Journal of Elementology ISSN 1644-2296

Urbanowicz, T., Spasenenko, I., Królak, J., Gabriel, K., Krasińska, B., Zawadzki, P., Grobelna, M., Zieliński, M., Staniszewski, R., Krasiński, Z., Tykarski, A. and Hanć, A. (2025) 'The relationship between hair manganese accumulation, obesity, and coronary artery disease: early observation', *Journal of Elementology*, 30(4), 777-787, available: https://doi.org/10.5601/jelem.2025.30.4.3691

RECEIVED: 29 October 2025
ACCEPTED: 17 November 2025

ORIGINAL PAPER

The relationship between hair manganese accumulation, obesity, and coronary artery disease: early observation*

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Abstract

Coronary artery disease (CAD) is a significant health problem worldwide. The prolonged, low-grade inflammatory activation in obese patients is believed to aggravate hemostasis disruption, resulting in premature mortality. The involvement of trace elements in excessive adipose tissue metabolism is postulated. Among trace elements, manganese (Mn) is an essential trace element involved in numerous biological processes, including inflammation regulation, mitochondrial function, metabolic enzyme activity, and antioxidant defense. This study aimed to assess the concentration of trace elements, especially manganese, in the hair of patients with chronic coronary syndrome, and to explore its relationship with obesity and coronary disease. Hair samples were analyzed using inductively coupled plasma mass spectrometry (ICP-MS). Patients were divided into groups based on the presence of coronary artery disease and body mass index (BMI). The results showed significantly higher manganese levels in the hair of obese patients, regardless of coronary disease status. This suggests that increased manganese concentration may reflect metabolic disturbances linked to obesity rather than being directly related to coronary disease. Higher manganese levels might be connected to increased oxidative stress and metabolic demands in obese individuals. A limitation of the study is the small number of participants, and therefore the results require confirmation on larger populations. In conclusion, manganese levels in hair may serve as a marker of metabolic changes related to obesity, but does not directly correlate with coronary artery disease risk. These findings underscore the need for additional research on the role of manganese in metabolic regulation.

Keywords: manganese, obesity, coronary artery disease, ICP-MS, trace elements

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* This work was supported by a research grant awarded by the Adam Mickiewicz University in Poznan, Faculty of Chemistry and Poznan University od Medical Sciences.

INTRODUCTION

Coronary artery disease represents one of the epidemiological challenges in the current era. Among well-established risk factors, such as arterial hypertension, dyslipidemia, nicotine, and hereditary lifestyle, a possible role of chronic immune system activation in atherosclerosis pathophysiology has been extensively investigated (Grajek et al. 2021, Shaya et al. 2022, Stone et al. 2023). Atherosclerosis is considered a chronic condition related to inflammatory activation, where the involvement of trace elements as enzymatic cofactors plays a significant role (Libby, 2021, Urbanowicz et al. 2021, d'Aiello et al. 2024, Urbanowicz et al. 2024).

Excessive calorie consumption in everyday diet, processed food, and inadequate physical activity result in excessive body mass gain, which contributes to global morbidity and mortality in developed countries. The prolonged, low-grade inflammatory activation in obese patients is believed to aggravate hemostasis disruption, resulting in premature mortality (Sanchez-Cabo et al. 2023, Urbanowicz et al. 2025). The involvement of trace elements in excessive adipose tissue metabolism is postulated. Among trace elements, manganese (Mn) is an essential one involved in numerous biological processes, including inflammation regulation, mitochondrial function, metabolic enzyme activity, and antioxidant defense. Manganese is a cofactor for antioxidant enzymes (e.g., Mn-SOD) that protect cells from oxidative stress. The emerging evidence suggests that either manganese deficiency or body accumulation may have a significant impact on cardiovascular morbidity. A reduced level of manganese may lead to increased oxidative stress, which promotes the development of atherosclerosis and cardiovascular diseases (Ou et al. 2024). In mice with manganese deficiency, cardiac oxidative damage and mitochondrial dysfunction were observed, which are attributed to the inhibition of the Nrf2 signaling pathway (Xu et al. 2025).

The interplay between this trace element and cardiovascular pathogenesis is complex, yet dualistic, highlighting the role of Mn in hemostasis.

The aim of this study was to assess the concentration of trace elements, with particular emphasis on manganese (Mn), in hair samples from patients with chronic coronary syndrome, in the context of their potential impact on the pathophysiology of atherosclerosis and metabolic disturbances associated with obesity. Hair as a biological material reflects long-term exposure to trace elements, making it a useful diagnostic tool. To precisely analyze the relationship between elemental status and the severity of coronary artery disease, coronary angiography results and body mass index (BMI) were used as criteria for dividing the study groups – an approach not commonly applied in previous research.

