Heliyon 10 (2024) e34062

Contents lists available at ScienceDirect

Heliyon

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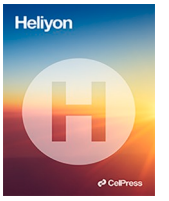

Research article

Association of carbon monoxide poisoning with cardiovascular disease risk: A systematic review and meta-analysis

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ARTICLE INFO

Keywords:
Carbon monoxide poisoning
Cardiovascular disease
A systematic review
Meta-analysis

ABSTRACT

Objective: This study aims to provide an updated overview of the relationship between carbon monoxide poisoning (COP) and cardiovascular disease.
Methods: A systematic literature search was conducted in PubMed, Embase, Cochrane, and Web of Science databases up to September 2023. The association between COP patients and cardiovascular adverse events was examined and summarized. The outcomes included arrhythmia, coronary heart disease, heart failure, myocardial infarction, major adverse cardiovascular events (MACE), carboxyhaemoglobin percent (COHB%), Pondus Hydrogenii (PH) electrocardiography (ECG) parameters.
Results: Eight eligible articles, involving a total of 251,971 patients, were included for evidence synthesis. The analysis revealed a heightened incidence of MACE in patients with COP. Additionally, COP exhibited an impact on specific ECG parameters. The incidence of MACE after COP was found to be similar in Korean and Chinese populations, and there was no significant effect of gender or underlying diseases on MACE incidence following COP. The incidence of MACE after COP did not differ significantly in individuals aged 50 years and older.
Conclusions: Considering the observed heterogeneity and potential biases in the selected studies, emergency physicians should be aware of the increased likelihood of cardiovascular events in patients diagnosed with COP.

1. Introduction

Carbon monoxide (CO), a potent toxic gas, is produced by the complete combustion of carbon-containing substances. Brief exposure to specific CO concentration can lead to poisoning [1]. Carbon monoxide poisoning (COP) primarily manifests as symptoms affecting the nervous and cardiovascular systems due to hypoxia, including dizziness, nausea, palpitations, chest tightness, weakness, lethargy, and, in severe cases, coma [2,3]. In the United States, approximately 50,000 COP cases present to emergency departments annually, with mortality rates ranging from 1 % to 3 % [4,5]. In Japan, COP accounts for 2000–5000 deaths yearly, making it the leading cause of poisoning-related fatalities [6–8].

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https://doi.org/10.1016/j.heliyon.2024.e34062
Received 18 March 2024; Received in revised form 17 May 2024; Accepted 3 July 2024
Available online 14 July 2024

CO has a 250-fold higher affinity for hemoglobin compared to oxygen, hindering the recombination of oxygen with hemoglobin once CO is absorbed into the human body. Consequently, hypoxia-induced damage is the primary mechanism of COP [1]. COP affects virtually all organs and tissues, with the cardiovascular and central nervous systems being the most impacted due to their heightened oxygen demands [9]. Studies indicate that CO's cardiotoxicity extends to direct effects on heart muscle contractility, as CO exhibits a higher affinity for myoglobin than oxygen [10–12]. A Chinese national study revealed that for every 1 mg/m3 rise in average daily and previous day CO concentration, there was a 1.12 % increase in cardiovascular disease mortality [13].

Further investigations support the link between COP and adverse cardiovascular outcomes. A Korean study, utilizing the National Health database, associated COP with an elevated risk of ischemic cardiomyopathy over a 6-year follow-up period [14]. In a Taiwanese cohort study, CO poisoning was linked to an increased risk of subsequent arrhythmias and coronary heart disease through 6 years of follow-up [5]. Despite a robust literature base on CO's impact on the nervous system, research on its cardiovascular effects is limited, with a comprehensive meta-analysis. This paper synthesizes existing clinical studies to conduct a meta-analysis, providing evidence-based insights into the impact of CO on cardiovascular diseases.

