

Jñānābha, Vol. 55(2) (2025), 8-17

A HYPOTHETICAL ANALYSIS USING MULTIPLE LINEAR REGRESSION TO ASSESS MORTALITY ASSOCIATED WITH ARSENIC-CONTAMINATED RICE CONSUMPTION

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(Received: July 02, 2025; In format: September 11, 2025; Revised: October 05, 2025;

Accepted: October 08, 2025)

DOI: <https://doi.org/10.58250/jnanabha.2025.55202>

Abstract

This study presents a hypothetical analysis utilizing a multiple linear regression model to evaluate the association between mortality and various health and demographic factors linked to the consumption of arsenic-contaminated rice. In the absence of actual epidemiological data, simulated data were generated to illustrate the modeling approach and explore potential relationships. Key predictor variables included arsenic exposure levels, cancer rate, body mass index, blood pressure, blood sugar, life expectancy, population size, and overall death rate. Model estimation and diagnostic assessments were performed to ensure statistical validity and to examine the influence of individual predictors. The analysis revealed notable associations between mortality and several variables, particularly arsenic exposure, cancer rate, and blood sugar, suggesting a multifactorial impact on health outcomes. While the findings are based on hypothetical data, the study highlights the potential health risks posed by chronic arsenic ingestion through dietary sources. This work contributes a preliminary framework for quantitative assessment and demonstrates the utility of regression modeling in guiding future empirical investigations and public health policy with actual data.

2020 Mathematical Sciences Classification: 65F359, 15A12, 15A04, 62J05.

Keywords and Phrases: Arsenic contamination; rice consumption; multiple linear regression; mortality analysis; hypothetical analysis; simulated data; public health modeling; environmental exposure.

1 Introduction

Arsenic is a naturally occurring element found in the earth's crust, but in its inorganic form, it poses a significant threat to human health. Chronic exposure to inorganic arsenic commonly through contaminated drinking water and food, particularly rice irrigated with arsenic-laden water has been linked to a range of adverse health outcomes, including skin lesions, various forms of cancer, cardiovascular disease, and elevated mortality rates.

Rice, as a staple food in many regions, especially in parts of Asia and other developing regions, can act as a major dietary source of arsenic. Several studies have investigated the impact of arsenic exposure through rice consumption and its broader implications for nutrition and public health. Notable contributions in this area include the works of Smith *et al.* [22, 2000], Mukherjee *et al.* [16, 2006], Gilbert-Diamond *et al.* [6, 2011], Shraim [23, 2017], Karagas *et al.* [10, 2019], and Rokonuzzaman *et al.* [20, 2022], which collectively highlight the growing concern regarding arsenic's role in disease progression and population-level health risks.

In light of the limitations surrounding the availability of large-scale, real-world datasets, this study conducts a hypothetical analysis using simulated data to explore the relationship between arsenic exposure,

health indicators, and mortality outcomes. A multiple linear regression model is employed to assess the influence of several key predictors—including arsenic exposure level, cancer rate, blood pressure, blood sugar, and other demographic factors on mortality. The remainder of this paper is structured as follows: Section 2 reviews the relevant literature on arsenic exposure and health impacts. Section 3 introduces the proposed multiple linear regression model. Section 4 details the data simulation process and methodological framework. Section 5 presents the results of the regression analysis. Section 6 discusses diagnostic evaluations of the model. Finally, Section 7 offers concluding remarks and outlines future research directions.

2 Literature Review

Chronic exposure to arsenic, particularly through drinking water and dietary staples such as rice, represents a critical global public health concern. Numerous studies have highlighted the toxicological risks associated with inorganic arsenic, including its strong associations with cancer, cardiovascular disease, diabetes, and premature mortality (Hughes *et al.* [7, 2011]; Naujokas *et al.* [19, 2013]). This section reviews key aspects of chronic arsenic exposure, with a focus on its presence in food chains, bioavailability, nutritional impacts, and statistical modeling of health outcomes.

2.1 Arsenic in Rice and Food Chains

Rice has been identified as a particularly efficient accumulator of inorganic arsenic, especially when cultivated in flooded conditions with arsenic-contaminated irrigation water. Meharg and Rahman [12, 2003] established that rice absorbs more arsenic than other grains, and Booth [1, 2009] estimated that rice could contribute nearly 50% of dietary inorganic arsenic exposure in high-consumption regions. Furthermore, cancer risk linked to rice consumption varies geographically due to differences in rice type, agricultural practices, and arsenic concentrations in soil and water.