MATERIALS AND METHODS

Patients and methods

The patients, with a median age of 66 (61-72) years, were meticulously selected for the study. They were referred by their cardiologist for coronary angiography presented for scheduled admissions at the Department of Hypertensiology, Angiology, and Internal Medicine of the Medical University in Poznań, in 2022. The demographic and clinical characteristics were examined and compared with the echocardiographic and coronary angiogram results. On admission, head hair samples were taken for chemical analysis, ensuring a comprehensive chemical analysis in relation to the obtained results. The stepwise analysis is presented in a flowchart (Figure 1).

Bioethics Committee Approval

The study was conducted in accordance with the principles of Good Clinical Practice and the Declaration of Helsinki. It was approved by the Local Ethics Committee of the Medical University of Poznań (approval number 875/22, dated November 3, 2022). All patients gave their informed consent for inclusion in the study.

Hair trace elements analysis

Hair samples (2-3 cm long), untreated and free from dye or chemical processing, were collected from the occipital region of the scalp, close to the skin. The samples were washed by stirring with different solvents in the sequence: acetone, deionized water, 0.5% Triton X-100 solution, and deionized water, and then dried according to a standardized procedure (Urbanowicz et al. 2023). Approximately 150-200 mg of dried hair was used for mineralization in a DigiTube system (SCP Science Ltd., Canada). For digestion, 4 mL of 65% nitric acid and 1 mL of 30% hydrogen peroxide were added. The digestion process was carried out using a block digestion system equipped with a temperature controller, which maintained a temperature of 150°C for 4 hours. After cooling to room temperature, the digested material was diluted 100-fold with ultrapure water.

Element concentrations were determined using inductively coupled plasma mass spectrometry (ICP-MS, Agilent 7700x, USA), a highly sensitive technique suitable for the analysis of trace elements. To ensure analytical accuracy, certified reference material (CRM) for human hair (NCS ZC 81002b, Beijing, China) was used. The method's reliability was confirmed by recovery rates ranging from 94% to 107% for the analyzed elements, indicating good trueness and precision. The limit of detection (LOD) for manganese was approximately 0.005 μg L^{-1}, and the limit of quantification (LOQ) was estimated at 0.015 μg L^{-1}, allowing for accurate measurement even at low concentrations. All measurements were performed under optimized