2. Materials and methods

2.1. Literature review

This evidence-based analysis adheres to the guidelines outlined in the PRISMA 2020 Statement [15], the Preferred Reporting Program for Systematic Review and Meta-analysis, and was prospectively registered with PROSPERO (CRD42023478507). A systematically literature search was conducted in PubMed, Embase, Cochrane, and Web of Science databases by September 2023 to explore the correlation between COP and cardiovascular disease, with subsequent publication in English. The search utilized terms such as "Monoxide, Carbon," "Cardiovascular Disease", "Disease", Cardiovascular", "Major Adverse Cardiac Events", "Cardiac Events", and others. Detailed search strategies are provided in Table S1. Additionally, we conducted a manual review of the reference lists of all eligible studies. Two investigators independently performed the search and evaluation of included studies, with any discrepancies resolved through consensus, Quality evaluation of the eligible studies shown in Table S2.

2.2. Study selection criteria

Studies were included if they met the following criteria: (1) Study subjects meeting the diagnostic criteria for carbon monoxide poisoning: ① Clear history of poisoning ② relevant clinical symptoms ③ Positive test for COHb [16]. (2) Cohort or case-control study design. (3) Outcome measures including major cardiovascular adverse events (MACE) or cardiovascular disease-related indicators, with a clear diagnosis or definition of MACE; and (4) Reported incidence of MACE or data allowing its calculation. Reviews, letters, editorial comments, case reports, conference abstracts, pediatric articles, unpublished articles, and non-English articles were excluded.

2.3. Data extraction

Two researchers independently performed data extraction, with any discrepancies resolved by a third researcher. The extracted data included first author, publication year, study period, study country, study design, sample size, inclusion criteria, age, sex, underlying diseases, hazard ratio (HR) for arrhythmia, HR for coronary heart disease, HR for heart failure, HR for myocardial infarction, HR for myocardial infarction MACE, COHB% levels, partial PH, QTc interval (msec), Tp−e interval (msec), Tp−e/QTc ratio, and Tp dispersion (msec). For continuous variable reported as median or interquartile range, the mean ± standard deviation was calculated using validated mathematical methods [17,18]. In cases of missing or unreported data, the corresponding authors were contacted for comprehensive information.

2.4. Quality assessment

The quality of included studies was independently assessed by two investigators using the Newcastle-Ottawa Scale (NOS) [19]. Studies scoring of 7–9 were considered high quality [20]. Disparities in quality assessment were resolved through discussion between the two researchers.

2.5. Statistical analysis

Evidence synthesis was conducted using Review Manager 5.3 version (Cochrane Collaboration, Oxford, UK). HR and odds ratios (OR) were employed for comparing continuous and dichotomous variables, respectively, with all metrics reported with 95 % confidence intervals (CIs). Heterogeneity among studies was evaluated using the chi-squared ($\chi 2$) test (Cochran's Q) and the inconsistency index (I^2) [21], with significant heterogeneity was defined as p value < 0.05 or $I^2 > 50$ %. A random-effect model was utilized to estimate combined HR or OR in the presence of significant heterogeneity; otherwise, a fixed-effect model was applied. Additionally, one-way sensitivity analyses were performed to assess the impact of included studies on combined results for outcomes with significant heterogeneity. For outcomes with 10 or more included studies, publication bias was visually evaluated through funnel plots in Review Manager 5.3 version and by conducting Egger's regression tests [22] using Stata 12.0 version (Stata Corp, College Station, TX, USA), with a p value < 0.05 indicating statistically significant publication bias.

3. Results

3.1. Literature search and study characteristics

Fig. 1 illustrates the flow chart of the search and selection process. A total of 1555 related articles were identified: Pubmed (n = 330), Embase (n = 284), Cochrane (n = 25), and Web of Science (n = 916). After removing duplicate papers, 1250 paper titles and abstracts were reviewed. Finally, eight full-text articles involving 251,971 patients were included in the pooled analysis [5,14,23–28], including one retrospective case-control study [14] and seven retrospective cohort studies [5,23–28]. Table 1 presents the characteristics, level of evidence, and quality score of each included study, with quality score ranging from 6 to 8. As the population of Huang 2018 and Huang 2021 originated from the same unit and had the same research period, the newer Huang 2021 was selected for data analysis, while Huang 2018 was used for system description.

3.2. Demographic characteristics

Data on patients' age, sex, and previous history of hypertension, diabetes, and hyperlipidemia were conducted and statistically analyzed at baseline. The results showed no statistically significant differences in age between COP patients and controls (HR: 0.02; 95 % CI: 0.11, 0.15; $p = 0.79$), sex (HR: 1.74; 95 % CI: 0.60, 5.09; $p = 0.31$), and hypertension (HR: 1.31; 95 % CI: 0.95, 1.82; $p = 0.10$). However, significant differences were found in diabetes (HR: 1.58; 95 % CI: 1.23, 2.03; $p < 0.00001$), hyperlipidemia (HR: 1.47; 95 % CI:1.15, 1.87; $p = 0.002$) were statistically significant (Table 2).

