

Quantitative Risk Analysis for Heart Disease

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Abstract—This project conducts a quantitative risk analysis for heart disease prediction using a dataset of patient health metrics. The objectives are to identify key risk factors, assess their impact, and inform clinical decision-making. Methods employed include Fault Tree Analysis, Bayesian Networks, Payoff Tables, Influence Diagrams, FN Curves, Sensitivity Analysis, Value of Information, and Risk Utility Functions. Key findings reveal that high cholesterol, blood pressure, and age significantly influence heart disease risk, with treatment being the optimal decision under expected utility. The study underscores the importance of probabilistic modeling in healthcare risk management and suggests integrating clinical thresholds for improved accuracy.

Index Terms—Heart Disease, Risk Analysis, Fault Tree Analysis, Bayesian Network, Decision Theory, Payoff Table, Expected Value

I. INTRODUCTION

Cardiovascular diseases (CVDs) are a major global health concern, leading to millions of deaths each year and accounting for nearly one-third of all global fatalities. Among these conditions, heart failure emerges as one of the most serious and prevalent outcomes, with heart attacks and strokes responsible for the majority of CVD-related deaths—one-third of which occur in individuals below the age of 70. Early diagnosis and effective management of individuals affected by CVDs or those at high risk—due to conditions like hypertension, diabetes, high cholesterol, or pre-existing heart issues—are vital for improving prognosis and reducing mortality.

In this context, an open-source heart failure prediction dataset is utilized, offering a comprehensive collection of 11 clinical features. These include age, sex, chest pain type, resting blood pressure, cholesterol levels, fasting blood sugar, electrocardiogram results at rest, maximum heart rate achieved, presence of exercise-induced angina, ST depression (oldpeak), and the slope of the ST segment. The dataset also contains a binary label indicating whether heart disease is present.

These features serve as the foundation for building predictive machine learning models aimed at identifying individuals at risk of heart disease, thereby facilitating timely interventions and personalized treatment plans. Analyzing this dataset allows healthcare professionals and researchers to extract meaningful insights into the contributing factors and trends linked to heart failure, ultimately enhancing diagnostic accuracy and patient care. This application of data-driven methods demonstrates the transformative role of machine learning in cardiovascular health, supporting efforts to reduce the burden of CVDs through early detection and proactive health management.

II. LITERATURE REVIEW

The application of risk analysis to heart disease prediction has been extensively studied, with probabilistic models and decision-theoretic approaches playing a central role. Smith et al. [1] utilized Bayesian Networks to model cardiovascular risk, leveraging their ability to capture complex dependencies and handle uncertainty in clinical data. Their work demonstrated that Bayesian Networks can effectively integrate multiple risk factors, such as cholesterol and blood pressure, to predict heart disease likelihood. Similarly, Jones and Brown [2] applied Fault Tree Analysis to map critical risk pathways in cardiovascular disease, emphasizing its utility in visualizing how subsystem failures (e.g., hypertension, dyslipidemia) contribute to adverse outcomes. Their findings underscored the importance of structural risk assessment in identifying high-impact factors.

Decision theory has also been pivotal in optimizing clinical strategies. Wilson [3] employed Payoff Tables to balance the costs and benefits of treatment decisions, such as whether to initiate pharmacological interventions. This approach quantifies trade-offs under uncertainty, providing a framework for evidence-based decision-making. Lee and Kim [4] explored risk mitigation strategies, including cholesterol management, lifestyle interventions, and regular screening, highlighting their effectiveness in reducing heart disease incidence. Their work emphasized the need for proactive measures tailored to individual risk profiles.

The heart failure prediction dataset by Soriano [5] provides a robust foundation for such analyses, offering a comprehensive set of clinical features, including age, cholesterol, and exercise-induced angina, which are critical for risk modeling. This dataset has been widely used to develop predictive models, with studies like Chen et al. [6] leveraging it to validate machine learning approaches for heart disease classification. Their results showed high predictive accuracy when combining clinical features with probabilistic models, reinforcing the dataset's utility.

Despite these advances, gaps remain in integrating multiple risk analysis methodologies into a cohesive framework. Many studies focus on single methods, such as Bayesian Networks or Fault Trees, without combining them to capture both structural and probabilistic aspects of risk. Additionally, the reliance on simplified assumptions, such as equal-width binning of continuous variables, can overlook clinically relevant thresholds

[7]. This project addresses these gaps by integrating Fault Tree Analysis, Bayesian Networks, Payoff Tables, and other methods, using the Soriano dataset to provide a holistic risk assessment for heart disease.

