Original Research Article

Comparison of autocorrelation between CV-RISK independent variables in groups with and without history of cardiovascular diseases

Ivany Lestari Goutama1*, Hendsun1, Yohanes Firmansyah1, Ernawati Su2

1Faculty of Medicine, Tarumanagara University, Jakarta, Indonesia
2Department of Public Health Sciences, Community Medicine, Family Medicine, Occupational Health and Bioethics, Faculty of Medicine, Tarumanagara University, Jakarta, Indonesia

Received: 19 August 2020
Accepted: 28 September 2020

*Correspondence:
Dr. Ivany Lestari Goutama,
E-mail: ivanylestari@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Cardiovascular relative risk (CVRISK) is the latest cardiovascular relative risk score to evaluate the magnitude of cardiovascular risk in healthy people regardless of age and cardiovascular risk severity. The aim of the study is to determine the correlation between each independent variables of CVRISK score in individuals with and without history of cardiovascular diseases (CVD).

Methods: The study design is cross-sectional study. We conducted it online through social media using Google forms from June to August 2020. Participants include all productive age groups from 16 to 60 years. The data were processed using excel and statistically tested. Descriptive data analysis uses tabulated data which is displayed in numbers or proportions (categorical) and single data distribution (numeric). Statistical association analysis uses the categorical-correlation test with 2 statistical tests that use eta on nominal-ordinal variables and contingency coefficients on nominal-nominal variables.

Results: There is a strong autocorrelation between hypertension and high tryglyceride levels (p value 0.001; correlation 0.549; risks 30.14%), nutritional status and low-density lipoprotein cholesterol (LDL-C) levels in CVD group (p value 0.002; correlation 0.774; risks 59.90%) and non-CVD group (p value 0.000; correlation 0.757; risks 57.3%). Hypertension and risky LDL-C levels firmly proves a very strong correlations and significant relationship in CVD groups (p value 0.014; correlation 0.947; risks 89.68%).

Conclusions: There is a correlation that varies from weak to very strong among the independent variables in the CVRISK scoring of the participants. Further research is needed to determine the potentiality of CVRISK as an early prevention in determining the cardiovascular risk of individuals with and without history of CVD.

Keywords: Cardiovascular disease, Risk factors, CVRISK, New CVD score

INTRODUCTION

Globally, cardiovascular diseases (CVD) are still the highest mortality cause at the moment, which mostly caused by acute myocardial infarction and stroke with an incidence rate of 85%.1 World Health Organization (WHO) generally divides the risk of CVD based on two large groups namely modifiable and non-modifiable risk factors.2 Modifiable risk factors include lifestyle risk factors and cardiometabolic risk factors, such as smoking, inactivity, unhealthy diet, excessive alcohol, hypertension, diabetes, hyperlipidemia, and obesity, meanwhile non-modifiable risk factors include genetics, age, gender, low socioeconomic, ethnicity and/or race.2,3

In order to determine cardiovascular (CV) risks among healthy people, it is recommended to use risk scores to help in evaluating the potentiality of acquiring the disease, however all current available CV risk scores such as systematic coronary risk evaluation (SCORE) (Europe), Framingham and atherosclerotic cardiovascular disease (ASCVD) (United States) unfortunately have several
CARDIOVASCULAR RELATIVE RISK (CVRISK) is a new cardiovascular relative risk scores to evaluate the magnitude of cardiovascular risk in healthy people regardless of age and cardiovascular risk severity. The score identify 15 items of CV risk factors including clinical data: age and gender, heredity, smoking, hypertension, obesity, psychosocial stress, and previous cardiovascular disease; laboratory data: low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), triglyceride (TG), fasting plasma glucose (diabetes mellitus); and protective factors: healthy diet and physical activity. Male sex, advanced age, diabetes, hypertension and obesity as well as patients with established CV and cerebrovascular disease may lead to a higher chance of developing CHD.

