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Evaluation of Coronary Heart Disease Risk Factors Using Cox and Stratified Cox Proportional Hazards Models Before and After Propensity Score Matching

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ABSTRACT: Coronary Heart Disease (CHD) remains a major cause of mortality globally and in Indonesia, with rising prevalence and healthcare burden. This study investigates the relationship between cardiovascular risk factors and CHD incidence using a Cox Proportional Hazards model, complemented by Propensity Score Matching (PSM) to address confounding. A retrospective cohort design utilized data from the Indonesian Ministry of Health's Non-Communicable Disease (NCD) Risk Factor Cohort Study (2011–2019), involving 2,984 participants from Bogor City. Cox regression estimated hazard ratios (HRs) for various risk factors, while Kaplan-Meier curves and log-rank tests were applied for survival analysis. PSM was conducted for sex and smoking status to improve group comparability.

Before matching, high blood pressure (HR ≈ 47.7), LDL (HR ≈ 33.3), cholesterol (HR ≈ 19.9), blood sugar (HR ≈ 13.4), and obesity (HR ≈ 11.0) were dominant predictors of CHD. After matching, sex (HR ≈ 1.43, $p = 0.03$) and smoking ($p < 0.001$) also showed significant associations. Non-controllable factors such as age (HR ≈ 1.60) and family history contributed notably. The proportional hazard assumption was violated for age and smoking, necessitating stratified Cox models.

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The findings underscore the significant influence of both controllable such as obesity, cholesterol, smoking and un-controllable such as age,and sex risk factors on CHD. The use of PSM enhanced the validity of risk estimates, highlighting the importance of robust analytical methods in epidemiological research. Targeted prevention focusing on controllable risks is essential to curb CHD incidence.

Keywords: *Coronary Heart Disease (CHD), Cardiovascular risk factors, Cox Proportional Hazards model, Propensity Score Matching (PSM), controllable risk factors, un-controllable risk factors, Survival analysis, cohort study, Indonesia.*

Introduction

Coronary Heart Disease (CHD) remains a major global health concern. According to the World Health Organization (WHO), cardiovascular diseases account for over 17 million deaths annually worldwide (1). In Indonesia, CHD is responsible for approximately 245343 of the 651481 cardiovascular-related deaths each year (2). The economic burden is significant, with global CHD treatment costs estimated at 21.7% of GDP per capita, exceeding the global average healthcare expenditure (3). In Indonesia, BPJS data from November 2022 reported IDR 10.9 trillion spent on cardiovascular care, representing nearly half of the total national healthcare expenditure (4).

CHD is driven by controllable factors such as hypertension, smoking, obesity, cholesterol, blood glucose and un-controllable factors such as age, sex, family history (5)(6). Controllable factors, which are related to behavioral and metabolic patterns that accelerate atherosclerosis and blood vessel inflammation (7). Unmodifiable factors such as age and sex. Postmenopausal women face increased risk due to decreased estrogen (8).

Survival analysis, particularly the Cox Proportional Hazards (CPH) model is widely used to estimate the effect of covariates on time-to-event outcomes such as CHD (9). The model is valuable for its interpretability but requires validation of the proportional hazards assumption (10). Propensity Score Matching (PSM) is often employed to reduce confounding bias in observational studies (11)(12).

Literature Review

The Cox Proportional Hazards model is a method used in survival analysis to understand how certain variables affect the time until an event happens, such as death or failure. It

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assumes that each individual has a baseline risk of the event, and that the influence of variables like age, treatment, or sex changes this risk by a constant proportion over time. This assumption is called the "proportional hazards" assumption. The model does not require you to know the exact shape of the baseline risk over time, which makes it flexible. The results are usually expressed as hazard ratios — for example, a hazard ratio of 2 means the event is twice as likely to happen at any time point for that group, compared to the reference (10).

The Stratified Cox model is an extension of the regular Cox model. It is used when one or more variables do not follow the proportional hazards assumption. For example, if survival patterns are very different between hospitals or sexes and those patterns change over time, this model allows each group (or “stratum”) to have its own baseline risk pattern. Instead of estimating the effect of the stratified variable, it controls for it by letting the baseline risk vary across strata. The effects of the other variables are still estimated, and these are assumed to apply equally across all strata (10).

