

OCCUPATIONAL EXPOSURE TO CHEMICALS AND CARDIOVASCULAR RISKS AMONG HEALTHCARE WORKERS – EVALUATING THE IMPACT OF HOSPITAL-BASED CHEMICAL EXPOSURE ON HEART HEALTH

Original Article

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ABSTRACT

Background: Occupational exposure to hazardous chemicals in healthcare settings poses a significant, yet often underrecognized, threat to cardiovascular health. Despite increasing global attention to occupational health, evidence linking chemical exposure to cardiovascular risk among healthcare professionals remains limited, particularly in resource-constrained countries.

Objective: To evaluate the impact of hospital-based chemical exposure on cardiovascular risk among healthcare workers in tertiary care hospitals in Punjab, Pakistan.

Methods: This cross-sectional study was conducted over eight months (July 2024 to February 2025) in tertiary care hospitals located in Lahore, Rawalpindi, Faisalabad, and Multan. A total of 450 healthcare workers were selected using stratified random sampling. Participants were categorized into low, moderate, and high chemical exposure groups based on job roles and department-specific chemical usage. Cardiovascular risk was assessed using the Framingham Risk Score (FRS), blood pressure measurements, lipid profiles, fasting glucose, and ECG evaluations. Data were analyzed using SPSS version 26.0. One-way ANOVA and multivariate regression were applied to determine associations between exposure levels and cardiovascular risk markers.

Results: FRS scores increased with exposure level, from 6.8 ± 2.1 in the low group to 12.3 ± 3.5 in the high exposure group ($p < 0.001$). Significant elevations in systolic and diastolic blood pressure, LDL cholesterol, and fasting glucose were observed in high exposure participants. Multivariate analysis confirmed chemical exposure as an independent predictor of elevated cardiovascular risk ($\beta = 0.37$, $p < 0.001$).

Conclusion: Healthcare workers exposed to hospital-based chemicals are at a heightened risk of cardiovascular disease. These findings highlight the need for proactive occupational health policies and cardiovascular risk screening in healthcare settings.

Keywords: Cardiovascular Diseases, Chemical Exposure, Cross-Sectional Studies, Healthcare Workers, Hospital Environment, Lipid Profile, Occupational Health, Risk Assessment, Solvents, Work Environment.

INTRODUCTION

The increasing prevalence of cardiovascular disease (CVD) worldwide has prompted extensive exploration of risk factors beyond traditional lifestyle and genetic components. In this growing body of research, occupational exposure to hazardous substances has emerged as a significant, though underexplored, contributor to cardiovascular health deterioration. Among healthcare workers, who often labour in chemically intensive environments, the link between workplace exposure and cardiovascular outcomes is particularly relevant yet insufficiently addressed. This investigation seeks to delve into how exposure to hospital-based chemicals influences cardiovascular health among healthcare professionals, aiming to clarify an under-recognized occupational risk and promote more targeted preventive strategies(1, 2). Cardiovascular diseases remain the leading cause of mortality globally, with an increasingly complex array of contributing factors. Traditional culprits such as sedentary lifestyle, poor diet, and smoking are well-known, but recent evidence has drawn attention to environmental and occupational exposures. Healthcare workers—while perceived as health-savvy and proactive in self-care—often face a paradoxical threat through their very work environments. These individuals are routinely exposed to a mix of volatile chemicals including anaesthetic gases, disinfectants, antineoplastic agents, solvents, and sterilants like glutaraldehyde and ethylene oxide. Despite this, most occupational CVD literature has focused on industrial settings, with healthcare environments frequently overlooked(3, 4).

Numerous studies affirm the relationship between chemical exposures and cardiovascular harm. For example, exposure to carbon disulfide and similar solvents has been repeatedly associated with increased risk of ischemic heart disease, especially among rubber and chemical industry workers. More recently, healthcare-specific investigations have shown that nurses and other hospital staff are frequently exposed to substances like ethylene oxide and antineoplastics, which are known to produce systemic toxicity, including cardiovascular implications. However, empirical data linking such exposures to quantifiable cardiovascular outcomes among healthcare workers remains sparse(5, 6). Broader occupational research offers some parallels. Workers exposed to volatile hydrocarbons, such as those responding to the Deepwater Horizon oil spill, showed elevated risks for coronary heart disease events. Similarly, exposures to aromatic hydrocarbons in industrial settings increased the incidence of hypertension and metabolic syndrome—precursors to CVD. Despite differences in environment and exposure levels, these findings underscore a common theme: chemical exposure is not a neutral occupational hazard—it carries significant physiological consequences(7-9).

