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ORIGINAL PAPER

Blood selenium concentrations and multi-metal biomarker profiles in current asthma: evidence from a nationally representative cross-sectional survey*

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Abstract

Trace element homeostasis increasingly emerges as a modifiable determinant of chronic airway disease, yet population-level multi-metal data in asthma remain limited. This study aimed to characterize whole-blood concentrations of five clinically relevant metals: lead, cadmium, total mercury, selenium, and manganese, in adults with and without current physician-diagnosed asthma, and to determine which metals independently predict asthma status after adjustment for key sociodemographic and lifestyle confounders. A cross-sectional analysis was conducted using data of adults (292 with current asthma, 225 controls) enrolled in a nationally representative health survey (2017-2020). Blood metals were quantified by inductively coupled plasma – mass spectrometry (ICP-MS). Group differences were examined with the Mann-Whitney *U* test, with effect sizes expressed as rank-biserial correlation coefficients. Multivariable binary logistic regression with receiver-operating characteristic (ROC) curve validation identified independent predictors. Blood selenium was the sole metal to differ significantly between groups: asthmatic participants exhibited lower concentrations than controls (median 174.5 vs. 178.9 μg L^{-1}, p=0.021, effect size r=0.10). Although the selenium effect was attenuated after full covariate adjustment (OR=0.995, 95% CI 0.978-1.013, p=0.605), the observed direction of association was similar to that reported in previous studies evaluating selenium and asthma. A borderline protective signal for total mercury, likely a surrogate marker of anti-inflammatory omega-3 fatty acid intake from fish, was also detected (OR=0.524, p=0.067). Lead, cadmium, and manganese did not differ between groups. The multimetal logistic model achieved modest discrimination (AUC=0.58, 95% CI=0.53-0.63). These findings highlight the complexity of selenium – asthma associations and the potential influence of dietary, socioeconomic, and sampling-related confounding. Survey-weighted analyses confirmed that the apparent association was not statistically significant after accounting for the NHANES complex sampling design (weighted p=0.401).

Keywords: selenium, asthma, trace elements, heavy metals, ICP-MS

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INTRODUCTION

Asthma is a heterogeneous chronic inflammatory airway disease, which affects an estimated 300 million people worldwide and remains among the leading preventable causes of morbidity and lost productivity (GBD 2019 Diseases and Injuries Collaborators 2020). Its pathogenesis is multifactorial, encompassing genetic predisposition, immune dysregulation, and a diverse array of environmental exposures (Lambrecht, Hammad 2015). While aeroallergens and ambient air pollutants are the most characterized triggers, accumulating evidence implicates trace metal homeostasis as a potentially modifiable dimension of asthma risk (Salam et al. 2005).

Two distinct categories of blood-borne metals are relevant to respiratory health. Toxic heavy metals, principally lead (Pb), cadmium (Cd), and mercury (Hg), are pervasive environmental contaminants that disrupt mitochondrial function, generate reactive oxygen species (ROS), and polarise immune responses towards a T-helper type 2 (Th2) phenotype, thereby recapitulating core immunological features of atopic asthma (Vieira 2002, Breysse et al. 2010). Contemporary NHANES-based analyses corroborate associations between blood heavy metal concentrations and eosinophilic inflammation and adverse outcomes in asthmatic populations (Wen et al. 2023, Liao et al. 2024), while serum cadmium and lead have separately been linked to wheeze and reduced lung function in U.S. adults (Yang et al. 2019). Conversely, the essential trace elements selenium (Se) and manganese (Mn) anchor critical antioxidant enzyme systems glutathione peroxidases (GPx) and superoxide dismutase that protect the respiratory epithelium from ROS-mediated injury (Rayman 2012).

