

# Associations Between Serum Creatinine and Cardiovascular Risk Parameters

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## Abstract

Within the framework of policies against climate change, the expectations of sustainability in the management and management of resources have been a focus of discussion and a central issue on the agenda of cities, but the corresponding studies have established a framework hegemonic theoretical consistent in political culture as a theoretical referent rather than as a scenario for observing the emergence of actions in favor of the environment. The objective of the work was to observe the structure of the variables that the literature identifies as determinants of pro-environmental behavior. A non-experimental study was carried out with a non-probabilistic selection of 400 students who responded to a self-report of their values, norms, perceptions, beliefs, attitudes, knowledge and actions to care for water resources. A structure was found that explained 67% of the variance and the determinant relationship between intention and behavior, but without the interrelation with the other variables, said discrepancies were discussed within the framework of optimization and innovation of organizations with corporate social responsibility.

**Keywords:** growth; development; organizations; communities; calendar

## Introduction

Some articles have reported that elevated serum creatinine (SCT) concentration levels may be an independent causal factor of all-cause of cardiovascular disease markers and mortality predictor variables [1-4]. It is well known that SCT level is associated with the elderly persons, myocardial infarction (or stroke) patients, and it increases mortality in hypertensive individuals [5-8]. An article has searched the relationship between SCT concentration levels and the risk factors of stroke events, major ischemic heart disease and all mortality causes of a middle-aged men population [9]. The article [9] has pointed out that there is a positive weak significant association between SCT concentration levels and diastolic blood pressure (DBP). However, it has been pointed out that SCT concentration levels ( $\geq 116 \mu\text{mol/L}$ ) is significantly associated with the increase in stroke both for hypertensive and normotensive men [9]. Recently an article [10] has examined the association between the ratio of SCT level and cystatin-C level (i.e., SCT/ Cystatin C ratio) and mortality in hypertensive patients. These researchers have studied a composite term SCT/ Cystatin C ratio instead of SCT level on heart disease parameters. The article [10] has pointed out that low muscle mass exhibited by lower SCT/Cystatin-C ratio was an independent causal factor for weak prognosis in hypertensive patients. However, the relationship between SCT level and cardiac (or heart) disease parameters is not definite or clear. Most of the earlier research articles regarding the relationship of SCT level and cardiac risk parameters are not definitively conclusive. In addition, it is better to

derive the relationship between two single factors than the composite factors. It is very difficult to conclude regarding composite factors. Actually, most of the earlier studies regarding the relationship between SCT level and cardiac risk factors are not based on proper probabilistic modeling, so all the earlier results invite some doubts and debates. This can be ensured with the help of the exact probabilistic model of SCT level with cardiac disease parameters such as blood pressure, systolic blood pressure, diastolic blood pressure, heart rate, ejection fraction, heart disease status along with the other heart disease explanatory variables/factors.

The current editorial report focuses on the following research hypotheses.

- Is there any relationship/ correlation of SCT level with high blood pressure (BP), ejection fraction (EFT) and heart disease subjects? If it is affirmative, what is the most probable SCT relationship model with cardiac parameters?
- How do we obtain the most probable SCT level model with cardiac causal factors?
- What are the associations of SCT level with BP, EFT and heart disease patients?

The above hypotheses are examined in the current report with the help of a real data set of 299 cardiac patients along with 13 factors, and the data set is reported in [11, 12], and it is available in the site

<https://archive.ics.uci.edu/ml/datasets/Heart+failure+clinical+records>,  
The 13 recorded factors in the data set are:

- Age,
- Sex (0=female, 1=male),
- Diabetes mellitus status (DMS) (0= no diabetes, 1= diabetes),
- Anaemia status (ANS) of subjects (0= no anaemia, 1= anaemia),
- Creatinine phosphokinase (CRP),
- High blood pressure (BP) (0= normal BP, 1=high BP),
- Serum creatinine (SCT),
- Ejection fraction (EFT),
- Serum sodium (SNa),
- Platelets count (PLC)
- Total follow-up time period (TTP),
- Death event (DEE) (0=alive, 1=death).
- Smoking habit (SMH) (0=no smoking, 1= smoking),

The above-mentioned heart patient's data set is multivariate form, heteroscedastic and non-normal. The response variable in the current study is SCT level that is an unequal variance continuous response variable. The variance of SCT's response variable is not stabilized by any suitable transformation, so it is modeled by using joint generalized linear models (JGLMs) that is illustrated in the book by Lee et al. [13]. The derived SCT's Log-normal fitted mean and variance models are as follows.