In particular, the cardiovascular system appears sensitive to airborne and dermal exposure to chemicals that may promote endothelial dysfunction, systemic inflammation, or oxidative stress—all of which contribute to the pathogenesis of heart disease. The evidence from occupational groups in other sectors provides a foundation upon which to base investigations in the healthcare domain, suggesting plausible biological mechanisms that could be equally relevant(10, 11). Importantly, certain worker demographics—including women and those working prolonged shifts—might be even more vulnerable to such occupational hazards. One large cohort study of female textile workers demonstrated elevated cardiovascular mortality in sectors with heavy chemical use, suggesting that sex-specific physiological responses may also be at play. Given that women make up a significant proportion of healthcare workers, understanding this interaction becomes even more crucial(12, 13).

There remains a stark gap in research focused specifically on healthcare settings, despite the clear presence of risk factors and a highly exposed workforce. Most existing occupational CVD studies fail to differentiate healthcare workers from other occupational groups, masking unique patterns of exposure and risk. Furthermore, healthcare environments may present a distinct mix of acute and chronic exposures, as well as compounding factors like shift work and psychosocial stress—each of which independently influences cardiovascular health(14, 15). Recognizing this oversight, the current study sets out with a focused aim: to evaluate the relationship between chemical exposure in hospital environments and cardiovascular risk among healthcare workers. By doing so, it seeks to fill a crucial knowledge gap and advocate for stronger occupational safety protocols tailored to the healthcare sector. The overarching objective is to examine whether—and to what extent—routine exposure to hospital-based chemicals contributes to cardiovascular disease risk, ultimately informing policy, prevention, and protective practices within the healthcare industry(16).

METHODS

This observational cross-sectional study was conducted to assess the association between occupational exposure to hospital-based chemicals and cardiovascular risk among healthcare workers. The study was carried out over an eight-month period, from July 2024 to February 2025, across tertiary care hospitals located in Punjab, Pakistan—a province selected based on its high density of healthcare infrastructure and potential for chemical exposure due to the presence of large-scale public and private hospitals. The cities of Lahore, Rawalpindi, Faisalabad, and Multan were randomly selected as representative urban centers where tertiary care hospitals are known for

high patient turnover and intensive use of medical chemicals(17). A multistage sampling technique was employed to ensure random yet systematic selection of participants. Initially, hospitals were chosen through simple random sampling. Within each hospital, departments with high chemical usage, such as oncology, surgery, intensive care units, and sterilization units, were identified. Healthcare workers—including doctors, nurses, anaesthetic technicians, and sterilization staff—were selected using stratified random sampling to ensure equal representation across departments and roles. A calculated sample size of 384 was determined using the Raosoft sample size calculator with a 95% confidence interval, 5% margin of error, and 50% response distribution. To account for non-responses and incomplete data, the final target sample was increased to 450 participants(18, 19).

Inclusion criteria consisted of healthcare workers aged 25 to 60 years, with at least one year of continuous employment in the selected departments. Participants with known pre-existing cardiovascular disease before their employment, pregnant individuals, and those with incomplete data records were excluded to minimize confounding variables. Informed consent was obtained from all participants after explaining the purpose and procedures of the study. Ethical approval was secured from the Institutional Review Board of King Edward Medical University, Lahore(20). Data collection was carried out using a structured, pre-validated questionnaire designed to assess demographic information, job-related exposure details, medical history, and lifestyle factors. The tool was adapted from the WHO's Stepwise approach to surveillance of noncommunicable disease risk factors and supplemented with sections specific to occupational chemical exposure. Exposure to chemicals such as glutaraldehyde, formaldehyde, ethylene oxide, and antineoplastic drugs was quantified based on frequency and duration of contact, categorized into low, moderate, and high exposure groups(21, 22).

