Mortality Prediction & Interpretation

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Introduction

Motivation

In the past half-century, society has made tremendous technical advancements in healthcare. Specifically in the realm of surgical techniques, optimized procedures ranging from open-heart surgeries and minimally invasive surgeries have emerged to deal with morbidities in quicker, safer, and more cost-effective ways (Gawande, 2012; Cooley Denton A. & Frazier O. H., 2000; M et al., 2017). However over this same period, almost ironically, public health officials have reported a steady decline of the rate of average life expectancy increase in the United States since the 1980s, which diverges from trends seen in other developed countries (Muennig et al., 2018). This paradoxical juxtaposition of technical advancements in healthcare but poorer overall health outcomes suggests a critical short-coming of our current healthcare system. While efforts to advance operative and post-operative techniques abound, far less rigor has been applied to optimizing pre-operative and preventative healthcare paradigms, which are comparatively more impactful from a public health perspective. Central to preventative healthcare models is an understanding of parameters -- of both the biological and socially-deterministic varieties -- that influence longitudinal healthcare outcomes.

Project Aims

To aid in the effective recommendation of preventative interventions, this project explores how a subset of parameters and data from the first National Health and Nutrition Examination Survey Epidemiologic Follow-up Study (NHEFS) relates to predicted mortality. These factors include both clinical and demographic data such as age, poverty index (PI), race, sex, pulse pressure, and red blood cell concentration (RBC), among other biological health measurements. From this data, we will build a model that predicts mortality to investigate which factors are most impactful to a patient's health and how these factors relate to the patient's risk of mortality.

Literature Review

Previous research may help inform the predictors we choose to examine. Prior public health research has found that age, income, race, and cardiovascular metrics can be possible predictors for mortality. Knowing that risk factors for morbidities increase as one grows older, age's link to mortality has been examined. In one study, researchers found that age, compared to 21 other variables, was the single best predictor for five-year mortality on ~80% of datasets from the U.S., Taiwan, and Costa Rica (Glei et al., 2016). Cardiovascular metrics have also been linked to risk of disease and mortality. A study conducted in Finland found that reduction of blood pressure and LDL cholesterol (more colloquially typed as "bad cholesterol") corresponded to ~90% decrease in cardiovascular-related mortality among middle-aged men and women (Jousilahti et al., 2016).

Though age and cardiovascular function are directly and self-explanatorily related to mortality, income and race impact health via more indirect, structural-level avenues. For example, lower-income individuals from African-American and Hispanic communities are far more likely to be housed near environmental waste-sites, in food deserts, and away from well-funded hospital services, which may manifest in poorer health outcomes (Fish et al., 2015). Additionally, minority stress is a well-documented phenomenon in which individuals from marginalized populations suffer from chronically high levels of stress, which impacts health and well-being more holistically (Wong et al., 2014).

Project Question

We aimed to answer what determinants are most important in predicting mortality and how these factors relate to health risk. Based on previous research, we expect to confirm that age, PI, race, serum cholesterol, and blood pressure are important risk factors. We further question how these factors might interrelate to contribute to mortality risk.

Social Impact

Understanding biological parameters that impact mortality certainly is of high clinical importance. From a preventative healthcare standpoint, knowing, for example, what deviations from normative blood profiles are most detrimental to health not only allows for earlier medical or lifestyle interventions (even before disease manifests), but also may inspire research into pathogenesis and treatments related to the investigated predictor. We also incorporate non-clinical factors (such as race, poverty, and sex) in an understanding that such factors impact healthcare outcomes not only by interacting with clinical aspects, but also by impacting more structural considerations, such as healthcare accessibility. It is our sincere hope that our offered analysis of both clinical and non-clinical factors, their interactions with each other, and the collective impact they bear on health and mortality will add urgency to health policy initiatives that help privilege traditionally under-served populations and promote preventative care measures. The long-term goal is to help elucidate specific health policies that can reduce risk factors in the population, and help reveal inequalities and biases in American society and government that lead to the ramifications we see.

Methodology

Overview of Data

In this project, we used the NHANES I Epidemiologic Follow-up Study (NHEFS), designed to investigate the relationships between clinical, nutritional, and behavioral factors assessed in the first National Health and Nutrition Examination Survey (NHANES I). The NHEFS cohort includes all persons 25-74 years of age who completed a medical examination at NHANES I in 1971-75 (n = 14,407). Participants were surveyed for a variety of demographic and medical data, including socio-economic data, various blood serum molecule concentrations, blood pressure readings, and other biological data. Our dataset is a filtered and moderately cleaned version of the NHEFS data; our base data contains 9932 entries across a subset of 18 predictors. A write-up describing these predictors can be found below **(Table A)**.

