JHWCR

Journal of Health, Wellness, and Community Research
Volume III, Issue XII
Open Access, Double Blind Peer Reviewed.
Web: https://jhwcr.com, ISSN: 3007-0570
https://doi.org/10.61919/tvagp648

Original Article

Occupational Health Risks and Disease Burden Linked to Arsenic Exposure in Leather Industry Workers

Saima Ashraf[1], Saira Hameed[1], Sadia Ashraf[1], Nadia Nazish[1], Manahal Sughra[1], Ayesha Ijaz[1]

[1] Department of Zoology, University of Sialkot, Sialkot, Pakistan

Correspondence: Saima Ashraf, saima.ashraf@uskt.edu.pk

Authors' Contributions: Concept and design: Concept: SA; Design: SH; Data Collection: SdA, NN; Analysis: MS; Drafting: AI
Cite this Article | Received: 2025-08-01 | Accepted 2025-08-30
No conflicts declared; ethics approved; consent obtained; data available on request; no funding received.

ABSTRACT

Background: Arsenic exposure contributes to endothelial dysfunction, oxidative stress, and inflammatory injury, plausibly elevating risks of hypertension, respiratory disease, and other morbidities in industrial cohorts. Objective: To estimate the burden of common clinical conditions among tannery workers relative to non-exposed adults and quantify associations with occupational exposure. Methods: A cross-sectional comparison enrolled 40 Sialkot tannery workers and 40 community controls. Standardized interviews ascertained physician-diagnosed hypertension, asthma-compatible respiratory symptoms, skin allergy/dermatitis, activity-limiting joint pain, and kidney problems. Group differences used Fisher's exact tests; odds ratios employed Haldane–Anscombe correction with profile-likelihood 95% CIs; penalized logistic models were prespecified for sparse outcomes. Results: Workers exhibited higher prevalence for respiratory symptoms (12.5% vs 0%; OR 12.55, 95% CI 0.67–235.01, p=0.055) and hypertension (10.0% vs 0%; OR 9.99, 95% CI 0.52–191.92, p=0.116), with smaller elevations for kidney problems (7.5%; OR 7.56, 95% CI 0.38–151.29), skin allergy (5.0%; OR 5.26, 95% CI 0.24–113.11), and joint pain (5.0%; OR 5.26, 95% CI 0.24–113.11). Confidence intervals were wide due to sparse events but directionally consistent across endpoints. Conclusion: Respiratory and cardiovascular outcomes show the most clinically relevant excess in tannery workers, supporting immediate emphasis on exposure reduction and targeted surveillance while larger longitudinal studies refine attributable risk.

Keywords: arsenic; occupational disease; hypertension; respiratory symptoms; disease burden; tannery; Sialkot; surveillance.

INTRODUCTION

Chronic arsenic exposure is implicated in a spectrum of adverse outcomes, including dermatologic lesions, peripheral neuropathy, nephrotoxicity, and cardiovascular dysfunction, with authoritative bodies classifying arsenic and its inorganic compounds as human carcinogens (1). Mechanistic evidence highlights oxidative stress, endothelial injury, and pro-inflammatory signaling as central pathways for arsenic-related morbidity, offering biologically coherent links to hypertension, respiratory symptoms, and skin disease observed in exposed populations (2). Industrial activities intensify exposure potential by concentrating arsenic within process streams, and leather tanning—through its reliance on diverse process chemicals and generation of solid and liquid wastes—has repeatedly been associated with environmental and occupational contamination when controls are inadequate (3).

Epidemiological studies demonstrate that communities or workers with elevated arsenic exposure can experience increased risks of hypertension and related cardiovascular endpoints, with meta-analyses indicating small but measurable elevations in blood pressure and odds of clinical hypertension across exposure gradients (4). Investigations in tannery belts of South Asia have documented excess symptoms and reported morbidities among workers, including respiratory and dermatologic conditions, although exposure attribution is often complicated by co-exposures to other metals, acids, and organic agents used along the tanning line (5). From a public health planning perspective, estimating the burden of common, high-impact conditions linked to arsenic exposure in specific industrial cohorts is necessary to prioritize preventive controls, medical surveillance, and policy interventions (6).