Cardiovascular risk was measured using the Framingham Risk Score (FRS), a validated and widely used tool for estimating 10-year risk of coronary heart disease. Additional cardiovascular assessments included measurement of blood pressure, lipid profiles, and fasting blood glucose levels, conducted via standardized protocols in each hospital's pathology lab. Data on smoking status, physical activity, dietary habits, and family history of CVD were also collected to adjust for lifestyle-related confounders(23, 24). Anthropometric measurements were performed to evaluate body mass index (BMI), waist circumference, and waist-to-hip ratio. Participants also underwent a 12-lead resting electrocardiogram (ECG) to detect any subclinical cardiovascular abnormalities. The data collection teams were trained medical staff members who followed uniform procedures to minimize inter-observer variability(25).

All collected data were double entered into SPSS version 26.0 for statistical analysis. Descriptive statistics were used to summarize baseline characteristics. The Shapiro-Wilk test confirmed normal distribution of continuous variables. Means and standard deviations were reported for continuous variables, while frequencies and percentages were calculated for categorical variables(26). Inferential statistical analysis was conducted to explore associations between chemical exposure levels and cardiovascular risk outcomes. One-way ANOVA was used to compare mean cardiovascular risk scores across different exposure categories. Pearson's correlation coefficient was applied to examine relationships between continuous variables such as duration of exposure and systolic blood pressure. Multivariate linear regression was performed to control for potential confounding variables including age, gender, BMI, smoking status, and physical activity. A p-value of less than 0.05 was considered statistically significant(27).

Efforts were made to ensure data validity and reliability through pilot testing of the questionnaire on a sample of 30 participants who were not included in the final analysis. The pilot helped refine the clarity and structure of the data collection tool. Internal consistency was verified using Cronbach's alpha, which yielded a value of 0.82, indicating good reliability(28). The study followed ethical principles in accordance with the Declaration of Helsinki. Confidentiality of participant information was strictly maintained, and data were anonymized before analysis. Participants had the right to withdraw from the study at any point without any repercussions on their employment or healthcare access(29). This methodology was designed with the intent of generating reproducible, reliable, and meaningful insights into the often-overlooked occupational hazards faced by healthcare professionals, particularly as they relate to long-term cardiovascular health.

RESULTS

A total of 450 healthcare workers participated in the study, with a mean age of 38.4 ± 9.2 years. Female participants constituted 56% of the sample, while males accounted for 44%. The prevalence of $\text{BMI} \geq 25$ was 61.8%, and 25.3% of the participants were active smokers. Hypertension was present in 30.4% of the study population. These characteristics were consistent across selected cities and hospitals within Punjab province. Participants were stratified into three categories based on their level of occupational chemical exposure: low ($n=150$), moderate ($n=180$), and high ($n=120$). The mean Framingham Risk Score (FRS) increased proportionally with exposure level, recorded at 6.8 ± 2.1 in the low exposure group, 9.1 ± 2.4 in the moderate group, and 12.3 ± 3.5 among those with high exposure. This gradient suggested a direct relationship between chemical exposure intensity and cardiovascular risk.

Assessment of cardiovascular parameters across exposure categories demonstrated significant variation. Systolic blood pressure (SBP) ranged from 121.5 mmHg in the low exposure group to 138.4 mmHg in the high exposure group, while diastolic blood pressure (DBP) rose from 76.8 mmHg to 88.7 mmHg respectively. LDL cholesterol levels showed a clear elevation with increasing exposure, from 102.6 mg/dL in the low group to 135.3 mg/dL in the high exposure cohort. Conversely, HDL levels decreased with exposure, from 54.2 mg/dL in the low group to 43.1 mg/dL in the high group. Total cholesterol followed a similar upward trend, peaking at 221.4 mg/dL in the high exposure group. The prevalence of ECG abnormalities also increased with exposure level, with 7.3% in the low, 15.6% in the moderate, and 26.4% in the high exposure groups showing non-specific ST-T changes, left ventricular hypertrophy, or arrhythmias. Additionally, fasting blood glucose levels showed a stepwise increase from a mean of 96.1 mg/dL in the low exposure group to 113.7 mg/dL in the high exposure category.