Table A: Overview of predictors and response variable

Variable	Type	Desc.	Max	Min
Age	QUANT	Age of participant at examination (years)	74	25
Poverty Index (PI)	QUANT	Family monthly poverty level index, a ratio of monthly family income to the HHS poverty guidelines specific to family size. Generally, lower PI corresponds to lower income.	998	2
Race	CAT	{1: White, 2: Black, 3: Other}	3	1
Sex	CAT	{1: Male, 2: Female}	2	1
Systolic BP	QUANT	Systolic blood pressure; how much pressure your blood is exerting against your artery walls when the heart beats. (mmHg)	270	80

Diastolic BP	QUANT	Diastolic blood pressure; how much pressure your blood is exerting against your artery walls while the heart is resting between beats. (mmHg)	180	34
Pulse Pressure	QUANT	calculated as: Systolic BP - Diastolic BP; Pulse pressure represents the force that the heart generates each time it contracts. (mmHg)	170	10
Serum Protein	QUANT	Concentration; amount of total protein in the bloodstream. (g/100mL)	11.5	4.4
Serum Albumin	QUANT	Concentration; Albumin is produced by the liver and is the most abundant blood protein in mammals. Albumin is essential for maintaining the oncotic pressure needed for proper distribution of body fluids between blood vessels and body tissues. (g/100mL)	5.7	2.7
Serum Cholesterol	QUANT	Concentration; Cholesterol composes about 30% of all animal cell membranes. It is required to build and maintain membranes and modulates membrane fluidity over the range of physiological temperatures. (mg/100mL)	537	78
Serum Magnesium	QUANT	Concentration; Magnesium is a cofactor in more than 300 enzyme systems that regulate diverse biochemical reactions in the body, including protein synthesis, muscle and nerve function, blood glucose control, and blood pressure regulation. (mEq/L)	2.89	0.82
Serum Iron	QUANT	Concentration; Iron is used to make hemoglobin, a protein in red blood cells that carries oxygen from the lungs to all parts of the body, and myoglobin, a protein that provides oxygen to muscles. (ug/100mL)	396	17
TIBC	QUANT	Total Iron Binding Capacity; measures the blood's ability to attach itself to iron and transport it around the body. (ug/100mL)	717	168
TS	QUANT	calculated as: Serum Iron/TIBC * 100%; Transferrin saturation represents the percentage of serum iron that is bound.	100	3.2
Red Blood Cells (RBC)	QUANT	Concentration; Red blood cells bind oxygen in the lungs and move it throughout the body. (millions/uL) NOTE: normal ranges of RBC range from ~4-6 million/uL, however our data is a magnitude of 10 greater.	69.9	25.5
White Blood Cells (WBC)	QUANT	Concentration; White blood cells are the cells involved in the body's immune response. (thousands/mm3)	51.2	2.1
Sedimentation Rate	QUANT	Rate at which red blood cells in anticoagulated whole blood descend in a standardized tube over a period of one hour; non-specific measure of inflammation.	72	1
BMI	QUANT	Body Mass Index; a person's weight in kilograms divided by the square of height in meters; A high BMI can be an indicator of high body fatness and can be used to screen for weight categories that may lead to health problems. (kg/m**2)	72.22	12.59
Response Variable	**Type**	**Desc.**	**Max**	**Min**
Y	QUANT	The time of death minus the time of their last exam. If the individual's year of death is unknown, their value is the negative value of the year they were last known alive minus the year of the last exam.	21.47	-22.06

Exploratory Data Analysis

Before building our predictive model, we first needed to clean and process our dataset. There were a significant number of missing values for WBC (10.4%) and Sedimentation Rate (8.2%), and a small number of missing values for Systolic BP (0.6%), Diastolic BP (0.6%), and Pulse Pressure (0.6%). To determine how to handle these missing values, we examined their distribution by plotting the marginal distributions of each predictor against the response variable and flagging all observations with NaNs in any column. Some examples of the plots are shown below **(Figure 1)**.

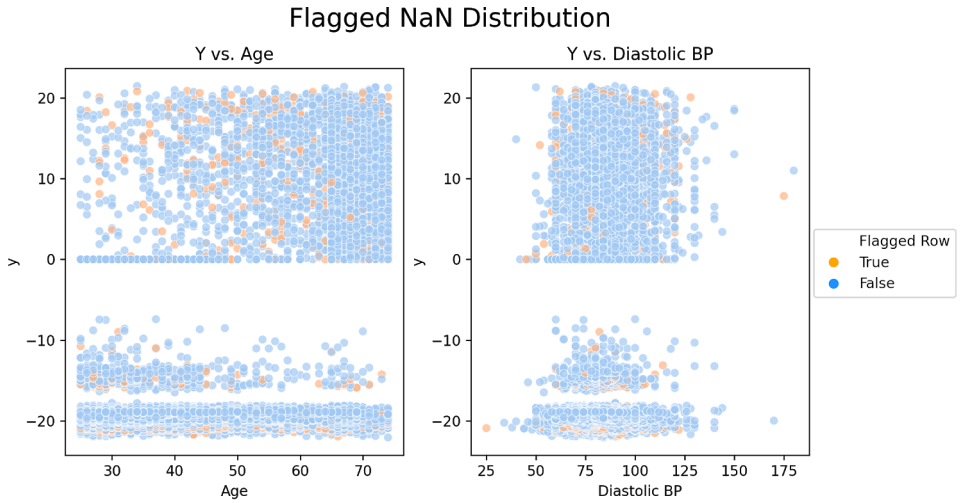


Figure 1: Example of random distribution of NaNs in NHANES data

In these plots, we see that the missing values appear randomly distributed throughout the data points, indicating that they are missing at random and suggesting that our choice of imputation method would likely not significantly alter our results. However, for the two specific predictors RBC and PI, we noticed vertical lines of data points, which were not necessarily missing data, that seemed strange at first **(Figure 2)**.