Selenium exerts its biological effects predominantly through selenoproteins, including GPx1-4 and thioredoxin reductases (TrxR), which constitute the primary antioxidant defense of airway epithelial cells. Selenium deficiency reduces the catalytic capacity of these enzymes, elevates intracellular ROS, activates nuclear factor kappa B (NF-κB) signaling, and augments interleukin-4 and interleukin-13 secretion the cardinal cytokines driving atopic airway inflammation. Furthermore, GPx4 protects against ferroptosis in airway epithelial cells, a recently identified non-apoptotic cell death pathway implicated in severe asthma exacerbations (Dixon et al. 2012). Meta-analytic evidence confirms that lower circulating selenium is significantly associated with increased asthma risk (Chen et al. 2020), a pattern reproduced in more recent clinical cohorts linking lower serum selenium to greater asthma severity and poorer airway control (Girdhar et al. 2022, Srivastava et al. 2023). However, most published studies have examined selenium in isolation without simultaneously controlling for the full spectrum of co-exposed metals, leaving open the question whether the association of selenium with asthma represents an independent biological signal or is partially explained by correlated dietary and socioeconomic exposures.

The unique contribution of the present study lies in applying a simultaneous multi-metal analytical strategy to a large, nationally representative sample. By profiling five metals concurrently and applying multivariable regression with ROC curve discrimination analysis and effect size quantification, this work moves beyond single-element descriptive reporting to provide an integrated picture of metal-asthma associations at the population level. The study aims were twofold: (i) to characterize whole-blood multi-metal profiles in U.S. adults with and without current physician-diagnosed asthma; and (ii) to identify which metals independently predict asthma status after adjustment for age, sex, race/ethnicity, body mass index (BMI), and household tobacco smoke exposure.

MATERIALS AND METHODS

Study design and population

This cross-sectional analysis used publicly available, de-identified data from a nationally representative, stratified, multistage probability health survey of U.S. non-institutionalized civilians conducted by the Centers for Disease Control and Prevention (CDC) over the period 2017 to early 2020 (CDC 2020). Of 15560 total participants across all age groups, 517 adults aged ≥18 years with complete data on asthma status, blood metal concentrations, and all prespecified covariates were retained in the final analytic sample using listwise deletion. The survey was conducted in accordance with the Declaration of Helsinki (1975, amended 2000), with protocols approved by the National Center for Health Statistics Research Ethics Review Board (Protocol #2018-01). All original participants provided written informed consent. This secondary analysis used entirely de-identified public-use data; no additional institutional ethics approval was required.

Case and control definition

Current asthma was ascertained using a validated two-step questionnaire approach. Participants responding affirmatively to both "Has a doctor or other health professional ever told you that you have asthma?" and "Do you still have asthma?" were classified as current asthma cases (n=292). Participants denying ever receiving an asthma diagnosis served as controls (n=225). Participants reporting a previous but no current diagnosis were excluded to avoid heterogeneity between active and remitted disease.

Blood metal biomarkers

Whole-blood concentrations of five metals were measured at the survey's central laboratory by ICP-MS, a reference analytical method with high sensitivity and specificity for trace-level quantification. Metals and measure-

ment units were lead (μg dL^{-1}), cadmium (μg L^{-1}), total mercury (μg L^{-1}), selenium (μg L^{-1}), and manganese (μg L^{-1}). Values below the assay's limit of detection (LOD) were imputed using the substitution value of LOD/√2, a recommended approach that preserves distributional characteristics without zero-inflation bias.

Covariates

Covariates selected a priori based on established associations with both asthma risk and trace metal metabolism were age (continuous, years), sex (male/female), race/ethnicity (non-Hispanic White, non-Hispanic Black, Mexican American, other), BMI (kg m^{-2}, continuous), and household tobacco smoke exposure (presence of ≥1 smoking household member, yes/no).