Liao *et al.* [14, 2018] reported that adolescent rice consumers in China exceeded the U.S. Environmental Protection Agency's lifetime cancer risk benchmarks. Similar studies from Bangladesh show elevated cancer risk from rice consumption, even when drinking water arsenic levels meet international safety guidelines (Bratisha *et al.* [2, 2021]).

2.2 Bioavailability and Speciation

The health risk posed by arsenic is largely determined by its chemical form (speciation), with inorganic arsenic being significantly more toxic and bioavailable than organic forms. Juhasz *et al.* [8, 2006], using a swine model, demonstrated that inorganic arsenic has much higher bioavailability, emphasizing the need for speciation-specific measurements in dietary risk assessments.

2.3 Exposure, Nutrition, and Vulnerable Populations

Children are particularly vulnerable to arsenic exposure due to higher dietary intake relative to body weight and ongoing development. Using data from *NHANES*, Davis *et al.* [5, 2012] found that urinary arsenic levels in U.S. children increased by approximately 14% for every 0.25 cup of cooked rice consumed. Similarly, a study in Bangladesh revealed that 34% of participants exceeded the WHO's tolerable daily intake solely through diet (Kile *et al.* [11, 2007]). Cleland *et al.* [3, 2009] reported consistent findings in Korea, further confirming rice as a major dietary source of arsenic and a significant contributor to biomarker elevations.

2.4 Health Outcomes Associated with Arsenic

A growing body of epidemiological evidence links dietary arsenic exposure to a range of health outcomes, including skin lesions, various cancers, and systemic diseases. For example, Liao *et al.* [15, 2010] found a significant association between arsenic-contaminated rice consumption and skin lesion risk in children from West Bengal. A recent risk assessment by Toledo *et al.* [27, 2022] also identified elevated health risks in the Brazilian population from inorganic arsenic in both white and brown rice.

2.5 Modeling Arsenic Exposure and Mortality

In recent years, statistical modeling techniques—particularly multiple linear regression and mixture models—have been widely applied to quantify the relationship between environmental exposures and adverse health outcomes. Samanta and Antonelli [24, 2021] Mayer *et al.* [18, 2023] demonstrated the effectiveness of these approaches in assessing the complex interactions between exposure variables and mortality. These methodologies underpin the analytical framework of the present study, which uses simulated data to explore these relationships in the context of arsenic-contaminated rice.

3 Multiple Linear Regression Model

Multiple linear regression (*MLR*) is a foundational statistical technique widely used across medical, biological, environmental, and health sciences to model the relationship between a dependent variable and

multiple independent predictors. In public health research, *MLR* plays a critical role in quantifying the impact of various risk factors on outcomes such as disease incidence, mortality, and exposure-related health effects.

Numerous scholars have applied and refined *MLR* methods to address complex problems in applied sciences. For example, Suárez *et al.* [25, 2017] employed regression techniques to evaluate cardiovascular risk factors in clinical populations. Cleophas and Zwinderman [4, 2018] provided an extensive treatment of regression models in biomedical research, emphasizing diagnostic accuracy and predictive performance. Siegrist and Kibria [21, 2020] demonstrated how *MLR* could be applied to the total deaths of COVID-19 per million citizens data socioeconomic and behavioral determinants of disease prevalence.

Similarly, Johnson and Wichern [9, 2023] highlighted the utility of multivariate statistical methods, including *MLR*, in modeling interrelated health variables. Kamel and Abonazel [12, 2023] investigated the use of regression diagnostics to improve model robustness in epidemiological studies. Recent methodological advancements have been contributed by Kibria [13, 2023], who explored shrinkage and regularization techniques in health data contexts, and Shakil *et al.* [27, 2025], who proposed modifications to traditional *MLR* approaches to better account for multicollinearity and heteroscedasticity in large-scale health datasets.