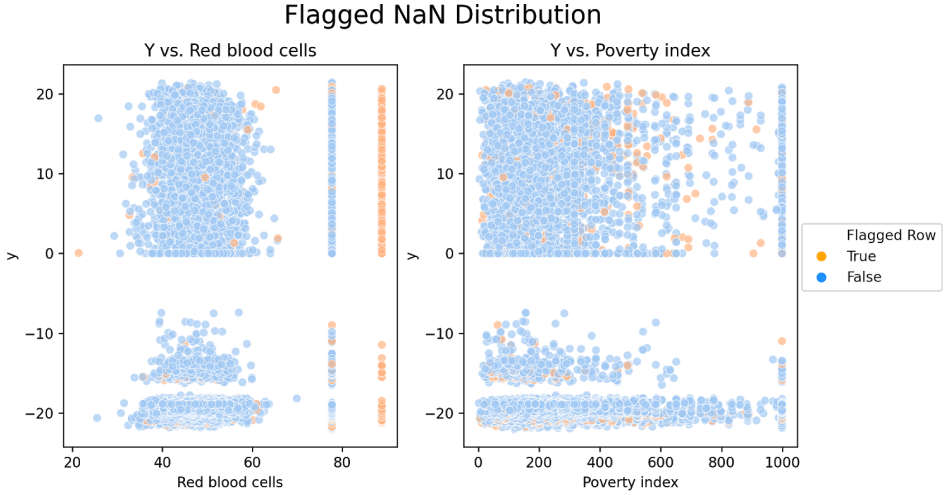


Figure 2: Labels designate NaNs for RBC and PI data

After further exploration into our dataset, we discovered that these vertical lines were labels in the NHANES survey, rather than numerical data. For RBC, 77.7 represented "Unacceptable Data", due to measuring errors, and 88.8 represented "Blank", when RBC measurements were not taken at all. For PI, 999 represented "No data", and 998 represented PI values greater or equal to 998 (*NHANES I (1971-1974)*, n.d.). Because these lines of points actually represented NaNs for those predictors, we see that all the missing values are generally missing at random. Then, to handle the missing data, including both the NaNs and the three representative values previously described, we performed mean imputation. To further prepare our dataset for modelling, we investigated whether predictors were significantly related to each other through a correlation matrix. The predictors did not exhibit very high collinearity **(Figure 3)**.