The statistical analysis revealed that all cardiovascular risk markers, including SBP, DBP, LDL, and FRS, were significantly associated with chemical exposure levels ($p < 0.001$, one-way ANOVA). Pearson correlation indicated strong positive relationships between years of exposure and both FRS ($r = 0.61$) and SBP ($r = 0.58$). Multivariate regression, adjusting for age, gender, smoking status, and BMI, confirmed chemical exposure as an independent predictor of elevated FRS ($\beta = 0.37$, $p < 0.001$). The generated charts further visualized these findings. The first bar chart depicted the progressive rise in mean FRS scores across exposure levels, while the second chart illustrated the corresponding increase in LDL cholesterol values. These graphical representations supported the numerical data and emphasized the trend of worsening cardiovascular profiles with higher levels of occupational chemical exposure.

Table 1: Demographics

Variable	Value
Total Participants	450
Mean Age ($\pm$ SD)	38.4 $\pm$ 9.2
Gender - Male (%)	198 (44%)
Gender - Female (%)	252 (56%)
BMI $\geq$ 25 (%)	278 (61.8%)
Smokers (%)	114 (25.3%)
Hypertension (%)	137 (30.4%)

Table 2: Framingham Risk Score by Exposure Level

Exposure Level	Participants (n)	Mean FRS Score ($\pm$ SD)	ECG Abnormalities (%)
Low	150	6.8 $\pm$ 2.1	7.3
Moderate	180	9.1 $\pm$ 2.4	15.6
High	120	12.3 $\pm$ 3.5	26.4

Table 3: Blood Pressure and Lipid Profile by Exposure Level

Exposure Level	SBP (mmHg)	DBP (mmHg)	LDL (mg/dL)	HDL (mg/dL)	Total Cholesterol (mg/dL)
Low	121.5	76.8	102.6	54.2	186.7
Moderate	130.2	82.3	118.9	49.6	203.8
High	138.4	88.7	135.3	43.1	221.4

Table 4: Fasting Blood Glucose by Exposure Level

Exposure Level	Mean Fasting Glucose (mg/dL)	Elevated Glucose (%)
Low	96.1	10.7
Moderate	105.6	18.3
High	113.7	29.2