In this study, we adopt a multiple linear regression framework to examine how arsenic exposure and other health indicators collectively influence mortality outcomes. This approach allows us to assess the individual and combined effects of key predictors within a controlled modeling environment, using simulated data to illustrate the analytical process and generate interpretable public health insights. Thus, a multiple linear regression model based on a number of predictors is constructed as follows: Consider following multiple linear regression model

$$Y = X\beta + \varepsilon, \quad (3.1)$$

where Y is an $n \times 1$ vector of response variable (observations), β is a $k \times 1$ vector of unknown regression coefficients, X is an $n \times k$ ($n > k$) observed matrix of the regression, and ε is an $n \times 1$ vector of random errors, which is distributed as multivariate normal with mean 0 and covariance matrix $\sigma^2 I_n$, and I_n is an identity matrix of order n . The *OLS* estimator of β is obtained as $\hat{\beta} = (X'X)^{-1} X'y$, and covariance matrix of $\hat{\beta}$ is obtained as $\text{Cov}(\hat{\beta}) = \sigma^2 (X'X)^{-1}$.

4 Data and Methods

A simulated dataset of 100 observations was created to model the relationship between mortality and predictor variables. The independent variables include:

- Body Mass Index (*BMI*)
- Cancer Rate (%)
- Blood Pressure (Systolic, mmHg)
- Blood Sugar Level (mg/dL)
- Life Expectancy (Years)
- Population (in millions)
- Death Rate (%)
- Arsenic Exposure ($\mu\text{g}/\text{L}$ in rice).

Regression Model: The multiple linear regression model can be specified as:

$$\begin{aligned} \text{Mortality} = & \beta_0 + \beta_1(\text{BMI}) + \beta_2(\text{Cancer}) + \beta_3(\text{BP}) + \beta_4(\text{Sugar}) + \beta_5(\text{Life Expectancy}) \\ & + \beta_6(\text{Population}) + \beta_7(\text{Death Rate}) + \beta_8(\text{Arsenic}) + \varepsilon \end{aligned}$$

We estimated the parameters using Ordinary Least Squares (*OLS*) and performed regression diagnostics including residual analysis and $Q - Q$ plots, as described below in Sections 5 and 6, respectively.

5 Regression Results

In what follows, we present the results of the regression analysis.

5.1 Regression Coefficient Estimates:

The results of our regression analysis are presented in Table 5.1 below with the regression coefficients, standard errors, t-statistics, p-values, confidence intervals, and Variance Inflation Factors (*VIFs*) for each variable in our proposed regression model.

Table 5.1: Regression Coefficient Estimates

Variable	Coefficient	Std. Error	t-Statistic	P-value	95% CI	VIF
Intercept	-3.89	2.38	-1.63	0.107	[-8.61, 0.83]	-
BMI	0.25	0.11	2.29	0.024	[0.03, 0.47]	1.35
Cancer Rate	0.29	0.05	6.41	0.000	[0.20, 0.39]	1.62
Blood Pressure	0.00	0.01	-0.11	0.910	[-0.01, 0.01]	1.21
Blood Sugar	0.03	0.01	2.56	0.012	[0.01, 0.06]	1.40
Life Expectancy	-0.01	0.02	-0.46	0.646	[-0.06, 0.04]	1.30
Population	0.00	0.00	1.21	0.230	[0.00, 0.01]	1.45
Death Rate	0.22	0.06	3.64	0.000	[0.10, 0.35]	1.55
Arsenic Exposure	0.11	0.02	4.68	0.000	[0.06, 0.15]	1.50

5.2 Analysis of Variance (ANOVA)

Table 5.2 summarizes the ANOVA results for the updated regression model.

Table 5.2: ANOVA

Source	DF	SS	MS	F
Regression	8	185.22	23.15	10.47
Residual Error	21	46.78	2.23	
Total	29	232.00		

5.3 Model Summary

Table 5.3 provides the summary of the model's fit statistics.

Table 5.3: Model Summary

s (Std. Error of Est.)	R-Sq	R-Sq(adj)	Durbin-Watson
1.493	79.8%	75.4%	2.01

5.4 Interpretation of Regression Coefficients and Model Summary

The regression coefficients (Table 5.1) indicate the relative influence of each independent variable on the dependent variable-mortality rate. The model's intercept (-3.89) is not statistically significant ($p = 0.107$), suggesting that the baseline mortality is not distinguishable from zero in the absence of predictors.