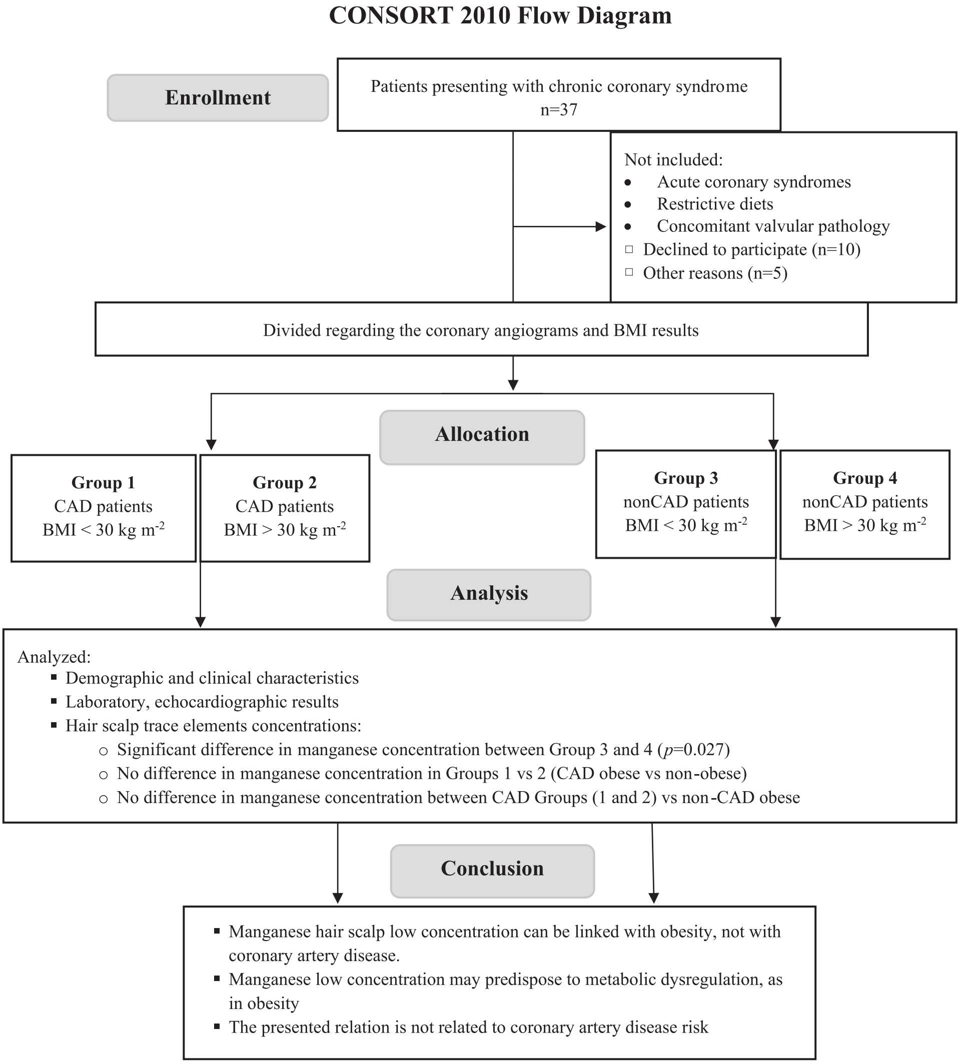

Fig. 1. Flow chart presenting patient enrolment, group division, and the results

laboratory conditions to minimize contamination and ensure reproducibility (Urbanowicz et al. 2025).

Statistical analysis

Since data did not follow normal distribution (the Shapiro-Wilk test), parameters were presented as medians and interquartile ranges (Q_1 - Q_3). The categorical data were presented as numbers and percentages. The comparison between groups was performed by the Kruskal–Wallis test with the post-hoc Dunn's tests. When a comparison included categorical data, the chisquare test of independence was used. Two-sample Wilcoxon (Mann-Whitney) tests were performed to present the differences between the measured parameters, including laboratory and clinical results.

RESULTS AND DISCUSSION

Out of 37 patients, the coronary epicardial disease was confirmed in 20 (54%). The median BMI in the analyzed group was 28.1 (26.3 - 33.5), including 15 (41%) patients with diagnosed obesity. The whole population was divided into two subgroups, CAD and non-CAD, and subdivided accordingly to the BMI ratio as presented in Table 1.

There were fourteen trace elements examined in hair samples. The results were compared within the analyzed groups as presented in Table 2, indicating the significant differences regarding the As hair concentration between group 1 vs 4 (p=0.045), Cu concentration between group 2 vs 4 (p=0.005), and Mn concentration between group 3 and 4 (p=0.027).

We observed significant differences in manganese hair concentration between the analyzed groups, a finding that could potentially lead to new insights in the field of trace element research and its relation to chronic coronary syndrome, as presented in Figure 2.

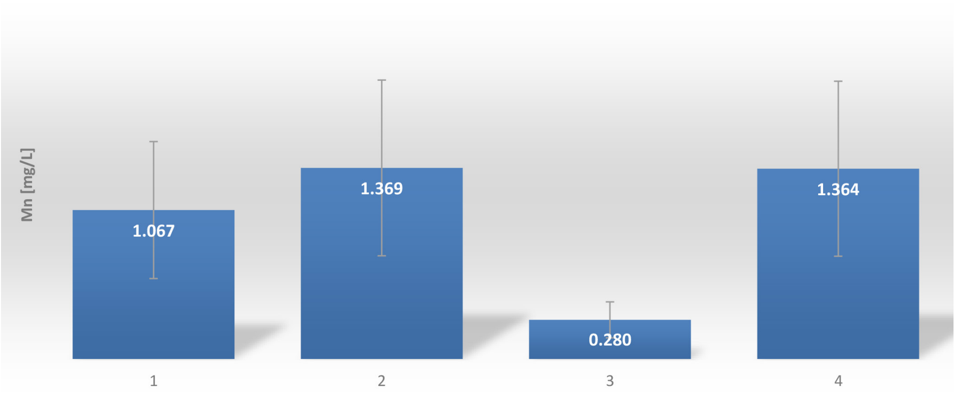

Fig. 2. Manganese hair concentration in Group 1 (CAD, BMI <30kg m^{-2}) vs Group 2 (CAD< BMI>30 kg m^{-2}) vs Group 3 (nonCAD BMI<30 kg m^{-2}) vs Group 4 (nonCAD BMI>30kg m^{-2})

