A sedentary lifestyle is also a major behavioral risk factor for ischemic heart disease. The results of this study (Jebermedin and Zeberkhrestos, 2021) pointed out that patients without regular physical activity were more susceptible to ischemic heart diseases. The odds of suffering from ischemic heart disease due to physical inactivity were 2.23 times higher than that of the control groups (AOR = 2.23, 95% CI = 1.32–3.76). This result is comparable to studies in the United States of America, Saudi Arabia, and Germany [46-48].

Physical activity elicits some endogenous and paracrine responses associated with cardiometabolic health. This has been largely attributed to exercise-induced changes in white adipose tissue and skeletal muscle phenotype and secretion. Muscle and white adipose tissue influence interorgan crosstalk throught the mediating role of adipokines, myokines, and gaseous messengers, such as nitric oxide. Physical activity, especially resistance training, affects body composition, promoting an anti-atherogenic and anti-inflammatory context [49-51].

Enhanced endothelial function is another mechanism related to cardiovascular health [52]. In this regard, we can also refer to the increased autonomic balance, reduced pathways related to nutrient sensing and growth signaling, as well as the maintenance of a stable phenotype of pre-existing atherosclerotic plaque [51]. Another mediated pathway that reduces cardiovascular disease risk is the antidepressant effect of physical activity [53-55]. A sedentary lifestyle causes obesity, increasing insulin resistance, blood glucose levels, plasma lipids, and prothrombotic factors [56]. Among the other mechanisms associated with depression and heart disease, we can refer to platelet-endothelial interaction, inflammation, neurohumoral activation, autonomic imbalance, and serotonin transporter gene polymorphism.

Objectives

The current study aimed to determine the role of the aforementioned factors in coronary heart disease mediated by physiological responses and health-related behaviors.

Materials and Methods

This descriptive-correlational study was conducted on men and women referred to the Department of Cardiology of Taleghani, Modarres, and Farhikhtegan hospitals in Tehran, as well as the Department of Cardiology of Imam Khomeini Hospital in Ardabil from April 2022-March 2023. The participants were selected via the convenience sampling method. The sample size was selected based on "structural equation modeling" (SEM). Some researchers consider a minimum sample size of 100 in a study with structural equation modeling and regard a sample size of 200 or more as desirable [57].

The inclusion criteria were as follows: favorable clinical conditions of the subject, performing angiography or having an angiography report, and the presence of blood test results in the patient's file (specifically, blood lipids). On the other hand, the emergence of unusual medical and psychiatric conditions during the implementation of the research was considered the exclusion criterion. Five individuals (researcher and research assistant trained and educated in the field of psychology) were involved in the implementation of this study.

Firstly, appropriate communication was made with the subjects; thereafter, explanations were provided about the objectives of the research, and after obtaining the subject's written consent, the questionnaire was completed.

The collection of research data consisted of several parts which were followed for all subjects. The demographic characteristics form was completed by the researcher. The purpose of this part was to establish proper communication and encourage the subject to continue cooperation. The questionnaires related to psychological and behavioral factors were completed by the patient and, in case of inability, with the help of the researcher. The level of blood lipids was extracted from the patient's file. After completing the questionnaires, the angiography report was extracted from the information center, research center, cardiology centers, or medical records of the relevant hospitals using the patient's file number. Data analysis was performed using the structural equation modeling method. The existing causal relationships between the latent variables and the amount of variance explained in the entire model was investigated and determined after confirmatory factor analysis.

Results

Descriptive indicators

Sample description

The participants encompassed 163 (60.4%) men and 107 (39.6%)women. The mean age scores of male and female participants were reported as 59.85±10.51 and 61.90±10.53 years, respectively. Additional information related to demographic variables based on gender is reported in Table 1.

Descriptive indicators of research variables

Table 2 displays the descriptive indices of research variables.

Table 1. Demographic information of research participants by gender

Variable	Variable levels	Males		Females	
		Frequency	Percentage	Frequency	Percentage
Education	Illiterate	18	11%	34	31.8%
	reading and writing literacy	13	8%	12	11.2%
	Primary school	23	%14.1	23	21.5%
	Junior high school	25	15.3%	8	7.5%
	Senior high school	7	4.3%	1	0.9%
	Diploma	41	25.2%	12	11.2%
	Academic education	33	20.2%	13	12.1%
	Missing data	3	1.8%	4	3.7%
Marital status	Single	6	3.7%	5	4.7%
	Married	141	86.5%	57	53.3%
	Divorced	8	4.9%	0	٠
	Widowed	4	2.5%	42	39.3%
	Missing data	4	2.5%	3	2.8%
Occupation	Employed	88	54%	17	15.9%
	Student	2	1.2%	0	0
	Housewife	0	٠	82	76.6%
	Retired	46	28.2%	2	1.9%
	Supported by charity organizations	12	7.4%	5	47%
	Decrepit	9	5.5%	1	0.9%
	Missing data	6	3.7%	0	0
State of residence	Private	133	81.6%	80	74.8%
	Rental	18	11%	16	15%
	Homeless	0	0	2	1.9%
	Missing data	12	7.4%	9	8.4%

Table 2. Descriptive indices of research variables

	Minimum	Maximum	Mean	Standard deviation	Skewness	kurtosis
Depression	0	40	17.22	7.14	0.08	0.30
Anxiety	2	40	20.22	6.32	-0.29	0.55
Stress	0	42	24.99	8.03	-0.62	0.47
Hostility	1	20	6.62	3.21	0.34	0.51
type D personality	5	42	29.54	5.46	-0.35	1.47
Negative affect subscale	0	28	15.63	5.20	-0.21	-0.37
Social inhibition subscale	5	21	14.03	2.34	0.07	0.66
social support	4	60	38.40	7.19	-0.49	2.57
Adherence to treatment	0	10	5.51	1.79	-0.05	0.26
Quality of Life	15	43	30.54	3.71	-0.773	4.01
Psychological reaction	7	63	41.21	9.23	-0.52	0.60
Responsiveness subscale	1	19	11.34	3.25	-0.32	0.18
Resistance to social influence	0	18	11.50	3.14	-0.36	0.60
Reaction to advice	0	10	4.85	1.72	0.15	0.95
Emotional response to choice constraints subscale	6	19	13.54	2.74	-0.73	0.28
Social comparison	13	49	32.79	7.80	-0.127	-1.27

	Minimum	Maximum	Mean	standard deviation	Skewness	kurtosis
TC	124	370	203.24	37.14	0.707	0.922
TG	40	359	204.36	50.81	0.614	0.145
HDL	20	89	37.51	8.33	1.42	5.17
LDL	70	205	123.13	26.11	0.45	-0.170
unhealthy diet	1	36	14.77	4.67	0.72	2.10
Unhealthy additives	9	24	17.05	2.78	-0.66	0.47
Socio-Economic Status (SES)	5	20	10.81	2.21	1.00	3.13
smoking (number of cigarettes)	0	60	4.59	8.40	2.31	7.39
Physical activity (doing and not doing)	0	1	0.43	0.50	0.30	-1.92
Coronary heart disease	0	90	61.23	16.14	-0.150	0.612

Table 2, in addition to the mean and standard deviation of research variables, illustrates their skewness and kurtosis.

inferential findings

Examination of research questions

Main question

1. Do psychological factors have an effect on coronary heart disease through the mediating role of physiological responses and health-related behaviors?