Propensity Score Matching (PSM) is a statistical method used to reduce bias when comparing two groups in observational studies, where random assignment (like in randomized controlled trials) is not possible. It helps estimate the causal effect of a treatment or intervention by ensuring that the treatment and control groups are similar in terms of observed characteristics (11)(12).

Propensity Score Matching (PSM) can be combined with the Cox Proportional Hazards model to analyze time-to-event data, especially in observational studies where treatment assignment is not random (11)(12).

In observational data, people who receive a treatment may differ from those who do not, which can create bias. Propensity Score Matching helps to reduce this bias by matching individuals in the treatment group with similar individuals in the control group based on observed characteristics such as age, sex, or health status. This matching creates two groups that are more comparable, as if they had been randomly assigned (11)(12).

Once the groups are matched, the Cox Proportional Hazards model can be used to estimate the effect of the treatment on the time until an event occurs, such as death or recovery. The Cox model takes into account the timing of the event and provides an estimate of the

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hazard ratio, which tells you how much more or less likely the event is to occur in the treatment group compared to the control group (11)(12).

This combined approach helps to improve the accuracy of the estimated treatment effect. By first reducing bias through matching and then analyzing time-to-event outcomes through the Cox model, researchers can make more reliable conclusions about the impact of a treatment. However, it's important to check that the matching worked well by comparing the characteristics of the groups after matching. Also, because the data is matched, researchers often use robust standard errors or stratification in the Cox model to account for the matched pairs (11)(12).

Materials and Methods

A retrospective cohort study was conducted using data from the Indonesian Ministry of Health's NCD Risk Factor Cohort Study (2011–2019) in Bogor City. After excluding 818 baseline CHD cases, 2,984 eligible participants remained, aged ≥40 years or <40 years with physician-

diagnosed hypertension (13). Diagnosis of heart disorders is based on the results of ECG (electrocardiogram) examinations. In this study, ECG examinations were only conducted on individuals aged 40 years and older, and on those under 40 years old who had been diagnosed with hypertension by a doctor. Respondents under 40 years old without hypertension were not subjected to ECG examination.

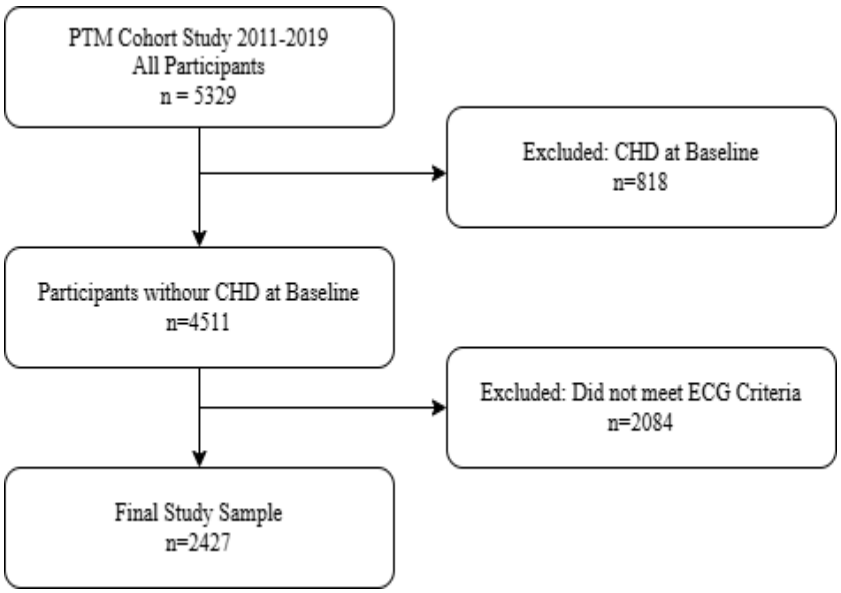

Figure 1. Study population screening flow chart

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CHD outcomes were tracked biennially. CPH models were used to estimate hazard ratios (HRs), adjusting for age, sex, BMI, blood pressure, cholesterol, blood glucose, smoking, and family history. Kaplan-Meier curves and log-rank tests assessed survival differences. PSM (1:1) was applied for sex and smoking. Schoenfeld residuals indicated violations for age and smoking; stratified Cox models were used. Analyses were conducted in R 4.3.2 using the survival and survminer packages (14)(15)(16). A p-value < 0.05 was considered statistically significant.