Our analysis points out the significantly higher manganese hair concentration in obese patients compared to standard controls. Our results concern hair scalp trace element concentrations, although samples of serum or whole blood are most commonly used in clinical studies. However, hair samples represent an interesting alternative and have attracted increasing attention from researchers as a valuable source of information. This is primarily because the elemental content in human blood can be influenced by factors

Table 1

Demographic and clinical characteristics, followed by laboratory and echocardiographic results in the analyzed groups

Parameter	CAD BMI<30 Group 1 *n*=13	CAD BMI>30 Group 2 *n*=7	nonCAD BMI<30 Group 3 *n*=9	nonCAD BMI>30 Group 4 *n*=8	*P* 1 vs 2	*P* 1 vs 3	*P* 1 vs 4	*P* 2vs 3	*P* 2 vs 4	*P* 3 vs 4
Demographics										
Age (years) median (Q1-Q3)	72 (65-73)	65 (53-68)	70 (65-75)	64 (52-66)	0.056	0.712	0.015	0.099	0.772	0.030
Sex – males (%)	8 (62)	7 (100)	6 (67)	3 (38)	0.114	0.841	0.316	0.213	0.026	0.267
BMI (kg m^{-2}) median (Q1-Q3)	26 (24-27)	34 (33-35)	27 (26-27)	37 (31-45)	<0.001	0.695	<0.001	0.001	0.685	<0.001
Co-morbidities:										
Arterial hypertension *n* (%)	13 (100)	7 (100)	8 (89)	6 (75)	1.000	0.409	0.133	1.000	0.467	0.513
Diabetes mellitus *n* (%)	4 (31)	2 (29)	6 (67)	4 (50)	0.960	0.060	0.414	0.095	0.454	0.349
Dyslipidemia *n* (%)	13 (100)	3 (43)	9 (100)	7 (88)	0.007	1.000	0.381	0.019	0.090	0.471
PAD (n (%)	1 (8)	0 (0)	1 (11)	1 (13)	1.000	0.776	0.776	1.000	1.000	1.000
Nicotine overall (n (%)	7 (54)	2 (29)	4 (44)	6 (75)	0.312	0.900	0.645	0.454	0.216	0.599
Laboratory results:										
Hb (mmolL^{-1}) median (Q1-Q3)	9.0 (8.5-9.2)	8.8 (8.7 -9.1)	8.4 (8.0-8.8)	8.9 (8.7-9.8)	0.690	0.298	0.488	0.182	0.815	0.208
Hct (%) median (Q1-Q3)	44 (41-45)	43 (42-45)	41 (40-47)	44 (43-49)	0.465	0.737	0.535	0.786	0.360	0.208
Crea (mm) median (Q1-Q3)	77 (71-90)	83 (74-118)	100 (89-111)	91 (70-115)	0.475	0.077	0.612	0.672	1.000	0.470
CRP (IUL^{-1}) median (Q1-Q3)	3.0 (2.0-4.0)	3 (2.0-4.0)	2.3 (2.1.-3.2)	2.5 (2.1-3.0)	0.879	0.546	0.643	0.216	0.215	0.267
TCH (mmolL^{-1}) median (Q1-Q3)	4.2 (3.6-4.7)	3.9 (3.6-5.1)	4.1 (3.8-4.3)	4.1 (3.4-4.2)	1.000	0.851	0.472	0.560	0.519	0.602
HDL (mmolL^{-1}) median (Q1-Q3)	1.2 (1.0-1.4)	1.1 (0.9-1.1)	1.0 (0.9-1.4)	1.0 (0.8-1.5)	0.205	0.734	0.704	0.698	1.000	0.728
LDL (mmolL^{-1}) median (Q1-Q3)	2.4 (2.3-3.2)	2.6 (2.5-3.9)	2.3 (2.0-2.5)	2.3 (2.2-2.7))	0.372	0.563	0.331	0.332	0.223	0.954
Echocardiography										
LV (mm) median (Q1-Q3)	48 (44-50)	54 (53-58)	49 (47-52)	50 (48-59)	0.056	0.569	0.162	0.090	0.521	0.596
RV (mm) median (Q1-Q3)	28 (27-32)	27 (23-29)	31 (30-33)	28 (25-39)	0.460	0.203	0.624	0.022	0.519	0.390
LVEF (%) median (Q1-Q3)	60 (55-60)	60 (50-60)	60 (55-60)	55 (43-58)	0.622	0.622	0.175	1.000	0.380	0.314

Abbreviations: BMI – body mass index, CAD – coronary artery disease, crea – serum creatinine, CRP – C-reactive protein, Hb – hemoglobin, Hct – hematocrit, HDL – high-density lipoprotein, LDL – low-density lipoprotein, LV – left ventricular diameter, LVEF – left ventricular ejection fraction, mm – millimeters, mmol – millimol, PAD – peripheral artery disease, P – probability value, RV – right ventricular diameter TCH – total cholesterol, Q – quartile.