3.3. MACE

MACE data were collected from four articles including 251,594 patients (71,375 COP patients and 180,219 control patients) [5,14,25,27]. Pooled analysis showed A higher incidence of MACE in COP patients compared to control patients (HR:2.02; 95 % CI: 1.892.16; $p < 0.00001$), with no statistically significant heterogeneity ($I^2 = 0$ %, $p = 0.68$),(Fig. 2). Visual evaluation of the funnel plot showed a slight publication bias (Fig. 3). However, Egger's test was not statistically significant ($p = 0.774$).

3.4. Coronary heart disease

Data on coronary heart disease were collected from 2 articles, including 98,131 patients (36,494 COP patients and 61,637 control

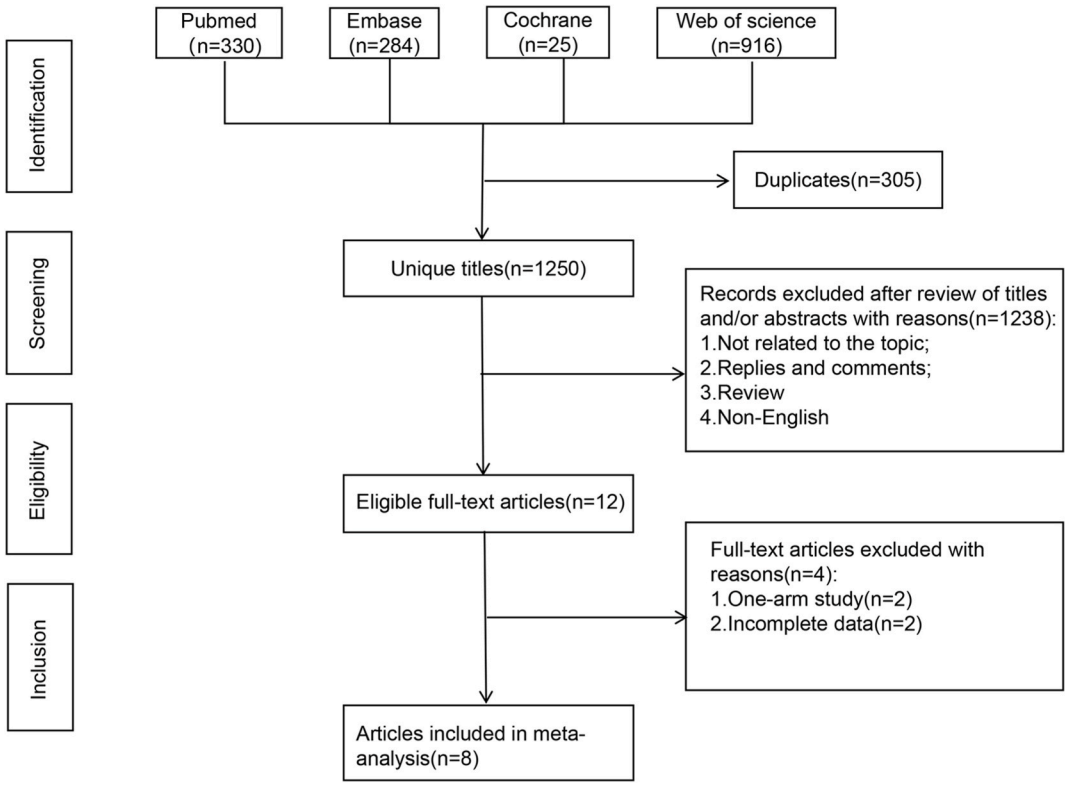

Fig. 1. Flowchart of the systematic search and selection process.

Table 1
Baseline characteristics of include studies and methodological assessment.