Findings and analysis

This study evaluated the impact of cardiovascular risk factors on coronary heart disease (CHD) progression using longitudinal data from 2011 to 2019. The number of participants decreased from 2984 to 2448 due to events (240) and censoring (2208), highlighting the complexities of long-term follow-up (Table 1).

Table 1. Risk, event and censoring

Time	2013	2015	2017	2019
Number at Risk	2984	2674	2584	2448
Cumulative Number of Events	141	40	136	240
Cumulative Number of Censoring	169	50	0	2208

From Table 2 at before PSM, Age, BMI, blood sugar, cholesterol, LDL, HDL, blood pressure, and family history were significantly associated with CHD ($p < 0.05$). CHD prevalence was higher in older adults (24% vs. 12%, OR ≈ 2.33), obese individuals (42% vs. 6%, OR ≈ 11), and those with high blood sugar (48% vs. 7%, OR ≈ 13.4). Elevated cholesterol (69% vs. 6%, OR ≈ 19.9) and LDL (42% vs. 2%, OR ≈ 33.3) also showed strong associations and normal HDL appeared protective (OR ≈ 0.23). Hypertension presented the highest risk (88% vs. 6%, OR ≈ 47.7).

Table 2. Demographic and medical history characteristics

		Before PSM				**P-value**					**P-value**
		Non-CHD		CHD			Non-CHD		CHD		
	N=2984	N=2427	%	N=557	%	χ^2	**N=850**	N=611	%	N=239	%	χ^2
Age (X1)						**0,000***						**0,01***
⩽ 45	1338	1177	88	161	12		267	209	78	58	22	
> 45	1646	1250	76	396	24		583	402	69	181	31	
Sex (X2)						**0,17**						**0,03***
Female	2063	1664	81	399	19		425	320	75	105	25	
Male	921	763	83	158	17		425	291	68	134	32	
BMI (X3)						**0,000***						**0,000***

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Normal	1943	1819	94	124	6	557	502	90	55	10
Obesity	1041	608	58	433	42	293	109	37	184	63
Blood sugar (X4)					**0,000***					
Normal	2124	1984	93	140	7	546	487	89	59	11
High	860	443	52	417	48	304	124	41	180	59
Cholestrol (X5)					**0,000***					**0,000***
Normal	2372	2238	94	134	6	627	568	91	59	9
High	612	189	31	423	69	223	43	19	180	81
LDL (X6)					**0,000***					**0,000***
Normal	1746	1712	98	34	2	466	454	97	12	3
High	1238	715	58	523	42	384	157	41	227	59
HDL (X7)					**0,000***					**0,000***
Low	1087	724	67	363	33	502	291	58	211	42
Normal	1897	1703	90	194	10	348	320	92	28	8
Blood pressure (X8)					**0,000***					**0,000***
Normal	2515	2372	94	143	6	646	598	93	48	7
Hypertension	469	55	12	414	88	204	13	6	191	94
Smoking (X9)					**0,07**					**0,000***
No	2221	1789	81	432	19	735	586	80	149	20
Yes	763	638	84	125	16	115	25	22	90	78
Family history of CHD (X10)					**0,000***					**0,000***
No	2826	2427	86	399	14	778	611	79	167	21
Yes	158	0	0	158	100	72	0	0	72	100

From Table 2 at after PSM the most associations remained significant. Age ($p = 0.01$, OR ≈ 1.60), sex ($p = 0.03$, OR ≈ 1.43), BMI ($p = 0.000$, OR ≈ 15.5), blood sugar (CHD: 59% vs. 11%), cholesterol (81% vs. 9%, OR $\approx$ 39), and LDL (59% vs. 3%, OR $\approx$ 48.3) continued to be strong predictors. Normal HDL remained protective (CHD: 8% vs. 42%, OR ≈ 0.13). Hypertension showed the strongest association (94% vs. 7%, OR ≈ 204.8),

Overall, the results support existing clinical knowledge that CHD is strongly associated with controllable factors such as obesity, hypertension, dyslipidemia, smoking, diabetus mellitus and uncontrolled factors such as age, sex, and family history. The findings highlight the importance of managing cardiovascular risk factors to prevent CHD and underline the utility of PSM in reducing confounding in observational data.

After adjusting for sex using Propensity Score Matching (PSM), we examined the survival probability of coronary heart disease (CHD) progression with Kaplan-Meier survival curves. Figure 2(a) shows the overall survival curve, where survival decreased over time—from

0.93 in 2013 to 0.70 in 2019. Figure 2(b) compares smokers and non-smokers. Before PSM, no clear difference was seen. After matching, smokers showed much lower survival, falling from 0.84 in 2013 to 0.19 in 2019. Non-smokers started at 0.94 and ended at 0.78. This difference was significant ($p < 0.0001$), showing smoking's harmful effect on heart health.