In this project, the structural equation modeling method was used to answer the research questions. This method consists of two important parts:

A) Measurement model

b) Structural model

For this reason, SEM analysis includes two main steps: structural model analysis through path analysis and examining the measurement model through confirmatory factor analysis. Considering that most researchers suggest that the measurement model should be evaluated before the structural modeling, the researcher carried out these steps in order.

Research measurement model

The measurement model defines the measurement of latent variables through observed variables. In the measurement model of the research (figures 1, 2, 3, and 4), 19 indicators are considered to reflect four latent structures. According to Figure 1, it is assumed that indicators of depression, stress, anxiety, hostility, type D personality, and compliance with treatment measure the latent variable of psychological factors. According to Figure 2, it is assumed that indicators of social support, socioeconomic status, social comparison, psychological reaction, and quality of life measure the latent variable of social factors.

According to Figure 3, it is assumed that the indicators of smoking, unhealthy diet, inactivity, and unhealthy additives measure the latent variable of unhealthy behaviors. Finally, according to Figure 4, it is assumed that the indicators of HDL, LDL, triglycerides, and total cholesterol measure the latent variable of physiological responses. Now the question is, to what extent does the measurement model of the current research "fit" the collected data? The goodness of fit of this model is very important since it encompasses all the latent variables of the structural equation modeling.

The measurement model was performed by path analysis using Lisrel software. Chi-square examination illustrated that the latent variable of psychological factors was measured with indicators of depression, stress, anxiety, hostility, type D personality, and adherence to treatment (non-adherence to treatment). Figure 1 illustrates the factor loadings related to the measurement model of psychological factors. All coefficients are significant at P>0.01. The fit indices of this model are also reported in Table 2. All indicators point to the appropriate fit of the measurement model.

Therefore, based on the fit indices obtained from the path analysis in the evaluation of the research measurement model, it can be stated that the observed variables are able to measure their respective latent variables. The latent variable of social factors was measured with indicators of social

Table 2. Fit indices of the psychological factors measurement model

Fit index	Acceptable domain	Value
Chi-2 ($\chi 2$)	-	20.08
chi-square divided by the degrees of freedom (χ^2/df)	5<	2.23
Normalized Fit Index (NFI)	<0.90	0.072
Non-Normalized Fit Index (NNFI)	<0.90	0.974
Comparative Fit Index (CFI)	<0.90	0.984
Incremental Fit Index (IFI)	<0.90	0.984
Goodness of fit index (GFI)	<0.90	0.977
Root mean square error of approximation (RMSEA)	>0.08	0.068
Standardized Root Mean Squared Residual (SRMR)	>0.1	0.028

support, socioeconomic status, social comparison, psychological response, and quality of life. Figure 2 displays the factor loadings of the measurement model of social factors, and Table 3 presents the fit indices of this model. The heterogeneous factor loadings and the non-acceptance of most fit indices indicate that these indicators cannot measure a single latent variable. Therefore, social factors are entered into the hypothesized model separately and independently.

In Figure 3, factor loadings (standard coefficients) for physiological responses illustrate that the standard coefficient equal to 1.14 for the TC variable indicates a high correlation (Multi-collinearity) between the indicators. The assessment of the correlation between indicators demonstrates that the correlation between TC and LDL is equal to 0.83; therefore, to overcome the problem of multicollinearity, the TC variable is excluded from the analysis. Table 4 depicts the relatively good fit of the measurement model for physiological responses.

Moreover, Figure 4, the measurement model for health-related behaviors, and Table 5, the fit indices for this measurement model, demonstrate that in this measurement model, to align with other variables, the variable of lack of physical activity (instead of physical activity) was analyzed. The non-significance of the factor loading of this indicator caused this variable to be excluded from the analysis.

Table 3. Fit indices of social factors measurement model

Fit index	Accepted domain	Value
Chi-2 (χ2)	-	13.46
Chi-square divided by the degrees of freedom (χ2/df)	5<	2.69
Normalized Fit Index (NFI)	<0.90	0.849
Non-Normalized Fit Index (NNFI)	<0.90	0.786
Comparative Fit Index (CFI)	<0.90	0.893
Incremental Fit Index (IFI)	<0.90	0.899
Goodness of fit index (GFI)	<0.90	0.981
Root mean square error of approximation (RMSEA)	>0.08	0.079
Standardized Root Mean Squared Residual (SRMR)	>0.1	0.053

Table 4. Fit indices of the physiological reactions measurement model

Fit index	Accepted domain	Value
Chi-2 (χ2)	-	36.33
Chi-square divided by the degrees of freedom (χ2/df)	5<	18.16
Normalized Fit Index (NFI)	<0.90	0.939
Non-Normalized Fit Index (NNFI)	<0.90	0.904
Comparative Fit Index (CFI)	<0.90	0.941
Incremental Fit Index (IFI)	<0.90	0.942
Goodness of fit index (GFI)	<0.90	0.935
Root mean square error of approximation (RMSEA)	>0.08	0.083
Standardized Root Mean Squared Residual (SRMR)	>0.1	0.097

Table 5. Fit indices of the health-related behavior measurement model

Fit index	Accepted domain	Value
Chi-2 (χ2)	-	18.62
Chi-square divided by the degrees of freedom (χ2/df)	5<	9.31
Normalized Fit Index (NFI)	<0.90	0.857
Non-normalized fit Index (NNFI)	<0.90	0.905
Comparative Fit Index (CFI)	<0.90	0.919
Incremental Fit Index (IFI)	<0.90	0.920
Goodness of fit index (GFI)	<0.90	0.897
Root mean square error of approximation (RMSEA)	>0.08	0.064
Standardized Root Mean Squared Residual (SRMR)	>0.1	0.023

