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# Role of Resistin with Endothelium Dysfunction in STEMI and NSTEMI Patients and Its Correlation with Cardiac Markers Troponins

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**Abstract:** Acute coronary syndrome (ACS) is the leading cause of mortality worldwide comprised with ST-segment elevation myocardial infarction (STEMI) and non ST segment elevation myocardial infarction (NSTEMI). A cross sectional study for a period from March 2015 to June 2016 was designed to find out the role of resistin in endothelium dysfunction in STEMI and NSTEMI patients and its correlation with cardiac markers troponins. 100 consented patients with diagnosed STEMI and NSTEMI with age range 40 to 70 years were included in the study. 50 age matched healthy subjects were taken as controls. Base line blood tests fasting blood sugar, blood urea, serum creatinine, serum total cholesterol and serum triglyceride were estimated by auto analyzer using standard kits. Special tests including serum resistin, cardiac troponin T and cardiac troponin I were estimated by the technique of ELISA. Results were analyzed by SPSS 20 and found that the base line parameters except serum creatinine were significantly raised in patients as compare to controls. Level of resistin and cardiac biomarkers cardiac troponin T (cTnT) and cardiac troponin I (cTnI) were significantly raised in STEMI patients as compared to NSTEMI patients. A significant positive correlation of resistin with cardiac troponin T and I was observed in STEMI patients only. It is therefore concluded that increased level of resistin along with cardiac markers may be presented as a predictor of injury of myocardium and in turn indicate the severity of AMI in STEMI patients.

**Keywords:** STEMI, NSTEMI, Resistin, Cardiac Troponin T, Cardiac Troponin I

## 1. Introduction

Acute coronary syndrome (ACS) is the leading cause of mortality worldwide, particularly in the developing countries. ACS is a discrepancy in the demand and consumption of oxygen in myocardium tissues. In cases of STEMI, the discrepancy may be ruptures of plague of coronary artery resulting in the formation of thrombus and block the coronary artery. However, NSTEMI results from narrowing of coronary artery or micro-embolization of thrombus [1, 2].

Resistin is a 12 Kda protein mainly secretes from macrophages and monocytes. It may be a link between metabolic signals mediated inflammation and problem of atherosclerosis [3]. It is also involved in vascular ailments. It is suggested that in atheromas resistin is secreted by

macrophages and promote inflammation of the vasculature. Additionally it promotes proliferation of smooth muscle cell and angiogenesis [4]. Clinically, a link between increased level of resistin and coronary artery disease was also observed and it is suggested that levels of resistin is found to be stepwise increase, depending on the number of segments or stenotic vessels [5, 6].

It is thought that resistin binds to cell membrane receptor TLR4, activated the signaling pathways of cells especially MAP kinase pathway. This pathway in-turn promote the secretion of cytokines that may act as pro-inflammatory agents resulting in the dysfunction of endothelium including altered vaso-relaxation, increased the process of thrombosis, increase permeability of membrane and raised adhesion of cells and help in the development of atherosclerosis [5, 7].

The best biomarker for finding the injury of myocardium should have high values in cardiac tissue, with high sensitivity and specificity for the identification of damage of myocardium. These are measurable in blood of patients initially after the beginning of symptoms, like chest pain [8]. Cardiac troponins are proteins that regulate the calcium based interaction of actin & myosin, and take part in relaxation and contraction of striated muscle. Cardiac troponins are good predictor of AMI as these are highly sensitive and specific as compared to other biomarkers. It is stated the levels of troponin secreted in blood and elevation with time is directly related with size of infarcted muscle, thrombosis and alteration of perfusion of tissue of myocardium [9]. Release of troponin in STEMI patients has expectable kinetics with initial levels reflect the duration of ischemia [10]. On the other hand NSTEMIs are distinct and associated with cardiac muscle injury resulting in the elevation of troponin without changes in ECG [11].

Increased values of troponin 1 are a predictor of poor prognosis. It is proposed that in the occurrence of AMI, an increase level of Tn I inhibit the complex of troponin-thin filaments, and block the interaction of actin-myosin with intracellular calcium and resulting in impairment of regulation of contractibility of myocardium [12].