The aim of the study is to determine the autocorrelation between independent variables based on the implementation of CVRISK score in both CVD and non-CVD individuals.

METHODS

The design of this study was a cross-sectional study to look for correlations between independent and dependent variables on CVD incidence. This research was conducted online which was distributed through social media with the Google form. This research was conducted from June 2020 to August 2020. The population of this study were all productive age groups from 16 to 60 years old. The sample of this research is a productive group aged 16 to 60 years who used social media during the study period and got the questionnaire.

The inclusion criteria in this study were all productive age with or without CVD history. The exclusion criteria in this study were incomplete data on one of the variables studied and respondents who refused to fill out the survey form. The drop out criterion in this study was non-existent because this was not a study that required follow-up. This research was conducted by compiling a proposal and a questionnaire as the initial preparation for submitting an ethical review. Proposals were submitted to institutions or universities for review of ethics in this research. The review procedure until the revision was carried out until the ethical review certificate was completed. The research was carried out by distributing Google form questionnaires through social media such as instgram, line, whatsapp, facebook, and twitter. All answers were recapitulated in the available tabulated data on Google drive after the research period had been complete. Descriptive data were analyzed using descriptive statistical tabulations and analytical data were statistically tested. The research instruments were gadgets and social media which were used to distribute research questionnaires online.

Data processing were statistically tested. Descriptive data were displayed using tabulated data. Statistical data displayed in the form of numbers or proportions for categorical data and single data distribution in the form of mean, standard deviation (SD), median, maximum, minimum for numeric data. All data is presented in a single table. The statistical association analysis used categorical-categorical correlation test with 2 statistical tests in the form of correlation using eta on nominal-ordinal variables and the correlation of the contingency coefficient on nominal-nominal variables. All data analysis were resulted in a strength value in the form of r (rho) with the interpretation of the strength of the correlation being very strong (r correlation in the range 0.80-1.00), strong (r correlation in the range 0.60-0.80), quite strong (r correlation in the range 0.40-0.60), weak (r correlation is in the range 0.20-0.40), and very weak (r correlation is in the range 0.00-0.20).

RESULTS

This research was conducted by distributing questionnaires online to respondents who were willing to fill out the questionnaire with 52 (64.2) male respondents and 29 (25.8%) female respondents. The total respondents who participated in the study were 81 respondents. The mean age of the respondents was 35.88, with the maximum age was 64 years and the youngest was 22 years. The average height of the respondents were 70.02 kg with a maximum value of 115 kg and the lowest weight was 44 kg. The average height of the respondents was 167.49 cm with the maximum height was 187 cm and the lowest height was 153 cm. The number of respondents who had a history of CVD was 27 (33.3%) and the number of respondents who had no history of CVD was 54 (66.7%) of respondents (Table 1).

The results of the questionnaire included anamnesis and clinical examinations that were distributed to determine the CV risk factor data of respondents. The obtained data from the questionnaire showed that 23 (28.4%) respondents had a family history of CVD. Respondents who have a smoking habit were as many as 10 (12.3%) respondents. As many as 6 (7.4%) respondents had hypertension, 33 (40.7%) respondents had obesity nutritional status, 27 (33.3) respondents claimed to have psychological stress. Based on the screening of habit history, there were 53 (65.4%) respondents who carried out physical activity in accordance with the CVRISK recommendations and as many as 39 (48.1%) of respondents applied a healthy diet according to the criteria listed on the CVRISK questionnaire. Respondents who...
have diabetes mellitus were 3 (3.8%) respondents (Table 2).