Authors	Study period	Country	Study design	Patients (n) CO/Control	Population	Quality score
Lee et al. [5]	2000–2011	China	retrospective	8381/33,524	No arrhythmia, coronary heart disease, heart failure. Age, gender information was complete.	8
Wong et al. [25]	2005–2013	China	retrospective	13,939/55,756	No arrhythmia, coronary heart disease, heart failure. Age, gender information was complete.	8
Huang et al., 2021 [27]	1999–2012	China	retrospective	20,942/62,826	No heart disease, complete information	8
Huang et al., 2018 [28]	1999–2013	China	retrospective	22,258/66,774	No heart disease, complete information	8
Bahng et al. [14]	2002–2017	Korea	retrospective	28,113/28,113	No coronary heart disease, correct registration information, no death within three months after poisoning	8
Abass et al. [24]	2013–2015	Egypt	retrospective	36/40	No heart disease, complete information	7
Eroglu et al. [23]	2005–2006	Turkey	retrospective	30/37	No heart disease, no cerebral infarction, no COPD, no tumor, no hyperbaric oxygen therapy	6
Temrel et al. [26]	2017–2019	Turkey	retrospective	166/68	>18 years old, no heart disease, no blood disease, COPD, mental illness, electrolyte disorder, infectious disease	6

Table 2
Demographics and clinical characteristics of included studies.

Outcomes	Studies	No. of patients CO/Control	HR or OR	95 % CI	p-value	Heterogeneity Chi^2	df	p-value	I^2 (%)
Age (years)	4	93,633/246,993	0.02	[-0.11, 0.15]	0.79	0.32	3	0.96	0
Gender (male)	4	48,085/122,615	1.74	[0.60, 5.09]	0.31	9947.72	3	<0.00001	100
T2DM	4	5625/9578	1.58	[1.23,2.03]	0.0003	118.59	3	<0.00001	97
Hypertension	4	7997/18,016	1.31	[0.95,1.82]	0.10	307.39	3	<0.00001	99
Hyperlipidemia	4	93,633/246,993	1.47	[1.15,1.87]	0.002	142.15	3	<0.00001	98

*Statistically significant; T2DM, diabetes mellitus type 2.

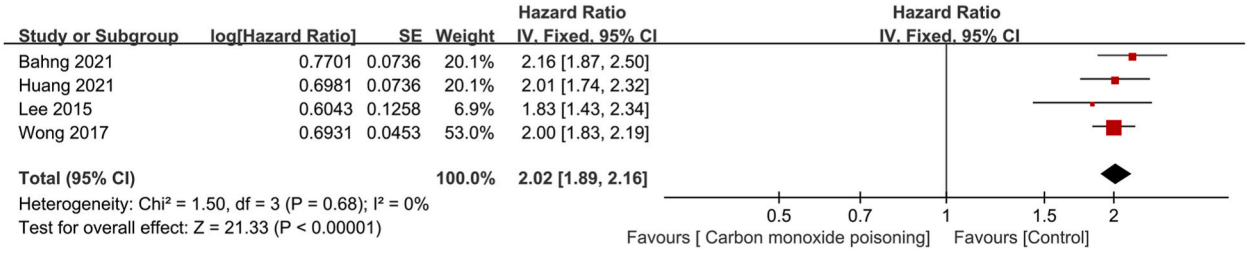

Fig. 2. Forest plots of MACE.

patients) [5,14]. Pooled analysis showed no statistically significant difference in the incidence of coronary heart disease between COP patients and the control group (HR: 1.58; 95 % CI: 0.84, 2.95; $p = 0.15$), with statistically significant heterogeneity ($I^2 = 96$ %, $p < 0.00001$) (Fig. 4).

3.5. Heart failure

Data on heart failure were collected from two aticles, including 125,673 patients (29,323 COP patients and 96,350 control patients) [5,27]. Pooled analysis showed no statistically significant difference in the incidence of heart failure between COP patients and control groups (HR: 1.49; 95 % CI: 0.81, 2.77; $p = 0.20$), with statistically significant heterogeneity ($I^2 = 92$ %, $p = 0.0006$) (Fig. 5).

3.6. Blood gas index

3.6.1. COHb% and PH

COHb% data were obtained from 2 articles, including 310 patients (202 in COP patients and 108 in control group) [24,26]. Combined analysis showed a significantly higher COHb% value in COP patients compared to the control group (HR: 21.57; 95 % CI: 9.66, 33.48; $p = 0.0004$), with statistically significant heterogeneity ($I^2 = 98$ %, $p < 0.00001$) (Fig. 6A).