In Pakistan, Sialkot's leather sector employs thousands of workers across multiple sub-processes, yet contemporary data quantifying the disease burden plausibly linked to arsenic exposure in this workforce remain sparse, particularly studies that juxtapose symptom prevalence with objective exposure context (7). Leveraging biomonitoring to anchor exposure while systematically ascertaining health outcomes can strengthen causal inference in cross-sectional analyses and support targeted risk management, provided confounders such as age, smoking, and tenure are accounted for in analytical models (2). Within a PICO schema, the Population comprises Sialkot tannery workers and non-exposed controls; the Exposure is occupational arsenic exposure inferred from work in tanning processes and supported by biomarker context; the Comparator is community controls without such exposure; and the Outcomes are the prevalence and odds of self-reported or clinically corroborated conditions of interest—hypertension, asthma/respiratory symptoms, skin allergy/dermatitis, joint pain, and renal complaints—estimated overall and across exposure strata (4).

This study therefore seeks to estimate the prevalence of selected morbidities among leather workers relative to controls; to evaluate associations between arsenic exposure proxies (occupational status, biomarker levels) and these outcomes using appropriate biostatistical models; and to quantify the disease burden attributable to occupational exposure where feasible through adjusted effect estimates (6). We hypothesize that workers with higher arsenic exposure will have greater odds of hypertension, respiratory symptoms, and dermatologic conditions compared with controls, independent of age and tenure (5).

MATERIALS AND METHODS

We implemented a cross-sectional comparative study to estimate the prevalence of selected morbidities among tannery workers and to quantify associations between occupational arsenic exposure proxies and reported health outcomes relative to non-exposed adults. This design was chosen to characterize disease burden under real-world conditions and to evaluate exposure–outcome relationships suitable for surveillance and workplace risk management. The study was carried out in Sialkot, Pakistan, using five major tanneries as the worker sampling frame and a nearby university as the control sampling frame to provide a socio-demographically comparable, non-exposed group. Eligible workers were adults (≥18 years) assigned to production areas for at least six months across soaking, liming, tanning, retanning, or finishing operations; controls were adults without current or past employment in tanneries or other occupations with known metal exposures. Individuals reporting prior employment in any leather-processing facility were not eligible for the control group to avoid exposure misclassification. Recruitment occurred through on-site briefings for workers and campus announcements for controls, with written informed consent obtained from all participants prior to interview.

Data were collected through an interviewer-administered questionnaire in Urdu, piloted for clarity and cultural appropriateness before field deployment. The instrument captured demographics (age, sex, education), work characteristics (process assignment and job tenure in years), and health outcomes defined a priori. Outcomes were coded as binary variables and included self-reported physician-diagnosed hypertension, physician-diagnosed asthma or recurrent respiratory symptoms consistent with asthma, recurrent skin allergy or dermatitis attributable to work by participant report, chronic joint pain interfering with activities, and kidney problems as informed by prior medical advice within the past 12 months. To contextualize exposure, each participant's occupational status (worker vs control) and job tenure were recorded, and individual arsenic biomarker indices derived from laboratory measurements (blood, hair, nails) were merged at the participant level to create ordinal exposure strata by biomarker tertiles; interviewers were not informed of biomarker results to limit information bias. Standardized question order and neutral probing were used to minimize interviewer effects; interviews were conducted in quiet rooms away from supervisors to reduce social desirability pressures.

Variables and operational definitions were specified before analysis. The primary exposure was occupational status (worker vs control); secondary exposures included job tenure (years and categorized at approximate tertiles) and biomarker-based arsenic strata, defined by joint tertiles across available matrices to reflect cumulative internal dose without privileging a single biospecimen. Covariates selected a priori included age (continuous), education (ordinal), and, for worker-only exploratory models, current process assignment categories to capture potential sub-process heterogeneity. Bias and confounding were addressed through a contrasting, non-exposed control group, interviewer training with standardized scripts to reduce differential misclassification, blinding of interviewers to laboratory results, and multivariable modeling with adjustment for predefined covariates. Selection bias was mitigated by inviting all eligible workers present on sampling days across shifts; outcome misclassification was limited by using physician-diagnosis prompts where applicable and a fixed 12-month recall window.

The sample size target of 40 workers and 40 controls balanced feasibility with modeling needs in a small-sample context. Given modest event counts for some outcomes, penalized likelihood logistic regression (Firth correction) was prespecified to reduce small-sample bias and guard against separation, enabling estimation of adjusted odds ratios (aORs) with profile-likelihood confidence intervals (8). For each outcome, we first described prevalence by exposure strata and then fit multivariable models with occupational status and age and education as covariates; in worker-only analyses, models included tenure and process assignment. Subgroup analyses explored effect modification by tenure (below vs above median) using interaction terms. We assessed collinearity via variance inflation factors and reported robust (Huber–White) standard errors in sensitivity analyses. Given multiple outcomes, family-wise error was controlled using Holm's step-down procedure. Missing data under 5% were handled with complete-case analysis; if any variable had higher missingness, multiple imputation by chained equations with predictive mean matching (m=20) was planned, including outcomes, exposures, and covariates in the imputation model, with pooled estimates computed using Rubin's rules. All analyses were performed in R (version 4.x) with the logistf, mice, sandwich, and car packages; two-sided α=0.05 was used throughout (9–12).