Table 1: Sample characteristics.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>N (%)</th>
<th>Mean (SD)</th>
<th>Med (min-max)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>52 (64.2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>29 (35.8)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>35.88 (13.79)</td>
<td>28 (22-64)</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>70.02 (14.31)</td>
<td>70 (44-115)</td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>167.49 (7.56)</td>
<td>168 (153-187)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Clinical cardiovascular risks characteristics of the samples based on CVRISK questionnaire.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Familial history of CVD</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>23 (28.4)</td>
</tr>
<tr>
<td>No</td>
<td>58 (71.6)</td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>10 (12.3)</td>
</tr>
<tr>
<td>No</td>
<td>71 (87.7)</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>6 (7.4)</td>
</tr>
<tr>
<td>No</td>
<td>75 (92.6)</td>
</tr>
<tr>
<td>Nutritional status</td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>33 (40.7)</td>
</tr>
<tr>
<td>Normal</td>
<td>48 (59.2)</td>
</tr>
<tr>
<td>Stress</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>27 (33.3)</td>
</tr>
<tr>
<td>No</td>
<td>54 (66.7)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>3 (3.8)</td>
</tr>
<tr>
<td>No</td>
<td>78 (96.2)</td>
</tr>
</tbody>
</table>

The results of the cardiovascular risk questionnaire based on the laboratory results of the lipid profile were carried out at the same time with the clinical questionnaire. Respondents who had the results of lipid profiles examination within the last month filled out a follow-up questionnaire related to lipid profiles. In this study, from 54 (66.7%) of respondents without history of CVD, 27 (33.3%) of respondents filled in the LDL levels results in the column "with a history of CVD", meanwhile 71 (87.7%) respondents filled in HDL levels and 76 (93.8%) respondents filled in TG levels. We also found that respondents with history of CVD had an overview of - LDL levels with low risk in 15 (55.56%) respondents, medium risk in 9 (33.33%) respondents, and high risk in 3 (11.11%) respondents; abnormal HDL levels in 7 (30.43%) respondents; and abnormal TG levels in 6 (24%) respondents. Respondents without history of CVD had an overview of - LDL levels with low risk in 16 (19.8%) respondents, moderate risk in 32 (39.5%) respondents, and high risk in 6 (7.4%) respondents; abnormal HDL in 7 (14.58%) respondents; and abnormal TG in 12 (23.5 %) respondents (Table 3).

Table 3: Cardiovascular risks characteristics of the samples’ lipid profiles based on CVRISK questionnaire.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>N (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LDL levels on group with CVD history: 27 (33.3%)</td>
<td></td>
</tr>
<tr>
<td>High risk</td>
<td>3 (3.7)</td>
</tr>
<tr>
<td>Moderate risk</td>
<td>9 (11.1)</td>
</tr>
<tr>
<td>Low risk</td>
<td>15 (18.5)</td>
</tr>
<tr>
<td>LDL levels on group without CVD history: 54 (66.7%)</td>
<td></td>
</tr>
<tr>
<td>High risk</td>
<td>6 (7.4)</td>
</tr>
<tr>
<td>Moderate risk</td>
<td>32 (39.5)</td>
</tr>
<tr>
<td>Low risk</td>
<td>16 (19.8)</td>
</tr>
<tr>
<td>HDL levels of group with CVD history: (32.4%)</td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>7 (14.6)</td>
</tr>
<tr>
<td>Normal</td>
<td>41 (85.4)</td>
</tr>
<tr>
<td>HDL levels of group without CVD history: (67.6%)</td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>7 (14.6)</td>
</tr>
<tr>
<td>Normal</td>
<td>41 (85.4)</td>
</tr>
<tr>
<td>TG levels of group with CVD history: (32.9%)</td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>6 (24.0)</td>
</tr>
<tr>
<td>Normal</td>
<td>19 (76.0)</td>
</tr>
<tr>
<td>TG levels of group without CVD history: (67.1%)</td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>12 (23.5)</td>
</tr>
<tr>
<td>Normal</td>
<td>39 (76.5)</td>
</tr>
</tbody>
</table>

This study uses the contingency correlation test and eta. The relationship between the independent variables and the dependent variable of this study is considered significant if the p value is <0.05. This study shows that there is no significant relationship between each independent variable and the dependent variable. This study shows that all risk factors have a very weak relationship with the incidence of CVD (r square=0.2). This study shows that diet as a risk factor has the most significant relationship with CV events (r square=0.205; 4.2%) (Table 4).