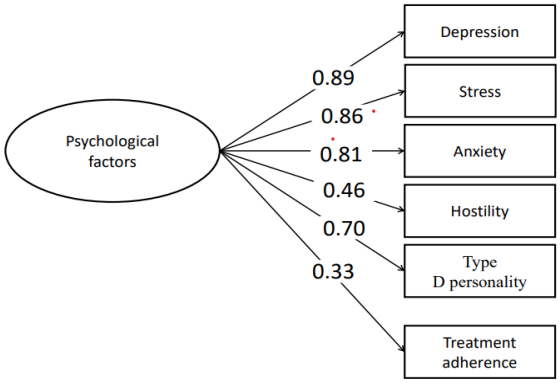

Figure 1. Measurement model for psychological factors

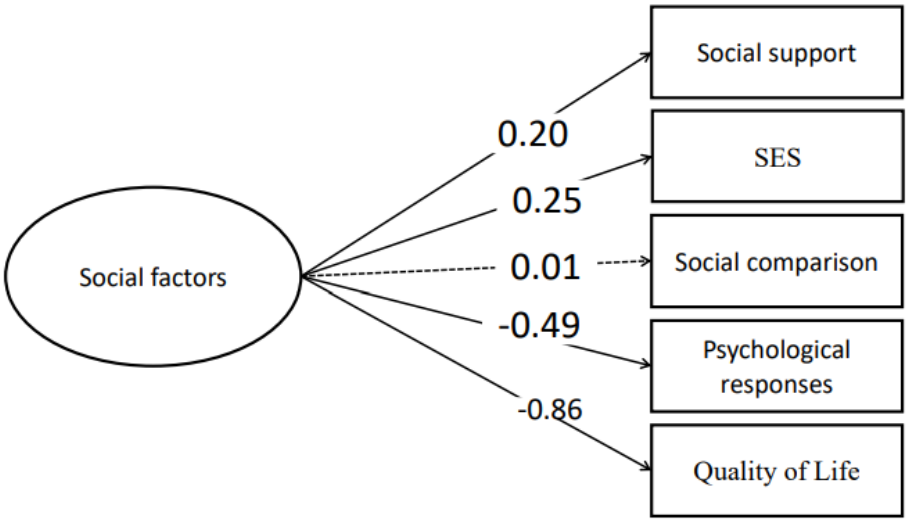

Figure 2. Measurement model for social factors

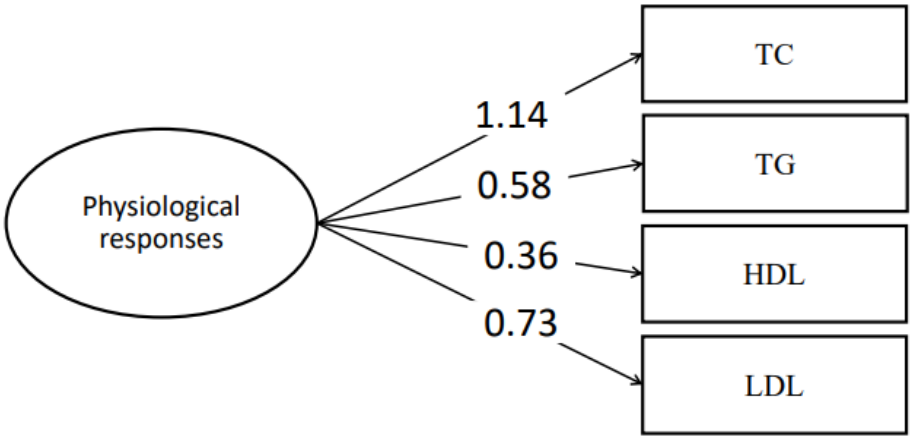

Figure 3. Measurement model for physiological responses

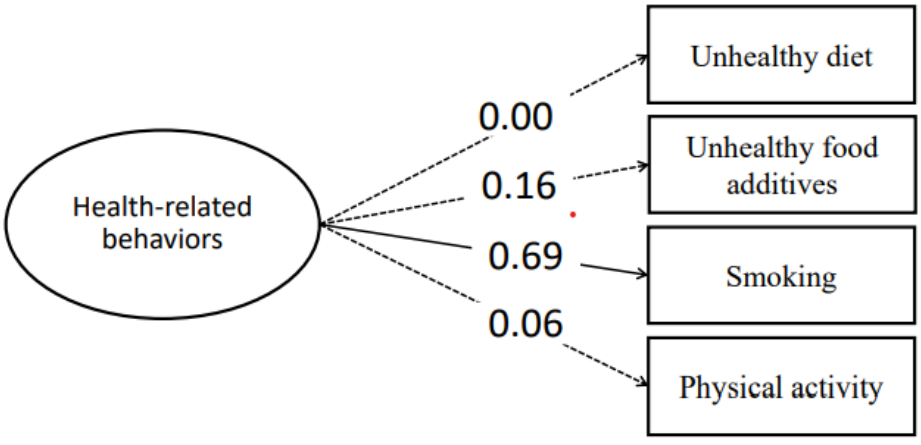

Figure 4. Measurement model for health-related behaviors

Structural modeling test

The structural model of the research was examined after ensuring the power of indicators in measuring psychological factors, social factors, physiological responses, and health-related behaviors (unhealthy behaviors). In this model (Figures 4, 5), it is assumed that the latent variable of psychological factors, social factors with the mediation of the latent variables of physiological responses, and unhealthy behaviors predict the severity of coronary heart disease. It can also be inferred that the researcher's assumption is that physiological factors predict the relationship between unhealthy behaviors and the severity of coronary heart disease.

Accordingly, it can be concluded that, in general, the structural model of the present research "fits" the collected data and the conceptual model of the research, in which the latent variables of psychological factors, social factors, physiological responses, and unhealthy behaviors in the form of a model predict the severity of coronary heart disease "fits" the collected data. The squared multiple correlations in the structural model of psychological factors explain 25.33% of the variance of coronary heart disease, signifying that the three variables of psychological factors, physiological responses, and unhealthy behaviors explain 25.33% of the variance of coronary heart disease.

In this model, psychological factors explain 41.8% of the variance of physiological responses and 18.49% of the variance of health-related behaviors (unhealthy behaviors). Moreover, physiological responses explain 10.89% of the variance of coronary heart disease, and health-related behaviors explain 14.14% of its variance. In addition, the squared multiple correlations in the structural model of social factors demonstrated that predictor variables explain 21.86% of the variance of coronary heart disease. In this model, social factors explain a total of 32.42% of the variance of physiological responses and 32.72% of the variance of health-related behaviors. Physiological responses explain 9.61% of the variance of coronary heart disease, and health-related behaviors explain 12.25% of its variance. In general, psychological and social factors explain 47.19% of the variance of coronary heart disease.