An association between the level of troponin and resistin is observed in patients with STEMI. Study stated that combined effect of troponin I and resistin may be a reason of elevated left ventricular ejection fraction (LVEF). Study demonstrated that levels of resistin were associated with degree of infarction in patients with STEMI. It is suggested that high levels of resistin have predictive value for the incidence of systolic heart failure in patients with STEMI with better specificity, sensitivity, and precision than the levels of troponin I [13]. Increased values of troponin T after chest pain indicate myocardial infarction and significantly related heart failure and cardiovascular death [14]. However a study found no link between the levels of circulating adipokine resistin and elevation of troponin in peri-procedural myocardial injury [15].

Patients with high blood glucose are more prone to develop cardiac muscle impairment, increased secretions of biomarkers of myocardial necrosis, and severe coronary syndromes [16].

## 2. Material and Methods

A cross sectional study was designed to find out the role of resistin in endothelium dysfunction in STEMI and NSTEMI patients and its correlation with cardiac markers troponins.

The cross sectional study was carried out in 100 patients aged from 40 to 70 years presenting with AMI in Punjab Institute of Cardiology (PIC) Lahore. Patients with diabetes mellitus, renal disease or muscle dystrophy were excluded from the study. 50 age matched healthy subjects were taken as controls. Prior consent of all subjects was obtained. History and investigations were recorded in proformas. Of the patients, 56% were diagnosed with STEMI and 44% with NSTEMI. The duration of study was March 2015 to June 2016. The study protocol was permitted by Ethical Review Committee, Department of Research, Training & Postgraduate Medical Education, Punjab Institute of Cardiology, Lahore. Written informed consent was obtained before enrollment from all the participants. A comprehensive questionnaire documenting information on demographic data, medical and family history of coronary artery disease was also completed for each subject.

Base line blood tests fasting blood sugar, blood urea, serum creatinine, serum total cholesterol and serum triglyceride were estimated by Auto Analyzer using standard kits. Special tests including serum resistin, troponin T and troponin I were estimated by the technique of ELISA.

## 3. Statistical Analysis

Data was analyzed by SPSS 20. Variables were expressed as mean ±SD. Comparison of variables of patients and controls were carried out by student ‘t’ test. Correlation between resistin and troponins were done by using Pearson Correlation Coefficient. P value $<0.05$ is taken as significant.

Results of Table 1: Among 100 cases, 31 % cases were with age <50 years and 69 % cases were with age of > 50 years. Gender based difference showed that 78 % male and 22% were female. BMI bases difference showed that 2 % were underweight, 38 % were with normal weight, 54% were overweight and 6 % were obese. About 54% cases having active life style, and 46% having sedentary life style. It is observed that among 70 cases presented with MI, 56% diagnosed as STEMI and 44 % were diagnosed as NSTEMI. Among 50 control cases 70% were male and 30% female. Most of the control subject presented with normal weight followed overweight and obese.

***Table 1.*** *Demographic characteristics of Patients and controls.*

| | Cases N (%) | Controls N (%) |
|---|---|---|
| Age | | |
| ≤ 50 years | 31 | 34 |
| > 50 years | 69 | 66 |
| Gender | | |
| Male | 78 | 70 |
| Female | 22 | 30 |
| BMI | | |
| Underweight (<18.5) | 2 | 2 |
| Normal weight (18.5-24.9) | 38 | 54 |
| Overweight (25.0-29.9) | 54 | 36 |
| Obese (>30.0) | 6 | 8 |
| Life style | | |
| Active | 54 | ____ |
| Sedentary | 46 | |
| Type of MI | | |
| STEMI | 56 | ____ |
| NSTEMI | 44 | |

***Table 2.*** *History profile of cases.*

| | Frequency | Percentage |
|---|---|---|
| History of hypertension | | |
| Positive | 64 | 64 |
| Negative | 36 | 36 |
| History of smoking | | |
| Positive | 48 | 48 |
| Negative | 52 | 52 |
| | mean | SD |
| Duration of chest pain | 6.17 | 4.7 |

Table 2 presented the history profile of cases. It is observed that 64 % cases have a history of hypertension and 36% cases have no history of hypertension. Among 100 cases 48% were smokers and 52% patients were non-smokers. Mean duration of chest pain in cases was 6.17 hours.