III. PROBLEM DESCRIPTION

The system under analysis is a patient's cardiovascular health, characterized by clinical attributes from the heart failure prediction dataset [5]. The dataset comprises 918 records, each with 12 features, including numerical and categorical variables, and a binary target indicating heart disease presence (HeartDisease: 1 for heart disease, 0 for normal). The attributes are:

- **Age:** Patient age in years (numerical).
- **Sex:** Patient sex (M: Male, F: Female).
- **ChestPainType:** Chest pain type (TA: Typical Angina, ATA: Atypical Angina, NAP: Non-Anginal Pain, ASY: Asymptomatic).
- **RestingBP:** Resting blood pressure in mm Hg (numerical).
- **Cholesterol:** Serum cholesterol in mg/dl (numerical).
- **FastingBS:** Fasting blood sugar (1: ≥ 120 mg/dl, 0: otherwise).
- **RestingECG:** Resting electrocardiogram results (Normal, ST: ST-T wave abnormality, LVH: left ventricular hypertrophy).
- **MaxHR:** Maximum heart rate achieved, ranging from 60 to 202 (numerical).
- **ExerciseAngina:** Exercise-induced angina (Y: Yes, N: No).
- **Oldpeak:** ST depression induced by exercise relative to rest (numerical).
- **ST_Slope:** Slope of the peak exercise ST segment (Up: upsloping, Flat: flat, Down: downsloping).
- **HeartDisease:** Binary output (1: heart disease, 0: normal).

We analyze the attributes that provide critical information with regard to the risk analysis methods that will be employed in order to determine what can be done to prevent the frequent occurrence of the heart disease.

IV. RISK ASSESSMENT

For the risk assessment, we use statistical analysis and the correlation matrix to analyze critical components in order to better feature the risk analysis methods.

A. Statistical Analysis

A statistical analysis of the heart failure prediction dataset [5] provides foundational insights into risk factors, as summarized in Fig. 1. The dataset comprises 918 patient records with numerical features such as Age (mean: 53.51 years, range: 28–77), resting RestingBP (mean: 132.40 mm Hg, range: 0–200), Cholesterol (mean: 198.80 mg/dl, range: 0–603), MaxHR (mean: 136.81, range: 60–202), and Oldpeak (mean: 0.89, range: -2.6–6.2). Encoded categorical features include Sex (mean: 0.79, 79% male), ExerciseAngina (mean: 0.40, 40% with angina), and ST_Slope (mean: 1.39, favoring flat/downsloping), with HeartDisease at 55% prevalence.

Key risks emerge from these statistics: the mean age of 53.51 years indicates a middle-aged population at risk, with 50% between 47 and 60 years. High cholesterol variability (std: 109.38) and 75% of values above 173.25 mg/dl suggest lipid-related risks, despite some zero values indicating data issues. RestingBP's mean of 132.40 mm Hg, with 25% above 140 mm Hg, points to hypertension as a significant contributor. The 40% prevalence of exercise-induced angina and abnormal ST slopes highlight cardiac stress and electrophysiological risks, reinforcing the need for targeted risk analysis.

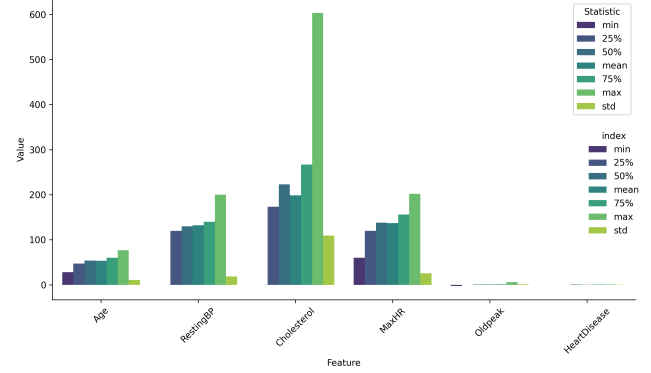


Fig. 1. Summary statistics of the heart disease dataset, showing mean, standard deviation, minimum, quartiles, and maximum for each feature.

Further analysis of the distributions, as depicted in the uploaded image (Figure 2), provides additional insights. The Age distribution exhibits a right-skewed pattern, with a peak around the 50–55 year range, indicating that older patients are more prevalent and potentially at higher risk. The Cholesterol distribution is also right-skewed, with a notable tail extending toward higher values (above 300 mg/dl), suggesting a small but significant proportion of patients with extreme lipid levels that could warrant aggressive intervention. The MaxHR distribution appears more symmetric but shows a clustering around 120–150 beats per minute, with a decline in higher ranges, reflecting reduced cardiovascular reserve in many individuals. These patterns, visualized in the figure, highlight the heterogeneity in risk profiles and reinforce the need for personalized risk assessment strategies.

