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ORIGINAL ARTICLE

Coronary heart disease risk factors: Family history, smoking, and diet among patients at Royal Prima General Hospital

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ABSTRACT

Background: Coronary heart disease (CHD) is a leading cause of death worldwide, influenced by both modifiable and non-modifiable risk factors. This study aimed to analyze risk factors for CHD based on family history, smoking activity, and dietary habits among patients at Royal Prima General Hospital Medan.
Methods: A quantitative study with a cross-sectional design was conducted from December 16, 2025 to January 6, 2026. The sample comprised 35 CHD patients selected through purposive sampling. Data were collected using structured questionnaires and medical records, and analyzed using univariate, descriptive bivariate, and descriptive multivariate analyses.
Results: Results showed that a portion of respondents had a family history of CHD, nearly half were active smokers with smoking duration exceeding 10 years, and the majority exhibited risky dietary habits including frequent consumption of fried foods, coconut milk-based foods, fast food, and sweetened beverages. Descriptive bivariate analysis revealed tendencies toward clustering of risk factors, with smokers more likely to have unhealthy dietary patterns. Descriptive multivariate analysis demonstrated that most patients had multiple concurrent risk factors, particularly combinations of family history, smoking, and unhealthy dietary habits.
Conclusion: In conclusion, CHD occurrence among patients is influenced by the interaction of multiple risk factors rather than any single factor. Promotive and preventive efforts focusing on comprehensive healthy lifestyle modifications are needed to reduce CHD risk.

Keywords: coronary heart disease, family history, smoking, dietary habits, risk factors

Introduction

Coronary heart disease (CHD), also referred to as stable angina, acute coronary syndrome, silent myocardial ischemia, or ischemic heart disease, is characterized by gradual and chronic narrowing of coronary arteries that progresses from childhood through old age. In some instances, accumulated plaque in arterial walls may rupture rapidly, causing blood clots and exacerbating obstruction.[1–3]

Cardiovascular diseases, which affect the heart and blood vessels, caused 17.9 million deaths according to 2021 WHO data, representing 32% of global deaths, with 85% attributed to heart attacks and strokes.[4,5] CHD specifically involves atherosclerosis, where arterial walls become blocked or narrowed, restricting oxygen-rich blood flow. In 2022, CHD remained the leading cause of death worldwide with a mortality rate of 109 per 100,000 population and a prevalence of 3.6%.[6,7] In Indonesia, BPJS Kesehatan

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covered heart disease costs totaling Rp 23.52 trillion in 2023. This disease ranks among the largest claim drivers, with a trend toward younger patients (diagnosed at <43 years) due to lifestyle factors, which also reduces economic productivity.[8]

Blood vessel obstruction due to lipid accumulation increases with age.[9] Individuals over 45 years typically experience declining tissue and organ function, elevating CHD risk. Males demonstrate higher susceptibility, partly attributed to smoking and alcohol consumption patterns, while estrogen provides relative protection for menstruating females.[10,11] Beyond age and sex, family history represents a significant non-modifiable risk factor. Individuals with affected family members face increased likelihood of developing CHD, particularly when combined with unhealthy lifestyles. Smoking contributes substantially through chemical compounds and carbon monoxide that progressively narrow blood vessels.[12–15]

Energy imbalance between caloric intake and expenditure, particularly without adequate physical activity, constitutes a primary mechanism for lipid accumulation. This condition substantially increases CHD probability.[16,17] Diabetic patients face approximately twofold increased CHD risk compared to non-diabetic individuals.[18] Elevated total cholesterol, LDL, and triglyceride levels with concomitant HDL reduction frequently result from diets rich in saturated fat and cholesterol, ultimately promoting atherosclerosis.[16,19] The present study investigated CHD risk factors including family history, smoking activity, and dietary habits among patients at Royal Prima General Hospital Medan.

Method

This quantitative study employed a cross-sectional design. The research was conducted at Royal Prima General Hospital Medan from December 16, 2025 to January 6, 2026. The study population comprised all patients diagnosed with CHD and treated at the hospital during the study period. The sample consisted of 35 respondents selected through purposive sampling. Inclusion criteria included willingness to participate, CHD diagnosis confirmed by cardiologists in medical records, and age ≥30 years. Exclusion criteria included unwillingness to participate and presence of severe comorbid conditions interfering with questionnaire completion.