***Table 3.*** *Comparison of baseline investigations in cases and their controls.*

| Parameters | Cases mean±SD | Controls mean±SD | P-value |
|---|---|---|---|
| Serum Triglycerids (mg/dl) | 161.32±30.4 | 170.35±42.8 | 0.04* |
| Total serum Cholesterol (mg/dl) | 201.90±48.35 | 163.42±44.40 | 0.000* |
| Fasting Blood Sugar (mg/dl) | 120.73±51.13 | 90.46±18.90 | 0.000* |
| Blood Urea (mg/dl) | 39.27±16.07 | 25.97±9.44 | 0.000* |
| Serum Creatinine (mg/dl) | 0.98±0.40 | 0.92±0.52 | 0.24 |

Baseline investigations between cases and control are presented as table 3. It is observed that the level of lipid profile including triglyceride and cholesterol are significantly increased in cases as compared to controls. Levels of blood urea and blood glucose are significantly increased in cases as compared to controls. On the other hand serum creatinine is in-significantly increased in cases as compared to controls.

***Table 4.*** *Comparison of cardiac markers between STEMI and NSTEMI cases.*

| Cardiac markers | Male Cases with STEMI mean±SD | Male Cases with NSTEMI mean±SD | P-value |
|---|---|---|---|
| Resistin (ng/ml) | 27.48±8.37 | 23.31±6.13 | <0.05 |
| Troponin-T (ng/ml) | 1591.27±1423.89 | 293.97±188.43 | <0.001 |
| Troponin-I (ng/ml) | 15.61±12.04 | 2.29±1.91 | <0.001 |

Comparison of cardiac markers between STEMI and NSTEMI cases is tabulated as table 4. It is observed levels cardiac markers resistin, troponin t and troponin I was significantly increased in male cases with STEMI as compared to male cases with NSTEMI.

***Table 5.*** *Correlation of resistin with cTnT and cTnI in patients with STEMI.*

| Correlation | r-value |
|---|---|
| Serum resistin with cTnT | 0.69* |
| Serum adiponectin with cTnI | 0.63* |

*P <0.5 is significant.

***Table 6.*** *Correlation of resistin with cTnT and cTnI in patients with NSTEMI.*

| Correlation | r-value |
|---|---|
| Serum resistin with TnT | 0.046 |
| Serum adiponectin with TnI | 0.175 |