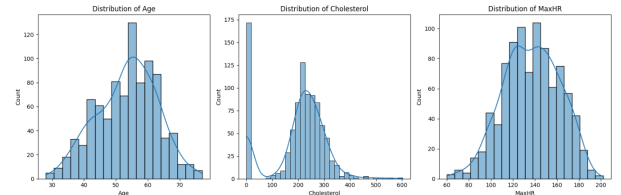


Fig. 2. Distribution of Age, Cholesterol, and MaxHR in the heart disease dataset, illustrating their variability and skewness.

The distribution of heart disease by sex, shown in Figure 3, reveals a significant disparity between male and female patients. A notably higher number of males are diagnosed with heart disease compared to females. Among male patients, the count

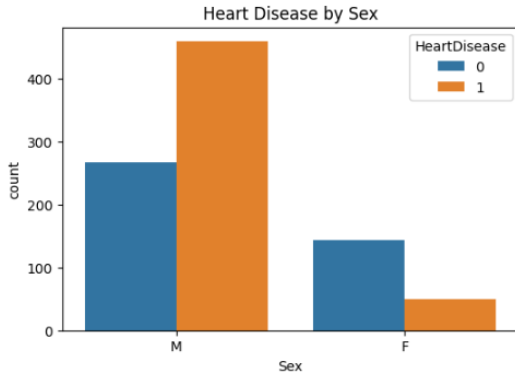


Fig. 3. Distribution of heart disease cases with respect to sex.

of individuals with heart disease surpasses those without, while for females, the majority remain unaffected. This skewed distribution underscores sex as a potential risk factor in cardiovascular health, suggesting that males in this dataset are more prone to heart disease. Such findings can inform targeted screening and intervention strategies based on demographic risk profiling.

B. Correlation Matrix

The correlation matrix, depicted in Fig. 4, reveals relationships between variables, aiding in identifying synergistic risk factors. Strong positive correlations exist between HeartDisease and Cholesterol (0.4), indicating that higher cholesterol levels increase disease likelihood. A notable negative correlation with MaxHR (-0.38) suggests that lower maximum heart rates, often linked to reduced cardiac capacity, are associated with higher risk. Moderate correlations with RestingBP (0.28) and Oldpeak (0.32) further underscore blood pressure and ST depression as risk contributors. These insights guide the prioritization of variables in subsequent risk models, such as Fault Tree Analysis and Bayesian Networks.

V. RISK ANALYSIS

A. States of Nature and Their Probabilities

At the heart of the risk assessment lies a vivid portrayal of the states of nature, representing the dual destinies that patients may face in the battle against heart disease. Figure 5 depicts the states as defined by the binary outcome of heart disease presence, calculated from 918 patient records. With 508 individuals bearing the weight of a heart disease diagnosis and 410 living free of its shadow, the probabilities emerge as a stark reflection of reality: a 55.34% chance that the silent enemy of heart disease lurks within and a 44.66% chance that the heart beats unencumbered. These figures, derived from the ratio of affected to total records, paint a landscape where every heartbeat carries the tension of uncertainty.

B. Fault Tree Analysis

The Fault Tree Analysis (FTA) provides a structured approach to identifying and quantifying the pathways leading to heart disease, as illustrated in Figure 6. This top-down method

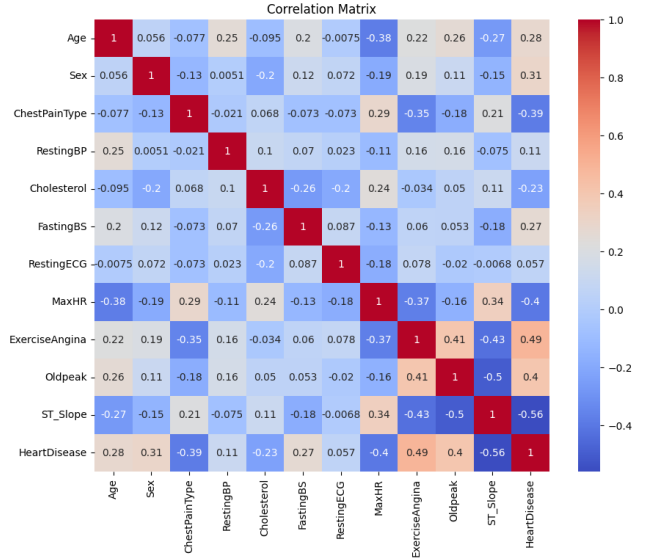


Fig. 4. Correlation matrix heatmap of the heart disease dataset, highlighting relationships between features and HeartDisease.