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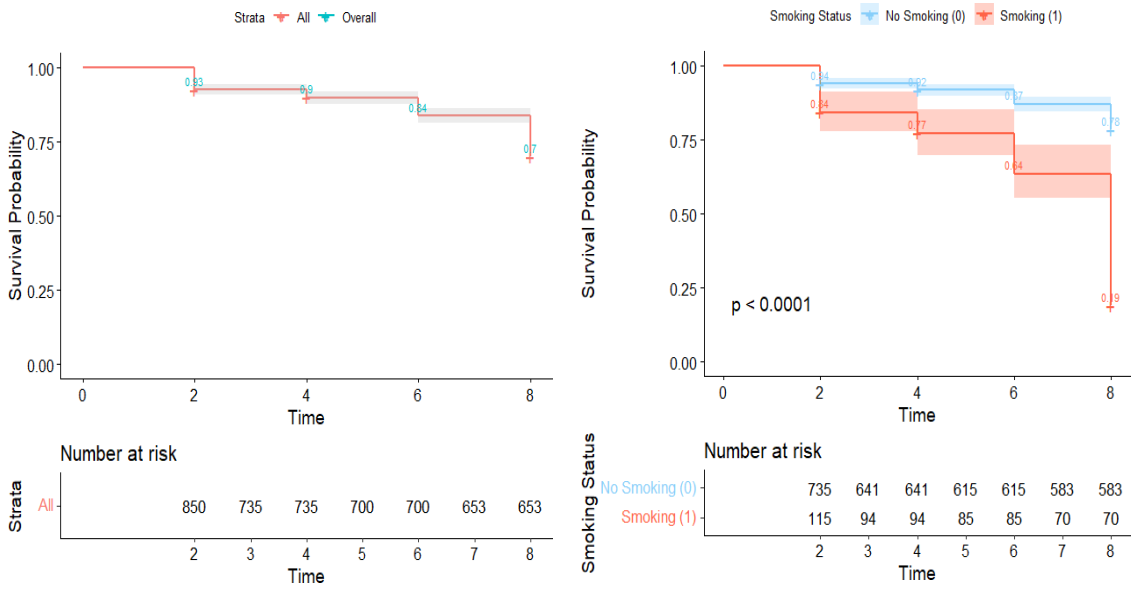

Figure 2. Kaplan Meier curves (a) general cohort (b) smoking status

We then used the Cox Proportional Hazards (CPH) model to assess the effect of risk factors. High LDL (HR = 6.79) and high blood pressure (HR = 4.92) were the strongest predictors of CHD. High cholesterol, blood sugar, smoking, and family history also increased risk significantly. HDL, the "good" cholesterol, reduced the risk by 46%. Age, sex, and BMI showed no significant effect (Table 3).

Table 3. Hazard ratios from the cox proportional hazards model

Variable	HR	95% CI (Lower, Upper)	p-value
Age	1.24	(0.91, 1.69)	0.166
Sex	1.06	(0.81, 1.39)	0.684
BMI	1.13	(0.75, 1.71)	0.559
Blood sugar	1.51	(1.07, 2.13)	0.020
Cholestrol	2.00	(1.41, 2.85)	0.0001
LDL	6.79	(3.58, 12.85)	$<2*10^{-16}$
HDL	0.54	(0.36, 0.85)	0.007
Blood pressure	4.92	(3.14, 7.73)	$<2*10^{-16}$
Smoking status	1.47	(1.09, 1.97)	0.011
Family history CHD	170	(1.25, 2.32)	0.001

In summary, the analysis shows that controlling LDL, blood pressure, and other controllable risk factors like smoking and blood sugar is vital for reducing CHD risk, while higher HDL levels provide protection.

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Before interpreting the final model, we tested the proportional hazards assumption. The Schoenfeld residuals test showed that this assumption was violated for smoking status ($p = 0.0021$) and age ($p = 0.00037$). This meant the hazard ratios for these variables changed over time. To address this, we used a stratified Cox regression model, stratifying the analysis by smoking status and age.