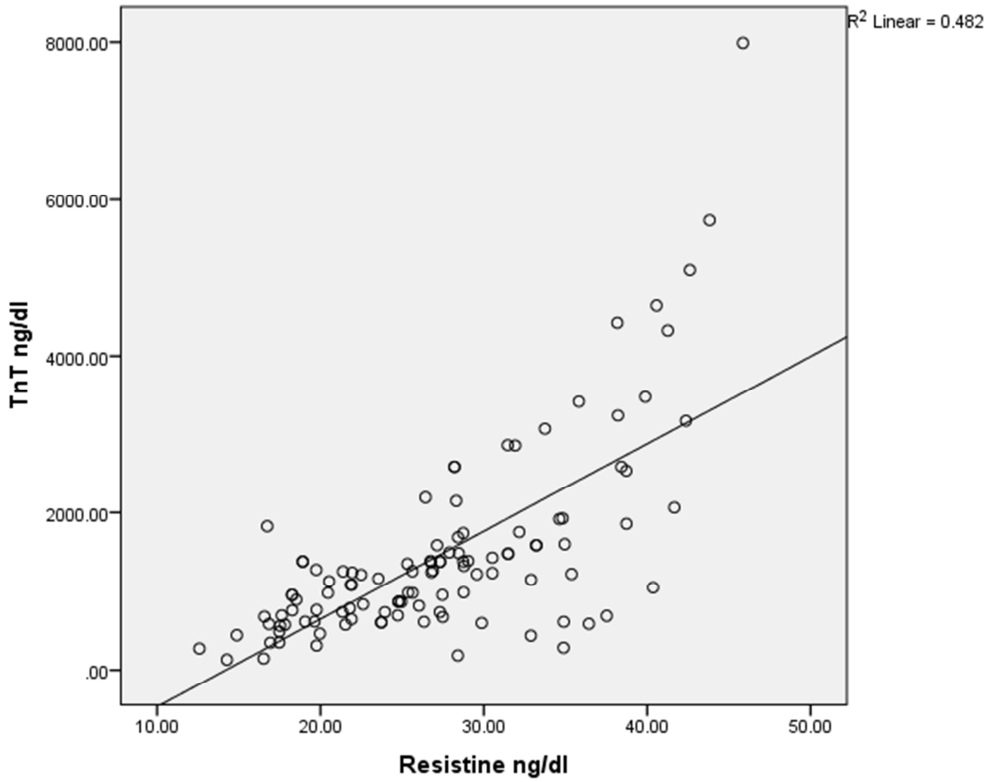


***Figure 1.*** *Significant positive correlation of resistin and cTnT in patients with STEMI.*

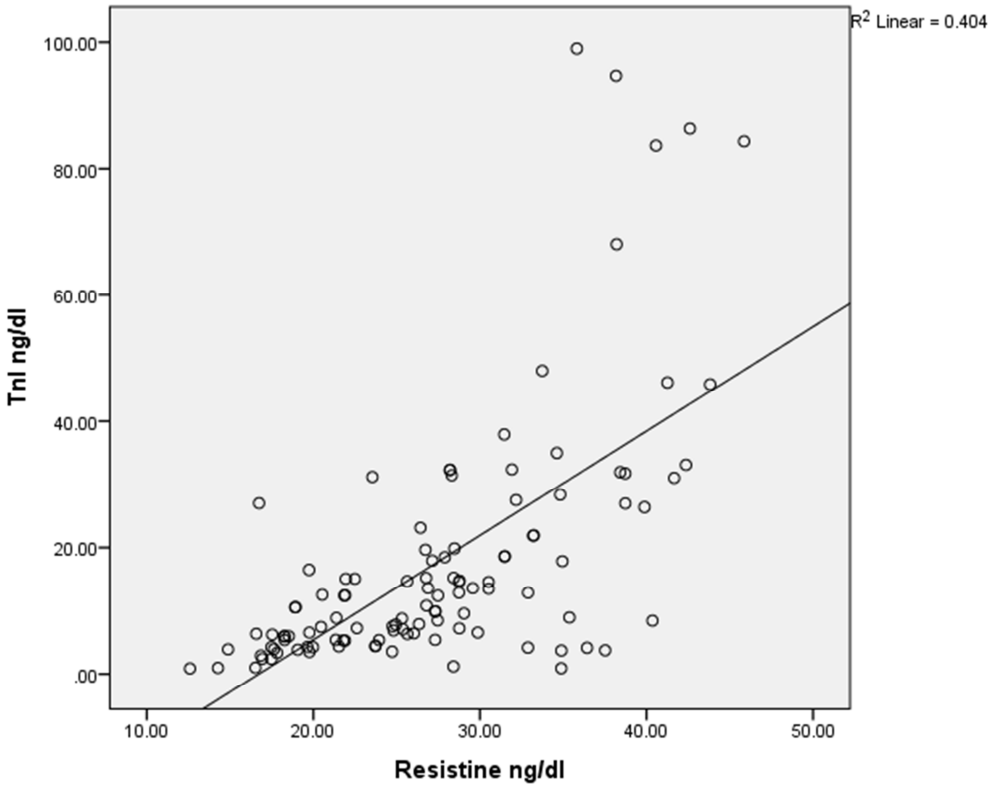


***Figure 2.*** *Significant positive correlation of resistin and cTnI in patients with STEMI.*

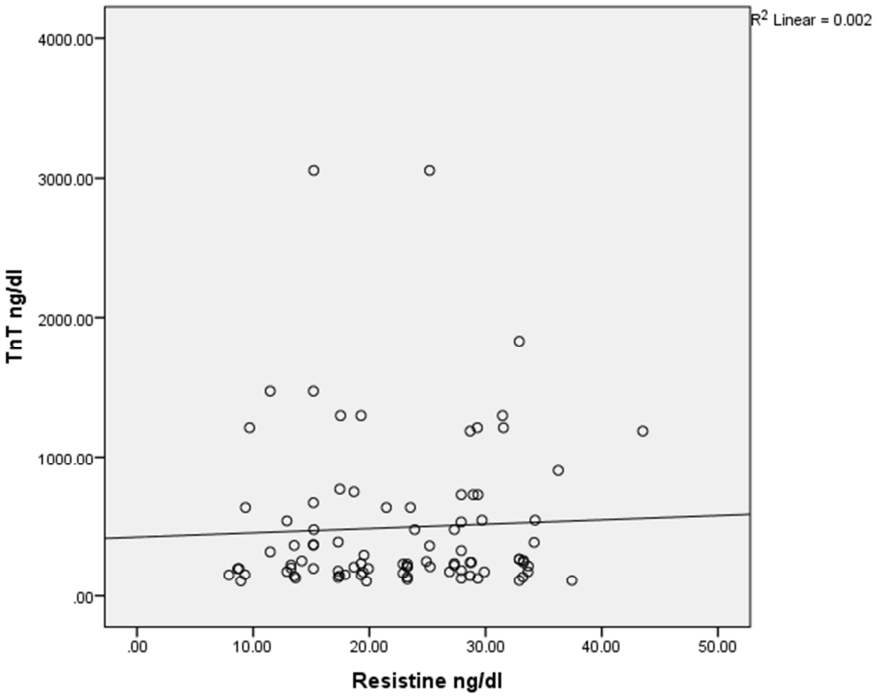


***Figure 3.*** *Positive correlation of resistin and cTnI in patients with NSTEMI.*

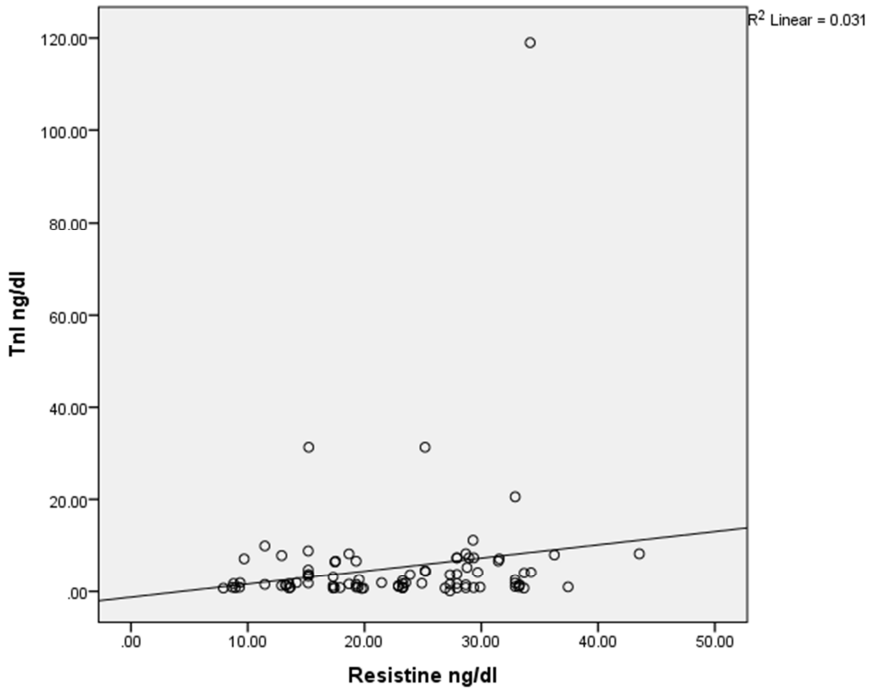


***Figure 4.*** *Positive correlation of resistin and cTnI in patients with NSTEMI.*

Correlation studies showed that significant positive correlation ($P<0.05$) of resistin with cTnT and cTnI was observed in patients with STEMI. However positive in-significant correlation of resistin with cTnT and cTnI was observed in patients with NSTEMI.

## 4. Discussion

According to our results, AMI is more prevalent in male with age > 50 years. Majority of patients are non-smoker but hypertensive. A study carried out on 484 Turkish male with first 3 to 6 hours of onset of symptom and found that AMI is more prevalent in age of more than 40 years (mean age 58 years) as compared to age less than 40 years onset were included in the study. [17].

Another study carried out on 100 ACS patients and revealed that hypertension is the furthermost known risk factor of developing ACS followed by type 2 diabetes and lastly smoking [18].

We observed that STEMI is more common in males as

compared to NSTEMI. We agreed with a study carried out in US that STEMI in male has higher incidence than males with NSTEMI [19]. According to Brazilian Society of Cardiology morality rate due to STEMI is higher than NSTEMI [20]. However a study observed that majority of patients was smoker while small number of patients was hypertensive and NSTEMI is more common than STEMI [18].