Statistical analysis

Distribution normality was assessed with the Shapiro-Wilk test. Because all metal variables showed significant departure from normality ($p<0.001$ for all), group differences were evaluated with the non-parametric Mann-Whitney *U* test, and effect sizes were quantified using the rank-biserial correlation coefficient (r), where $r=0.10$, 0.30, and 0.50 correspond to small, medium, and large effects, respectively. Categorical variables were compared with Pearson chi-square or Fisher's exact test. Multivariable binary logistic regression was performed with simultaneous entry of all five metals and all five covariates (Enter method). Model fit was evaluated with the Omnibus chi-square test and the Hosmer-Lemeshow goodness-of-fit test. Overall model discrimination was assessed using ROC curve analysis, with the area under the curve (AUC) and its 95% CI reported. Multicollinearity was evaluated with variance inflation factors (VIF; threshold VIF > 10). All analyses were performed using IBM SPSS Statistics version 24.0 (IBM Corp., Armonk, NY, USA). Statistical significance was set at $p<0.05$; borderline trends were noted at $p<0.10$. The participant selection flow is presented in Figure 1. Survey-weighted analyses were additionally performed using NHANES complex sampling design variables: sampling weight (WTMEC2YR), stratification variable (SDMVSTRA), and primary sampling unit (SDMVPSU), implemented via the IBM SPSS Complex Samples module. Weighted group comparisons used the Complex Samples General Linear Model (CS-GLM); weighted multivariable analysis used Complex Samples Logistic Regression (CS-LR). Both unweighted and weighted results are reported.

RESULTS AND DISCUSSION

Participant characteristics

The analytic sample comprised 517 adults: 292 with current asthma (56.5%) and 225 asthma-free controls (43.5%). The two groups were