Log-normal fitted SCT mean ( $\hat{\mu}_z$ ) model (Table 1) is

$$\hat{\mu}_z = 2.83 - 0.0029 \text{ EFT} + 0.007 \text{ AGE} + 0.0519 \text{ DEE} - 0.0019 \text{ CPK} + 0.0069 \text{ EFT*DEE} - 0.0215 \text{ SNa} - 0.1113 \text{ BP} + 0.0001 \text{ CPK*SNa} - 0.0003 \text{ TFP} + 0.0602 \text{ ANS} + 0.049 \text{ SEX} - 0.0008 \text{ TFP*ANS} - 0.2591 \text{ SMS} +$$

0.0015 TFP\*SMS, and the fitted SCT variance ( $\hat{\sigma}^2_z$ ) model is

$$\hat{\sigma}^2_z = \exp.(1.6286 - 4.0776 \text{ ANS} - 0.0224 \text{ AGE} - 0.0006 \text{ CPK} + 0.0518 \text{ AGE*ANS} + 0.0005 \text{ CPK*ANS} - 0.5959 \text{ DMS} - 0.0355 \text{ EFT} + 0.0006 \text{ CPK*DMS} + 0.0332 \text{ EFT*ANS} - 0.0001 \text{ PLC} - 2.4493 \text{ DEE} - 1.4723 \text{ BP} + 0.0001 \text{ PLC*BP} + 0.0767 \text{ EFT*DEE} + 0.4080 \text{ SEX} - 0.0028 \text{ TFP} - 0.0032 \text{ TFP*ANS} - 0.2012 \text{ SMS}).$$

The above data set contains only two heart disease related risk factors such as ejection fraction (EFT) and high blood pressure status (BP) (0= normal BP, 1=high BP). From the above SCT level fitted mean Log-normal model, it is derived that mean SCT level is positively associated with AGE ( $P < 0.0001$ ). It indicates that the mean SCT levels are higher at older ages, which supports the earlier reports [2,3,5,7]. From the mean model, it is noted that mean SCT level is partially inversely associated with ejection fraction (EFT) ( $P = 0.097$ ) and partially directly associated with the joint interaction effects (JIEs) of EFT\*DEE (0=alive, 1=death) ( $P = 0.116$ ). This implies that SCT level increases as the joint effect EFT\*DEE increases. In other words, SCT level is higher for the heart patients who are close to death than the surviving heart patients. This is not reported in any previous articles. Mean SCT level is inversely associated with high blood pressure (BP) ( $P = 0.003$ ), indicating that SCT level rises as the BP decreases. This outcome is different from the results of the earlier articles [1, 4, 8]. This implies that higher SCT levels can decrease BP, which may invite stroke for the heart patients. From SCT variance model, it is noted that SCT variance is inversely associated with EFT ( $P = 0.002$ ) and anaemia status (ANS) ( $P = 0.004$ ), while it is directly associated with their JIEs of EFT\*ANS ( $P = 0.023$ ). This reveals that SCT level's scatteredness increases for the anaemia patients with higher EFT levels. Also, SCT variance is inversely associated with PLC ( $P = 0.054$ ) and BP ( $P = 0.013$ ), while it is directly associated with their JIEs of PLC\*BP ( $P = 0.035$ ). This indicates that SCT level's scatteredness increases for the BP patients with higher PLC levels. Again SCT's variance is inversely associated with DEE

( $P < 0.001$ ) and EFT ( $P = 0.002$ ), while it is directly associated with their JIEs of EFT\*DEE ( $P < 0.001$ ). It implies that SCT level's scatteredness increases for the heart patients close to death with higher EFT levels. Note that variance related associations of SCT level with the cardiac factors are not reported to any previous articles. The editorial note focuses on the above mean and variance associations of SCT levels with the cardiac parameters high BP and ejection fraction (EFT) only. The data set does not contain separately the systolic BP, diastolic BP, basal BP, mean arterial BP, heart rate, basal heart rate, peak heart rate, maximum heart rate, myocardial infarction etc. So, it is not possible to examine the associations of SCT levels on these cardiac parameters. Hope future researchers will consider all these parameters for evaluating the associations of SCT levels on these cardiac parameters. The report has pointed out that SCT level increases at older ages, and SCT level is higher for the heart patients who are close to death than the surviving heart patients, and also higher SCT levels can decrease BP, that may invite stroke for the heart patients. The editorial note will be helpful for the heart patients, practitioners and researchers to know the effect of SCT levels. Care should be taken for the SCT levels at older ages.

**Conflict of interest:** The authors confirm that this article content has no conflict of interest.

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