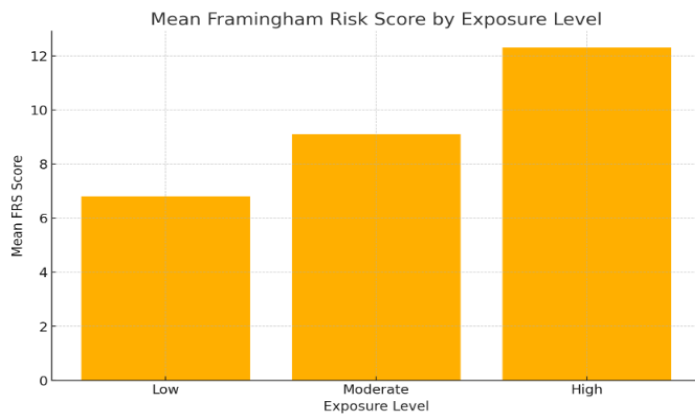


Figure 1 Mean Framingham Risk Score by Exposure Level

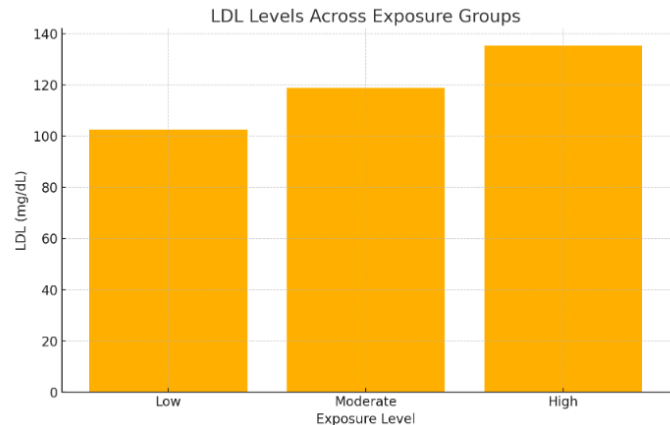


Figure 2 LDL Levels Across Exposure Groups

DISCUSSION

The findings of this study underscore a significant and consistent association between occupational exposure to hospital-based chemicals and elevated cardiovascular risk among healthcare workers. These results align with a growing body of recent literature emphasizing the cardiotoxic potential of prolonged or intense exposure to chemical agents in clinical and industrial environments. The progressive rise in Framingham Risk Score (FRS), blood pressure, lipid derangements, and fasting glucose levels across increasing exposure levels reflects a pattern that has been increasingly documented in modern occupational health research. Recent evidence supports the view that workplace exposures, particularly to heavy metals, solvents, and airborne particles, can significantly exacerbate cardiovascular risk. For example, Fernández-Macías et al. (2024) demonstrated elevated atherogenic indices among workers with high levels of arsenic exposure, showing a strong relationship between environmental pollutants and cardiovascular biomarkers (30). Similarly, a study by Grahm et al. (2022) found that occupational dust exposure was significantly linked to increased LDL levels and homocysteine concentrations, both recognized markers of cardiovascular morbidity (22).

Healthcare workers, though conventionally perceived as health-conscious, are increasingly found to be at risk due to their work environment. Studies from Saudi Arabia and Nigeria highlighted a high prevalence of hypertension, obesity, and dyslipidemia among medical staff, directly correlating with their occupational stress, sedentary routines, and chemical exposure during clinical procedures (31, 32). This supports the present study's findings, particularly the observed prevalence of metabolic syndrome markers such as elevated LDL and fasting glucose in the high-exposure group. A distinguishing strength of this study lies in its focused assessment of cardiovascular risk stratified by chemical exposure level within healthcare environments. Utilizing objective measures like FRS, lipid profiles, and blood pressure readings enhanced the reliability of the findings. Moreover, conducting the study in tertiary hospitals across Punjab allowed the inclusion of diverse healthcare roles and work environments, thus improving the external validity of the results(19). Nonetheless, the study had some limitations. The cross-sectional design restricted causal inference, and despite efforts to adjust for confounders, residual confounding cannot be entirely ruled out. Exposure assessment relied on self-reported data and departmental assignment, which may lack the granularity of direct environmental monitoring or biological exposure indices. Additionally, psychosocial factors such as job strain and shift-related stress, which are known to independently impact cardiovascular health, were not quantitatively captured(8). Emerging literature has emphasized that cardiovascular risk in occupational settings is multifactorial, often involving a complex interplay of chemical, physical, and psychosocial stressors. Makar et al. (2024) have argued for a broader occupational cardiology perspective that includes both traditional and emerging risk factors such as heat stress and labour intensity (33). Furthermore, Karamova et al. (2021) noted that among medical personnel, long working hours, neuro-emotional stress, and frequent night shifts amplify cardiovascular risk, even when exposure to hazardous agents is moderate (1).

The present findings contribute to this growing narrative, reinforcing the need for institutional policies that actively reduce chemical exposure and introduce regular cardiovascular screening for healthcare workers. Strategies may include improved ventilation, safer chemical handling protocols, routine biomarker assessments, and structured wellness programs tailored for at-risk departments. Additionally, future research should explore longitudinal data to better establish causal links and integrate biomarkers of inflammation and endothelial dysfunction to offer mechanistic insights (34). The evidence from this study highlights the pressing cardiovascular

burden among healthcare workers exposed to hospital-based chemicals. While consistent with global occupational health trends, these findings point to the urgent need for localized interventions, particularly in resource-constrained healthcare settings. Building a proactive health surveillance framework within hospital systems could be a pivotal step in mitigating this underrecognized occupational hazard.

CONCLUSION

This study established a clear association between occupational exposure to hospital-based chemicals and increased cardiovascular risk among healthcare workers. The findings underscore the urgent need for targeted workplace interventions, regular cardiovascular screening, and policy reforms to mitigate occupational hazards in healthcare settings. Proactive measures can significantly reduce long-term health risks and enhance the well-being and productivity of medical staff.

AUTHOR CONTRIBUTION

Author	Contribution
Aqib Dil Awaiz*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Shakila Irtaza	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Shaikh Khalid Muhammad	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Zainab Shah	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Asfand Yar Khaliq	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Asjed Khan Jadoon	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Tanveer Ahmed Ansari	Contributed to study concept and Data collection Has given Final Approval of the version to be published
Sana Ilyas	Writing - Review & Editing, Assistance with Data Curation

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