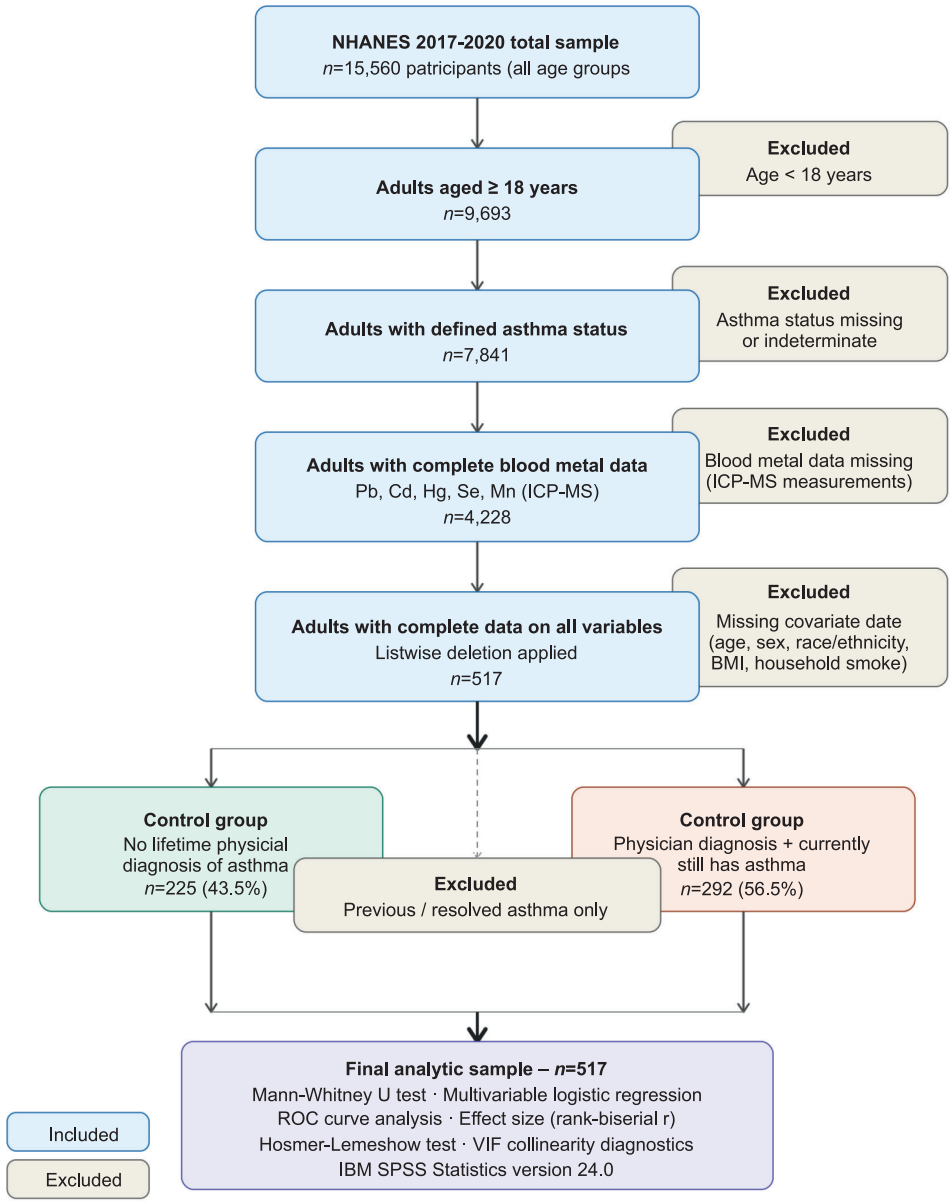

Fig. 1. Flowchart of participant selection from the NHANES 2017-2020 dataset: ICP-MS – inductively coupled plasma – mass spectrometry, BMI – body mass index

well-matched across all prespecified demographic and lifestyle covariates (all $p>0.05$, Table 1), confirming that observed differences in blood metal concentrations cannot be attributed to measured confounders. The mean age was 44.3±21.6 years overall. The female sex accounted for 48.0% of partici-

Table 1

Baseline characteristics of study participants stratified by current asthma status

Characteristic	Total (n=517)	Control (n=225)	Asthma (n=292)	p value
Age (years), mean ± SD	44.3±21.6	44.3±18.0	44.3±21.6	0.652
Female sex, n (%)	248 (48.0)	111 (49.3)	137 (46.9)	0.586
Race/Ethnicity, n (%)				0.552
Non-Hispanic White	303 (58.6)	133 (59.1)	170 (58.2)	
Non-Hispanic Black	39 (7.5)	17 (7.6)	22 (7.5)	
Mexican American	64 (12.4)	28 (12.4)	36 (12.3)	
Other	111 (21.5)	47 (20.9)	64 (21.9)	
BMI (kg m^{-2}), mean ± SD	29.6±7.2	29.1±6.8	30.0±7.5	0.120
Household smoke exposure, n (%)	110 (21.3)	44 (19.6)	66 (22.6)	0.410

BMI – body mass index, SD – standard deviation, p values from Pearson chi-square or independent-samples t-test as appropriate

pants, and the predominant racial/ethnic group was Non-Hispanic White (58.6%). Mean BMI was 29.6±7.2 kg m^{-2}, and household tobacco smoke exposure was present in 21.3% of participants.

Blood trace element and metal concentrations by asthma status

Table 2 presents blood metal concentrations stratified by asthma status. Blood selenium was the sole metal to differ significantly between groups: asthmatic participants had lower concentrations compared with controls (median 174.5 µg L^{-1} [IQR 110.4] vs. 178.9 µg L^{-1} [IQR 86.0]; p=0.021, effect size r=0.10) – Figure 2, Figure 3. The small effect size is consistent with the

Table 2

Blood trace element and metal concentrations by asthma status (Mann-Whitney U test)

Metal (unit)	Control median (IQR)	Asthma median (IQR)	p value	Effect size r	Normal range	Weighted p
Lead (µg dL^{-1})	0.630 (0.32-0.78)	0.680 (0.40-0.83)	0.946	r=0.00	< 3.5	0.474
Cadmium (µg L^{-1})	0.280 (0.12-0.46)	0.320 (0.12-0.42)	0.207	r=0.05	< 1.0	0.396
Mercury (µg L^{-1})	0.290 (0.12-0.51)	0.280 (0.12-0.46)	0.930	r=0.01	< 5.8	0.809
Selenium (µg L^{-1})*	178.9 (86.0)	174.5 (110.4)	0.021*	r=0.10	70-150	0.401
Manganese (µg L^{-1})†	9.460 (3.84)	9.980 (3.75)	0.080†	r=0.08	4.0-14.0	0.419

IQR – interquartile range, r – rank-biserial correlation coefficient. * p<0.05, † p<0.10 (borderline trend)