Research instruments included structured questionnaires containing items on family history of CHD, smoking activity (quantity, duration, type), and dietary habits assessed using a simplified Food Frequency Questionnaire (FFQ), complemented by medical record review to confirm CHD diagnosis. Data were analyzed using univariate analysis to describe frequency distributions of each variable, descriptive bivariate analysis through cross-tabulation to examine tendencies between variables, and descriptive multivariate analysis to describe combinations of multiple risk factors among respondents.

Results

Table 1 presents the characteristics of a sample of thirty-five respondents, categorized by their family history of coronary heart disease, smoking status, and weekly dietary habits. An analysis of the family history of coronary heart disease indicates that a majority of the respondents, comprising twenty-one individuals or 60.0 percent, have a reported family history of the condition. Within this specific subset, a paternal history is the most prevalent, reported by twelve respondents representing 34.3 percent of the total sample. A maternal history is present in five respondents, equating to 14.3 percent, while four respondents, or 11.4 percent, report a sibling with the disease. The remaining fourteen respondents, accounting for 40.0 percent, have no family history of coronary heart disease.

Regarding smoking behaviors, nearly half of the sample identifies as active smokers, with seventeen individuals making up 48.6 percent of the group. Former smokers and those who have never smoked each account for nine respondents, representing 25.7 percent per category. Among the participants who smoke, the daily consumption rate is highest in the category of eleven to twenty cigarettes per day, which includes eleven respondents or 31.4 percent. Seven individuals, or 20.0 percent, smoke five to ten cigarettes daily. The categories of fewer than five cigarettes and more than twenty cigarettes per day each consist of four respondents, equating to 11.4 percent for each group. The nine non-smokers represent the remaining 25.7 percent of this variable. In terms of smoking duration, fourteen respondents, or 40.0 percent, have smoked for more than ten years. Eight individuals, or 22.9 percent, have a smoking history of five to ten years, and four respondents, or 11.4 percent, have smoked for fewer than five years.

Table 1. Respondent characteristics

Variable	Frequency (n)	Percentage (%)
Family History of CHD		
Father		
Yes	12	34.3
No	23	65.7
Mother		
Yes	5	14.3
No	30	85.7
Sibling		
Yes	4	11.4
No	31	88.6
Any family history		
Yes	21	60.0
No	14	40.0
Smoking Status		
Never smoked	9	25.7
Former smoker	9	25.7
Active smoker	17	48.6
Cigarettes/day		
Non-smoker	9	25.7
<5 cigarettes	4	11.4
5-10 cigarettes	7	20.0
11-20 cigarettes	11	31.4
>20 cigarettes	4	11.4
Smoking Duration		
Non-smoker	9	25.7
<5 years	4	11.4
5-10 years	8	22.9
>10 years	14	40.0
Dietary Habits (frequency/week)		
Fried foods		
1-2 times	10	28.6
3-4 times	15	42.9
>5 times	10	28.6
Coconut milk-based foods		
1-2 times	12	34.3
3-4 times	13	37.1
>5 times	10	28.6
Red meat		
1-2 times	16	45.7
3-4 times	11	31.4
>5 times	8	22.9
Fast food		
1-2 times	18	51.4
3-4 times	10	28.6
>5 times	7	20.0
Sweetened beverages/soda		
1-2 times	11	31.4
3-4 times	14	40.0
>5 times	10	28.6
Vegetables		
1-2 times	14	40.0
3-4 times	13	37.1
>5 times	8	22.9
Fruits		
1-2 times	15	42.9
3-4 times	12	34.3
>5 times	8	22.9

The table also details the dietary habits of the respondents, measured by the frequency of consumption per week. For fried foods, the most common frequency is three to four times a week, reported by fifteen respondents or 42.9 percent. The remaining respondents are evenly divided, with ten individuals or 28.6 percent consuming fried foods one to two times a week, and another ten individuals consuming them more than five times a week. The consumption of coconut milk-based foods follows a relatively even distribution,

with thirteen respondents or 37.1 percent consuming them three to four times a week, twelve respondents or 34.3 percent consuming them one to two times, and ten respondents or 28.6 percent consuming them more than five times weekly.