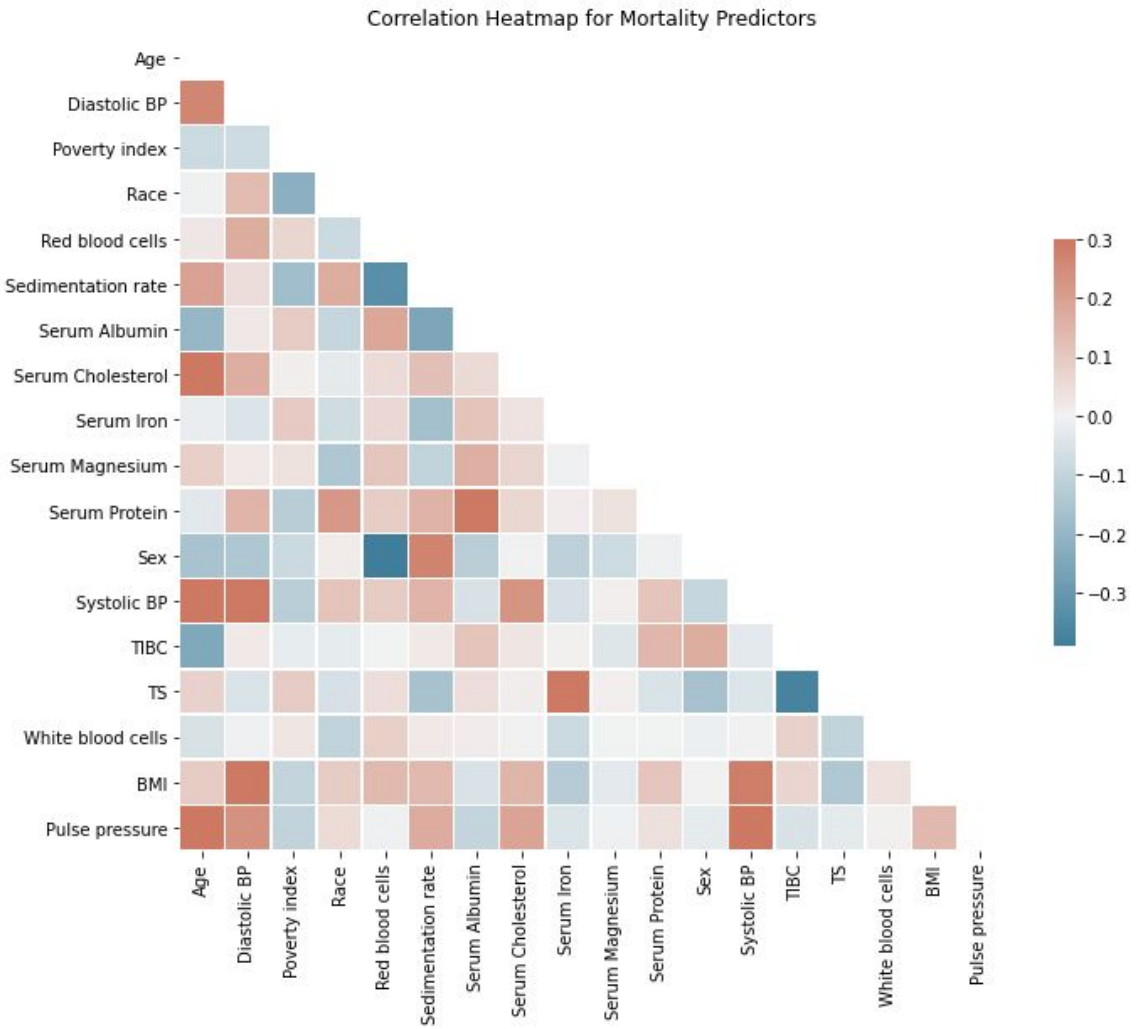


Figure 3: Correlation heatmap for predictors shows no significant correlations

Furthermore, although the correlations were moderate, we dropped the variables TS and Pulse Pressure from our dataset. We dropped them because these variables were derived from the variables "Serum Iron", "TIBC", "Systolic BP", and "Diastolic BP", and it is helpful clinically to have fewer features. TS = Serum Iron / TIBC. Pulse Pressure = Systolic BP - Diastolic BP. The clear relationships between these raw and derived variables were confirmed by plotting the variables against each other **(Figure 4)**.

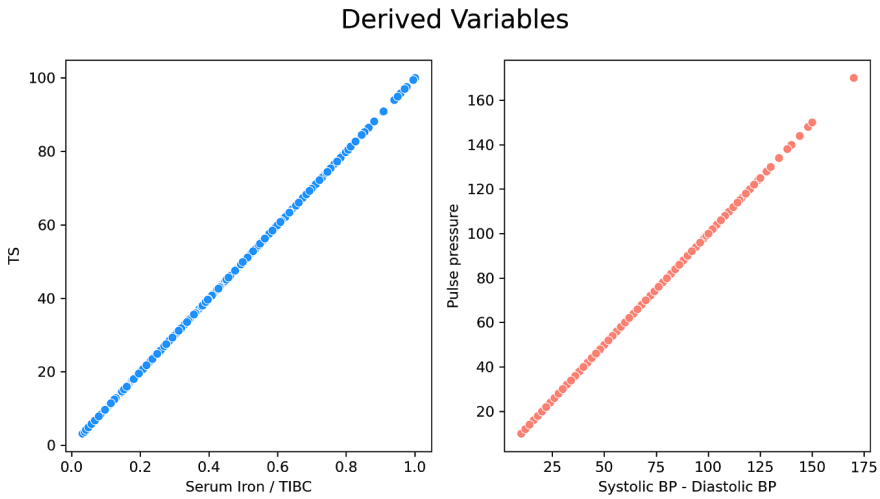
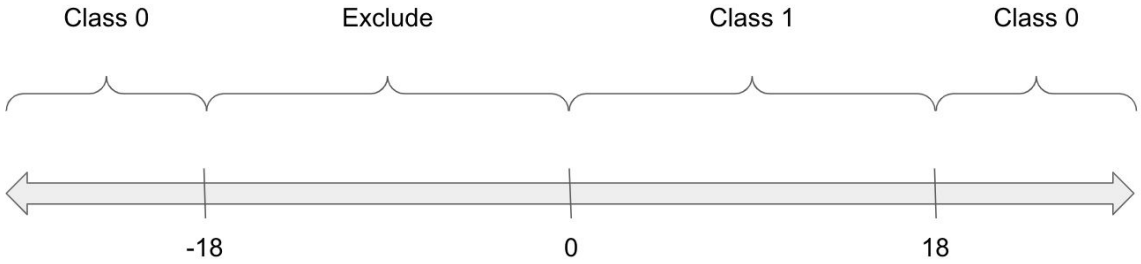


Figure 4: Verifying relationships of derived variables

Baseline Model

For our baseline model, we employed a logistic classification model for mortality. Though we pondered whether regression might be appropriate for predicting the number of years an individual would live past their last exam, regression would necessitate dropping all of the observations with negative values for y. That is, only positive values for y tell us for certain the number of years an individual lived before passing away. A total of 6077 data points of the available 9932 data points would be dropped under this regime. We instead employed a classification model to preserve as much of our data as possible.



In designing our classification task, we first modified the response variable to classify mortality within a threshold number of years. Specifically, we dropped values of y within the interval $-threshold \leq y < 0$, determining that such observations yielded too much uncertainty as to whether the individual had died or lived. For values of y that were positive and less than or equal to the defined threshold ($0 \leq y \leq threshold$), the individual is known to have died within the threshold number of years after their last exam. We labeled this scenario "class 1." For any value of y greater than the threshold or less than the negative threshold ($y > threshold$ or $y < -threshold$), the individual is known to have lived *at least* that number of years after the last offered exam. We labeled this scenario "class 0."

We performed logistic regression several times using different mortality thresholds to classify the standardized data, evaluating the performance of our model each time with Area Under the Receiver Operating Characteristic Curve (AUC). AUC is a performance metric that quantifies true-false positive trade-off and thereby indicates the degree of separability between the target classes. Not only is AUC the most common metric employed in public health research into mortality predictors (Hajian-Tilaki,

2013), it is also largely preferred to classification accuracy for binary classification tasks. That is, models assessed with classification accuracy may represent the data well but not necessarily properly discriminate between the two classes. For example, a model that only predicts class 0 on data composed of 99% class 0 and 1% class 1 will certainly have a high classification accuracy though it has not learned anything more fundamental about how to differentiate the two classes. AUC helps balance data representation and categorical discrimination, and is thereby a better performance metric for our classification task.