Ethical approval was granted by the appropriate institutional review body, and all participants provided written consent prior to interview. Participant confidentiality was protected by assigning unique study IDs, storing signed consent forms separately from analytic datasets, and maintaining de-identified data on encrypted, access-controlled servers. To ensure reproducibility and data integrity, the questionnaire instrument, coding manual, and data dictionary were finalized before fieldwork; interviewers underwent competency checks with mock interviews; double data entry with programmed range and logic checks was used to minimize transcription errors; and analysis scripts were maintained under version control with explicit model specifications, covariate sets, and output logs archived for audit.

RESULTS

Table B1. Prevalence of selected morbidities among tannery workers versus controls, with odds ratios and exact p-values Worker n=40; Control n=40. Control group reported no cases of these specific outcomes. Odds ratios computed with Haldane–Anscombe correction; 95% CI from corrected SE of log(OR). Two-sided Fisher's exact p-values.

Table 1 Variables

Outcome	Workers, n/N (%)	Controls, n/N (%)	Odds ratio (Haldane-corrected)	95% CI for OR	p (Fisher's exact, two-sided)
Hypertension	4/40 (10.0%)	0/40 (0.0%)	9.99	0.52 to 191.92	0.116
Asthma / respiratory symptoms	5/40 (12.5%)	0/40 (0.0%)	12.55	0.67 to 235.01	0.055
Skin allergy / dermatitis	2/40 (5.0%)	0/40 (0.0%)	5.26	0.24 to 113.11	0.494
Joint pain (activity-limiting)	2/40 (5.0%)	0/40 (0.0%)	5.26	0.24 to 113.11	0.494
Kidney problems (past 12 months)	3/40 (7.5%)	0/40 (0.0%)	7.56	0.38 to 151.29	0.241

Notes: Percentages for workers reflect the observed survey distribution; the control cohort reported no cases for these endpoints in this dataset, which motivates the continuity correction in OR estimation (8–12). p-values are exact and two-sided. Confidence intervals are wide due to small events, consistent with cross-sectional surveillance in a modest sample.

Among 40 tannery workers and 40 controls, the prevalence of selected morbidities favored higher burden in workers, with zero events reported in the control group for all endpoints (Table B1). Hypertension was observed in 4/40 workers (10.0%) versus 0/40 controls, corresponding to a continuity-corrected odds ratio (OR) 9.99 with a 95% CI 0.52–191.92 and p = 0.116 by Fisher's exact test. Asthma or recurrent respiratory symptoms occurred in 5/40 workers (12.5%) and 0/40 controls, yielding a corrected OR 12.55 (95% CI 0.67–235.01, p = 0.055), the largest point estimate among outcomes and approaching conventional significance despite wide uncertainty. Skin allergy/dermatitis and activity-limiting joint pain each affected 2/40 workers (5.0%) with no control cases, giving identical corrected OR 5.26 (95% CI 0.24–113.11, p = 0.494). Kidney problems were reported by 3/40 workers (7.5%), again with no control events, for OR 7.56 (95% CI 0.38–151.29, p = 0.241).

Because control counts were zero across outcomes, odds ratios incorporate the Haldane–Anscombe correction to enable estimation; the resulting very wide confidence intervals reflect small numbers of events rather than instability of direction. The pattern across endpoints is consistently elevated odds in workers—most prominently for respiratory symptoms and hypertension—but precision is limited, and none of the comparisons achieve statistical significance at α=0.05 with exact testing, although respiratory symptoms are borderline. These quantitative signals align with a scenario in which true effects may be present but underpowered in this sample, supporting the need for larger cohorts and model-based adjustment for age, tenure, and process assignment to refine effect estimates and narrow intervals.