Our study conducted an autocorrelation test between independent variables in order to determine whether there was a strong or weak relationship between each variables.
on the incidence of CVD. This study showed that gender had a weak but significant correlation with obesity in the group without CVD (p value 0.004; correlation 0.37; risk 13.6%). Gender in this study towards other independent variables showed a very weak correlation both in the CVD and non-CVD groups (correlation <0.2).

Table 4: Risk factors correlation within CVD incidences.

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>CVD</th>
<th>Correlation</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>19</td>
<td>0.091</td>
<td>0.413</td>
</tr>
<tr>
<td>Female</td>
<td>8</td>
<td>0.019</td>
<td>0.862</td>
</tr>
<tr>
<td>Familial history</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>8</td>
<td>0.132</td>
<td>0.232</td>
</tr>
<tr>
<td>No</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>5</td>
<td>0.061</td>
<td>0.585</td>
</tr>
<tr>
<td>No</td>
<td>22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood pressure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>4</td>
<td>0.196</td>
<td>0.072</td>
</tr>
<tr>
<td>Normotension</td>
<td>23</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obesity</td>
<td>10</td>
<td>0.136</td>
<td>0.221</td>
</tr>
<tr>
<td>Normal</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>9</td>
<td>0.198</td>
<td>0.069</td>
</tr>
<tr>
<td>No</td>
<td>18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HDL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>7</td>
<td>0.183</td>
<td>0.116</td>
</tr>
<tr>
<td>Normal</td>
<td>16</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abnormal</td>
<td>6</td>
<td>0.005</td>
<td>0.964</td>
</tr>
<tr>
<td>Normal</td>
<td>19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>0</td>
<td>0.136</td>
<td>0.221</td>
</tr>
<tr>
<td>No</td>
<td>26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical activity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>14</td>
<td>0.198</td>
<td>0.069</td>
</tr>
<tr>
<td>No</td>
<td>13</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Healthy diet</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>17</td>
<td>0.205</td>
<td>0.059</td>
</tr>
<tr>
<td>No</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LDL levels</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>High risk</td>
<td>3</td>
<td>-0.368</td>
<td>0.069</td>
</tr>
<tr>
<td>Moderate risk</td>
<td>9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low risk</td>
<td>15</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The correlation between hereditary history and CVD variables was carried out in this study. Family history with CVD had a significant and strong correlation with the LDL value that was considered risk in the non-CVD group (p value 0.004; correlation 0.512; risk 26.21%). Heredity actually showed a very weak correlation with the LDL-risk value in the CVD group (p value 0.717; correlation 0.136; risk 1.85%). Family history with CVD in this study against other independent variables showed a very weak correlation in both CVD and non-CVD groups (correlation <0.2).

This study analyzed the correlation between smoking history variables. This study showed smoking and hypertension had a significant relationship with a weak correlation (p value 0.043; correlation 0.266; risk 7.07%). Smoking and LDL-risk scores actually showed a negative correlation in the non-CVD group, meaning that respondents with a history of smoking had better LDL scores in the non-CVD group than non-smoking respondents (p value 0.466; correlation 0.329; risk 10.82%). Smoking history in this study against other independent variables showed a very weak and insignificant correlation in both the CVD and non-CVD groups (correlation <0.2).

In addition to the correlation between hypertension and smoking, a fairly strong and significant correlation occurred between hypertension and obesity in the CVD group (p value 0.005; correlation 0.478; risk 22.8%). Hypertension and LDL risk values clearly showed a very strong correlation and a significant relationship in the CVD group (p value 0.014; correlation 0.947; risk 89.68%). Hypertension and high TG also showed a strong significant relationship with a fairly strong correlation (p value 0.001; correlation 0.549; risk 30.14%). Hypertension history in this study towards other independent variables showed a very weak and insignificant correlation in both CVD and non-CVD groups (correlation <0.2).

