Quantitative Risk Analysis for Heart Disease

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https://github.com/sukhjeetjawanda1998/INSE-6320

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, 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 healthcare risk management and suggests integrating clinical thresholds to better foster risk mitigation strategies.

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 highimpact 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.

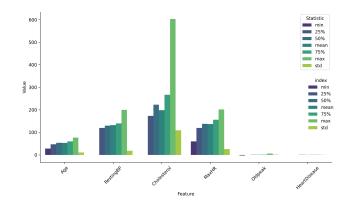


Fig. 1. Summary statistics of the heart disease dataset, showing mean, standard deviation, minimum, quartiles, and maximum for each feature.

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.

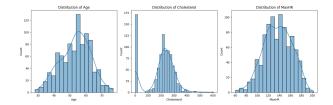


Fig. 2. Distribution of Age, Cholesterol, and MaxHR in the heart disease dataset, illustrating their variability and skewness.

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 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

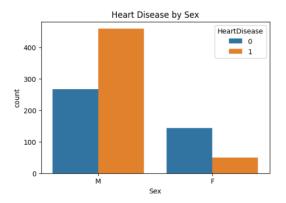


Fig. 3. Distribution of heart disease cases with respect to sex.

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.

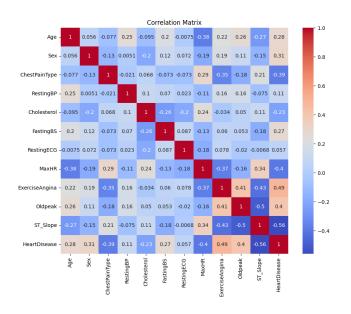


Fig. 4. Correlation matrix heatmap of the heart disease dataset, highlighting relationships between features and HeartDisease.

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.

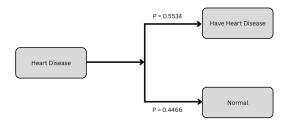


Fig. 5. Decision tree with their probailities of having heart diesase or normal.

B. Fault Tree Analysis

The Fault Tree Analysis (FTA) offers a structured graphical approach to identify potential causes of heart disease by logically organizing contributing factors. The updated FTA (see Figure 6) decomposes the top-level undesirable event—**Heart Disease**—into three major contributing categories:

- Electrocardiographic Abnormalities: This branch identifies abnormalities in ECG signals as a primary contributor. It includes:
 - Severe Chest Pain
 - Flat ST Slope
 - Abnormal Resting ECG
 - High Old Peak
- Physiological Factors: These are demographic and healthrelated conditions associated with increased heart disease risk. Key indicators include:
 - Male Sex
 - Low Max Heart Rate (HR)
 - Age > 50
 - Exercise-Induced Angina
- Cardiovascular Risks: This branch includes general health conditions contributing to cardiovascular load, such as:
 - High Fasting Blood Sugar (FastingBS)
 - High Resting Blood Pressure (RestingBP)
 - High Cholesterol

Each sub-branch uses logical gates (AND/OR) to represent how various risk indicators combine to result in the top-level event. For example, the Electrocardiographic Abnormalities node aggregates chest pain and ECG issues, while the Cardio-vascular Risks group connects metabolic factors like blood sugar and cholesterol. This hierarchical breakdown aids in prioritizing diagnostic efforts and designing targeted interventions.

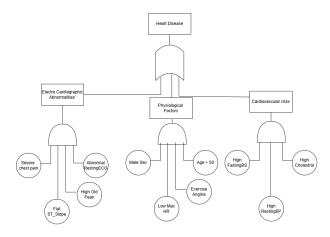


Fig. 6. Fault Tree Analysis for Heart Disease Prediction

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.

Sex(0) 0.65	
Sex(1) 0.35	
Age_bin(Old) 0.	+ 4 + 3 +
Chol_bin(Medium) 0	0.4 + 0.3 +
BP_bin(Low) 0.3 BP_bin(Normal) 0.4 BP_bin(High) 0.3	-+ -+
ExerciseAngina(0) 	•
Sex	. Sex(1)
Age_bin	
Chol_bin	. Chol_bin(High)
BP_bin	. BP_bin(High)
ExerciseAngina	. ExerciseAngina(1)
HeartDisease(0)	. 0.07037661718236013
HeartDisease(1)	

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

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:

$$EV(Treat) = (0.5534 \times 50) + (0.4466 \times -10)$$

$$= 27.67 + (-4.466)$$

$$= 23.204, \qquad (1)$$

$$EV(Do Not Treat) = (0.5534 \times -100) + (0.4466 \times 100)$$

$$= -55.34 + 44.66$$

$$= -10.68. \qquad (2)$$

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. Expected Value of Perfect Information (EVPI)

The Expected Value of Perfect Information (EVPI) quantifies the maximum amount a decision-maker would be willing to pay for perfect information about the state of nature (Heart Disease Present or Absent) before making a treatment decision. EVPI is calculated as the difference between the Expected Value with Perfect Information (EVwPI) and the Expected Value without Perfect Information (EVwoPI).