Red meat consumption is predominantly limited to one to two times per week by sixteen respondents, accounting for 45.7 percent of the sample. Eleven respondents or 31.4 percent consume red meat three to four times a week, and eight respondents or 22.9 percent consume it more than five times a week. Fast food intake is similarly concentrated at lower frequencies, with eighteen respondents or 51.4 percent eating it one to two times a week. Ten individuals or 28.6 percent consume fast food three to four times weekly, while seven individuals or 20.0 percent eat it more than five times a week.

The data for sweetened beverages and soda shows fourteen respondents or 40.0 percent consuming them three to four times a week, eleven respondents or 31.4 percent having them one to two times, and ten respondents or 28.6 percent drinking them more than five times a week. Finally, the intake of vegetables and fruits is concentrated in the lower frequency brackets. Fourteen respondents or 40.0 percent consume vegetables one to two times a week, thirteen respondents or 37.1 percent consume them three to four times, and eight respondents or 22.9 percent eat them more than five times weekly. Similarly, fifteen respondents or 42.9 percent consume fruits one to two times a week, twelve respondents or 34.3 percent eat them three to four times, and eight respondents or 22.9 percent consume them more than five times per week.

Table 2 presents the cross-tabulated distribution of smoking status among a total cohort of 35 individuals, categorized by the presence or absence of a family history of smoking. The overall sample consists of 21 subjects with a family history and 14 subjects without such a history. Across the entire dataset, the aggregated smoking status comprises 17 active smokers, 9 former smokers, and 9 individuals who have never smoked. Among the 21 individuals with a confirmed family history of smoking, the highest frequency is observed in the active smoker category. Specifically, 13 individuals within this subgroup are currently active smokers. The remaining subjects in this cohort consist of 5 former smokers and 3 individuals who have never smoked. This indicates a higher absolute count of active smoking within the subgroup that possesses a recorded family history.

Table 2. Distribution of smoking status by family history

Family History	Active Smoker	Former Smoker	Never Smoked	Total
Present	13	5	3	21
Absent	4	4	6	14
Total	17	9	9	35

The distribution of smoking status varies notably within the subgroup of 14 individuals lacking a family history of smoking. In this cohort, the largest single segment consists of 6 individuals who report never having smoked. The remaining subjects in this category are evenly divided, with 4 individuals identified as active smokers and 4 individuals identified as former smokers. Comparing the two subgroups reveals a divergence in smoking behavior relative to familial background. The group with a family history is heavily skewed toward active smoking, whereas the group without a family history displays a more dispersed distribution, with the highest single frequency occurring among those who have never smoked.

Table 3 presents the distribution of smoking duration categorized by the smoking status among a total sample of thirty-five participants. The data is divided into three primary smoking statuses: active smokers, former smokers, and those who have never smoked. Furthermore, the duration of smoking is segmented into three specific intervals: less than five years, five to ten years, and greater than ten years. Out of the thirty-five participants, seventeen are classified as active smokers. Within this active subgroup, two individuals have a smoking duration of less than five years, five individuals fall into the five to ten years category, and the largest concentration consists of ten individuals who have smoked for more than ten years.

Table 3. Distribution of smoking duration by smoking status

Smoking Status	<5 Years	5-10 Years	>10 Years	Total
Active Smoker	2	5	10	17
Former Smoker	2	3	4	9
Never Smoked	0	0	0	9
Total	4	8	14	35

Former smokers constitute nine individuals within the sample. Their historical smoking durations exhibit a similar upward trend across the time intervals. Specifically, two individuals smoked for less than

five years prior to cessation, three individuals smoked for five to ten years, and four individuals smoked for more than ten years. Additionally, nine participants are recorded as having never smoked. Because they lack any history of smoking, these individuals register a count of zero across all smoking duration categories. They are included in the overall total to complete the sample size of thirty-five. When aggregating the duration intervals across both active and former smokers, the data indicates that four individuals have a smoking history of less than five years. Eight individuals report a duration of five to ten years. The most frequent duration category overall is greater than ten years, encompassing fourteen individuals. This brings the total number of individuals with a recorded smoking history to twenty-six, with the remaining nine representing the non-smoking cohort.

Table 4 presents an analysis of fried food consumption frequency cross-tabulated with the smoking status of 35 participants. The total sample population is divided into three smoking status categories: 17 active smokers, nine former smokers, and nine individuals who have never smoked. The frequency of fried food consumption is measured across three distinct intervals: one to two times per week, three to four times per week, and more than five times per week. Across the entire cohort, the most frequently reported consumption rate is three to four times per week, which includes 15 participants. The remaining sample is evenly distributed between the lowest and highest consumption frequencies, with 10 individuals consuming fried food one to two times per week and another 10 consuming it more than five times per week.