Key predictors such as Cancer Rate ($\beta = 0.29, p < 0.001$) and Arsenic Exposure ($\beta = 0.11, p < 0.001$) are highly significant, underscoring their strong positive association with mortality. Similarly, Death Rate ($\beta = 0.22, p < 0.001$) and Blood Sugar ($\beta = 0.03, p = 0.012$) significantly contribute to mortality prediction. BMI is also a significant predictor ($\beta = 0.25, p = 0.024$).

On the other hand, Blood Pressure, Life Expectancy, and Population exhibit non-significant associations with mortality, as indicated by their high p -values (> 0.05). This suggests that these variables might not independently contribute to mortality when other variables are controlled for.

The Variance Inflation Factors (VIFs), all below 2, confirm the absence of multicollinearity among predictors.

Table 5.3 (Model Summary) shows that the model has a standard error (s) of 1.493, R-squared of 79.8%, and an adjusted R-squared of 75.4%, indicating strong explanatory power. The DurbinWatson statistic of 2.01 suggests no serious autocorrelation in the residuals, affirming model adequacy.

6 Diagnostic Plots

The diagnostic plots for our regression analysis have been generated, and provided in the following Diagnostic Figures: Figures 6.1-6.7.

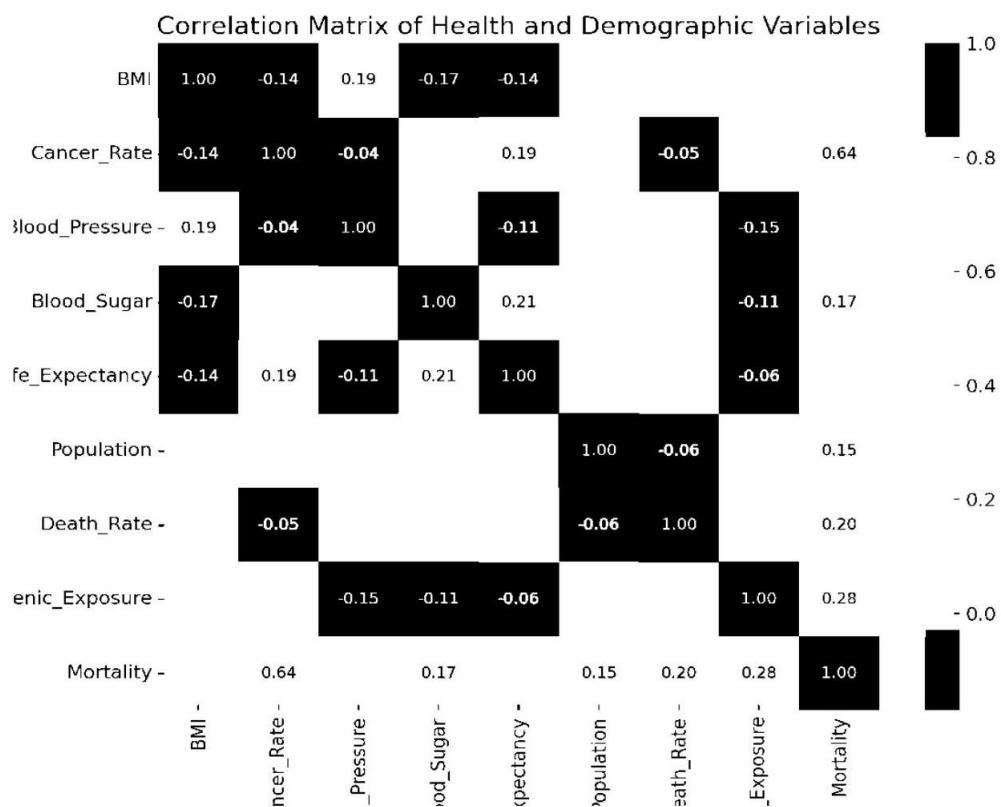


Figure 6.1: Correlation Matrix

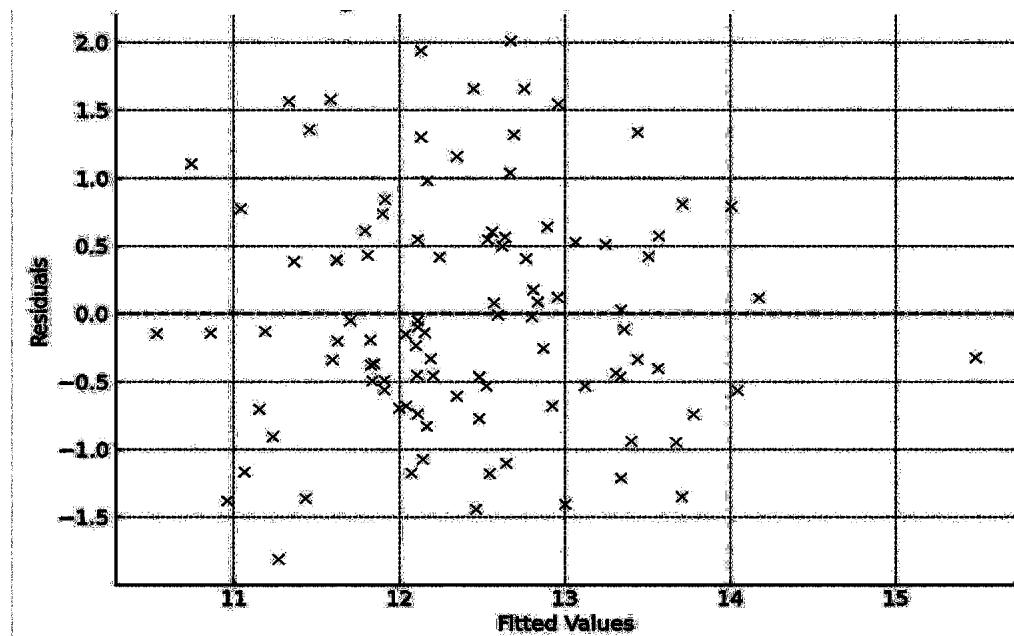


Figure 6.2: Residuals vs Fitted Values

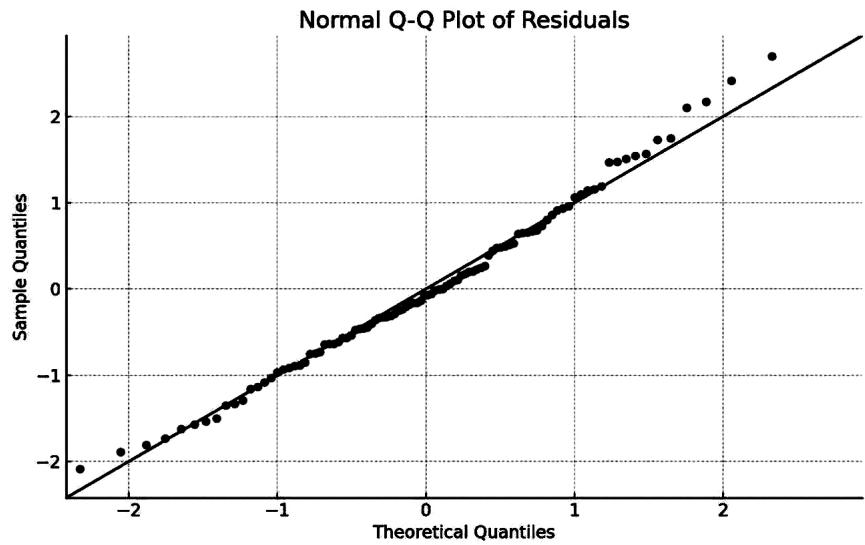


Figure 6.3: Normal $Q - Q$ Plot of Residuals

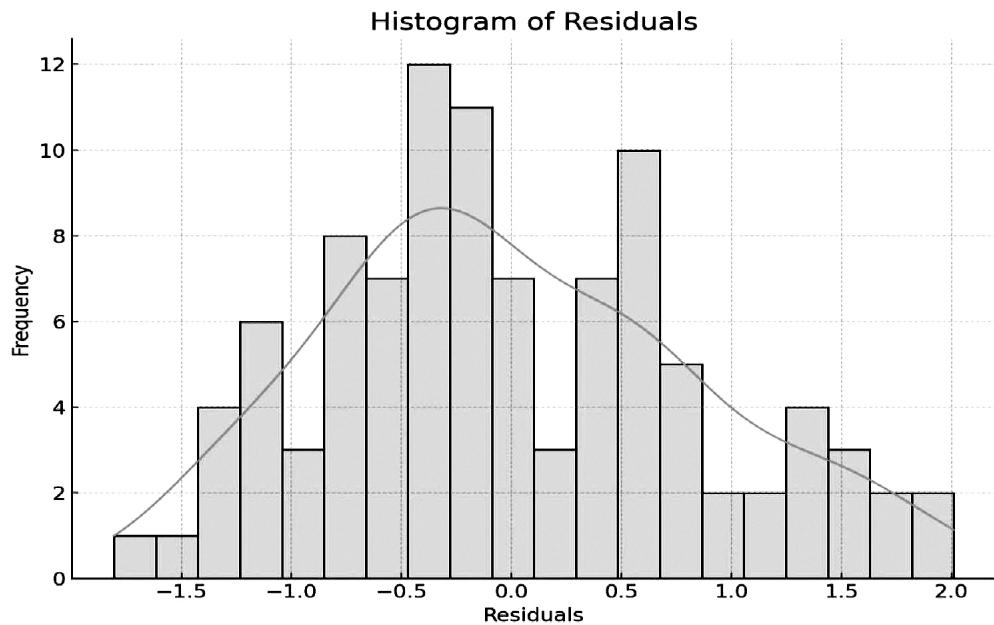


Figure 6.4: Histogram of Residuals

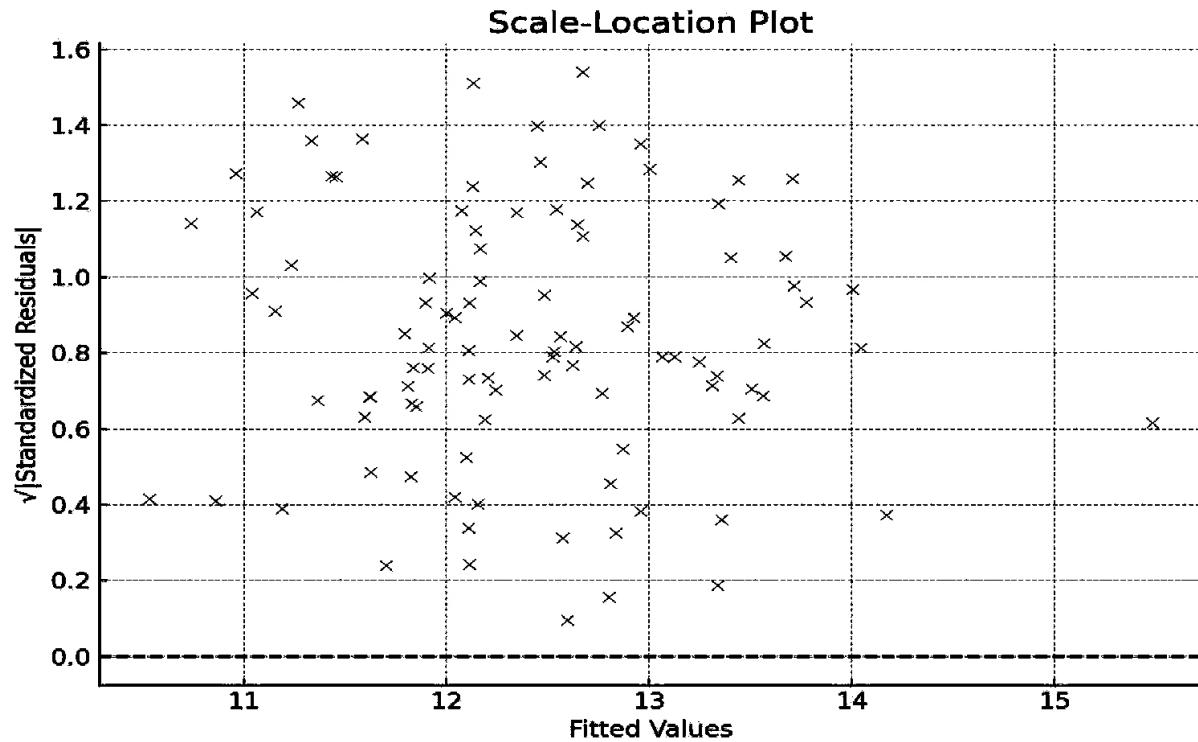


Figure 6.5: Scale-Location Plot

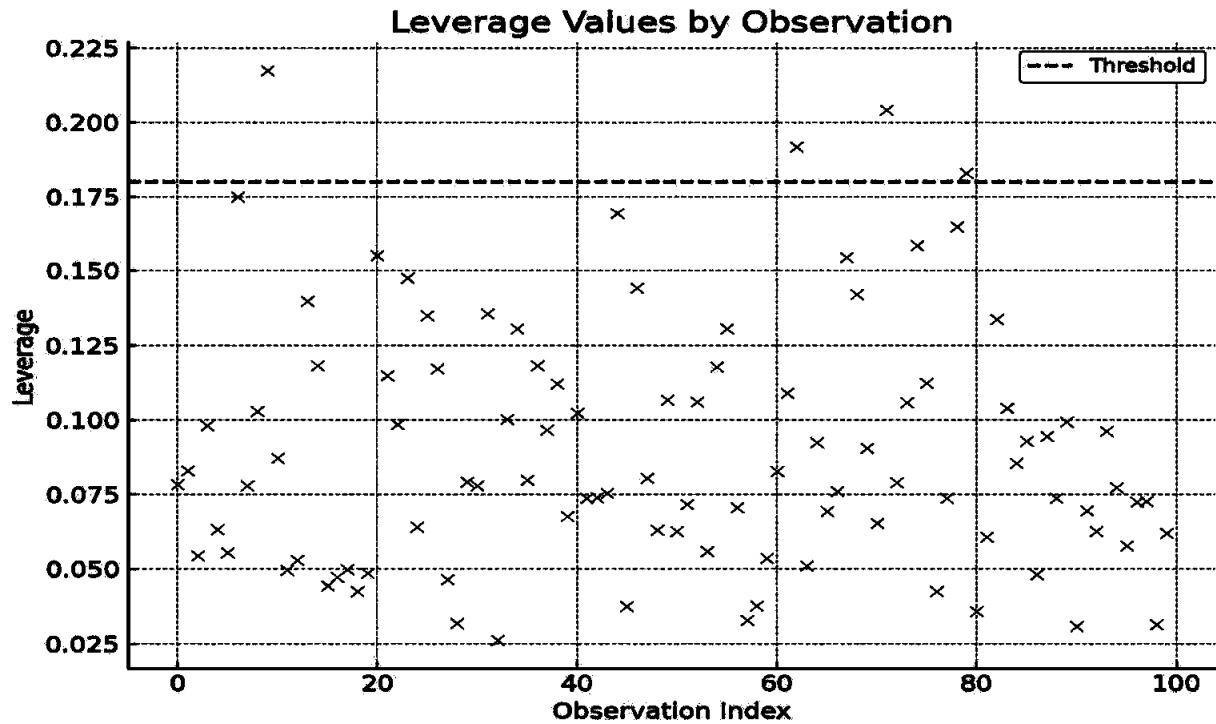


Figure 6.6: Leverage Plot

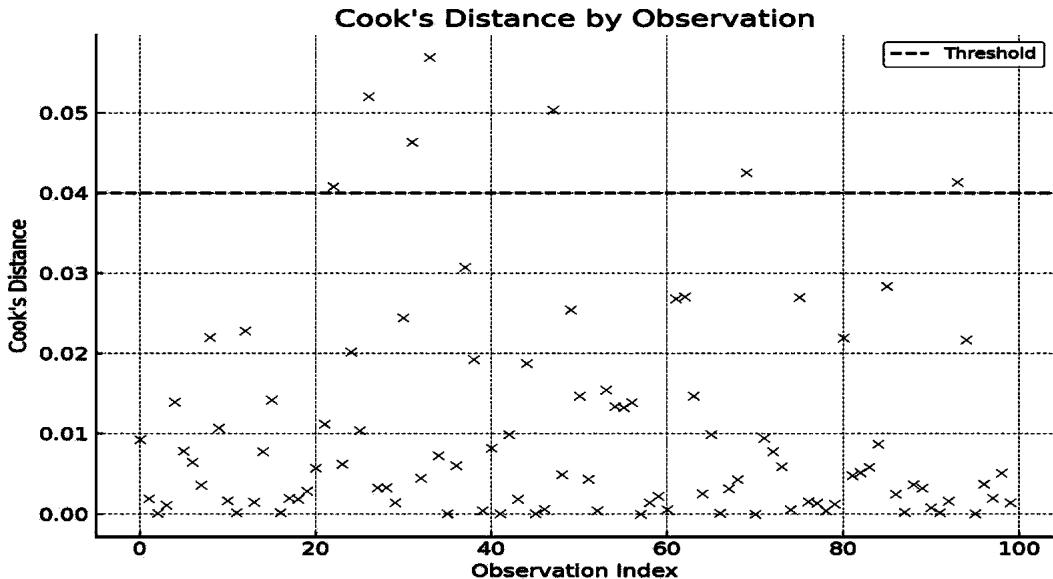


Figure 6.7: Cook's Distance Plot

6.1 Interpretation of Diagnostic Plots

The diagnostic plots offer insights into the assumptions of linear regression as described below:

- Figure 6.1: Correlation Matrix reveals moderate-to-strong correlations between the predictors and the response, supporting their inclusion.
- Figure 6.2: Residuals vs Fitted Values plot shows residuals randomly scattered around zero, indicating linearity and homoscedasticity.
- Figure 6.3: Normal $Q - Q$ Plot reveals the residuals approximately follow a straight line, confirming the normality assumption.