We found that a threshold of 18 years yielded the highest AUC score on test performance and stratified the data into 6048 class 0 and 3470 class 1 observations. That is, a threshold of 18 years optimized model performance while only dropping 414 data points of the available 9932 observations. Importantly, however, thresholding the data as such resulted in a large class-size imbalance. That is, our training set was composed of 4858 class 0 and 2755 class 1 observations, nearly a two-fold class-size imbalance. To accommodate, we re-weighted class 1 such that it held twice as much weight as that of class 0.

After such modifications, the AUC score for our logistic regression model using all 16 predictors was 83.8%. We wanted to assess whether performance on this classification task might increase by changing the model to different ensemble-based methods (Decision Tree, Random Forest, Adaboost). While logistic regression and the listed ensemble-based methods all perform classification, ensemble-based methods generate decision boundaries that are far more extensive and flexible than the single boundary generated through logistic regression. That is, ensemble-based methods might better represent the highly complex, multi-dimensional data of our project. The AUC scores of these different ensemble-based methods (using the 18-year thresholded and re-weighted data as before) were as follows: Simple Decision Tree, 68.2%; AdaBoost, 84.0%; Random Forest, 84.3%.

Final Model

As our Random Forest classifier yielded the highest model performance as assessed by AUC, we decided to optimize the Random Forest classifier further for our final model. First, we investigated the hyper-parameter max_depth, which is the maximum number of layers into which the decision tree will branch before stopping. Max_depth is a critical hyper-parameter as too low of a depth will fail to encompass the complexity of the data. The impact of too high of a depth is slightly more involved. For a Random Forest model, we want the max_depth to be set such that the individual decision trees slightly over-fit to the data; aggregating the responses of many over-fit trees helps the collective Random Forest model attenuate the impact of over-fitting while still preserving model complexity. However, if the individual trees are too over-fit (that is, the max_depth is set too high), aggregating the responses will not be enough to prevent over-fitting. In the graph below, we see that validation score is optimized at depth = 4 for a single Decision Tree. For our Random Forest model, we chose depth = 8 as it slightly overfits the decision tree to the data **(Figure 5)**.

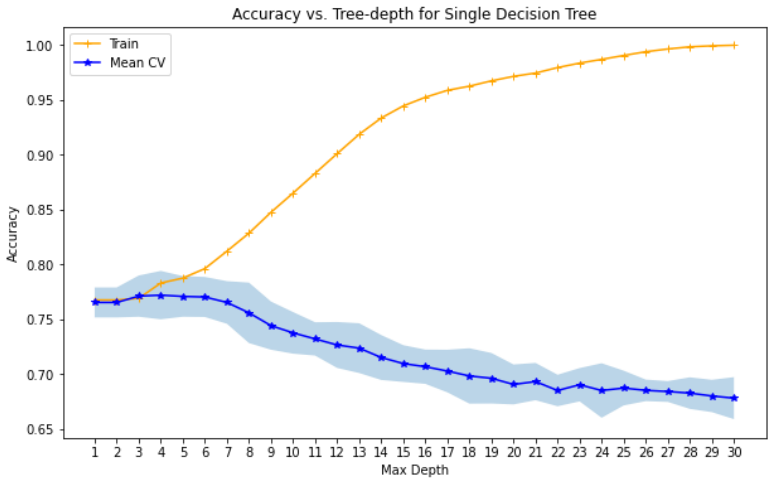


Figure 5: Examining overfitting of single decision Tree to data

We additionally optimized other hyper-parameters (min_samples_split, min_samples_leaf, and max_features) using Randomized Search with Cross Validation. The value of the tuned hyper-parameters were as follows: min_samples_split=50, min_samples_leaf=1, and max_features='sqrt'. As branching in tree-based methods implicitly incorporates interaction effects between predictors, we decided not to explicitly engineer and introduce interaction terms to our data. Additionally, though we considered standardizing our data with StandardScaler(), we opted to keep our data unstandardized. Standardization did not impact model AUC performance, and keeping our data on their original scales would allow for easier feature interpretability. Our final model incorporating all such modifications yielded an AUC score of 84.8%

Discussion

Model Interpretation

Once we established our best predictive model, we examined the relative importance of each feature to the model prediction using SHAP values (SHapley Additive exPlanations). SHAP values can be used for global model interpretability to illustrate how much each predictor contributes, either positively or negatively, to the target variable. A SHAP summary plot reveals that each predictor we included had some impact on the model and are thereby important for mortality prediction **(Figure 6)**.