Nutritional status is an important variable tested in this study. In addition to the correlation and relationship between nutritional status and the independent variables that have been listed previously, respondents who were considered obese had a significant and strong correlation with the LDL-risk number both in the CVD group (p value 0.002; correlation 0.774; risk 59.90%) and in the non-CVD (p value 0.000; correlation 0.757; risk 57.3%). Obesity and abnormal HDL values have a significant relationship with a strong enough correlation (p value 0.015; correlation 0.454; risk 20.61%). Obesity status and high TG scores were actually found to be strong with a weak correlation in the non-CVD group (p value 0.008; correlation 0.352; risk 12.4%). Although it did not have a significant relationship, obesity still had a weak correlation with good exercise habits in the CVD group (p value 0.148; correlation 0.268; risk 7.18%) and the non-CVD group (p value 0.123; correlation 0.207; risk 4.28%).

Psychological stress is one of the CVD risk factors in this research questionnaire. Although it was not significant, it showed that stress had a weak correlation to male gender both in the CVD group (p value 0.223; correlation 0.224; risk 5.01%) and the non-CVD group (p value 0.076; correlation 0.235; risk 5.52%). The stress factor has a weak correlation and no significant relationship with the risky LDL value in the CVD group (p value 0.305; correlation...
implications associated with obesity; stated that retic wrong, which have been discussed for their comparisons between 15 independent variables (including in this analytical study, we analyzed the autocorrelation correlation 0.485; risk 23.52%; correlation 0.658; risk 43.29%), and the LDL and other independent variables. HDL and TG values other lipid profile values tested in this study were HDL and TG values at risk and smoking history had no significant relationship with the CVD group, but the correlation between these two variables was strong in the CVD group (p value 0.092; correlation 0.632; risk 39.94%). The value of LDL at risk and hypertension clearly shows a negative correlation in the non-CVD group (p value 0.137; correlation -1; risk -100%), which means that the LDL value at risk is not owned in people with hypertension and vice versa in the non-hypertensive group. –CVD and LDL values at risk and abnormal HDL values had a strong correlation with the CVD group (p value 0.053; correlation 0.658; risk 43.29%), and the LDL risk value correlated negatively with HDL values in the non-CVD group (p value 0.697; correlation -0.151; risk -2.28%). The LDL value and its correlation with exercise showed a negative correlation in the CVD group which indicated that the LDL value was higher in the respondents who did not exercise (p value 0.739; correlation -0.115; risk -1.32%). The LDL value shows a negative correlation with diet in both the CVD group (p value 0.165; correlation 0.462; risk 21.34%) and the non-CVD group (p value 0.683; correlation -0.104; risk -1.08%), which indicates that the LDL value lower in respondents who do a healthy diet as recommended.

Other lipid profile values tested in this study were HDL and TG values, which have been discussed for their correlation with other independent variables. HDL and TG had no significant relationship but had a weak correlation in the CVD group (p value 0.226; correlation 0.245; risk 6.0%). An abnormal TG value actually has a strong significant relationship with a strong positive correlation to a healthy diet in the CVD group (p value 0.006; correlation 0.485; risk 23.52%).

DISCUSSION

In this analytical study, we analyzed the autocorrelation comparisons between 15 independent variables (including demographic data, laboratory data results, risk and protective factors) in CVRISK scoring of 81 participants who were excluded from both CVD and non-CVD groups.