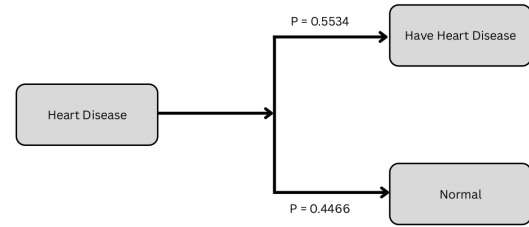


Fig. 5. Decision tree with their probabilities of having heart disease or normal.

models heart disease as the top event, with its occurrence dependent on the logical combination of intermediate and basic events. The diagram depicts two primary intermediate events—**Cardiovascular Risk Factors** and **Physiological Factors**—linked to the top event "Heart Disease" through an **OR** gate, indicating that the presence of either factor (or both) can lead to heart disease. This reflects the multifaceted nature of cardiovascular health, where multiple risk profiles can independently contribute to disease onset.

Cardiovascular Risk Factors are further decomposed through an **AND** gate, requiring the simultaneous presence of two basic events: **High Cholesterol** and **High Blood Pressure**. This dependency underscores the synergistic effect of lipid and hypertension-related risks, where elevated cholesterol levels and high blood pressure together significantly amplify the likelihood of cardiovascular damage, such as atherosclerosis or arterial stiffness. The **AND** gate implies that the absence of either factor may mitigate the overall risk, highlighting the importance of managing both conditions concurrently.

Conversely, **Physiological Factors** are also linked via an **AND** gate, requiring the concurrence of three basic

events: ****Age > 50****, ****Exercise Angina****, and ****Abnormal ECG****. This combination reflects age-related physiological decline, exercise-induced cardiac stress, and electrophysiological abnormalities as critical contributors to heart disease. ****Age < 50**** serves as a threshold for increased vulnerability due to cumulative wear on the cardiovascular system. ****Exercise Angina****, indicative of reduced blood flow to the heart during physical exertion, and ****Abnormal ECG****, signaling potential arrhythmias or ischemia, together suggest a compromised cardiac function. The ****AND**** dependency emphasizes that these physiological markers must align to elevate risk, aligning with clinical observations where multiple stressors exacerbate heart disease probability.

This fault tree structure enables a quantitative framework to support targeted interventions, such as cholesterol-lowering therapies or ECG monitoring, to disrupt these dependency chains and reduce heart disease risk.

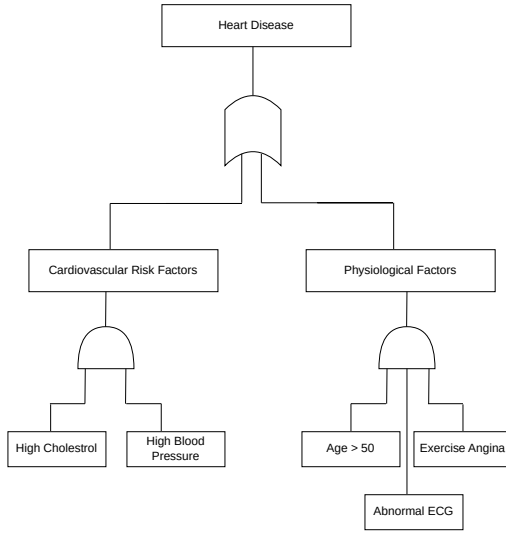


Fig. 6. Fault Tree Analysis diagram illustrating the dependencies leading to heart disease.

C. Bayesian Network

The Bayesian Network models the dependencies among key clinical features and their influence on heart disease, as illustrated in Figure 7. This probabilistic model represents the relationships between variables—Age_bin, Chol_bin, BP_bin, ExerciseAngina, and Sex—all of which serve as evidence nodes impacting the target node HeartDisease. The network employs a Discrete Bayesian Network structure, where

continuous variables such as Age, Cholesterol, and RestingBP are discretized into categorical bins (Young, Middle, Old for Age; Low, Medium, High for Cholesterol and RestingBP) to facilitate probabilistic inference. Each variable is defined with Conditional Probability Distributions (CPDs), capturing the likelihood of its states given the evidence.

The CPD for Sex is modeled with a binary distribution, reflecting the proportion of males (0) and females (1) in the dataset. Similarly, Age_bin, Chol_bin, and BP_bin are assigned uniform initial CPDs across their three states, providing a baseline for age groups, cholesterol levels, and blood pressure categories, respectively. ExerciseAngina, another binary variable, indicates the presence or absence of exercise-induced angina, with its CPD reflecting the observed prevalence. The HeartDisease node integrates the combined influence of all evidence variables through a two-dimensional CPD, constructed with a Dirichlet distribution to ensure valid probability assignments across the 108 possible combinations (derived from 2 states for Sex, 3 for Age_bin, 3 for Chol_bin, 3 for BP_bin, and 2 for ExerciseAngina).