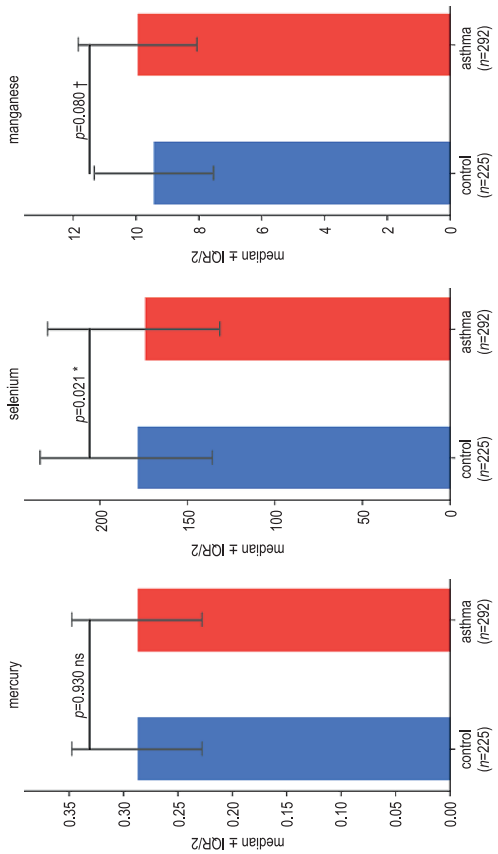

Fig. 2. Comparison of median blood metal levels between control and current asthma groups. Error bars represent IQR/2;
* $p<0.05$, † $p<0.10$, ns – not significant

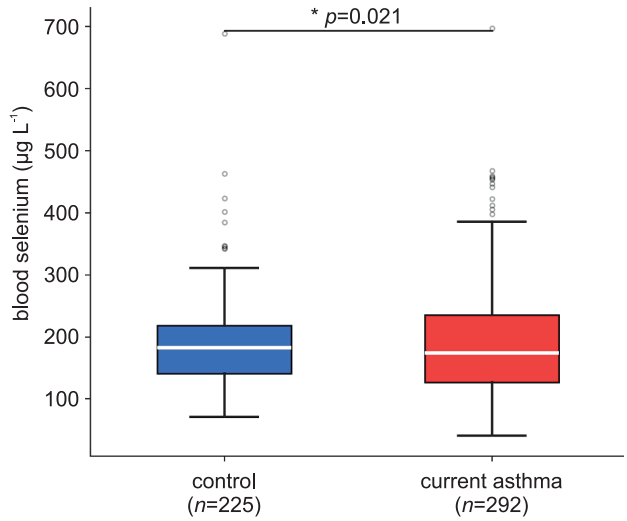

Fig. 3. Box plot of blood selenium concentrations by asthma status. The horizontal line inside each box represents the median; boxes indicate the IQR; whiskers extend to 1.5×IQR

expected magnitude of micronutrient-disease associations in epidemiological samples without frank deficiency. A borderline difference in blood manganese was also observed (p=0.080), with slightly higher concentrations in asthmatics. No significant differences were detected for lead, cadmium, or total mercury (all p>0.20). Survey-weighted analyses (CS-GLM) confirmed no significant group differences: selenium-weighted p=0.401 (weighted means: control 179.0 µg L^{-1}, asthma 177.2 µg L^{-1}), lead p=0.474, cadmium p=0.396, mercury p=0.809, and manganese p=0.419. The attenuation of the unweighted selenium finding after survey weighting is presented in revised Table 2. All metals demonstrated multicollinearity within acceptable limits (VIF range 1.04-1.22).

Multivariable logistic regression and model discrimination

Table 3 and Figure 4 present results of multivariable binary logistic regression. After adjustment, no blood metal emerged as an independent statistically significant predictor of current asthma. The selenium effect was considerably attenuated (OR=0.995, 95% CI 0.978-1.013, p=0.605), consistent with partial confounding by dietary and lifestyle factors that concurrently influence both selenium status and asthma risk. In survey-weighted complex samples logistic regression, blood mercury emerged as a statistically significant inverse predictor (OR=0.428, 95% CI 0.206-0.893, p=0.027), strengthening from borderline significance in unweighted analysis (OR=0.524, 95% CI 0.263-1.045, p=0.067). Overall model fit was acceptable (Omnibus χ^2=9.95, df=10, p=0.445, Hosmer-Lemeshow χ^2=6.82, df=8, p=0.556). The multi-metal model achieved an AUC of 0.58 (95% CI 0.53-0.63), indicat-