PH data were obtained from 2 articles, including 310 patients (202 in COP patients and 108 in control group) [24,26]. Pooled

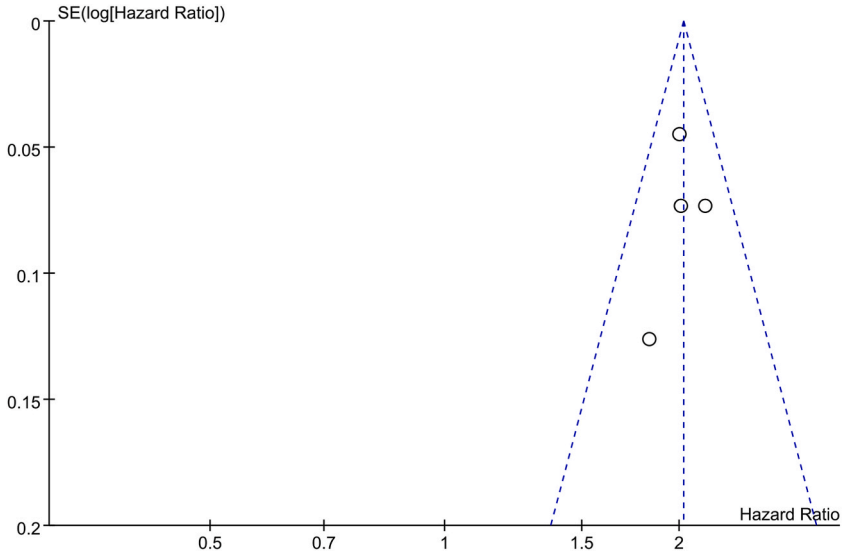

Fig. 3. Funnel plots of MACE.

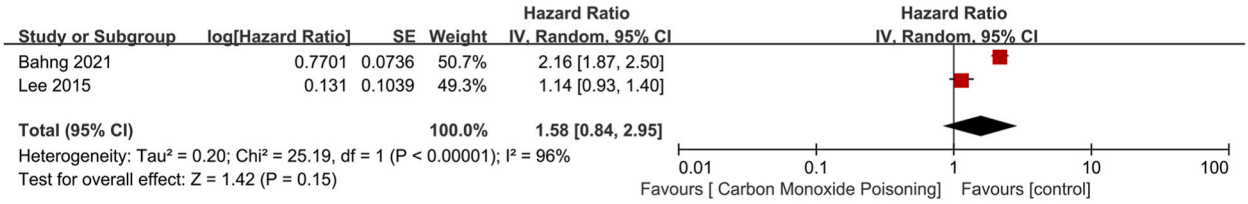

Fig. 4. Forest plots of coronary heart disease.

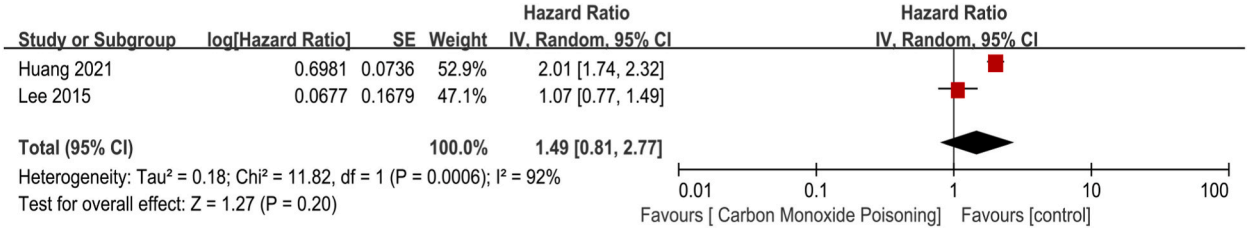

Fig. 5. Forest plots of heart failure.

analysis showed no statistically significant difference in PH between COP patients and control group (HR: 0.10; 95 % CI: 0.25, 0.04; $p = 0.17$), with statistically significant heterogeneity ($I^2 = 98$ %, $p < 0.00001$) (Fig. 6B).