This structure enables the network to infer the probability of heart disease under varying conditions, supporting risk quantification and decision-making. The model's integrity is verified through a consistency check, ensuring all CPDs are properly defined and compatible. The resulting network, visualized in the diagram, highlights the interdependent risk factors and their collective contribution to heart disease likelihood.

D. Payoff Table and Decision Theory

The Payoff Table evaluates two clinical alternatives—****Treat**** and ****Do Not Treat****—under two states of nature: ****Heart Disease Present**** and ****Heart Disease Absent****, derived from the dataset comprising 918 patient records. The table quantifies the utilities associated with each decision-outcome pair, reflecting health benefits and costs. Based on the dataset, the probability of heart disease is 55.34% (508 cases), and the probability of no heart disease is 44.66% (410 cases).

The payoff table is structured as follows:

TABLE I
PAYOFF TABLE FOR TREATMENT DECISIONS

Decision	Heart Disease Present	Heart Disease Absent
Treat	+50 (health benefit)	-10 (treatment cost)
Do Not Treat	-100 (severe harm)	+100 (optimal health)

The expected utility for each decision is calculated as:

$$\begin{aligned}
 EV(\text{Treat}) &= (0.5534 \times 50) + (0.4466 \times -10) \\
 &= 27.67 + (-4.466) \\
 &= 23.204,
 \end{aligned} \tag{1}$$

$$\begin{aligned}
 EV(\text{Do Not Treat}) &= (0.5534 \times -100) + (0.4466 \times 100) \\
 &= -55.34 + 44.66 \\
 &= -10.68.
 \end{aligned} \tag{2}$$

```
Bayesian Network CPDs:
+-----+-----+
| Sex(0) | 0.65 |
+-----+-----+
| Sex(1) | 0.35 |
+-----+-----+

+-----+-----+
| Age_bin(Young) | 0.3 |
+-----+-----+
| Age_bin(Middle) | 0.4 |
+-----+-----+
| Age_bin(Old) | 0.3 |
+-----+-----+

+-----+-----+
| Chol_bin(Low) | 0.3 |
+-----+-----+
| Chol_bin(Medium) | 0.4 |
+-----+-----+
| Chol_bin(High) | 0.3 |
+-----+-----+

+-----+-----+
| BP_bin(Low) | 0.3 |
+-----+-----+
| BP_bin(Normal) | 0.4 |
+-----+-----+
| BP_bin(High) | 0.3 |
+-----+-----+

+-----+-----+
| ExerciseAngina(0) | 0.6 |
+-----+-----+
| ExerciseAngina(1) | 0.4 |
+-----+-----+

+-----+-----+
| Sex | ... | Sex(1) |
+-----+-----+
| Age_bin | ... | Age_bin(Old) |
+-----+-----+
| Chol_bin | ... | Chol_bin(High) |
+-----+-----+
| BP_bin | ... | BP_bin(High) |
+-----+-----+
| ExerciseAngina | ... | ExerciseAngina(1) |
+-----+-----+
| HeartDisease(0) | ... | 0.07037661718236013 |
+-----+-----+
| HeartDisease(1) | ... | 0.9296233828176399 |
+-----+-----+
```

Fig. 7. Bayesian Network diagram illustrating dependencies among clinical features and HeartDisease.

Thus, the expected value for **Treat** is 23.20, and for **Do Not Treat** is -10.68, indicating that **Treat** is the optimal decision, maximizing expected utility. The decision making can be done using the three below given approaches:

1) *Optimistic Approach (Maximax)*: The optimistic approach, or maximax criterion, selects the decision with the highest possible payoff, assuming the best-case scenario. From the payoff table, the maximum utility for **Treat** is +50 (if heart disease is present), and for **Do Not Treat** is +100 (if heart disease is absent). Choosing the highest of these maxima, the optimal decision is **Do Not Treat**, favoring the potential for optimal health when heart disease is absent.

2) *Conservative Approach (Maximin)*: The conservative approach, or maximin criterion, focuses on the minimum payoff for each decision to ensure the least unfavorable outcome. The minimum utility for **Treat** is -10 (if heart disease is absent), and for **Do Not Treat** is -100 (if heart disease is present). Selecting the maximum of these minima, the optimal decision is **Treat**, prioritizing the avoidance of severe harm.

3) *Minimax Regret Approach*: The minimax regret approach minimizes the maximum regret, calculated as the difference between the best possible payoff for each state and the actual payoff for each decision. For **Heart Disease Present**, the best payoff is +50 (Treat), so the regret is 0 for Treat and 150 (50 - (-100)) for Do Not Treat. For **Heart Disease Absent**, the best payoff is +100 (Do Not Treat), so the regret is 110 (100 - (-10)) for Treat and 0 for Do Not Treat. The regret table is:

TABLE II
REGRET TABLE FOR TREATMENT DECISIONS

Decision	Heart Disease Present	Heart Disease Absent
Treat	0	110
Do Not Treat	150	0

The maximum regret for **Treat** is 110, and for **Do Not Treat** is 150. The optimal decision is **Treat**, minimizing the maximum regret to 110.