Table 3

Multivariable binary logistic regression: independent predictors of current asthma

Variable	B	SE	Wald	OR (95% CI)	*p* value	VIF	Weighted OR (95% CI)	Weighted *p*
Age (years)	-0.005	0.012	0.204	0.995 (0.972-1.018)	0.652	1.07	0.976 (0.946-1.007)	0.115
Sex (female)	0.239	0.447	0.286	1.270 (0.529-3.050)	0.593	1.11	0.560 (0.127-2.469)	0.416
Race/ethnicity	-0.131	0.220	0.354	0.877 (0.570-1.350)	0.552	1.09	0.698 (0.320-1.522)	0.340
BMI (kg m^{-2})	0.042	0.027	2.423	1.043 (0.989-1.100)	0.120	1.08	1.007 (0.921-1.100)	0.876
Household smoke exposure	-0.131	0.441	0.088	0.877 (0.369-2.083)	0.766	1.06	0.310 (0.057-1.688)	0.160
Blood lead (μg dL^{-1})	0.138	0.243	0.323	1.148 (0.713-1.847)	0.570	1.14	1.441 (0.709-2.926)	0.288
Blood cadmium (μg L^{-1})	0.060	0.404	0.022	1.062 (0.481-2.345)	0.881	1.08	1.571 (0.332-7.442)	0.543
Blood mercury (μg L^{-1})†	-0.646	0.352	3.367	0.524 (0.263-1.045)	0.067†	1.22	0.428 (0.206-0.893) †	0.027 †
Blood selenium (μg L^{-1})	-0.005	0.009	0.267	0.995 (0.978-1.013)	0.605	1.15	1.014 (0.989-1.039)	0.241
Blood manganese (μg L^{-1})	0.003	0.057	0.004	1.003 (0.897-1.123)	0.952	1.04	0.992 (0.796-1.237)	0.940

OR – odds ratio, CI – confidence interval, SE – standard error, VIF – variance inflation factor, BMI – body mass index. † $p<0.10$ (borderline trend). Omnibus $\chi^2=9.95$, df=10, $p=0.445$, Hosmer-Lemeshow $\chi^2=6.82$, df=8, $p=0.556$, AUC=0.58 (95% CI 0.53-0.63)

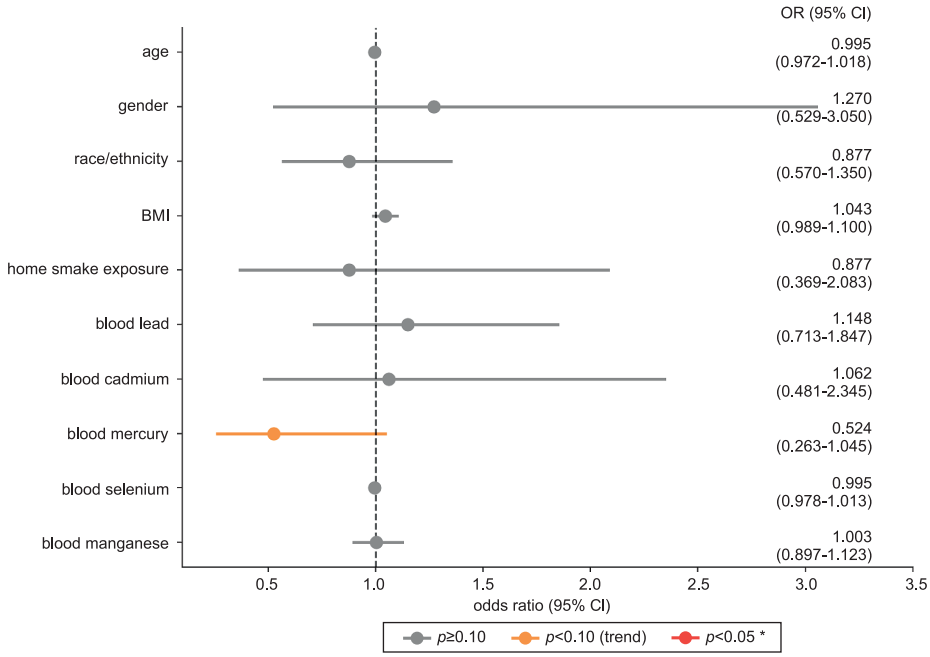

Fig. 4. Forest plot of odds ratios from multivariable logistic regression for current asthma. Squares indicate point estimates; horizontal lines represent 95% CIs. The vertical dashed line indicates OR=1.0 (no association). † $p<0.10$

ing modest but above-chance discrimination. All VIF values were <1.25, confirming the absence of problematic multicollinearity.