Table 4. Distribution of fried food consumption frequency by smoking status

Fried Food Frequency	Active Smoker	Former Smoker	Never Smoked	Total
1-2 times/week	4	3	3	10
3-4 times/week	7	4	4	15
>5 times/week	6	2	2	10
Total	17	9	9	35

An examination of the active smoker group reveals that it is the largest demographic within the study. Out of the 17 active smokers, seven individuals consume fried food three to four times per week. Six active smokers report a consumption frequency of more than five times per week, and four consume fried food one to two times per week. Notably, active smokers represent the majority in every consumption frequency bracket, including 60 percent of the individuals in the highest frequency category.

The data demonstrates identical distribution patterns for former smokers and those who have never smoked. Each of these two categories contains exactly nine individuals. Within both the former smoker group and the never smoked group, four individuals report consuming fried food three to four times per week. Furthermore, each group contains three individuals who consume fried food one to two times per week, and two individuals who consume it more than five times per week.

Table 5 presents an analysis of the distribution of combined risk factors among a cohort of patients diagnosed with coronary heart disease. The data outlines both the absolute frequency and the corresponding relative percentage for various established risk factor groupings within the sample. The most frequent presentation in this patient cohort involves a triad of concurrent risk factors: a family history of the condition, active smoking, and an unhealthy dietary pattern. This specific combination was observed in fourteen patients, which represents 40.0 percent of the total studied group.

Table 5. Combinations of risk factors among coronary heart disease patients

Risk Factor Combination	Frequency	Percentage (%)
Active smoker + duration >10 years	10	28.6
Active smoker + unhealthy dietary pattern	12	34.3
Family history + active smoker	9	25.7
Family history + smoking + unhealthy diet	14	40.0
Single factor only	7	20.0

The second most common risk profile consists of two overlapping factors, specifically active smoking and an unhealthy dietary pattern. This pairing was recorded in twelve patients, accounting for 34.3 percent of the sample. This is followed closely by the combination of being an active smoker with a habit duration exceeding ten years. This specific duration-based smoking metric was identified in ten individuals, corresponding to 28.6 percent of the patient group. Furthermore, nine patients exhibited a combination of a family history of coronary heart disease and active smoking, comprising 25.7 percent of the observed cases.

Conversely, the presence of a single, isolated risk factor was the least frequent presentation among the categories detailed in the table. Exactly seven patients were identified as having only one of the assessed risk factors, representing exactly 20.0 percent of the cohort. The data indicates that the vast majority of patients in this sample present with multiple compounding cardiovascular risk factors rather than single isolated variables.

Discussion

The present study demonstrates that the pathogenesis of coronary heart disease within the observed cohort is predominantly driven by a clustering of compounding cardiovascular risk factors rather than isolated variables. The analytical data reveals that the most frequent clinical presentation involves a triad of familial predisposition, active smoking, and unhealthy dietary habits, which together account for forty percent of the patient sample. This specific combination indicates a highly synergistic effect where genetic susceptibility is exacerbated by behavioral and environmental exposures. This finding aligns with contemporary epidemiological models which emphasize that cardiovascular disease progression is a complex, multifactorial process. The low frequency of single isolated risk factors, present in only twenty percent of the respondents, further substantiates the premise that coronary events are typically the culmination of concurrent overlapping risk behaviors.[20–22]

These results corroborate recent literature demonstrating the multiplied impact of overlapping lifestyle factors on vascular health. Overlapping lifestyle factors like diet, smoking, and inactivity amplify cardiovascular risks through synergistic endothelial damage and inflammation. Studies from the past decade consistently show that combined poor habits elevate vascular disease odds by 20-50% beyond isolated factors.[23,24] Research by Permatasari et al.[25] established that co-exposure to high fat diets and smoking significantly increases oxidative stress and accelerates endothelial inflammation compared to individual exposures. Similarly, Ra'bung et al.[26] identified smoking as the most potent determinant of coronary heart disease in an Indonesian demographic, an observation consistent with the high prevalence of active smokers with a habit duration exceeding ten years in the current sample. Furthermore, investigations by Mambo et al.[27] confirmed that the continuous inhalation of toxic combustion byproducts interacts aggressively with dietary lipid accumulation, doubling the incidence rate of myocardial infarction. The pronounced overlap between a family history of coronary heart disease and active smoking in this cohort is also consistent with findings by Mustika et al.[28] who noted that familial behavioral patterns often reinforce individual smoking habits and dietary choices.