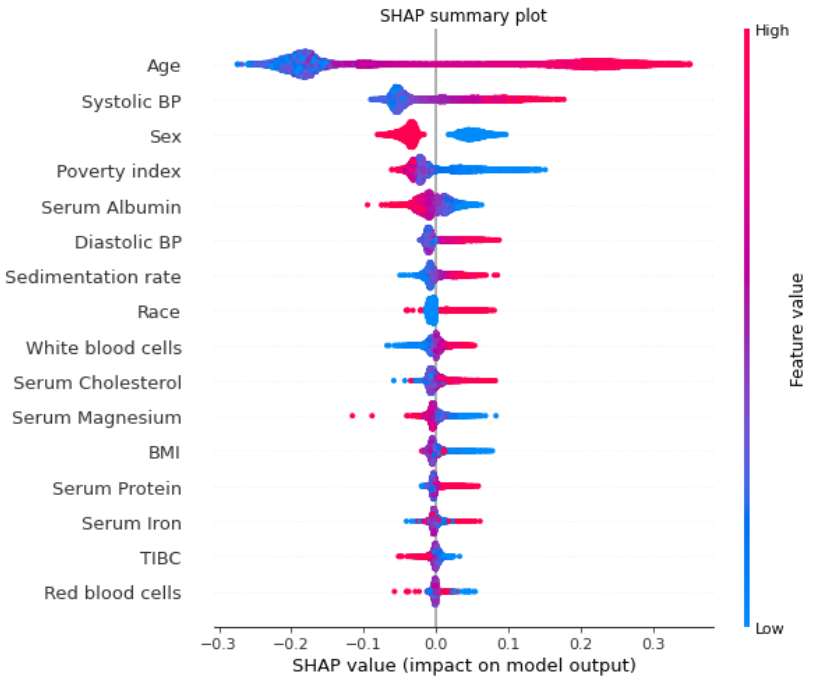


Figure 6: Age is the predictor with the most significant SHAP values

The summary plot illustrates feature values and their respective SHAP values. Positive SHAP values indicate that the corresponding feature values contribute to a higher risk of mortality within 18 years from the last date of examination, and negative SHAP values indicate lower risk.

From the plot, we see that higher age, systolic BP, diastolic BP, sedimentation rate, WBC, serum cholesterol, serum protein, and serum iron are all associated with higher probability of mortality within 18 years. Lower PI, serum albumin, serum magnesium, BMI, TIBC, and RBC are also associated with increased risk of mortality within 18 years. Additionally, being male and being non-white are associated with higher risk of mortality within 18 years.

The apparent relationships between mortality in 18 years and age, systolic and diastolic BP, WBC, serum cholesterol, PI, RBC, TIBC, male, and non-white all make intuitive sense. Furthermore, because albumin and magnesium are essential to maintaining the healthy distribution of body fluids and regulating biochemical reactions respectively, individuals with low serum albumin or magnesium could be more likely to be unhealthy and thus suffer mortality within 18 years. Being underweight (low BMI) may also be associated with mortality-inducing diseases such as dementia or Alzheimer's Disease (Bhaskaran et al., 2018). Moreover, because iron is an essential nutrient for bacterial survival, higher serum iron levels could be associated with bacterial infections and increased mortality risk (Lan et al., 2018). High sedimentation rates may also indicate the presence of cancerous tumors or autoimmune disease, which may lead to mortality (*Erythrocyte Sedimentation Rate Test (ESR Test)*, 2018). High serum protein might be indicative of chronic infection, inflammation, or autoimmune disorder.

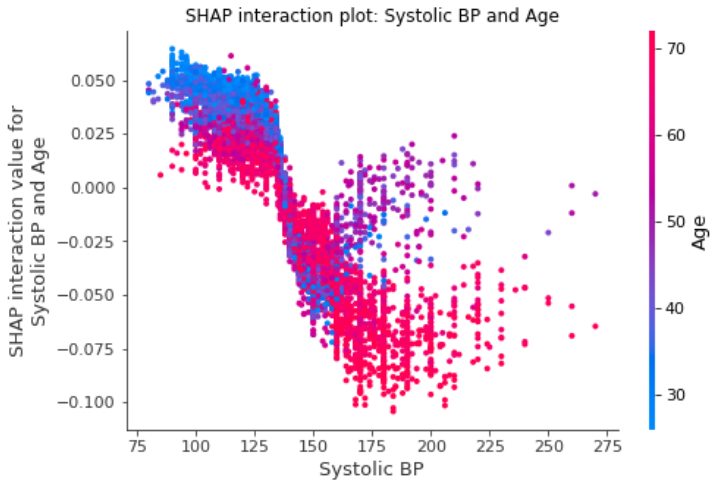
By verifying that the model produces sensible relationships between predictors and mortality within 18 years, we illustrate that our model may be valid for clinical applications. This model could be utilized by healthcare providers or policymakers to predict the impact of various medical measurements and demographic data on health and inform how doctors recommend treatments or preventative care

options to patients. Educational or awareness programs may also be devised to inform the public about these important biological metrics and how to maintain healthy values.

Interaction Plots

Beyond the global relative feature importance, we were interested in exploring hidden interactive effects between predictors that impact mortality risk and might further inform healthcare and policy strategies. To understand the distribution of the effects, we plotted SHAP interaction plots. These plots graph the interactive SHAP values versus the two predictors. The SHAP interaction value is the additional combined feature effect after accounting for the individual feature effects.