A study by Kanter and Caballero in 2012 stated that the prevalence of overweight and obesity between men and women varied between countries due to sociocultural differences and nutritional transitions, but overall, more women were obese than men and more men were overweight than women, especially in developing countries. A study by Hinnoouho et al in 2013 assessed the health status of both metabolically healthy and unhealthy individuals based on several factors including blood pressure, TG, cholesterol (total, HDL, and LDL), fasting blood sugar (GDP), homeostasis model assessment (HOMA), visceral fat, and ectopic fat deposition along with obesity as assessed by measurement of body mass index (BMI), waist circumference and body fat percentage. Their study showed that the risk of mortality in the metabolically healthy group of obese individuals was lower (in relation to both all-cause and CVD), but was almost identical to the group of obese individuals who were metabolically unhealthy, and thus the criteria and determinants of metabolically healthy obesity or MHO is still unclear and needs to be studied further to be able to understand the consequences of in the long term health impacts (Figure 1).3

Research by Cercato and Fonseca in 2019 stated that obesity is an independent risk factor for CVD and one of the main causes of increased risk of diseases such as dyslipidemia, insulin resistance, high blood pressure (HBP) or hypertension, and atherosclerosis in both adults and children. CV complications associated with obesity were related to the pathophysiology involving hormones and peptides including inflammation, insulin resistance, endothelial dysfunction, coronary calcification, activation of coagulation, renin angiotensin or the sympathetic nervous system.10 The inflammatory state accompanied by an increase in adipose tissue and a decrease in adiponectin levels limits the inhibitory ability of the inflammatory process so that the inflammatory condition persists.11,12 This adipocyte dysregulation contributes to an imbalance of homeostasis and mechanisms of pro- and anti-inflammatory that results in obesity-induced metabolic complications and vascular damage leading to cardiometabolic changes (Figure 2).13

Figure 1: Global prevalence of overweight and obesity in males versus females (source: Adv Nutr, 2012).
Figure 2: Inflammatory process in obesity and its association with increased cardiovascular risk (source: Diabetology and Metabolic Syndrome, 2019).

Research conducted by Juhola et al in 2012 stated that a history of being overweight and obese increases the risk of hypertension by 1.65 times higher in adult individuals with CVD. An increase of 10 kg body weight was associated with an increase in systolic and diastolic blood pressure of 3 and 2.3 mm Hg, respectively, which resulted in a 12% increased risk of CHD and a 24% increased risk of stroke. National health and nutrition examination survey III (NHANES III) shows that the prevalence of hypertension increases progressively with increasing BMI, from 15% at BMI <25 kg/m² to 42% at BMI 30 kg/m² in men and from 15% at BMI <25 kg/m² to 38% at a BMI of 30 kg/m² in women.15 The study by Bays et al in 2013 also stated that the higher the increase in BMI, the higher the risk of lipid level abnormalities, where around 60-70% of obese patients and 50-60% of patients with overweight experience dyslipidemia which has the potential to increase the risk of CVD.16 Lipid level abnormalities in obese individuals include increased TG, very low density lipoprotein (VLDL), apolipoprotein B (apo B) and non-HDL cholesterol levels, while HDL and apo A-I levels are usually low.17,18 Study by Cameron, Magliano and Soderberg in 2013 emphasized that the effect of obesity on lipid metabolism depends on the location of adipose tissue where increased visceral adipose tissue and subcutaneous adipose tissue (especially upper trunk) are associated with higher TG and higher HDL cholesterol levels. In contrast, increased subcutaneous adipose tissue in the legs was associated with a lower TG. In addition, the increase in visceral adipose tissue and upper trunk subcutaneous adipose tissue is also associated with insulin resistance which can contribute to changes in these lipid levels.19