3.7. Electrocardiogram parameter

Ecg parameters QTc (msec), Tp-e (msec), Tp-e/QTc, Tp disp (msec) were obtained from 2 articles, including 304 patients (196 COP patients and 108 control patients) [23,26].No significant difference was foud in QTc (msec) between COP patients and the control group (HR: 1.65; 95 % CI: 43.92, 47.22; $p = 0.94$), with statistically significant heterogeneity ($I^2 = 99$ %, $p < 0.00001$) (Fig. 7A). Significant differences were observed in Tp-e (msec) between COP patients and the control group (HR: 8.93; 95 % CI: 1.38, 16.47; $p = 0.02$), with statistically significant heterogeneity ($I^2 = 70$ %, $p = 0.07$). (Fig. 7B). Tp-e/QTc also showed significant difference between COP patients and control group (HR: 0.06; 95 % CI: 0.05, 0.07; $p < 0.00001$), with statistically significant heterogeneity ($I^2 = 3$ %, $p = 0.31$) (Fig. 7C). No significant difference was found in Tp disp (msec) between COP patients and the control group (HR: 5.25; 95 % CI: 0.13, 10.62; $p = 0.06$), with statistically significant heterogeneity ($I^2 = 66$ %, $p = 0.09$).(Fig. 7D).

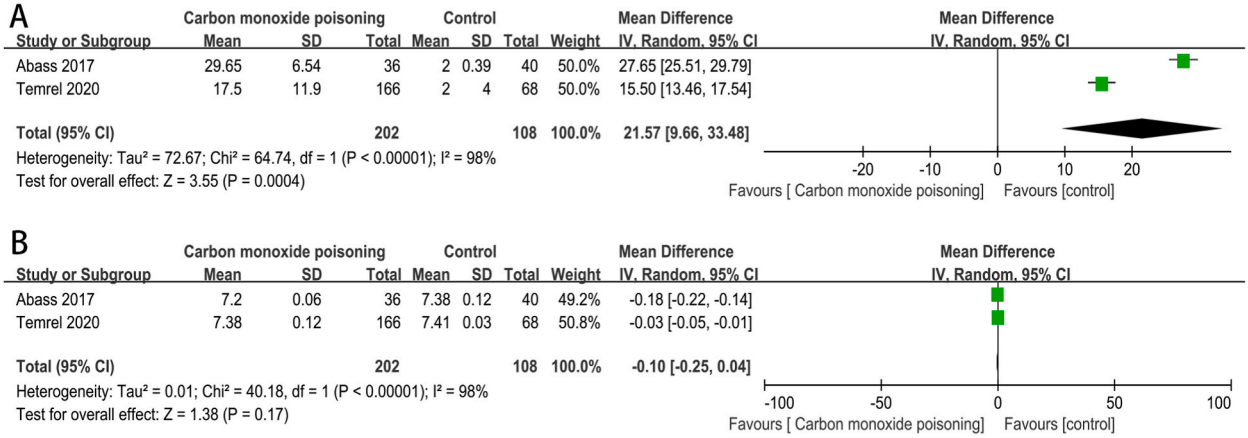

Fig. 6. Forest plots of Blood gas index: **(A)** COHb% **(B)** PH.