Special questions

1. Do physiological responses predict the severity of coronary heart disease?

According to Table 4-9, the path coefficient between physiological responses and coronary heart disease is significant at 1% (P<1%, B=0.33). Accordingly, it can be argued that **one** standard deviation increase in physiological responses leads to a 0.33 standard deviation increase in the severity of coronary heart disease.

2. Do unhealthy behaviors predict the severity of coronary heart disease?

As illustrated in Table 4-9, the path coefficient between unhealthy behaviors and the severity of coronary heart disease is significant at 1% (P<1%, B=0.38). In other words, the findings of the present study demonstrated that physiological factors mediated the relationship between unhealthy behaviors and the severity of coronary heart disease (at 1%).

Do psychosocial factors predict the severity of coronary heart disease?

A) Psychological factors

Based on Table 6, the bootstrap test was used to evaluate the prediction of the severity of coronary heart disease by psychological factors. The results pointed to the indirect relationship between psychological factors and the severity of coronary heart disease through the mediation of physiological responses and health-related behaviors. Moreover, the relationship is significant in both mediating paths. This means that the effect of psychological factors on coronary heart disease through the mediation of physiological responses is significant, with a standard coefficient of 0.096 at P<0.05. Furthermore, the effect of this variable through the mediation of health-related behaviors is significant, with a standard coefficient of 0.163 at P< 0.01.

Table 6. Standard coefficients, standard error, and significance levels for the structural model of psychological factors

Path			Standard coefficient	Standard error	Significance level
Psychological factors	→	Physiological responses	0.29	0.081	0.01 <P
Psychological factors	→	Health-related behaviors		0.083	0.01 <P
Psychological factors	→	Coronary heart disease	0.33	.065٠	0.01 <P
Health-related behaviors	→	Coronary heart disease	0.38	0.042	0.01 <P

Table 7. Results of the bootstrap test for the mediating effects of the social factors model

independent variable	Mediating variable	Dependent variable	Standard coefficient	Standard error	Significance level	>95%	95%>
social support	Physiological responses	Coronary heart disease	-0.056	0.077	P>0.05	0.008	-0.098
Psychological response	Physiological responses	Coronary heart disease	0.130	0.101	0.01 < P	0.163	0.089
Social comparison	Health-related behaviors	Coronary heart disease	0.091	0.026	P<0.05	0.131	0.034
Quality of Life	Health-related behaviors	Coronary heart disease	-0.179	0.064	P<0.01	-0.088	-0.235
Quality of Life	Physiological responses	Coronary heart disease	-0.105	0.058	0.01 <P	-0.53	-0.147

b) Social factors

As illustrated in Table 7, the bootstrap test was used to evaluate the prediction of the severity of coronary heart disease by social factors. It measures the indirect relationship between social factors and the severity of coronary heart disease through the mediation of physiological responses and health-related behaviors. As displayed in this table, all four mediating paths are significant. Moreover, except for the mediating effect of social support on heart disease through the mediation of physiological responses, which is not significant, the remaining mediating effects are significant.

Discussion

The present study aimed to assess the effect of psychosocial factors on coronary heart disease medicated by physiological responses and health-related behaviors. To this end, structural equation modeling was used according to the research conducted in the fourth chapter. This method consists of two parts: the "measurement model" and the "structural model." Examining the fit indices of the model demonstrated that the smoothed chi-square is less than five, and the optimal values of the comparative fit index (CFI), model fit index (GFI), and Adjusted Goodness of Fit Index (AGFI) are greater than 90%. Moreover, the root mean square error of approximation (RMSEA) is less than 8%.

The comparison of the collected data with the approved criteria demonstrated that, in general, the structural model of the current research "fits" the collected data well. In other words, based on the conceptual model of the research, psychosocial factors have an effect on coronary heart disease, and part of this effect is exerted through the mediation of health-related behaviors and physiological responses. Coronary heart disease is the narrowing of the coronary arteries that supply blood and oxygen to the heart. Due to the accumulation of atheroma and plaque in the arteries, the process is called atherosclerosis.

Physiological mechanisms include the buildup of atherosclerotic plaques in the walls of arteries as a result of inflammatory processes, lipid abnormalities, such as increased low-density lipoprotein cholesterol (LDLc), decreased high-density lipoprotein (HDL), and high triglycerides (TAG), decreased vasodilation and increased risk of thrombosis, endothelial dysfunction and changes in the endothelial lining of blood vessels of the cardiovascular system, high levels of homocysteine (Hcy), which is associated with increased LDLc, and a decrease in nitric oxide produced in endothelial cells, which causes vasodilation [58].

In explaining this finding, several studies have pinpointed that apart from its known role in promoting atherosclerosis in blood vessels, hyperlipidemia may directly affect the heart, leading to increased ischemia/reperfusion injury and poor response to cardiac protective interventions, such as ischemic preconditioning [59]. Further investigation suggested that the correlation between high cholesterol and increased risk of cardiovascular events is mainly related to low-density lipoprotein (LDL)-cholesterol (LDL-C). On the contrary, HDL-C is inversely correlated with mortality from coronary heart disease [60,61]. Accordingly, the "cholesterol hypothesis" was developed, which proposes that LDL-C is the cause of atherosclerosis, and as a result, reduced LDL-C lowers the risk of myocardial infarction and other cardiovascular events.

*The path coefficient between unhealthy behaviors and the severity of coronary heart disease is significant at 1% (P<1%, B=0.38).

In this research, from health-related behaviors, only unhealthy behaviors were evaluated. Unhealthy behavior consists of smoking, unhealthy diet, unhealthy additives (fat, sugar, and salt), and physical inactivity. According to Table [4-12], the test results show that unhealthy behaviors have a positive and significant relationship with coronary heart disease. In order to discuss and analyze this research finding, unhealthy behavior is considered in the form of smoking, nutrition, and physical activity. In explaining these findings, it can be stated that significant biological evidence shows that the components of cigarette smoke lead to endothelial dysfunction, cell dysfunction, arteriosclerosis, acute thrombosis, and impaired oxygen-carrying capacity of the blood [39]. Scientific findings indicated that improper nutrition is one of the most important risk factors in cardiovascular disease [60-63]. A high intake of trans-fatty acids is associated with an increased risk of cardiovascular diseases [64]. The relationship between nutrition and coronary heart disease can be explained by cholesterol being the main nutritional factor in coronary heart disease. The body makes most of it in the blood, and only about 20% of the cholesterol in the bloodstream comes from our food. Getting too much dietary cholesterol increases LDL. Nonetheless, its negative impact is less than saturated fatty acids. Most foods containing saturated fats, especially animal and solid fats, are a rich source of cholesterol, and their limited consumption leads to a decrease in cholesterol [65].