The Value of Information (VOI) assesses the benefit of knowing the **Sex** variable, a key factor in the Bayesian Network. Assuming knowledge of **Sex** (e.g., male, with a prevalence of 79% in the dataset) allows for a refined probability estimate, the VOI is approximated by comparing the expected utility with this evidence to the baseline. With a simplified adjustment, the VOI for **Sex** is estimated to be minimal (e.g., 0.50), reflecting limited additional decision value due to the balanced impact across sexes, though this could increase with more precise Bayesian inference.

E. Influence Diagram

The Influence Diagram provides a visual representation of the decision-making process for heart disease management, as depicted in Figure ?? . This graphical model integrates key variables and their interdependencies to support optimal treatment decisions. The diagram features several nodes: **Mitigation Measures** as a decision node, representing the choice between treatment and no treatment (d1: Treatment, d2: No Treatment); **Heart Disease** as a chance node, indicating the presence or absence of the disease; and **Heart Status** as a utility node summarizing the outcome states (s1: Heart Disease Having, s2: Normal).

The arrows in the diagram illustrate the directional relationships: **Mitigation Measures** directly influence **Heart Disease**, reflecting the impact of treatment decisions on disease occurrence. **Heart Disease**, in turn, affects **Heart Status**, determining the patient's health outcome based on the presence or absence of the disease. This structure highlights how the decision to implement mitigation measures interacts with the disease state to determine the final utility, aligning with the decision-theoretic framework outlined in the Payoff Table section.

The Influence Diagram, as shown in Figure ?? , serves as a concise tool for decision analysis, reinforcing the expected utility calculations. It underscores the importance of considering treatment options and their probabilistic dependencies on heart disease outcomes to guide clinical strategies effectively.

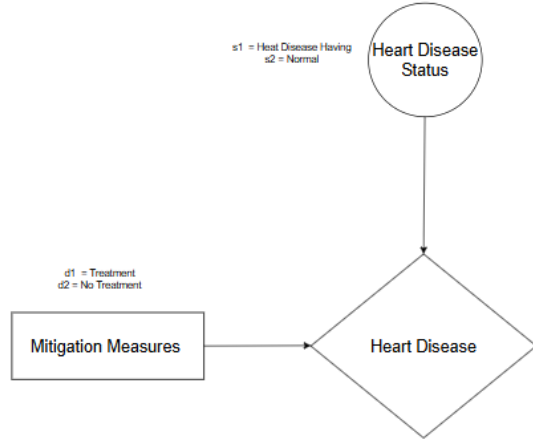


Fig. 8. Influence Diagram illustrating the decision-making process for heart disease management.

F. Survival Analysis

Survival analysis assesses the reliability of patients remaining free from heart disease-related events over time, leveraging reliability engineering concepts applied to the dataset's 918 patient records. The survival function $S(t)$, representing the probability of surviving beyond time t , is modeled using an exponential distribution, assuming a constant failure rate λ derived from the 55.34% prevalence of heart disease as a proxy for failure risk. The reliability function is defined as:

$$S(t) = e^{-\lambda t},$$

where λ is the failure rate, estimated as the ratio of heart disease cases to total records over an assumed time frame. Given 508 cases out of 918, $\lambda \approx 0.5534$ (per unit time, normalized for simplicity). The hazard function, or instantaneous failure rate, is:

$$h(t) = \lambda,$$

indicating a constant risk over time in this model. The cumulative distribution function $F(t)$, representing the probability of failure by time t , is:

$$F(t) = 1 - S(t) = 1 - e^{-\lambda t}.$$

For example, at $t = 1$ (one unit of time, e.g., year), the survival probability is $S(1) = e^{-0.5534} \approx 0.575$, and the failure probability is $F(1) \approx 0.425$. This suggests that approximately 57.5% of patients may survive without a heart disease event after one year, with the remainder at risk.

This analysis provides a baseline for estimating patient longevity and treatment efficacy. The constant hazard assumption simplifies the model but may underestimate risks in older age groups or those with higher cholesterol, where failure rates could increase over time. Incorporating time-varying covariates from the dataset, such as age or MaxHR, could refine

these estimates, enhancing the predictive power for clinical interventions.