Selenium and selenoprotein-mediated airway defense

The selenium-asthma relationship identified in univariate analysis is grounded in well-characterized selenoprotein biology. GPx1 and GPx4, expressed abundantly in bronchial epithelium and alveolar macrophages, use selenium as a catalytic cofactor to neutralize hydrogen peroxide and lipid hydroperoxides generated during allergen-driven inflammation (Rayman 2012). Selenium deficiency reduces the catalytic capacity of these enzymes, elevates intracellular ROS, activates NF-κB, and promotes Th2 cytokine secretion, precisely the immunological cascade driving atopic asthma. Clinical corroboration comes from the meta-analysis by Chen et al. (2020), who confirmed that lower circulating selenium was significantly associated with increased asthma risk across multiple study populations. Pediatric data from Kocyigit et al. (2004) further showed significantly reduced plasma selenium alongside elevated oxidative stress markers in asthmatic children, confirming that selenium deficiency is not confined to adult populations. More recently, Girdhar et al. (2022) reported markedly lower serum selenium in asthmatic patients relative to controls, and Srivastava et al. (2023) identified low

serum selenium, alongside zinc and vitamin D3, as a biomarker of airway inflammation and poor asthma control, reinforcing the biological plausibility of the present findings.

The attenuation after both unweighted adjustment and NHANES survey weighting (weighted p=0.401) indicates that this association was substantially driven by the complex sampling structure, underscoring the importance of survey weights in NHANES research. Chen et al. (2025), using Mendelian randomization, similarly reported that dietary antioxidant intake was inversely associated with asthma risk but substantially influenced by dietary quality confounding. Shaheen et al. (2001) likewise reported attenuation of the antioxidant-asthma relationship after comprehensive covariate adjustment.

Mercury, dietary omega-3 fatty acids, and clinical implications

The borderline inverse association of blood total mercury with asthma (OR=0.524, p=0.067) merits cautious interpretation. Organic methylmercury from fish consumption is the predominant form of total mercury in the general U.S. population. Fish intake simultaneously provides long-chain omega-3 polyunsaturated fatty acids, eicosapentaenoic acid and docosahexaenoic acid, which suppress leukotriene-mediated bronchoconstriction and reduce airway hyperresponsiveness (Mickleborough, Lindley 2013), consistent with more recent meta-analytic evidence linking higher omega-3 intake to a reduced risk of asthma and wheeze (Wu, Li 2022, Bärebring et al. 2022). Accordingly, the inverse mercury signal likely represents surrogate capture of protective dietary exposure rather than a direct biological effect. This observation should be interpreted cautiously and may reflect unmeasured dietary factors rather than a direct biological effect of mercury. Cross-sectional data do not permit dietary recommendations**.** Disentangling these pathways requires studies incorporating mercury speciation and detailed dietary intake data.

Null findings for lead, cadmium, and manganese

The absence of a lead association likely reflects the very low concentrations observed (median ≤0.68 μg dL^{-1}), well below the CDC action threshold of 3.5 μg dL^{-1}. Min et al. (2008) demonstrated that elevated blood lead independently predicted increased bronchial responsiveness at higher exposure levels, implying significant respiratory effects may only emerge above a toxicological threshold not represented in this contemporary low-exposure cohort, a threshold effect also suggested by Yang et al. (2019), who found no association between serum lead and current wheeze or asthma in a nationwide U.S. sample despite significant associations with reduced lung function. For cadmium, Wang et al. (2024) documented a significant association with chronic obstructive pulmonary disease (COPD), but pulmonary disease specificity may differ between COPD and asthma, reflecting divergent pathomechanisms of emphysema versus eosinophilic airway inflammation.