Table 4. Stratified Cox Proportional Hazards model

Variable	HR	95% CI (Lower, Upper)	p-value
Sex	1.06	(0.81, 1.39)	0.684
BMI	1.07	(0.75, 1.71)	0.559
Blood sugar	1.63	(1.07, 2.13)	0.020
Cholestrol	1.92	(1.41, 2.85)	0.0001
LDL	6.59	(3.58, 12.85)	$<2*10^{-16}$
HDL	0.58	(0.38, 0.90)	0.007
Blood pressure	4.72	(3.14, 7.73)	$<2*10^{-16}$
Family history CHD	1.70	(1.25, 2.32)	0.001

Table 4 presents the results. After stratification, high LDL, high blood pressure, and high cholesterol remained strong and significant risk factors for coronary heart disease (CHD). Blood sugar and family history of CHD also showed significant associations. HDL was protective, while sex and BMI were not significant predictors. The hazard ratio for LDL was especially high (HR = 6.59), highlighting its key role in CHD risk. These findings support the importance of managing cholesterol, blood pressure, and blood sugar to prevent CHD, especially in individuals with a family history or other known risk factors.

The analysis in Tables 3 and 4, the Cox model provides unadjusted hazard ratios (HRs), while the Stratified model adjusts for age and smoking status. Age was not a significant risk factor in the Cox model (HR = 1.24, p = 0.166) and was stratified in the second model to reduce confounding. Sex and BMI were not significant in either model. Blood sugar, cholesterol, LDL, blood pressure, and family history showed consistent and statistically significant associations with increased CHD risk across both models. HDL consistently acted as a protective factor. The impact of smoking was significant in the Cox model but omitted in the stratified model due to stratification. Overall, both models confirmed that

 DOI: *10.5281/zenodo.22093419*

high LDL, blood pressure, and cholesterol are key risk factors for CHD, even after adjusting for time-dependent effects.

Discussion

The results presented before that underscore key risk factors for coronary heart disease (CHD), particularly age, sex, BMI, and elevated blood sugar levels, each of which is supported by well-established physiological mechanisms and epidemiological literature. Age remains a prominent non-modifiable factor, with CHD prevalence markedly higher among older individuals (e.g., 24% vs. 12%; 31% vs. 22%). This aligns with (17) , who highlighted vascular and myocardial aging—through endothelial dysfunction and arterial stiffness—as critical contributors to CHD. Endothelial cells produce less nitric oxide with age, impairing vasodilation and increasing inflammation and thrombosis. Arterial stiffening further increases cardiac workload, promoting left ventricular hypertrophy and elevating CHD risk.

Sex differences, although variably significant in the data ($p = 0.17$ and $p = 0.03$), are biologically grounded. It explained that estrogen provides cardiovascular protection in premenopausal women via enhanced nitric oxide availability and anti-inflammatory effects. CHD risk rises post-menopause, closing the sex gap—consistent with your observed male-female differences (32% vs. 25%). Sampling variation, age distribution, and hormonal status could explain initial non-significance (18).

Results highlights a consistently strong and statistically significant association between Body Mass Index (BMI) and coronary heart disease (CHD) across both unmatched and matched datasets, reinforcing the role of obesity as a key modifiable risk factor for cardiovascular disease. Before Propensity Score Matching (PSM), chi-square analysis indicated a significant relationship ($p = 0.000$), with 42% of obese individuals having CHD compared to just 6% of those with normal BMI. This stark difference reflects a strong link between excess body weight and CHD prevalence. However, given the observational nature of the data, the association could be influenced by confounding variables such as age, hypertension, diabetes, physical inactivity, poor diet, and socioeconomic status. After adjusting for these through PSM, the association became even stronger—63% of obese individuals had CHD compared to 10% with normal BMI ($p = 0.000$), suggesting a direct

 DOI: *10.5281/zenodo.22093419*

relationship. This reinforces that obesity may contribute independently to CHD development. Prior studies (19)(20)(21)(22) support this through evidence of biological mechanisms such as insulin resistance, systemic inflammation, dyslipidemia, and hypertension. Moreover, central adiposity has been shown to elevate myocardial infarction risk, and long-term cohort data confirm that rising BMI correlates with a greater CHD risk, even after adjusting for key metabolic factors.

Hyperglycaemia showed a strong association with CHD both before and after propensity score matching, with an increased risk ranging from 48% to 59%. Diabetes has been shown to significantly elevate the risk of CHD, with some studies reporting a two- to fourfold increase (23). Mechanistically, hyperglycaemia contributes to endothelial dysfunction and oxidative stress, which in turn accelerate the development and destabilization of atherosclerotic plaques (24).