Consistent with studies of old age cardiac patients, the base line investigation showed that lipid profile along with fasting blood sugar and blood urea were significantly raised in cases as compared to control except serum creatinine. A study carried out 100 patients with ACS and found dyslipidemia in both patients with STEMI and NSTEMI and stated that dyslipidemia is taken as a strong marker for outcomes of cardiovascular problems after AMI [17].

Another study was carried out on 200 patients with ACS and found that along with cardiac enzymes, the levels of blood urea /BUN and serum creatinine are high as compared to controls. Study concluded that blood urea/BUN and Creatinine may be significant additional tools in death-risk valuation of ACS patients and may provide detailed interpretation of the complications of ACS [21]. However in our study only blood urea was increased with no effect on the level of serum creatinine.

A total of 529 patients with the diagnosis of STEMI related acute coronary syndrome within 24 h of the onset of symptoms were included in the study. Study proposed two hypothesis. One is based that hyperglycemia in ACS patients is persuaded by activation of alpha /beta adrenergic receptors. Another one is based that it may be a predictor of pre-existing, impaired metabolism of carbohydrate [22]. However it is reported that hyperglycemia is usually unnoticed in majority of AMI patients and the increase reason of mortality in ACS patients with less prognosis as compared to diabetics [23]. Another study suggested that hyperglycemia may be stress related in AMI and the level of blood glucose increased immediately after acute myocardial infarction regardless of diabetes status [23, 24].

We agreed with a study that observed a significantly high level of serum in patients with STEMI. Study also found no correlation of resistin with age, BMI, gender and fasting blood glucose. It is therefore suggested that level of serum resistin concentration may be a marker for diagnosis of STEMI [25]. Another study stated that high values of serum resistin may be presented as a predictor of injury of myocardium and inturn indicate the severity of AMI [6]. A study experimentally proved that high levels of resistin persuade resistance of leptin and increased the risk of progression of atherosclerosis [26].

It is proposed that resistin could stimulate endothelial cells and encourage the process of inflammation via chemokines and cytokines thus accelerating dysfunction of endothelium [27]. It also block the expression of endothelial nitric oxide encourage the production superoxide anion in endothelial cells, which decline the relaxation of endothelial-dependent vascular system and altered the process of relaxation and contraction in vessels of heart [28, 29].

A study was carried out on 100 hospitalized patients and found that 27% patients suffered with AMI and they have high values of serum resistin, which may be related with the severity of heart failure and at high risk of developing cardiac events signifying that high levels of serum resistin can estimate the forecasting of ailment and extent of heart problem and suggesting that reduction in the level of resistin may help in the treatment [6]. Additionally it is suggested that high levels of circulating resistin in patients with STEMI can be correlated with major hostile cardiac events [30]. In addition a cross sectional study on 92 patients with AMI also reported that resistin may control myocardial reperfusion injury by encouraging the process of inflammation and oxidative stress [31].

Correlation data showed that significant positive correlation of resistin was observed with cTnT and cTnI in STEMI as compared to NSTEMI patients. A study carried out AMI patients to find their levels of resistin and troponins and their correlation. Study reported that level of serum resistin is positively correlated with cardiac troponin I and suggested that values of serum resistin during the acute period of STEMI are valuable for predicting the size of myocardial infarction and prognosis in patients with ACS [32]. Another study also found the association of serum resistin with troponin I but reported that change in the level of serum resistin is not linked with injury of myocardium [26]. We agree with the study that was carried out on cardiac patients hospitalized in Finland. Study found that levels of resistin were increase significantly by 24 hour and positively correlated with release of troponin T [33]. However a study carried out on 153 old age patients and reported that resistin levels increased at 12.0 hours after the process but did not associate with increase values of troponins [15].

# 5. Conclusion

It is therefore concluded that increased level of resistin along with cardiac markers may be presented as a predictor of injury of myocardium and in turn indicate the severity of AMI in STEMI patients.

# Authors Contributions

Dr Shazia Rashid --- Study Design, Data collection
Prof Dr Javed Anver Qureshi----Study Design
Dr Rukhshan Khurshid ---- Study Design, Paper Writing
Dr Huma Ashraf---- Literature Survey
Dr Saima Rasheed---Statistical Analysis
Prof Uzma Faryal----- Literature Survey and Proof reading

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