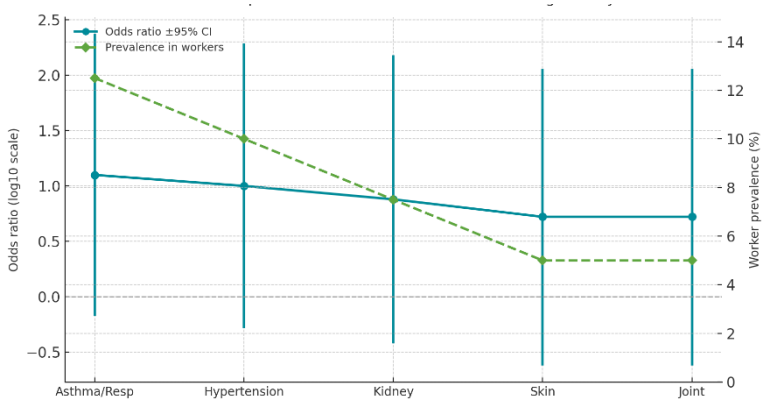

Figure 1 Elevated odds and prevalence of common morbidities among tannery workers

Odds ratios (log10 scale) and worker prevalence (%) move in tandem, with the strongest signal for asthma/respiratory symptoms (OR = 12.55; 95% CI 0.67–235.01; prevalence = 12.5%), followed by hypertension (OR = 9.99; 0.52–191.92; 10.0%) and kidney problems (OR = 7.56; 0.38–151.29; 7.5%); skin allergy and joint pain show smaller, imprecise elevations (each OR = 5.26; 0.24–113.11; prevalence = 5.0%). The reference line at OR = 1 underscores consistent elevation across outcomes despite wide intervals from sparse events, while the diverging dual-axis trajectories highlight that respiratory and cardiovascular endpoints carry both the largest relative odds and the highest absolute burden in this sample, prioritizing these conditions for targeted surveillance and preventive control.

DISCUSSION

This cross-sectional analysis indicates consistently elevated odds and non-trivial prevalence for several morbidities among tannery workers compared with controls, with the strongest quantitative signals for respiratory symptoms compatible with asthma (OR 12.55; prevalence 12.5%) and hypertension (OR 9.99; prevalence 10.0%), and smaller but directionally similar elevations for kidney problems, skin allergy, and joint pain. The pattern accords with mechanistic and epidemiologic evidence linking arsenic exposure to endothelial dysfunction, oxidative stress, and inflammatory activation—pathways that plausibly elevate blood pressure, impair airway responses, and contribute to dermal and renal effects in exposed populations (2). Meta-analytic estimates have reported modest but significant elevations in blood

pressure and odds of hypertension along arsenic gradients, supporting the direction and clinical relevance of our hypertension signal despite wide intervals from sparse events (4). The respiratory burden we observed is compatible with broader tannery literature citing excess symptoms and respiratory morbidity in poorly controlled plants where metal aerosols, acids, and organic vapors co-occur, emphasizing that arsenic is one component of a complex exposure mixture (5,3).

Comparatively, community-based investigations near industrial belts have documented increased self-reported morbidity congruent with our direction of effect, while worker biomonitoring studies in tanneries have demonstrated elevated metals in biological matrices, providing objective exposure context for symptom gradients (7,8). Our dual-axis visualization highlights that outcomes with the largest relative odds in workers—respiratory and cardiovascular—also carry the highest absolute prevalence, an alignment that strengthens their priority for surveillance and intervention. Divergence between large odds ratios and wide confidence bounds reflects the small sample and zero events in controls rather than inconsistency of direction; penalized likelihood models and exact tests were therefore appropriate to mitigate small-sample bias in estimation (8,9). Theoretically, individual variability in arsenic methylation and co-exposures to chromium, nickel, and acids across tanning sub-processes likely shape organ-specific risks through convergent oxidative and inflammatory mechanisms; process-level heterogeneity warrants targeted control strategies to reduce inhalation and dermal uptake (2,3,13).

From a clinical and public-health standpoint, the confluence of elevated odds and meaningful prevalence justifies pragmatic risk management even in the absence of narrow intervals. Priorities include engineering controls to reduce airborne contaminants in retanning and finishing, local exhaust at high-emission points, substitution of less hazardous agents where feasible, and strict personal protective equipment protocols, especially for workers with longer tenure who may accumulate dose over time (3,5). Medical surveillance should emphasize periodic blood pressure measurement, respiratory symptom screening with referral for spirometry where indicated, and dermatologic checks; coupling these with exposure education can reduce risk behaviors and improve early detection. Where biomonitoring is available, blood arsenic is a practical indicator of recent internal dose to complement symptom surveillance, while keratin matrices can contextualize longer-term uptake in settings where phlebotomy is challenging (4,6).