The consistent finding that individuals with high cholesterol levels are significantly more likely to develop coronary heart disease (CHD) underscores the critical role of dyslipidemia as a modifiable risk factor in cardiovascular health. Notably, the strength of this association becomes even more pronounced after applying Propensity Score Matching (PSM), indicating that cholesterol's contribution to CHD risk is not only significant but also independent of other confounding variables.

Cholesterol, particularly low-density lipoprotein (LDL), is a central contributor to the pathogenesis of atherosclerosis, the primary underlying mechanism of CHD. Elevated total and LDL cholesterol levels promote fatty plaque accumulation within arterial walls, which narrows and stiffens coronary vessels, reducing myocardial perfusion and elevating the risk of ischemic events.

High levels of total cholesterol significantly contribute to the development of atherosclerosis. Identifying this condition early and initiating lipid-lowering treatment are crucial steps in lowering the risk of coronary heart disease (25).

The importance of statin therapy has been further emphasized, establishing it as a key component in both the prevention of initial cardiovascular events and the reduction of recurrence. Evidence from large-scale cohort studies has demonstrated that a 1% decrease in cholesterol levels is associated with a 2–3% reduction in the risk of coronary heart

 DOI: *10.5281/zenodo.22093419*

disease, underscoring the dose- dependent relationship between cholesterol and CHD risk (26)(27).

The consistent and statistically significant associations between dyslipidaemia, hypertension, smoking, and family history with coronary heart disease (CHD), both before and after Propensity Score Matching (PSM), emphasize the robust and independent roles of these factors in cardiovascular risk. Among these, elevated LDL cholesterol remains a central causal factor. Before PSM, 42% of CHD patients had high LDL compared to 2% without CHD ($p = 0.000$), and after PSM, the figures were 59% and 3%, respectively. These findings corroborate LDL's atherogenic role through endothelial infiltration, oxidation, and plaque formation (28). Mendelian randomization and clinical trials further affirm LDL's causal role and the effectiveness of statins and PCSK9 inhibitors in reducing events(29).

Similarly, low HDL-C was significantly associated with CHD before (33% vs. 10%) and after PSM (42% vs. 8%), reinforcing its protective functions including reverse cholesterol transport and anti-inflammatory actions (30)(31). Although its causal role is debated by (32), HDL remains a critical marker of cardiovascular health.

Hypertension also showed a strong association with CHD, rising from 88% vs. 6% pre-PSM to 94% vs. 7% post-PSM. This finding aligns with extensive evidence that elevated blood pressure damages vasculature, promotes atherosclerosis, and increases myocardial workload(33)(34). Smoking showed a weak association before PSM ($p = 0.07$), but a strong one after (78% vs. 20%, $p < 0.001$), supporting the known pathophysiological pathways including endothelial dysfunction, oxidative stress, and thrombogenesis (35).

Family history had a perfect correlation with CHD in this dataset (100% vs. 0%, $p = 0.000$), highlighting genetic predispositions such as familial hypercholesterolemia and early subclinical atherosclerosis(36)(37). The persistence of this association after PSM underscores its role as an independent risk factor.

These findings support current ACC/AHA guidelines that prioritize LDL control, blood pressure management, and smoking cessation in CHD prevention (ACC/AHA, 2018), reinforcing the critical need for early screening and intervention strategies.

 DOI: *10.5281/zenodo.22093419*

Recommendations

Based on the findings discussed, several important recommendations can be drawn to strengthen the prevention and management of coronary heart disease (CHD). To begin with, early identification of individuals at risk is essential. Regular screening for major risk factors—such as age, sex, body mass index (BMI), blood glucose levels, blood pressure, and cholesterol—should be implemented as a routine part of preventive healthcare. While age and sex are non-modifiable, recognizing these factors can help guide the intensity of monitoring and intervention efforts.

Obesity stands out as a critical modifiable risk factor. Given its strong and consistent association with CHD across both unmatched and matched datasets, public health strategies must prioritize weight control through the promotion of healthy eating habits and regular physical activity. Interventions at both the individual and community level are needed to address the rising burden of obesity-related cardiovascular risk.