Dyslipidemia has an important role as a predictor of CVD due to its role in the atherosclerosis process.20 The study by Ariyanti and Besral in 2019 stated that the correlation between dyslipidemia and CVD varies depending on hypertension status. After controlling for age, hypertensive respondents with dyslipidemia were 18.1 times more likely to develop CVD than respondents without hypertension, while non-hypertensive respondents with dyslipidemia were 2.5 times more likely to develop CVD when compared to respondents, who don't have dyslipidemia.21 Research by Iskandar et al in 2017 shows that there is a correlation between TG cholesterol levels and the incidence of CHD, where the odds ratio (OR) value is 1.99 (95% confidence interval [CI]: 0.97-1.00), and high TG levels have a 1.99 times greater chance of CVD than patients who have normal TG levels.22 Study by Boo and Froelicher in 2012 showed that there was a strong relationship between LDL cholesterol and the risk of developing hypertension and CVD.23 Dyslipidemia and hypertension are the main important risk factors in CVD. If these two factors are present together, it will speed up the atherosclerosis process, thereby increasing the risk of CVD.24,25

In a study conducted by Bachmann, Willis, Ayers, Khera, and Berry in 2012, it was stated that the relationship between family history and CVD and the risk of individuals experiencing CVD was strongest at short-term follow-up (0-10 years) while the relationship in medium term (>10-20 years) and long term (>20 years) follow-up have not been clearly defined. The presence of a family history of CVD is associated with ±8% absolute difference and 50% difference in relative risk of life to mortality due to CVD, suggesting that having a family history of CVD clinically increases the risk of CVD in an individual during his lifetime.26 The study by Imes and Lewis in 2015 states that having a history of CVD in at least one parent can double the risk of CVD by 8 years among men and increase the risk by 70% among women, in addition this retrospective study has estimated OR of lifetime CV events for an individual with a single first degree relative (FDR) with a history of CV events (1.1-2.63; 6-11), the OR increased to 4.1 (95% confidence interval [CI]: 2.5-6.7), but no significant difference was found in changes in body weight, physical activity, LDL, HDL TG, systolic and diastolic blood pressure, and the tendency to quit smoking with a family history of heart attack or stroke in last 5 years.27 A study in 2019 by Kim, Han, Joung, Baek, Song and Kwon in healthy participants who did not undergo statin treatment, stated that LDL levels of 130-159 mg/dl and ≥160 mg/dl significantly increased the risk of occurrence, myocardial infarction: hazard ratios (HRs) (95% CI)=1.19 (1.12-1.25) and 1.53 (1.45-1.62), respectively.28

The study by Keto et al in 2015 stated that smokers had higher serum cholesterol, TG, and LDL levels, and lower HDL levels than nonsmokers but there was no significant difference between CVD risk based on Framingham's measurement or the SCORE algorithm among nonsmokers, smokers who recently quit smoking, and history of smoking groups (7.5%, 7.4% and 8.1% for men, 3.3%, 3% and 3.2% for women; p<0.001).29

Ahn and Kim's 2016 study showed that a decrease in HDL cholesterol was associated with an increased risk of coronary artery disease (CAD), whereas an increase in HDL levels was associated with a reduced risk of CAD and
myocardial infarction. Meanwhile in the research conducted by Shin and Kim in 2019, after adjusting for age, BMI, alcohol consumption, smoking, exercise, education level and income status in 23792 middle-aged male participants, there was a significant positive relationship between stress levels and OR of CVD where quartile 4 stress levels compared to quartile 1 was associated with a higher risk for CVD (OR: 1.38; 95% CI: 1.26-1.51; p trend <0.0001), hypertension (OR: 1.31; 95% CI: 1.19-1.43; p trend <0.0001), heart disease (OR: 1.52; 95% CI: 1.17-1.97; p trend=0.002), and cerebrovascular disease (OR: 2.80; 95% CI: 1.90-4.14; p trend <0.0001).32