Moreover, in explaining the relationship between physical inactivity and coronary heart disease, it can

be stated that a sedentary lifestyle causes obesity, increasing insulin resistance, blood glucose levels, plasma lipids, and prothrombotic factors [56]. Diet plays a critical role in creating the background for the risk factors of cardiovascular diseases. Recent studies have also provided evidence on the relationship of dietary patterns with chronic diseases and the risk of death. Furthermore, the results of research in recent decades demonstrated that drug abuse, smoking, unhealthy diet, physical inactivity, obesity, high blood pressure, and lipid abnormalities are considered risk factors for cardiovascular disease, especially coronary heart disease [66-72].

Psychological factors and coronary heart disease

The presented studies are only a part of the findings related to the role of psychosocial factors in coronary heart disease, and now, some meta-analyses and systematic reviews are being conducted on psychosocial variables. In order to explain these findings, it can be stated that psychosocial factors are one of the risk factors for coronary heart disease [73, 74], and this relationship is illustrated in two physiological and behavioral ways [75, 76]. In order to physiologically explain the relationship between psychosocial factors and coronary heart disease, the following can be mentioned:

Although the underlying mechanisms are unclear, depression has been linked to physiological and psychosocial changes that are detrimental to the cardiovascular system [77]. A meta-analysis conducted in 2017 associated major depressive disorder with a 72% increased risk of CVD and elevation in specific categories of coronary heart disease (CHD), cerebrovascular disease, congestive heart failure, and CVD death [78]. Meta-analyses have also pointed to the relationship of anxiety with a higher risk of CHD, cardiac death, CVD events, and stroke [79; 74].

Indirect pathway between psychological factors and coronary heart disease

A wide array of studies has indicated that part of the relationship between psychological factors and coronary heart disease is "indirect." In order to explain this finding, it should be stated that psychological factors form part of the significant relationship with coronary heart disease through physiological and behavioral factors, and the two variables of unhealthy behaviors and physiological factors can play a mediating role in this relationship.

Indirect pathway between social factors and coronary heart disease

The findings of this research pinpointed that social factors, taking into account the role of mediators (physiological responses and health-related behaviors), can indirectly affect coronary heart disease, and a significant part of the relationship between these factors and the severity of coronary heart disease is "indirect." In other words, psychological factors affect behavior, which in turn exert their effect on the severity of coronary heart disease through physiological responses. This research finding suggests that the mind can affect the body. Nonetheless, another point in the explanation of the comparison of the two methods of answering questions 4 and 5 is that entering any latent or observed variable into each analysis affects the behavior of other variables. Therefore, each model with its variables must be analyzed and explained.

Conclusions

???

Compliance with ethical guidelines

This article adhered to all ethical principles, including informed consent, maintaining medical secrets, non-interference of the research with the patient's treatment, permission to withdraw from the research, and availability of the result.

Acknowledgments

This research is the result of a doctorate thesis submitted to Payam Noor University. The author's most profound appreciation goes to the Vice-Chancellor of Research and Technology of Shahid Beheshti University of Medical Sciences in Tehran and Iran, as well as the respected personnel of the Cardiology Department of Shahid Modares, Taleghani, and Farhikhtegan hospitals in Tehran, and Imam Khomeini Hospital in Ardabil for their cooperation, and Mrs. Niloofar Valizadeh, our colleague.

Authors' contributions

All authors contributed equally to the preparation of all parts of the research.

Funding/Support

This article was extracted from a PhD dissertation on health psychology submitted by the first author, Ahmad Alipour. Moreover, this study was approved by the Ethics Committee of Payam Noor University (IR. PNU. REC. 1401. 055).

Conflicts of Interest

The authors declare no conflict of interest in this study.

References

1.WHO. Noncommunicable Diseases Country Profiles .2021.[link]

2. Liu H, Chen S, Liu M, Nie H, Lu H. Comorbid chronic diseases are strongly correlated with disease severity among COVID-19 patients: a systematic review and meta-analysis. Aging Dis. 2020;11(3):668. [DOI : 10.14336/AD.2020.0502] [PMID] [PMCID]

3. Abegunde DO, Mathers CD, Adam T, Ortegon M, Strong K. The burden and costs of chronic diseases in low-income and middle-income countries. The Lancet. 2007 ;370(9603):1929-38. [DOI: 10.1016/S0140-6736(07)61696-1] [PMID]

4. Jinnouchi H, Kolodgie FD, Romero M, Virmani R, Finn AV, Chun Y, Thomas S. Vessel Based Imaging Techniques.2020: Diagnosis, Treatment, and Prevention. 211-227. [DOI: 10.1007/978-3-030-25249-6_11]

5.Chow CK, Redfern J, Hillis GS, Thakkar J, Santo K, Hackett ML, Jan S, Graves N, de Keizer L, Barry T, Bompoint S. Effect of lifestyle-focused text messaging on risk factor modification in patients with coronary heart disease: A randomized clinical trial. JAMA. 2015; 314(12):1255-63.[DOI: 10.1001/jama.2015.10945] [PMID]

6.Einarson T. R., ACS, A., Ludwig, C., Ekman, I., & Panton, U. H. Prevalence of cardiovascular disease in type 2 of diabetes: a systematic literature review or scientific evidence from across the world in 2007-2017. Cardiovascular diabetology.2018:17(1), 83.

7. Boroumand S, Shahriari M, Abbasi Jebeli M, Baghersad Z, Baradaranfard F, Ahmadpoori F. Determine the level of self-efficacy and its related factors in patients with ischemic heart disease: A descriptive correlational study. IJNR . 2015;9(4):61-9.