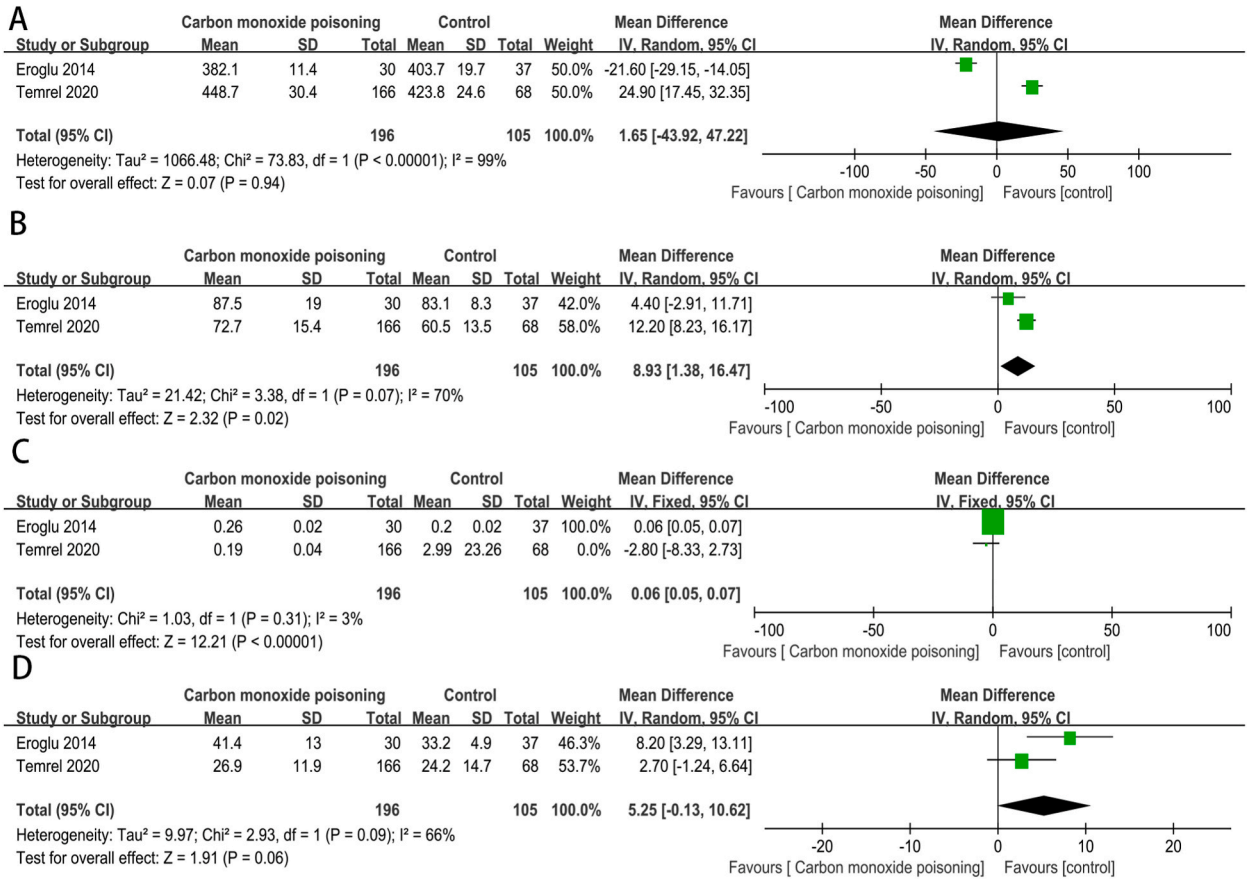

Fig. 7. Forest plots of Electrocardiogram parameter: (**A**) QTc (msec), (**B**) Tp−e (msec), (**C**) Tp−e/QTc, (**D**) Tp disp (msec).

3.8. Subgroup analysis

3.8.1. Region (China, Korea)

Subgroup data by region were obtained from 4 articles, including 251,594 patients (71,375 COP patients and 180,219 control patients) [5,14,25,27]. The analysis showed a statistically significant difference in the incidence of MACE between Chinese COP patients and the control group (HR: 1.99; 95 % CI: 1.85, 2.14; $p < 0.00001$), with no statistically significant heterogeneity ($I^2 = 0$ %, $p < 0.00001$). Similarly, the incidence of MACE in Korean COP patients was significantly different from that in the control group (HR:

2.16; 95 % CI: 1.87, 2.50; $p = 0.17$). (Fig. 8). The incidence of MACE in Korea COP patients was higher than that in Chinese COP patients.

3.8.2. Sex

Subgroup data by sex were obtained from 4 articles including 251,594 patients (71,375 COP patients and 180,219 control patients) [5,14,25,27]. The analysis showed a statistically significant difference in the incidence of MACE between male COP patients and the control group (HR:2.00; 95 % CI: 1.84, 2.18; $p < 0.00001$), with no statistically significant heterogeneity ($I^2 = 0$ %, $p = 0.58$). Similarly, the incidence of MACE in female COP patients was significantly different from that in the control group (HR: 2.07; 95 % CI: 1.88, 2.28; $p < 0.00001$), with no statistically significant heterogeneity ($I^2 = 0$ %, $p = 0.97$). The incidence of MACE was slightly higher in female COP patients compared to male COP patients (Fig. 9).