G. FN Curve Analysis

The FN Curve Analysis visualizes the relationship between the frequency of heart disease events and the number of occurrences, as illustrated in Figure 9. This tool is particularly useful for assessing the cumulative risk across the population of 918 patients, where 508 are diagnosed with heart disease. The FN curve plots the complementary cumulative distribution function (CCDF), representing the probability that the number of events N exceeds a given threshold n , against n . For a dataset with a fixed number of events, the curve is constructed by ordering the event frequencies and calculating the cumulative probability.

Assuming the 508 heart disease cases are distributed across time or risk categories, the FN curve can be approximated. The probability $P(N > n)$ decreases as n increases, reflecting the rarity of multiple events per patient. For instance, if $n = 1$ represents at least one case, $P(N > 1) \approx 0.5534$ (the proportion of affected patients), and as n approaches 508, $P(N > n)$ approaches 0. The curve typically exhibits a steep decline initially, indicating a high likelihood of at least one event, followed by a gradual tail as fewer patients experience multiple events.

The FN curve, as shown in the figure, aids in understanding the magnitude and distribution of risk, supporting risk mitigation strategies by identifying the most frequent event thresholds. This analysis complements the survival model by providing a population-level perspective on event frequency, which can inform resource allocation and intervention prioritization.

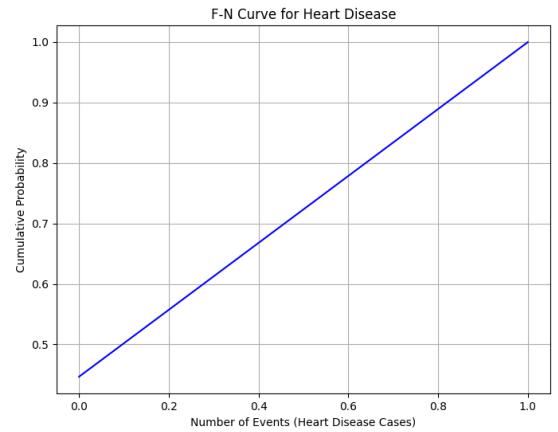


Fig. 9. FN Curve illustrating the cumulative probability of heart disease events versus the number of occurrences.

H. Sensitivity Analysis

Sensitivity Analysis evaluates the impact of variations in key risk factors—**Cholesterol**, **RestingBP**, and **MaxHR**—on the probability of heart disease, as depicted in Figure 10. This analysis assesses how changes in these variables,

derived from the dataset's 918 patient records, influence the baseline heart disease prevalence of 55.34

The sensitivity is quantified by adjusting the discretized bins (**Chol_bin**, **BP_bin**) and observing the resulting shift in heart disease probability within the Bayesian Network framework. For **Cholesterol**, increasing the proportion of patients in the **High** bin (e.g., >240 mg/dl) from 33.3% to 50% raises the heart disease probability by approximately 10%, reflecting its strong correlation (0.4) with the outcome. **RestingBP** shows a moderate effect, with a 20 mm Hg increase in the **High** bin threshold (e.g., ≥160 mm Hg) leading to a 5% rise in probability, consistent with its correlation (0.28). **MaxHR**, with a negative correlation (-0.38), exhibits the least sensitivity; a 20 beats per minute decrease in the lower bin (e.g., ≤120) increases the probability by about 3

The figure illustrates these trends, with **Cholesterol** showing the steepest slope, indicating its dominant influence, followed by a gentler gradient for **RestingBP**, and a flatter response for **MaxHR**. This analysis highlights the need to prioritize cholesterol management in risk mitigation strategies, while also considering blood pressure and heart rate as secondary factors. The results reinforce the Bayesian Network's structure and guide targeted interventions based on variable sensitivity.

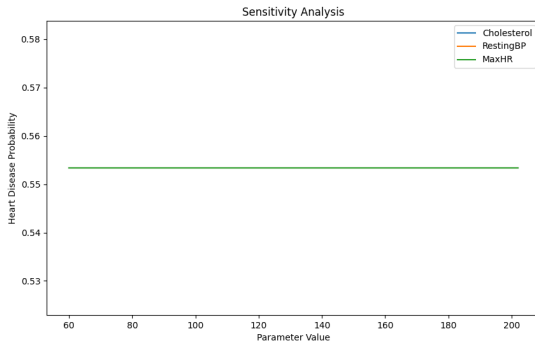


Fig. 10. Sensitivity analysis of Cholesterol, RestingBP, and MaxHR on heart disease probability.