Age vs. Systolic BP

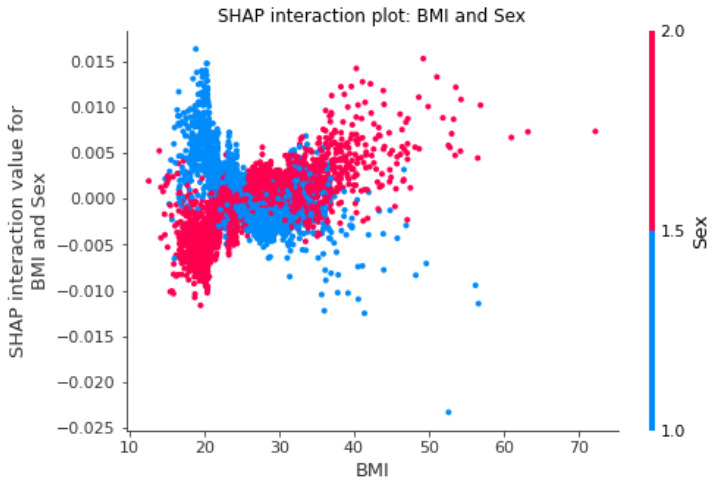


We note a clear effect of the interaction between age and systolic BP. At nearly all respective values of systolic BP, we see that observations with younger ages typically have higher SHAP interaction values compared to older ages, with the difference most prominent at lower and higher extremes of systolic BP. This suggests that younger individuals who deviate from normative systolic BP are more at risk for mortality compared to older individuals with similar cardiovascular metrics. Clinically, what this might suggest is while deviant systolic BP is a common manifestation of old age (due to natural, age-related structural changes in cardiovascular vessels), abnormal systolic BP measurements in younger individuals is more likely an indication of an underlying health condition and thereby increased risk for mortality. From a public health standpoint, these results inform a necessary paradigm shift to add more urgency to cardiovascular abnormalities presented by younger individuals. That is, if a younger individual presents with deviant cardiovascular metrics, more rigorous interventions should be applied earlier to potentially stave off increased risk for mortality.

Sex vs. Age

Interestingly, the interaction between age and sex follows a clear time-course. At ages less than 50, female individuals bear higher SHAP interaction values compared to their male counterparts, suggesting that female individuals of this age range are at higher risk for mortality. Such sex-stratified difference diminishes as age 50 is approached for both cohorts. From ages 50 - 70, we note that the trend has flipped, with male individuals now presenting with higher risks for mortality. The difference is most prominent at age 60, with male and female individuals bearing, respectively, the highest and lowest risks for mortality compared to any other age group. For ages above 70, female individuals once again bear the higher risk for mortality. While it is difficult to assess why such a time-course is exhibited, the results confirm the importance of considering both age and sex in healthcare considerations. That is, for example, a 60-year-old male who presents with low blood pressure, which we know from our previous Age/Systolic BP analysis increases risk for mortality, may require more interventions compared to a female counterpart with the same diagnosis.

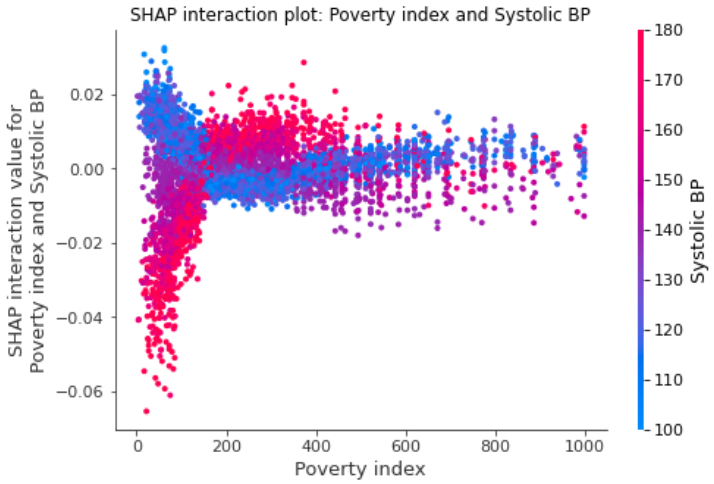
Sex vs. BMI



Sex impacts the relationship between BMI and risk for mortality. Specifically, we see that female individuals with low BMI are at lower risk for mortality, whereas male individuals at the same BMI are, in contrast, at higher risk for mortality. The relationship flips at higher BMIs, where female individuals bear

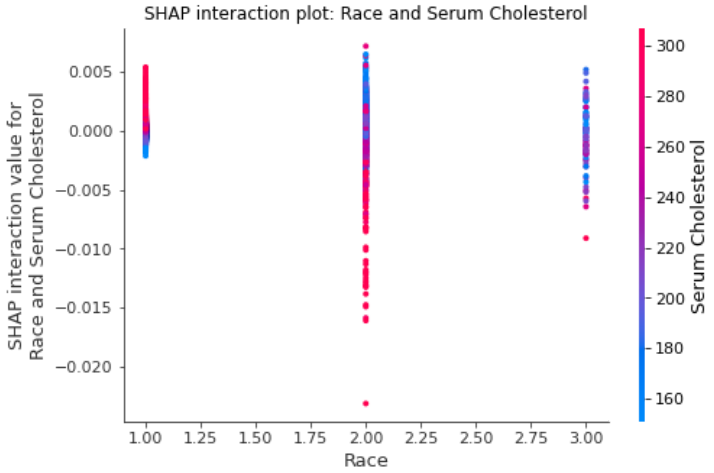
higher mortality risk relative to their male counterparts. From a public health standpoint, what this may suggest is a need to emphasize differential interventions in lifestyle based on one's sex. That is, public health officials may emphasize to men methods to increase BMI (within medically-informed, reasonable bounds), whereas BMI-reduction interventions might be better aimed at female populations.