The borderline increase in blood manganese in asthmatic participants (p=0.080, r=0.08) is biologically plausible given that manganese-superoxide dismutase (MnSOD) activity may be upregulated as a compensatory response to heightened oxidative stress in asthmatic airways; however, the small effect size precludes definitive conclusions.

Strengths and limitations

Principal strengths include: (i) the large nationally representative sample with objectively validated ICP-MS multi-metal quantification; (ii) the two-item asthma definition minimizing misclassification; (iii) comprehensive multivariable adjustment; (iv) effect size quantification and ROC curve discrimination analysis extending interpretive value beyond statistical significance; and (v) simultaneous profiling of five biologically relevant metals. Limitations include the cross-sectional design precluding causal inference; a single blood draw not reflecting chronic exposure; residual confounding by dietary patterns and inhaled corticosteroid use; and the modest AUC (0.58) confirming that blood metal profiles alone provide limited discriminatory capacity for asthma, which is a complex, multideterminant disease.

CONCLUSIONS

In this nationally representative sample, the apparent association between blood selenium and current asthma observed in unweighted analyses (p=0.021) was attenuated after multivariable adjustment and NHANES survey weighting (weighted p = 0.401). These findings suggest that previously reported selenium – asthma associations may be influenced by dietary, socioeconomic, and sampling-related factors rather than a direct independent biological effect. Blood mercury showed a statistically significant inverse association with current asthma in survey-weighted analyses (OR=0.428, 95% CI 0.206-0.893, p=0.027), although this finding should be interpreted cautiously because of potential dietary confounding from omega-3 fatty acid intake. Further prospective studies incorporating selenium and mercury speciation and comprehensive dietary assessment are needed to clarify the role of trace elements in asthma pathogenesis.

Author contributions

E.C. – conceptualization, data curation, formal analysis, methodology, visualization, writing – original draft preparation, writing – review & editing. The author has read and agreed to the published version of the manuscript.

Ethics statement

This study is a secondary analysis of entirely de-identified, publicly available data from the National Health and Nutrition Examination Survey (NHANES), a nationally representative cross-sectional survey administered by the Centers for Disease Control and Prevention (CDC), the United States. The original NHANES protocols covering the 2017-2020 survey cycles were reviewed and approved by the National Center for Health Statistics (NCHS) Research Ethics Review Board (ERB), an institutional review board operating in accordance with the provisions of 45 CFR 46 (Protocol Reference #2018-01). Written informed consent was obtained from all original survey participants by the NCHS prior to data collection. All procedures performed in the original survey were in accordance with the ethical standards of the NCHS ERB and with the 1964 Declaration of Helsinki and its later amendments.

The present secondary analysis was conducted exclusively on de-identified public-use data files, which are freely available to the research community at https://wwwn.cdc.gov/nchs/nhanes/ (accessed 25 April 2026). Under United States federal regulations (45 CFR 46.101(b)(4)) and the guidelines of the Office for Human Research Protections (OHRP), secondary analyses of pre-existing, publicly available, de-identified data do not constitute human subjects research and are therefore exempt from additional institutional review board (IRB) oversight. Accordingly, no separate ethics approval was sought or required for this analysis. No participant could be identified from the data used in this study. The author did not contact any survey participants, did not collect any new biological or personal data, and did not conduct any animal experiments**.** This study is fully compliant with the ethical principles governing secondary analyses of public health surveillance data.

Data availability statement

All data used in this study are publicly available from the National Health and Nutrition Examination Survey (NHANES) repository maintained by the Centers for Disease Control and Prevention, accessible at https://wwwn.cdc.gov/nchs/nhanes/ (accessed 25 April 2026). No proprietary or restricted datasets were used. The SPSS analytical syntax employed in this study is available from the corresponding author upon reasonable request.

Informed consent statement

This study did not involve direct contact with human participants. Informed consent was obtained from all original NHANES survey participants by the National Center for Health Statistics prior to data collection. The secondary analysis used only de-identified public-use data files; individual participant consent for this analysis was neither required nor applicable.

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Conflicts of interest

The author ensures that she has neither professional nor financial connections related to the manuscript sent to the Editorial Board. This research received no external funding.

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