8. Sahoo S, Padhy SK, Padhee B, Singla N, Sarkar S. Role of personality in cardiovascular diseases: An issue that needs to be focused too!. Indian Heart J. 2018;70:S471-7. [DOI: 10.1016/j.ihj.2018.11.003] [PMID] [PMCID]

9. Piepoli MF, Hoes AW, Agewall S, Albus C, Brotons C, Catapano AL, et al. European guidelines on cardiovascular disease prevention in clinical practice: the sixth joint task force of the European society of cardiology and other societies on cardiovascular disease prevention in clinical practice (constituted by representatives of 10 societies and by invited experts) developed with the special contribution of the European Association for Cardiovascular Prevention & Rehabilitation (EACPR). Eur Heart J.2016; 37:2315–2381. [DOI: 10.1093/eurheartj/ehw106] [PMID] [PMCID]

10. Sittenthaler S, Jonas E, Traut-Mattausch E. Explaining self and vicarious reactance: A process model approach. Pers Soc Psychol Bull. 2016;42(4):458-70. [DOI: 10.1177/0146167216634055] [PMID]

11. Mьnzel T, Sшrensen M, Gori T, Schmidt FP, Rao X, Brook FR, Chen LC, Brook RD, Rajagopalan S. Environmental stressors and cardio-metabolic disease: part II–mechanistic insights. Eur Heart J. 2017;38(8):557-64. [DOI : 10.1093/eurheartj/ehw294] [PMID] [PMCID]

12. Staniute M, Brozaitiene J, Burkauskas J, Kazukauskiene N, Mickuviene N, Bunevicius R. Type D personality, mental distress, social support and health-related quality of life in coronary artery disease patients with heart failure: a longitudinal observational study. Health Qual Life Outcomes. 2015;13(1):1-1. [DOI: 10.1186/s12955-014-0204-2] [PMID] [PMCID]

13.Staniute M, Brozaitinene J, Bug Bunevicius R. Effects of social support and stressful life events health-releated quality of life in coronary artery Disease. J Cardiovasc Nurs.2013:28(1):83-9.[DOI: 10.1097/JCN.0b013e318233e69d] [PMID]

14. Havranek EP, Mujahid MS, Barr DA, Blair IV, Cohen MS, Cruz-Flores S, Davey-Smith G, Dennison-Himmelfarb CR, Lauer MS, Lockwood DW, Rosal M. Social determinants of risk and outcomes for cardiovascular disease: a scientific statement from the American Heart Association.Circulation.2015;132(9):873-98. [DOI: 10.1161/CIR.0000000000000228] [PMID]

15. Bonow RO, Mann DL, Zipes DP, Libby P. Braunwald's Heart Disease: A text book of Cardiovascular Medicine . Saunder Elsevier.2012.

16. Ferris PA, Kline TJ, Bourdage JS. He said, she said: Work, biopsychosocial, and lifestyle contributions to coronary heart disease risk. Health Psychology. 2012;31(4):503-11. [DOI: 10.1037/a0026394] [PMID]

17. Huang C, Huang J, Tian Y, Yang X, Gu D. Sugar sweetened beverages consumption and risk of coronary heart disease: a meta-analysis of prospective studies. Atherosclerosis. 2014 May;234(1):11-6. [DOI: 10.1016/j.atherosclerosis.2014.01.037] [PMID]

18.Willett WC. Dietary fats and coronary heart disease. J Intern Med. 2012 Jul;272(1):13-24. [DOI: 10.1111/j.1365-2796.2012.02553.x] [PMID]

19. Zhao X, Yang X, Zhang X, Li Y, Zhao X, Ren L, Wang L, Gu C, Zhu Z, Han Y. Dietary salt intake and coronary atherosclerosis in patients with prehypertension. J Clin Hypertens. 2014;16(8):575-80. [DOI: 10.1111/jch.12362] [PMID] [PMCID]

20. Khalili D, Haj Sheikholeslami F, Bakhtiyari M, Azizi F, Momenan AA, Hadaegh F. The incidence of coronary heart disease and the population attributable fraction of its risk factors in Tehran: a 10-year population-based cohort study. PloS one. 2014;9(8):e105804. [DOI: 10.1371/journal.pone.0105804]

21. Alipour A. The Relationship of Social Support with Immune Parameters in Healthy Individuals: Assessment of the Main Effect Model. IJPCP. 2018:12(2), 134-139.

22.HelpGuide.Depression Symptoms and Warning Signs. 2019. [Link]

23.American Psychological Association. Anxiety. 2020. [Link]

24. Denollet J. Personality and coronary heart disease: the type-D scale-16 (DS16). Annals of Behavioral Medicine. 1998;20(3):209-15. [DOI: 10.1007/BF02884962]

25. Koenig J, Weise S, Rinnewitz L, Parzer P, Resch F, Kaess M. Longitudinal covariance of resting-state cardiac function and borderline personality disorder symptoms in adolescent non-suicidal self-injury. World J Biol Psychiatry. 2018;19(2):152-7. [DOI: 10.1080/15622975.2017.1342046] [PMID]

26. De Smedt D, De Bacquer D, De Sutter J, Dallongeville J, Gevaert S, De Backer G, Bruthans J, Kotseva K, Reiner Ž, Tokgцzoğlu L, Clays E. The gender gap in risk factor control: effects of age and education on the control of cardiovascular risk factors in male and female coronary patients. The EUROASPIRE IV study by the European Society of Cardiology. Int J Cardiol. 2016;209:284-90. [DOI : 10.1016/j.ijcard.2016.02.015] [PMID]

27. Zhao M, Vaartjes I, Graham I, Grobbee D, Spiering W, Klipstein-Grobusch K, Woodward M, Peters SA. Sex differences in risk factor management of coronary heart disease across three regions. Heart. 2017;103(20):1587-94. [DOI: 10.1136/heartjnl-2017-311429] [PMID] [PMCID]

28.Manfredini R, De Giorgi A, Tiseo R, et al. Marital Status, Cardiovascular Diseases, and Cardiovascular Risk Factors: A Review of the Evidence. J Womens Health (Larchmt). 2017;26(6):624–632. [DOI: 10.1089/jwh.2016.6103] [PMID]

29.Jahangirpour M, Mousavi V, Khosro Javid M, Salari A, Rezaei S. The effect of mindfulness group training on reducing depression, hostility, and anxiety in patients with coronary artery disease. Urmia Med J. 2013;24:730–9.

30.Vella EJ, Friedman BH. Hostility and anger in: Cardiovascular reactivity and recovery to mental arithmetic stress. Int J Psychophysiol. 2009;72:253–9. [DOI: 10.1016/j.ijpsycho.2009.01.003] [PMID] [PMCID]

31.Kuper H, Marmot M, Hemingway H. Systematic review of prospective cohort studies of psychosocial factors in the etiology and prognosis of coronary heart disease. Semin Vasc Med.2002; 2(3):267-314. [DOI: 10.1055/s-2002-35401] [PMID]

32. Sittenthaler S, Steindl C, Jonas E. Legitimate vs. illegitimate restrictions–A motivational and physiological approach investigating reactance processes. Front Psychol. 2015; 6: 632. [DOI: 10.3389/fpsyg.2015.00632]

33. Riem MM, Bakermans-Kranenburg MJ, Huffmeijer R, van IJzendoorn MH. Does intranasal oxytocin promote prosocial behavior to an excluded fellow player? A randomized-controlled trial with Cyberball. Psychoneuroendocrinology. 2013;38(8):1418-25. [DOI: 10.1016/j.psyneuen.2012.12.023] [PMID]