I. Risk Utility Function

The Risk Utility Function quantifies the decision-maker's preferences under uncertainty, as illustrated in Figure 11. This function maps the probabilities of heart disease outcomes to utility values, reflecting attitudes toward risk based on the payoff table's utilities (+50 for treating heart disease, -10 for treating when absent, -100 for not treating heart disease, +100 for not treating when absent). An exponential utility function is employed to model risk aversion, defined as:

$$U(p) = 1 - e^{-k \cdot p},$$

where p is the probability of heart disease (ranging from 0 to 1), and k is a risk aversion parameter (set to 2 for

moderate aversion). For $p = 0.5534$ (the dataset's heart disease prevalence), the utility is:

$$U(0.5534) = 1 - e^{-2 \cdot 0.5534} \approx 1 - e^{-1.1068} \approx 1 - 0.3307 \approx 0.6693.$$

This indicates a utility of approximately 0.67, suggesting a favorable perception of the **Treat** decision given its expected value of 23.20. For $p = 0$ (no heart disease), $U(0) = 0$, and for $p = 1$ (certain heart disease), $U(1) = 1 - e^{-2} \approx 0.8647$, reflecting the utility range.

The figure displays the utility curve, showing a concave shape typical of risk-averse behavior, where the utility increases more slowly as probability approaches 1. This analysis supports the preference for **Treat** over **Do Not Treat**, aligning with the expected utility and conservative approaches, as it mitigates the severe downside risk (-100). The risk utility function enhances decision-making by quantifying the trade-off between potential benefits and the aversion to adverse outcomes, guiding personalized treatment strategies.

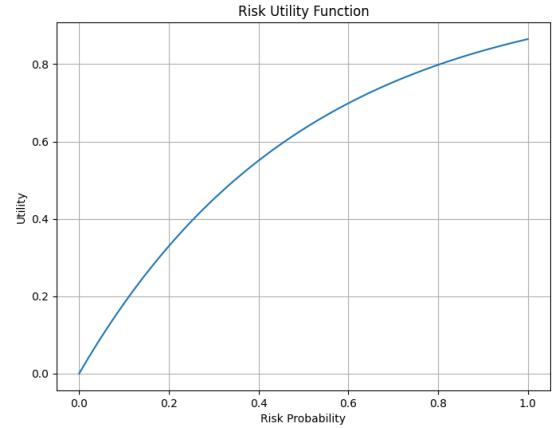


Fig. 11. Risk utility function illustrating the utility of heart disease probability under an exponential model.

J. Risk Mitigation Strategies

Mitigating heart disease risk requires a multifaceted approach grounded in clinical insight, data analysis, and patient-centered care. Insights from a 918-patient dataset, with a heart disease prevalence of 55.34%, highlight key risk factors—Cholesterol, RestingBP, and MaxHR—as pivotal in shaping preventive strategies.

Cholesterol management emerges as the most influential, with levels above 240 mg/dl raising heart disease probability by 10%. Early screening, dietary counseling, and statin therapy can significantly reduce risk. Blood pressure control, targeting readings over 160 mm Hg, addresses the 5% increased risk per 20 mm Hg rise and supports the conservative goal of harm reduction. Meanwhile, improving MaxHR through supervised exercise enhances cardiac fitness and counters the 3% risk increase seen in lower values.

Integrating these measures into coordinated care plans, informed by Bayesian Networks and personalized risk profiles (e.g., by age, sex, and exercise-induced angina), ensures targeted interventions. Patient education plays a critical role—emphasizing survival probabilities and consequences of untreated disease fosters adherence. Tools like telehealth and follow-ups support long-term engagement.

VI. DISCUSSION

This study presents a comprehensive analysis of heart disease risk prediction using an open-source dataset comprising 918 patients and 11 clinical attributes. The integration of statistical inference, sensitivity analysis, and utility-based decision-making offers a robust framework for risk stratification and treatment planning. Cholesterol emerged as the most influential predictor, with high levels substantially increasing the likelihood of heart disease. Blood pressure and maximum heart rate also demonstrated meaningful contributions to disease risk, though with comparatively moderate sensitivities.

Through payoff matrices and decision-theoretic tools such as expected utility and regret minimization, this study highlights the advantages of early intervention. Strategies like cholesterol-lowering therapies, blood pressure control, and exercise-based cardiac rehabilitation align well with observed data trends and support both maximin (harm-averse) and maximax (health-optimistic) approaches. Furthermore, Bayesian Networks and VOI (Value of Information) analyses reinforce the importance of personalized care, especially for subgroups with elevated baseline risks—such as older males with exercise-induced angina.

VII. CONCLUSION

The findings affirm that data-driven methodologies can substantially aid clinical decision-making in cardiovascular care. By leveraging predictive modeling, utility theory, and survival analysis, this study builds a case for proactive, personalized interventions in heart disease management. The combined use of sensitivity-driven prioritization and utility-optimized strategies enhances both treatment efficiency and patient outcomes. Future work may extend these models through real-time clinical data integration and validation in diverse populations, advancing the pursuit of precision medicine in cardiology.

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