In addition, the management of elevated blood sugar levels is equally important. Hyperglycemia contributes to vascular damage and the progression of atherosclerosis, thereby increasing the risk of CHD. Individuals with prediabetes or diabetes should receive comprehensive care focused on blood sugar control, including lifestyle modification, dietary counseling, and appropriate medical therapy.

Another central factor is dyslipidemia, particularly elevated LDL cholesterol. The evidence strongly supports the causal role of LDL in atherosclerosis and CHD. Therefore, lipid-lowering treatments such as statins—and when necessary, PCSK9 inhibitors—should be widely used, especially in high-risk groups. At the same time, maintaining healthy HDL cholesterol levels through lifestyle measures can offer additional cardiovascular protection, even though its direct causal role is still debated.

Hypertension, which is highly prevalent among CHD patients, must also be addressed. High blood pressure leads to increased cardiac workload and damages blood vessels over time. Effective blood pressure management through lifestyle change and antihypertensive medication should be a cornerstone of CHD prevention strategies.

 DOI: *10.5281/zenodo.22093419*

Smoking, although showing weaker associations before adjustment, emerged as a strong predictor of CHD after correcting for confounders. This finding reinforces the urgent need for robust smoking cessation programs, supported by policy, education, and access to cessation aids.

Family history of CHD also proved to be a strong and independent risk factor. Individuals with a family history should be considered high priority for early screening and preventive counseling, as they may carry genetic risks such as familial hypercholesterolemia or early-onset atherosclerosis. Together, these findings support current international guidelines, including those from the ACC/AHA, which emphasize the importance of controlling cholesterol and blood pressure, promoting healthy lifestyles, and eliminating tobacco use. Public health policies and clinical practices should align with these evidence-based recommendations to effectively reduce the burden of CHD.

Lastly, raising awareness among both the public and healthcare professionals is essential. Education campaigns, community-based programs, and continued medical training can play a vital role in improving early detection, encouraging healthier behavior, and ultimately reducing the incidence of CHD at the population level.

Conclusion

This study highlights the significant association between multiple cardiovascular risk factors and coronary heart disease (CHD), using robust analysis before and after Propensity Score Matching (PSM). Before matching, high blood pressure, LDL, cholesterol, blood sugar, and obesity emerged as the strongest CHD predictors, while HDL was protective. After PSM, additional variables such as sex and smoking became significant, demonstrating the importance of adjusting for confounding. Age and sex showed moderate associations, with older individuals and males at higher risk. Persistently strong predictors like BMI, blood sugar, cholesterol, LDL, HDL, blood pressure, and family history remained significant post-matching, reinforcing their independent roles. The biological plausibility of these findings is supported by existing research, linking these factors to mechanisms such as inflammation, atherogenesis, and vascular dysfunction. These results emphasize the need to target controllable risk factors,especially obesity,

 DOI: *10.5281/zenodo.22093419*

hyperglycemia, and dyslipidemia,while promoting preventive strategies like hypertension control and smoking cessation for effective CHD risk reduction.

Ethical Statement

This study was conducted using secondary data from the Indonesian Ministry of Health's Non-Communicable Disease (NCD) Risk Factor Cohort Study (2011–2019). Ethical approval for the cohort study was obtained from the Health Research Ethics Committee of the Ministry of Health of the Republic of Indonesia (approval number: LB.02.01/2/KE.706/2018. March 1, 2018 and LB.02.01/2/KE.102/2019, March 26, 2019. All participants provided written informed consent at baseline. The present analysis used anonymized data and was performed in accordance with the Declaration of Helsinki.

Authors' Contributions

All authors made substantial contributions to this research and approved the final manuscript. Muhammad Nur Aidi, Fitrah Ernawati , Alfons M Latelay contributed to the data analysis and interpretation, writing and review. Muhammad Nur Aidi, Fitrah Ernawati contributed to English translate, and data interpretation. Fitrah Ernawati, Alfons M Latelay, Efriwati Efriwati, Nunung Nurjanah contributed to every step (research concept, design, investigation, data interpretation, writing, editing, and review). Hanung Safriza, Sachnaz Desta Oktarina, Fifi Retiaty, Rika Rachmawati, Nuzuliyati Nurhidayati, Elisa D. Julianti contributed to writing, data interpretation

and review. Salimar Salimar, Aya Y. Arifin, Dian Sundari, Budi Setyawati, Nazarina-Nazarina contributed to investigation, data curation, writing, and editing.

Conflicts of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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