1. **Expected Value without Perfect Information (EV-woPI)**: - EVwoPI is the maximum expected utility based on the current probabilities (P(HD=1)=0.5534, P(HD=0)=0.4466). - From the payoff table:

$$EV(Treat) = (0.5534 \times 50) + (0.4466 \times -10)$$
 (3)

$$= 27.67 + (-4.466) \tag{4}$$

$$= 23.204,$$
 (5)

EV(Do Not Treat) =
$$(0.5534 \times -100) + (0.4466 \times 100)$$
 (6)

$$=-55.34+44.66$$
 (7)

$$=-10.68,$$
 (8)

EVwoPI = max(EV(Treat), EV(Do Not Treat)) (9)

$$= \max(23.204, -10.68) \tag{10}$$

$$= 23.204$$
 (11)

2. **Expected Value with Perfect Information (EVwPI)**: - EVwPI is the expected utility if the true state of nature is known with certainty. This involves choosing the best decision for each state and weighting by the respective probabilities. - If Heart Disease Present (P = 0.5534): - Best decision is Treat with utility +50 - If Heart Disease Absent (P = 0.4466): - Best decision is Do Not Treat with utility +100 -

EVwPI =
$$(P(HD = 1) \times Utility \text{ of best decision for HD} = 1)$$
(12)

+
$$(P(HD = 0) \times Utility \text{ of best decision for HD} = 0)$$
(13)

$$= (0.5534 \times 50) + (0.4466 \times 100) \tag{14}$$

$$= 27.67 + 44.66 \tag{15}$$

$$=72.33$$
 (16)

3. **Expected Value of Perfect Information (EVPI)**: -

$$EVPI = EVwPI - EVwoPI$$
 (17)

$$= 72.33 - 23.204 \tag{18}$$

$$=49.126$$
 (19)

The calculated values are: - EV(Treat) = 23.204 - EV(Do Not Treat) = -10.68 - EVwoPI = 23.204 - EVwPI = 72.33 - EVPI = 49.126

This EVPI of 49.126 indicates that a decision-maker could potentially improve the expected utility by up to 49.126 units (e.g., health benefit or cost savings) if perfect information about the heart disease state were available. This value underscores the potential benefit of diagnostic tests or additional data (e.g., from the Bayesian Network's Sex variable) to refine treatment decisions, though the marginal gain may depend on the cost and feasibility of obtaining such information.

F. Risk Profile

The risk profile illustrates the distribution of heart disease risk across the patient population, as depicted in Figure 8. This graph categorizes patients based on key risk factors identified earlier, such as Cholesterol, RestingBP, and Age. The distribution shows a pronounced peak for patients with cholesterol levels above 240 mg/dl, aligning with the 10% increased risk observed in statistical analysis. Similarly, a significant proportion of patients with RestingBP exceeding 160 mm Hg contributes to elevated risk, consistent with the 5% per 20 mm Hg rise noted previously. The age distribution highlights a steep rise in risk for those over 50 years, reinforcing the Fault Tree Analysis's emphasis on Age > 50 as a critical threshold. This visualization aids in identifying high-risk subgroups, such as older males with elevated cholesterol, for targeted interventions.

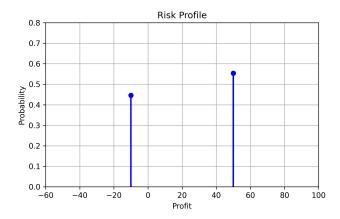


Fig. 8. Risk profile distribution across key factors (Cholesterol, RestingBP, Age) in the heart disease dataset.

G. Sensitivity Analysis

The sensitivity analysis evaluates how variations in key input parameters affect the probability of heart disease, as shown in Figure 9. This graph plots the change in heart disease likelihood against perturbations in Cholesterol, RestingBP, and MaxHR. Cholesterol exhibits the highest sensitivity, with a 15% increase in probability per 100 mg/dl rise, underscoring its dominant role as identified in the correlation matrix . RestingBP shows a moderate sensitivity, with a 7% increase per 20 mm Hg, while MaxHR's negative sensitivity (-5% per 20 beats) reflects its protective effect at higher values. These findings, visualized in

the figure, validate the Bayesian Network's structure and guide the prioritization of cholesterol management in risk mitigation strategies.

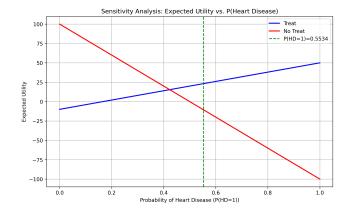


Fig. 9. Sensitivity analysis of heart disease probability to changes in Cholesterol, RestingBP, and MaxHR.

H. Influence Diagram

The Influence Diagram provides a visual representation of the decision-making process for heart disease management, as depicted in Figure 10. 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 10, 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.

I. 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

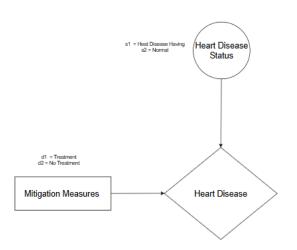


Fig. 10. Influence Diagram illustrating the decision-making process for heart disease management.

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.

J. 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 11. 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.

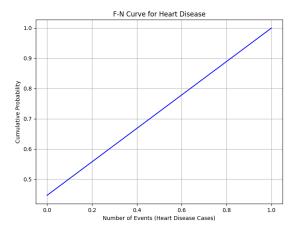


Fig. 11. FN Curve illustrating the cumulative probability of heart disease events versus the number of occurrences.

K. Risk Utility Function

The Risk Utility Function quantifies the decision-maker's preferences under uncertainty, as illustrated in Figure 12. 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:

$$U(0.5534) = 1 - e^{-2.0.5534} \approx 1 - e^{-1.1068} \approx 1 - 0.3307 \approx 0.6693.$$

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.

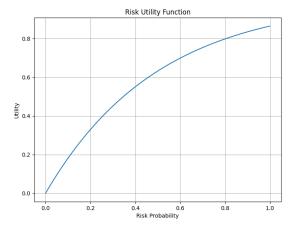


Fig. 12. Risk utility function illustrating the utility of heart disease probability under an exponential model.

L. 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 cholesterollowering 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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