Systolic BP vs. Poverty Index



We observe a "bottleneck" effect between the interaction between PI and systolic BP. That is, as poverty indices grow above 200, the relationship of different systolic BPs to mortality risk becomes increasingly ablated. What this speaks to is a compensatory effect of money and socioeconomic status; that is, having money (for a variety of reasons including increased access to well-funded healthcare structures) helps to lessen the longitudinal impact of abnormal cardiovascular conditions. Interestingly, at poverty indices below 200, high systolic BP decreases one's risk for mortality, whereas low systolic BP increases it. Though more research is needed to corroborate this relationship, these results preliminarily suggest a clinical need to increase cardiovascular diagnostic efforts for underserved, low-income populations. Efforts to help low-income individuals presenting with low systolic BP obtain access to prescription medication and other interventions should be emphasized.

Serum Cholesterol vs. Race



Surprisingly, we observe differential impacts of serum cholesterol levels based on race. For individuals who are White, high serum cholesterol corresponds to an increased risk of mortality within 18 years. In contrast, lower serum cholesterol levels correspond to an increased risk of mortality for Black and Other individuals. More research might be warranted to corroborate this differential impact of cholesterol based on race, but the model suggests that healthcare providers may tailor their recommendations to patients according to this factor. That is, a doctor might recommend further testing and lifestyle accommodations for Whites with high cholesterol and for non-Whites with low cholesterol.

Feature Selection

To further quantify the importance of the top four predictors in our model, we selected the top four features (age, systolic BP, sex, and PI) as informed by the SHAP summary plot and trained a new Random Forest Classifier model to see if it would achieve a similar performance as our original Random Forest Classifier. Building as parsimonious a model as possible is clinically important because a stream-lined feature subset employed in clinical spaces allows physicians to make quicker decisions while still preserving diagnostic accuracy. Age, systolic BP, sex, and PI are also data that can be acquired without invasive, time-consuming, or expensive methodologies. The resulting AUC of this parsimonious model scored similarly, albeit slightly lower with an AUC of 84.5%.

Conclusion

Age, systolic BP, sex, and PI proved to be the most influential predictors impacting risk for mortality. Notably, three of these four predictors are demographic considerations, which emphasizes a clinical need to synthesize biological measurements with a holistic understanding of patient narratives and relevant non-clinical, social determinants.

Strengths and Limitations

By building a decision tree model, not only were we able to build a model to predict mortality within 18 years with a reasonably high accuracy, we were also able to incorporate interpretability into our RandomForest model by using the SHAP library, allowing us to pinpoint how each feature impacts the probability of death. This model could help healthcare professionals develop more targeted health interventions.

However, our project still had limitations, particularly in the data we used to train, evaluate, and add interpretability in our model. To enhance interpretability, we had to transform our response variable into a classification of mortality using thresholding, which limited our interpretation to mortality within 18 years. Approximately 60% of the observations we were working with were participants for whom NHANES did not have an exact date-time of death; this meant that in the dataset, those values had time last known alive as a proxy for time of death, giving us inherently less precise data to work with and thereby limiting longitudinal analysis. Furthermore, certain predictors had a significant amount of missing values that we estimated through mean imputation.

Future Directions

As we encountered some counterintuitive results, we hope to conduct further research to corroborate some of these findings. First, we found that low serum cholesterol levels corresponded to a higher risk of mortality in Black and Other individuals. As it is unclear whether the cholesterol investigated in this study regards HDL (“good cholesterol”) or LDL (“bad cholesterol”), we would like to run our model on

new data clearly specifying cholesterol type in order to confirm our expectation that high LDL and HDL levels, respectively, lead to increased and decreased mortality risk.

Additionally, to our surprise, PI seemed to bear little to no interaction with other predictors. In contrast, however, most epidemiological studies suggest that poverty impacts biological factors including blood pressure and BMI, which was not reproduced in our own study. We suspect that PI may not be the best metric to include, as PI has been shown to under-estimate true levels of poverty (Hutto et al., 2011). To more properly represent poverty in our dataset, we hope to investigate the relationship annual household income might bear with other predictors.

As relatively few individuals in our dataset identified with the racial "other" category, we hope to increase recruitment efforts to diversify our subject population. In doing so, we additionally hope to explicitly include more racial categories in our model, rather than lumping non-White, non-Black individuals into a single broad category.

Beyond the predictors included in our dataset, we wish to further incorporate additional non-biological predictors and investigate their impact on mortality risk. For example, it would be interesting to see whether the nutritional data collected from the original NHANES I survey impacts mortality in alignment with our expectations. One motivating question might be "What type of diet most increases one's risk for mortality?", which may help inform health initiatives such as those related to public school cafeteria food offerings. Behavioral component data from the NHANES I survey too may be interesting to explore to assess how factors ranging from mental health to exercise impact mortality risk.

Despite the aforementioned benefits that might come with enhancing our dataset, we have still successfully pinpointed several recommendations of public health initiatives moving forward. It is our sincere hope that such recommendations may help to personalize and improve preventative healthcare for individuals as well as identify broader strategies to promote health consciousness in the general population.

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