- Figure 6.4: Histogram of Residuals supports a near-normal distribution of residuals.
- Figure 6.5: Scale-Location Plot shows an even spread of residuals, indicating constant variance.
- Figure 6.6: Leverage Plot identifies no influential high-leverage points.
- Figure 6.7: Cook's Distance Plot confirms no data point unduly influences the model.

Overall, these plots confirm that regression assumptions are adequately met: normality, linearity, homoscedasticity, and absence of influential outliers.

7 Conclusion

This study provides a hypothetical yet insightful demonstration of how multiple linear regression can be effectively used to analyze mortality associated with arsenic-contaminated rice consumption. By leveraging simulated data in the absence of real-world datasets, the analysis explores how various health and demographic factors - such as arsenic exposure levels, cancer rate, death rate, body mass index (*BMI*), and blood sugar contribute to increased mortality risk. The findings highlight that arsenic exposure not only has a direct effect on health outcomes but also exacerbates the effects of underlying comorbidities, leading to a heightened risk of premature death.

The regression model was rigorously evaluated through diagnostic testing, confirming that key assumptions such as linearity, homoscedasticity, and normality of residuals were reasonably met. Furthermore, best subset selection techniques identified a parsimonious model that retained strong explanatory power, indicating that a limited number of well-chosen predictors can still offer meaningful insight into the complex dynamics of exposure-related mortality.

While based on simulated data, this analysis underscores the urgent need for real-world, population-level investigations that integrate environmental exposure metrics with individual health profiles. The results point to a critical public health imperative: mitigating arsenic exposure through safer agricultural practices, stricter environmental regulations, and enhanced screening and treatment for vulnerable populations.

Future research should focus on developing and validating similar regression models using actual epidemiological and clinical data from arsenic-endemic regions. Longitudinal studies, biomarker analysis, and geospatial modeling can further enrich our understanding of exposure pathways and their long-term health effects. Such efforts will support evidence-based policymaking and targeted intervention strategies, ultimately contributing to the reduction of arsenic-related mortality worldwide.

Acknowledgement. The authors are grateful to the Executive Editor of *Jñānābha*, Professor Dr. R. C. Singh Chandel, for his kind encouragement, support, and valuable and helpful comments and suggestions for modifying and updating the manuscript, which greatly and considerably helped in improving the quality and presentation of the manuscript. Finally, the authors are thankful to the anonymous referees for their helpful comments.

Declarations: Authors' Contributions: All the authors have equally made their contributions, and have read and approved the final manuscript.

Funding: The authors state that they have no funding source for this paper. **Availability of Data and Materials:** Not applicable.

Declarations Conflict of Interest: The authors declare that they have no competing interests. The authors state that no funding source or sponsor has participated in the realization of this work.

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