Table 2

Trace elements hair concentration in the analyzed groups

Hair trace elements concentration (median Q1-Q3)	CAD BMI<30 Group 1 n=13	CAD BMI>30 Group 2 n=7	nonCAD BMI<30 Group 3 n=9	nonCAD BMI>30 Group 4 n=7	P 1 vs 2	P 1 vs 3	P 1 vs 4	P 2vs 3	P 2 vs 4	P 3 vs 4
Li	0.08 (0.05 0 .10)	0.06 (0.04-0.14)	0.10 (0.06-0.18)	0.11 (0.06-0.20)	0.692	0.601	0.595	0.203	0.118	0.815
Na	438 (362-1769)	362 (258-859)	591 (301-1240)	720 (467-1292)	0.204	0.601	0.374	0.672	0.118	0.423
Mg	56.9 (36.0-124.3)	62.3 951.3-97.1)	28.9 (12.7-39.8)	133 (67-146)	1.000	0.164	0.238	0.112	0.147	0.059
Al.	54.6 (21.4-116.1)	24.0 (23.1-86.7)	20.1 (11.4-45.8)	48.7 (32.6-109.2)	0.579	0.209	0.860	0.458	0.451	0.236
K	173 (135-346)	129 (58-280)	150 (99-218)	185 (156-450)	0.267	0.512	0.645	0.496	0.271	0.277
Ca	1283 (346-2538)	458 (376-938)	423 (270-1068)	1193 (861-2245)	0.132	0.082	1.000	0.832	0.093	0.167
Cr	1.7 (1.2-7.6)	1.7 (0.8-2.8)	1.4 (0.9-2.3)	2.5 (1.5-4.4)	0.341	0.209	0.645	0.916	0.182	0.200
Mn	1.1 (0.8-2.1)	1.4 (0.8-1.6)	0.28 (0.23-1.19)	1.36 (1.16-3.47)	0.751	0.082	0.268	0.458	0.324	0.027
Fe	26.9 (13.9-34.9)	17.7 (15.8-27.0)	16.1 (8.2-24.5)	23.4 (18.5-60.9)	0.692	0.357	0.697	0.458	0.223	0.093
Ni	0.02 (0.00-1.10)	0.00 (0.00 0.76)	20.7 (17.7-23.8)	27.0 (20.0-43.8)	0.611	0.231	0.147	0.916	0.334	0.772
Cu	20.1 (15.3-21.8)	17.4 (16.5-18.0)	103 (0-114)	110 (11-129)	0.526	0.744	0.185	0.289	0.005	0.200
Zn	45.4 (0.00-129.4)	65.5 (51.2-159.2)	0.15 (0.12-0.31)	0.28 (0.18-0.31)	0.224	0.628	0.527	0.631	0.522	0.661
As	0.12 (0.00-0.20)	0.19 (0.17-0.25)	0.49 (0.37-0.64)	0.87 (0.21-1.56)	0.315	0.347	0.045	0.672	0.685	0.277
Se	0.36 (0.00-0.64)	0.34 (0.33 -1.25)	0.03 (0.01-0.05)	0.05 (0.01-0.14)	0.301	0.315	0.177	0.916	1.000	0.815

Abbreviations+: Al – aluminum, As – arsenic, BMI – body mass index, Ca – calcium, CAD – coronary artery disease Cr – chromium, Cu – copper, Fe – iron, K – potassium, Li – lithium, Mg – magnesium, Mn – manganese, n – number, Na – sodium, Ni – nickel, nonCAD – no coronary disease, P – probability value, Se – selenum, Q – quartile, Zn – zinc such as the time of day, food intake, serum protein concentration, etc. (Pozebon et al. 2017, Fanni et al. 2021), whereas hair is independent of homeostatic mechanisms. In patients treated and operated on due to cardiovascular diseases, numerous factors also disrupt the natural levels of elements in blood or plasma (e.g., anesthesia). For this reason, hair was selected as the biological material for analysis.