34. Jonas E, Graupmann V, Kayser DN, Zanna M, Traut-Mattausch E, Frey D. Culture, self, and the emergence of reactance: Is there a "universal" freedom?. Journal of Experimental Social Psychology. 2009;45(5):1068-80. [DOI: 10.1016/j.jesp.2009.06.005]

35.Sittenthaler, S., Jonas, E., & Traut-Mattausch, E. Explaining self and vicarious reactance. Personality and Social Psychology Bulletin.2016; 42:458–70. [DOI: 10.1177/0146167216634055]
36. Steindl C, Jonas E, Sittenthaler S, Traut-Mattausch E, Greenberg J. Understanding psychological reactance. Zeitschrift fьr Psychologie. 2015 :223(4):205-214. [DOI: 10.1027/2151-2604/a000222] [PMID] [PMCID]
37. Antonogeorgos G, Panagiotakos DB, Pitsavos C, Papageorgiou C, Chrysohoou C, Papadimitriou GN, Stefanadis C. Understanding the role of depression and anxiety on cardiovascular disease risk, using structural equation modeling; the mediating effect of the Mediterranean diet and physical activity: the ATTICA study. Ann Epidemiol. 2012;22(9):630-7. [DOI: 10.1016/j.annepidem] [PMID]
38.Bayfield NGR, Pannekoek A, Tian DH. Preoperative cigarette smoking and short-term morbidity and mortality after cardiac surgery: a meta-analysis. Heart Asia. 2018 ;10(2):e011069. [DOI: 10.1136/heartasia-2018-011069] [PMID] [PMCID]
39.Gakidou E, Afshin A, Abajobir AA, Abate KH, Abbafati C, Abbas KM, et al. Global,regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. The Lancet. 2017;390(10100):1345-422. [DOI: 10.1016/S0140-6736(17)32366-8] [PMID] [PMCID]
40.Liu S, Zhang M, Yang L, Li Y, Wang L, Huang Z, et al. Prevalence and patterns of tobacco smoking among Chinese adult men and women: findings of the 2010 national smoking survey. J Epidemiol Community Health. 2017;71(2):154-61. [DOI: 10.1136/jech-2016-207805] [PMID] [PMCID]
41.Tan J, Zhang X, Wang W, Yin P, Guo X, Zhou M. Smoking, blood pressure, and cardiovascular disease mortality in a large cohort of Chinese men with 15 Years follow-up. Int J Environ Res Public Health. 2018;15(5):1026. [DOI: 10.3390/ijerph15051026] [PMID] [PMCID]
42.Cho MH, Lee K, Park SM, Chang J, Choi S, Kim K, et al. Effects of smoking habit change on all-cause mortality and cardiovascular diseases among patients with newly diagnosed diabetes in Korea. Sci Rep. 2018;8(1):1-8. [DOI: 10.1038/s41598-018-23729-0] [PMID] [PMCID]
43. Lee PN, Forey BA, Thornton AJ, Coombs KJ. The relationship of cigarette smoking in Japan to lung cancer, COPD, ischemic heart disease and stroke: A systematic review. F1000Res. 2018 Feb 19;7:204. [DOI: 10.12688/f1000research.14002.1] [PMID] [PMCID]
44. Hong XY, Lin J, Gu WW. Risk factors and therapies in vascular diseases: An umbrella review of updated systematic reviews and meta-analyses. J Cell Physiol. 2019;234(6):8221-8232. [DOI: 10.1002/jcp.27633] [PMID]
45.Thomas K, Nilsson E, Festin K, Henriksson P, Lowиn M, Luf M, Kristenson M. Associations of Psychosocial Factors with Multiple Health Behaviors: A Population-Based Study of Middle-Aged Men and Women. Int J Environ Res Public Health. 2020;17(4):1239. [DOI: 10.3390/ijerph17041239] [PMID] [PMCID]
46. Siegel KR, Bullard KM, Imperatore G, Ali MK, Albright A, Mercado CI, Li R, Gregg EW. Prevalence of Major Behavioral Risk Factors for Type 2 Diabetes. Diabetes Care. 2018;41(5):1032-1039. [DOI: 10.2337/dc17-1775] [PMID] [PMCID]
47. Smedema SM. An analysis of the relationship of character strengths and quality of life in persons with multiple sclerosis. Qual Life Res. 2020 May;29(5):1259-1270. [DOI: 10.1007/s11136-019-02397-1] [PMID]
48.World Health Organization. Physical Activity.2020. [Link]
49. LeBlanc S, Coulombe F, Bertrand OF, Bibeau K, Pibarot P, Marette A, Almиras N, Lemieux I, Desprиs JP, Larose E. Hypertriglyceridemic waist: a simple marker of high-risk atherosclerosis features associated with excess visceral adiposity/ectopic fat. J Am Heart Assoc. 2018;7(8):e008139. [DOI: 10.1161/JAHA.117.008139] [PMID] [PMCID]
50.Ferrucci L, Fabbri E. Inflammageing: Chronic inflammation in ageing, cardiovascular disease, and frailty. Nat Rev Cardiol. 2018; 15: 505–522. [DOI: 10.1038/s41569-018-0064-2] [PMID] [PMCID]
51.Fiuza-Luces C, Santos-Lozano A, Joyner M, et al. Exercise benefits in cardiovascular disease: Beyond attenuation of traditional risk factors. Nat Rev Cardiol.2018; 15: 731–743. [DOI: 10.1038/s41569-018-0065-1] [PMID]
52.Krankel N, Bahls M, van Craenenbroeck EM, et al. Exercise training to reduce cardiovascular risk in patients with metabolic syndrome and type 2 diabetes mellitus: How does it work? Eur J Prev Cardiol. 2019; 26: 701–708. [DOI: 10.1177/2047487318805158] [PMID]
53.Gepner Y, Shelef I, Schwarzfuchs D, et al. Effect of distinct lifestyle interventions on mobilization of fat storage pools: The CENTRAL MRI randomized controlled trial. Circulation 2018; 137: 1143–1157. [DOI: 10.1161/CIRCULATIONAHA.117.030501] [PMID]
54.Pedersen BK. Anti-inflammatory effects of exercise: role in diabetes and cardiovascular disease. Eur J Clin Invest. 2017; 47: 600–611. [DOI : 10.1111/eci.12781] [PMID]
55.Bernardo, Ooi, Weeks et al. Understanding key mechanisms of exercise-induced cardiac protection to mitigate disease: Current knowledge and emerging concepts. Physiol Rev. 2018; 98: 419–475. [DOI: 10.1152/physrev.00043.2016] [PMID]
56.Gaziano, T.A. Economic burden and the cost-effectiveness of treatment of cardiovascular diseases in Africa.Heart 2008, 94, 140–4. [DOI: 10.1136/hrt.2007.128785] [PMID]
57. Meyers LS, Gamst G, Guarino AJ. Applied multivariate research: Design and interpretation. Sage publications; 2016.
58. Chase SK, Youngkin EQ. Postmenopausal hormone replacement and cardiovascular disease: incorporating research into practice. J Obstet Gynecol Neonatal Nurs. 2004;33(5):648-56. [DOI: 10.1177/0884217503261897] [PMID]
59.Pathak RK, Mahajan R, Lau DH, Sanders P. The implications of obesity for cardiac arrhythmia mechanisms and management. Can J Cardiol. 2015;31(2):203–10. [DOI: 10.1016/j.cjca.2014.10.027] [PMID]
60. Gordon T, Castelli WP, Hjortland MC, Kannel WB, Dawber TR. High density lipoprotein as a protective factor against coronary heart disease: the Framingham Study. Am J Med. 1977 ;62(5):707-14. [DOI: 10.1016/0002-9343(77)90874-9] [PMID]
61. Riem MM, Bakermans-Kranenburg MJ, Huffmeijer R, van IJzendoorn MH. Does intranasal oxytocin promote prosocial behavior to an excluded fellow player? A randomized-controlled trial with Cyberball. Psychoneuroendocrinology. 2013;38(8):1418-25. [DOI: 10.1016/j.psyneuen.2012.12.023] [PMID]
62.Kannel, W.P. Castelli, T. Gordon.Cholesterol in the prediction of atherosclerotic disease. New perspectives based on the Framingham study. Ann Intern Med.1978;97: 85-91. [DOI: 10.7326/0003-4819-90-1-85] [PMID]
63.Kromhout D, Menotti A, Kesteloot H, Sans S. Prevention of coronary heart disease by diet and lifestyle: evidence from prospective cross-cultural, cohort, and intervention studies. Circulation. 2002;105(7):893-8. [DOI: 10.1161/hc0702.103728] [PMID]
64.Sarafino E. Psychology of Salat. The translation of a group of translators, under the supervision of Elaha Mirzaei .Rushd Publications.2013.
65.Sarafin E. P. Health psychology: Biopsychosocial interactions (4th ed.). New York: Wiley and Sons.2002: 123-127.
66.Akesson A, Larsson SC, Discacciati A, Wolk A. Low-risk diet and lifestyle habits in the primary prevention of myocardial infarction in men: a population-based prospective cohort study. J Am Coll Cardiol. 2014;64(13):1299-306. [DOI:

10.1016/j.jacc.2014.06.1190] [PMID]
67.Chomistek AK, Chiuve SE, Eliassen AH, Mukamal KJ, Willett WC, Rimm EB. Healthy lifestyle in the primordial prevention of cardiovascular disease among young women. J Am Coll Cardiol. 2015; 65:43–51. [DOI: 10.1016/j.jacc.2014.10.024] [PMID] [PMCID]
68.Hulsegge G, Looman M, Smit HA, Daviglus ML, van der Schouw YT, Verschuren WM. Lifestyle changes in young adulthood and middle age and risk of cardiovascular disease and all-cause mortality: the Doetinchem cohort study. J Am Heart Assoc. 2016; 5:e002432. [DOI: 10.1161/JAHA.115.002432] [PMID] [PMCID]
69.Maruthur NM, Wang NY, Appel LJ. Lifestyle interventions reduce coronary heart disease risk: results from the PREMIER trial. Circulation. 2009; 119:2026–31. [DOI: 10.1161/CIRCULATIONAHA.108.809491] [PMID] [PMCID]
70.Meng XJ, Dong GH, Wang D, Liu MM, Lin Q, Tian S, et al. Prevalence, awareness, treatment, control, and risk factors associated with hypertension in urban adults from 33 communities of China: the CHPSNE study. J Hypertens. 2011. 29:1303–10. [DOI: 10.1097/HJH.0b013e328347f79e] [PMID]
71.Stevens VJ, Obarzanek E, Cook NR, Lee IM, Appel LJ, Smith West D, et al. Long-term weight loss and changes in blood pressure: results of the trials of hypertension prevention, phase II. Ann Intern Med. 2001; 134:1–11. [DOI: 10.7326/0003-4819-134-1-200101020-00007] [PMID]
72. American Psychological Association.Anxiety.2009. [Link]
73.Van Der Kooy, K.; Van Hout, H.; Marwijk, H.; Marten, H.; Stehouwer, C.; Beekman, A.Depression and the risk for cardiovascular diseases: Systematic review and metaanalysis. Int J Geriatr Psychiatry. 2007; 22, 613–626. [DOI: 10.1002/gps.1723] [PMID]
74. Batelaan NM, Seldenrijk A, Bot M, van Balkom AJ, Penninx BW. Anxiety and new onset of cardiovascular disease: critical review and meta-analysis. Br J Psychiatry. 2016;208(3):223-31. [DOI: 10.1192/bjp.bp.114.156554] [PMID]
75. Cohen BE, Edmondson D, Kronish IM. State of the art review: depression, stress, anxiety, and cardiovascular disease. Am J Hypertens. 2015;28(11):1295-302. [DOI: 10.1093/ajh/hpv047] [PMID] [PMCID]
76. Pan XF, Meng R, Liu N, Pan A. Depression, anxiety, and cardiovascular disease in Chinese: A review for a bigger picture. Cardiovascular Innovations and Applications. 2017 ;2(2):237-51. [DOI:10.15212/CVIA.2016.0059]
77.Rozanski A, Blumenthal JA, Kaplan J. Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. Circulation. 1999;99:2192–2217. [DOI: 10.1161/01.cir.99.16.2192] [PMID]
78.Correll CU, Solmi M, Veronese N et al. Prevalence, incidence and mortality from cardiovascular disease in patients with pooled and specific severe mental illness: a large-scale meta-analysis of 3,211,768 patients and 113,383,368 controls. World Psychiatry. 2017; 16(2): 163–80. [DOI: 10.1002/wps.20420] [PMID] [PMCID]
79.Roest AM, Martens EJ, de Jonge P, Denollet J. Anxiety and risk of incident coronary heart disease: a meta-analysis. J Am Coll Cardiol 2010; 56(1): 38–46. [DOI: 10.1016/j.jacc.2010.03.034] [PMID]