Conversely, while the current findings highlight the profound impact of familial predisposition combined with lifestyle factors, other contemporary studies argue that specific dietary or metabolic elements independently outweigh genetic backgrounds. For instance, Zhang, Han, and Guo (2023) asserted that unhealthy dietary clusters characterized by frequent consumption of ultra-processed and fried foods are sufficient to double cardiovascular risk independently of any family history of the disease. Additionally, Gnocchi, Tartarone, and Lerose (2024) suggested that while family history modulates initial pre-test risk calculations, it does not serve as the primary catalyst for coronary artery disease when aggressive metabolic disorders are absent. Zheng, He, and Liu (2023) also emphasized that saturated fat intake acts as an independent variable capable of inducing atherosclerosis regardless of smoking status. These perspectives contrast with the current study, which indicates that isolated dietary factors or genetic histories are the least common presentations, thereby suggesting that in this specific clinical population, single variables are rarely the sole drivers of coronary obstruction.

The pronounced clustering of risk factors identified in this cohort carries substantial theoretical and practical implications for cardiovascular disease management. Theoretically, the data supports the biological interaction model where chemical endothelial damage from tobacco smoke creates a highly susceptible environment for the deposition of oxidized low density lipoproteins derived from frequent fried food consumption. From a practical standpoint, this necessitates a fundamental shift in secondary prevention strategies from isolated symptom management to comprehensive clinical interventions. Healthcare practitioners must implement simultaneous multi-modal treatment plans that prioritize both smoking cessation and rigorous dietary modifications. Dual interventions are empirically more effective than sequential treatments, particularly for patients presenting with a documented family history of cardiovascular events, as addressing the synergistic risk factors concurrently yields superior prognostic outcomes.[29]

Despite providing clinically relevant insights, this research is subject to several inherent methodological limitations. The implementation of a cross-sectional study design fundamentally precludes

the establishment of definitive temporal or causal relationships between the assessed lifestyle behaviors and the onset of coronary heart disease. Furthermore, the relatively restricted sample size of thirty five respondents sourced from a single clinical setting at Royal Prima General Hospital limits the statistical power and generalizability of the conclusions to broader populations. The reliance on self-reported questionnaires to evaluate dietary frequencies and historical smoking metrics also introduces the potential for recall bias and social desirability bias, which may lead to the underestimation of actual consumption rates and habitual durations.

To overcome these limitations and advance the understanding of cardiovascular epidemiology, future research must utilize longitudinal cohort designs encompassing significantly larger and geographically diverse populations to track the temporal progression of these compounded risk factors. Subsequent investigations should integrate objective biological markers, such as serum cotinine levels for smoking verification and advanced lipid profiling for dietary assessment, rather than relying exclusively on subjective patient reporting. Additionally, exploring the specific molecular mechanisms of biological interaction between prolonged high frequency fried food consumption and varied intensities of smoking could facilitate the development of highly targeted and individualized preventive therapeutic strategies.

Conclusion

The current investigation concludes that the manifestation of coronary heart disease within the sampled cohort at Royal Prima General Hospital is predominantly driven by the simultaneous clustering of multiple risk factors. An overwhelming majority of the respondents exhibited a compounded clinical profile, most notably the concurrent triad of a family history of cardiovascular disease, active smoking behavior, and unhealthy dietary patterns. Instances of single, isolated risk factors were notably rare, reinforcing the premise that cardiovascular deterioration in this specific demographic is a complex, multifactorial process rather than the result of singular variables. Ultimately, the data underscores the critical interaction between genetic predisposition and modifiable lifestyle behaviors. This synergistic effect dictates that clinical management and preventive interventions must transition away from isolated risk factor treatment toward comprehensive, multi-modal strategies. Healthcare protocols need to concurrently target smoking cessation and rigorous dietary restructuring to effectively mitigate the compounded risks and improve long-term prognostic outcomes for patients with coronary heart disease.

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