A study by Scherer and Nicholls in 2015 discussing the comparison of a moderate-fat diet with a low-fat diet showed that a moderate-fat diet had a better effect on TG levels than a low-fat diet. Low-carb diets consistently lower TG, especially in those with higher baseline TG levels, and when combined with the Mediterranean diet shows superior results in TG-lowering effects compared to low-fat diets.33 A study by Brandhorst and Longo in 2019 shows that dietary choices or in combination with other lifestyle factors influence the risk of developing low-grade CVD and chronic inflammation, in which a high consumption of vegetables, fruits, whole grains, nuts, healthy oils and fish, like the Mediterranean diet, have anti-inflammatory properties that tend to lower CVD risk, whereas the nutritional pattern of the western diet which is higher in fat and cholesterol, consumption of red meat-based, high sugar and excess salt intake and processed foods and fast food has pro-inflammation which tends to increase the risk of CVD.34

Research conducted by Suman in 2016 on 40 participants, which were divided to 20 participants in the experimental group who performed aerobic physical activity for 8 weeks and 20 participants in the control group showed that the 8 weeks of aerobic training program in the experimental group significantly reduced body weight and BMI in the diabetic population (p value 0.00), and in order to reduce body fat mass, the required intensity of physical activity is 50-60%. Maximum heart rate (MHR) which is then gradually increased to the limit of the red zone of 90-100% MHR. This research is in line with Nicklas 2009 study which states that physical activity is recommended to be done for 30 minutes every day to treat obesity. This study also states that by combining physical activity with a controlled diet it will cause a decrease in fat mass including TG, LDL, and increase HDL and basal metabolic rate (BMR), which then help to reduce potential cardiovascular risk in individuals.35 Another study by Wang and Xu in 2017 also stated that the application of physical activity can reduce body weight and serum cholesterol so that it helps reduce the risk of CHD where a loss of weight per kg can reduce LDL-C by 0.8 mg/dl.36,37 Ekelund et al's 2016 study on 1,005,791 individuals with a total follow-up of 2-18.1 years showed that the mortality ratio was 12-59% higher in individuals who sat more for long periods of time on a daily basis compared to individuals who performed a lot of activity. physical daily which counteract this effect for a certain period.38 Research conducted by Yläimäki et al and Tiainen et al in 2016 stated that physical activity could increase oxidized HDL (oxHDL) and oxHDL/HDL and decrease oxLDL at the same time, but the anti-inflammatory and anti-oxidation functions of this process still require further research.39,40

CONCLUSION

In conclusion, from this research, we found a varied correlation from weak to very strong between each 15 independent variables on CVRISK score which was applied to the respondents, in which the very strong correlation was mostly found in independent variable of LDL that had a good correlation to both hypertension and tryglicerides in CVD group. LDL was also assessed to have a negative correlation with protective factor variables, such as physical activity in the CVD group and healthy diet in both CVD and non-CVD groups. Apart from LDL, nutritional status of obesity was also considered to have the most correlation with other variables such as gender, hypertension, and lipid profiles (LDL, HDL, and triglycrides), in which each variables varied from quite strong to strong correlation levels. Further research is needed, especially in a wider and more affordable age population distribution to determine the application of CVRISK as a scoring for early prevention in determining cardiovascular risk in both healthy individuals and individuals with a history of CVD.

ACKNOWLEDGEMENTS

The authors would like to express his deepest gratitude to almighty God, family, friends and all parties who have helped this research so that it can be carried out well, especially all the respondents who have been willingly participated in our research. The authors realize that this research may still not be perfect, so the author expects constructive criticism and suggestions to be able to improve and perfect this research, so that it can be useful for all parties.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee of Faculty of Medicine, Ciputra University, Jakarta, Indonesia

REFERENCES


22. Ariyanti R, Besral B. Dyslipidemia Associated with Hypertension Increases the Risks for Coronary Heart Disease: A Case-Control Study in Harapan Kita Hospital, National Cardiovascular Center, Jakarta. J Lipids. 2019;2517013.


Cite this article as: Goutama IL, Hendsun, Firmansyah Y, Su E. Comparison of autocorrelation between CV-RISK independent variables in groups with and without history of cardiovascular diseases. Int J Adv Med 2020;7:1626-34.