In patients with coronary disease, the high manganese concentration is comparable in both obese and normal-weight patients. Recent reports have highlighted altered trace element homeostasis in cardiovascular disorders.

Manganese represents an essential trace element involved in various enzymatic antioxidant defense and metabolic processes (Peng et al. 2022, Sołek et al. 2025). Several possible mechanisms may underlie the presented phenomenon. The higher manganese concentration in obese patients and coronary disease patients involves physiological adaptations, but also may be related to pathological dysregulation.

Manganese is reported to be a crucial cofactor for the mitochondrial antioxidant enzyme manganese superoxide dismutase (MnSOD), which neutralizes reactive oxygen species (ROS) – Liu et al. (2022). The possible upregulation of MnSOD in obese and CAD patients may be explained by the relationship between those pathological conditions and chronic low-grade inflammation and oxidative stress. Elevated hair manganese concentration may reflect a homeostatic response to increased oxidative burden. In both conditions, obesity and coronary atherosclerosis, elevated Mn may enhance MnSOD activity (Ma et al. 2022).

One of the possible explanations of increased manganese accumulation in obese individuals may be the upregulation of divalent metal transporter 1 (DMT1) – Zhang et al. (2024). The increased concentration of DMT1 in intestinal cells, directed to iron absorption, may increase Mn body concentration as a secondary effect (Citelli et al. 2015). Manganese competes with iron, zinc, and copper for binding sites in enzymes. Imbalances in these elements may affect metabolic and oxidative functions (Horning et al. 2015).

Previous studies (Zhang et al. 2023, Wang et al. 2024) suggested that Mn acts as a cofactor in enzymatic reactions involved in gluconeogenesis and insulin secretion. The involvement of manganese in glucose metabolism and insulin signaling may lead to increased Mn retention to support metabolic enzyme function. Genes that code for metal transporters (such as SLC30A10 and SLC39A8) can affect how much manganese the body absorbs and uses. In people with certain genetic traits, manganese may build up in the body even if their intake is normal. Because of this, genetic differences in the activity of these transporters can influence manganese levels in the body, including in hair, which reflects long-term exposure (Wahlberg et al. 2018). In contrast, Horning et al. (2015) emphasize that the cellular mechanisms regulating manganese homeostasis are poorly understood compared to other metals. Manganese is essential as a cofactor for enzymes like arginase and glutamine syntheses, but too much of it can be toxic, especially to the nervous system. In conditions such as cardiovascular disease and obesity, where metabolic imbalances and deficiencies in other trace elements are common, these interactions may have important clinical relevance.

We may suggest that the elevated manganese concentrations observed in obese and CAD patients are most likely multifactorial and related to the increased oxidative stress, and Mn accumulation may be the result of compensatory antioxidant demands. The observed manganese accumulation may be explained by its possible role in metabolic regulation and insulin sensitivity. In future studies, researchers plan to investigate the correlation between manganese levels in hair and its concentration in plasma and urine, aiming to better understand the mechanisms of manganese distribution and retention in the body.

STUDY LIMITATION

The study was conducted with a relatively small number of participants as a single-center, prospective study. Large volume studies are required to confirm the presented results.

CONCLUSIONS

Manganese present at low concentrations in the hair scalp can be linked with obesity rather than with coronary artery disease. Manganese may predispose to metabolic dysregulation, such as obesity. The presented relation is not linked to coronary artery disease risk. The results of the prospective preliminary report analysis need further confirmation in large-scale studies. The data suggest that elevated manganese levels in hair may reflect a broader physiological response to metabolic stress, rather than serving as a direct indicator of cardiovascular disease. The observed link with obesity, in the absence of a similar association with coronary artery disease, suggests that manganese may play a role in metabolic regulation. Given these findings, future research on metabolic health and micronutrient interactions should consider the manganese status.

Author contributions

T.U. and A.H. – conceptualization, A.H. – methodology, T.U – software, A.H and T.U. – validation, T.U. and A.H. – formal analysis, T.U. and A.H. – investigation, E.S., J.K., K.G., B.K., P.Z., M.G., R.S. – resources, T.U., A.H, – data curation, T.U. and A.H. – writing – original draft preparation, A.H. and T.U. – writing – review and editing, E.S., J.K., K.G., B.K., P.Z., M.G., M.Z., R.S. – visualization, T.U., A.H., Z.K., A.T. – supervision. All authors have read and agreed to the published version of the manuscript.

Conflicts of interest

The authors declare no competing interests.

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