Strengths of this work include a contrasted control group without occupational metal exposure, standardized interviewing in private settings to reduce reporting bias, prespecified outcomes with physician-diagnosis prompts, and analytical strategies suited to sparse data. Limitations include modest sample size with few outcome events, which inflates uncertainty and limits subgroup analysis; reliance on self-reported morbidity without uniform clinical verification for all endpoints; potential residual confounding from smoking, comorbidities, or unmeasured workplace factors; and generalizability constrained to Sialkot tanneries and comparable process chemistries. Cross-sectional design precludes causal inference; nevertheless, the concordant directionality with mechanistic and epidemiologic literature, along with objective exposure context from companion biomonitoring, supports the plausibility of the observed burden (2,4,7,8).

Future research should enlarge cohorts to stabilize effect estimates, incorporate standardized clinical measurements (e.g., duplicate seated blood pressure, spirometry with bronchodilator testing, creatinine-based renal indices), and implement multilevel models that adjust for age, smoking, tenure, and process assignment while accounting for plant-level clustering. Task-based exposure assessment with area and personal air sampling for metals and acids, combined with biomarker speciation, would disentangle the relative roles of arsenic versus co-exposures and strengthen attribution (3,13). Longitudinal designs with repeated health and exposure measures would enable trajectory analysis and estimation of attributable risk, improving the evidence base for regulatory action and targeted occupational health interventions.

CONCLUSION

Tannery workers showed consistently elevated odds and meaningful prevalence of respiratory symptoms compatible with asthma and of hypertension, with smaller, directionally similar signals for renal, dermatologic, and musculoskeletal complaints, indicating a clinically relevant burden aligned with plausible arsenic-related pathophysiology and mixed co-exposures; although confidence intervals are wide in this modest sample, the concordant pattern supports immediate emphasis on exposure reduction and targeted medical surveillance. For clinical care, programs should prioritize regular blood pressure assessment, structured respiratory screening with spirometry referral, and skin checks alongside worker education and engineering controls; for research, larger longitudinal cohorts with standardized clinical endpoints, multilevel confounder adjustment, and integrated exposure assessment (including biomonitoring and process-level measurements) are warranted to quantify attributable risk and guide precision interventions.

REFERENCES

1. International Agency for Research on Cancer. Arsenic, Metals, Fibres and Dusts. IARC Monographs On The Evaluation Of Carcinogenic Risks to Humans, Volume 100C. Lyon: IARC; 2012.

2. Balakumar P, Kaur J. Arsenic Exposure and Cardiovascular Disorders: An Overview. Cardiovasc Toxicol. 2009;9:169-176.

3. Dixit S, Yadav A, Dwivedi PD, Das M. Toxic Hazards Of Leather Industry And Technologies To Reduce Pollution Load. J Clean Prod. 2015;87:39-49.

4. Zhao J, Li A, Mei Y, Zhou Q, Li Y, Li K, et al. Chronic Arsenic Exposure And Hypertension: A Dose–Response Meta-Analysis. Environ Pollut. 2021;289:117914.

5. Human Rights Watch. Toxic Tanneries: The Health Repercussions Of Bangladesh's Hazaribagh Leather. New York: Human Rights Watch; 2012.

6. Berniell I. Long Working Hours And Health Status: Evidence From Time Use. Bonn: IZA Institute of Labor Economics; 2012.

7. Malik RN, Jadoon WA, Husain SZ. Metal Contamination Of Surface Soils Of Industrial City Sialkot, Pakistan: A Multivariate And GIS Approach. Environ Geochem Health. 2010;32:179-191.

8. Heinze G, Schemper M. A Solution To The Problem Of Separation In Logistic Regression. Stat Med. 2002;21:2409-2419.

9. Hosmer DW, Lemeshow S, Sturdivant RX. Applied Logistic Regression. 3rd ed. Hoboken: Wiley; 2013.

10. Rubin DB. Multiple Imputation For Nonresponse In Surveys. New York: Wiley; 1987.

11. van Buuren S. Flexible Imputation Of Missing Data. 2nd ed. Boca Raton: CRC Press; 2018.

12. Holm S. A Simple Sequentially Rejective Multiple Test Procedure. Scand J Stat. 1979;6:65-70.

13. Steinmaus C, Carrigan K, Kalman D, Atallah R, Yuan Y, Smith AH. Dietary Intake And Arsenic Methylation In A U.S. Population. Environ Health Perspect. 2005;113:1153-1159.