3.8.3. Hypertension, diabetes, hyperlipidemia

Data for subgroups with underlying diseases, including hypertension, diabetes, and hyperlipidemia, were obtained from two articles, including 153,263 patients (34,881 COP patients and 118,582 control patients) [25,27].The incidence of MACE in COP patients with hypertension was significantly different from that in the control group (HR: 1.51; 95 % CI: 1.34, 1.71; $p < 0.00001$), with no statistically significant heterogeneity ($I^2 = 0$ %, $p = 0.92$). Similarly, the incidence of MACE in COP patients with diabetes mellitus was significantly different from that in the control group (HR: 1.49; 95 % CI: 1.27, 1.75; $p < 0.00001$), with no statistically significant heterogeneity ($I^2 = 0$ %, $p = 0.89$). The incidence of MACE in COP patients with hyperlipidemia was also significantly different from that in the control group (HR: 1.58; 95 % CI: 1.33, 1.88; $p < 0.00001$), with no statistically significant heterogeneity ($I^2 = 0$ %, $p = 0.52$) (Fig. 10).

3.8.4. Age

Subgroup data for patients aged ≥65 years were obtained from four articles including 251,594 patients (71,375 COP patients and 180,219 control patients) [5,14,25,27]. Subgroup data for patients aged 50–64 years were obtained from two articles, including 125, 673 patients (29,323 COP patients and 96,350 control patients) [5,27], No significant difference was found in the incidence of MACE between COP patients aged ≥65 years and the control group (HR: 1.40; 95 % CI: 0.99, 1.99; $p = 0.06$), with statistically significant heterogeneity ($I^2 = 76$ %, $p = 0.005$). Similarly, no significant difference was observed in the incidence of MACE between COP patients aged 50–64 and the control group (HR: 1.23; 95 % CI: 0.44, 3.43; $p = 0.70$), with statistically significant heterogeneity ($I^2 = 89$ %, $p = 0.002$). (Fig. 11).

3.9. Sensitivity analysis

A one-way sensitivity analysis was conducted to evaluate the impact individual studies on the pooled HR for major adverse cardiovascular events (MACE) by sequentially excluding each study. The pooled HR remained unaltered after excluding any individual study. (Fig. 12).

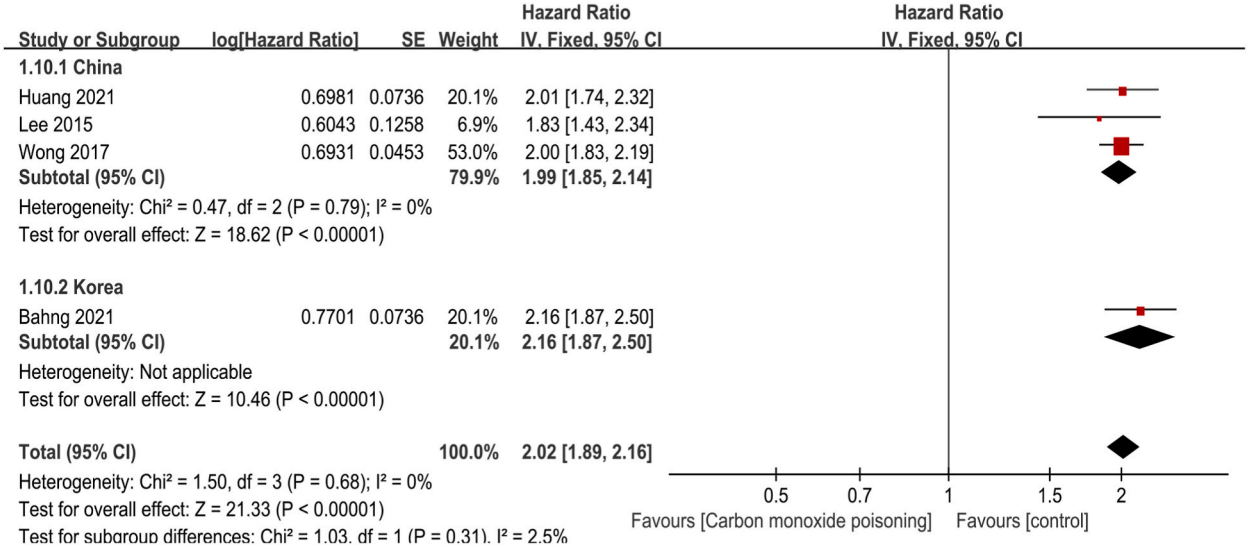

Fig. 8. Forest plots of Region (China, Korea).

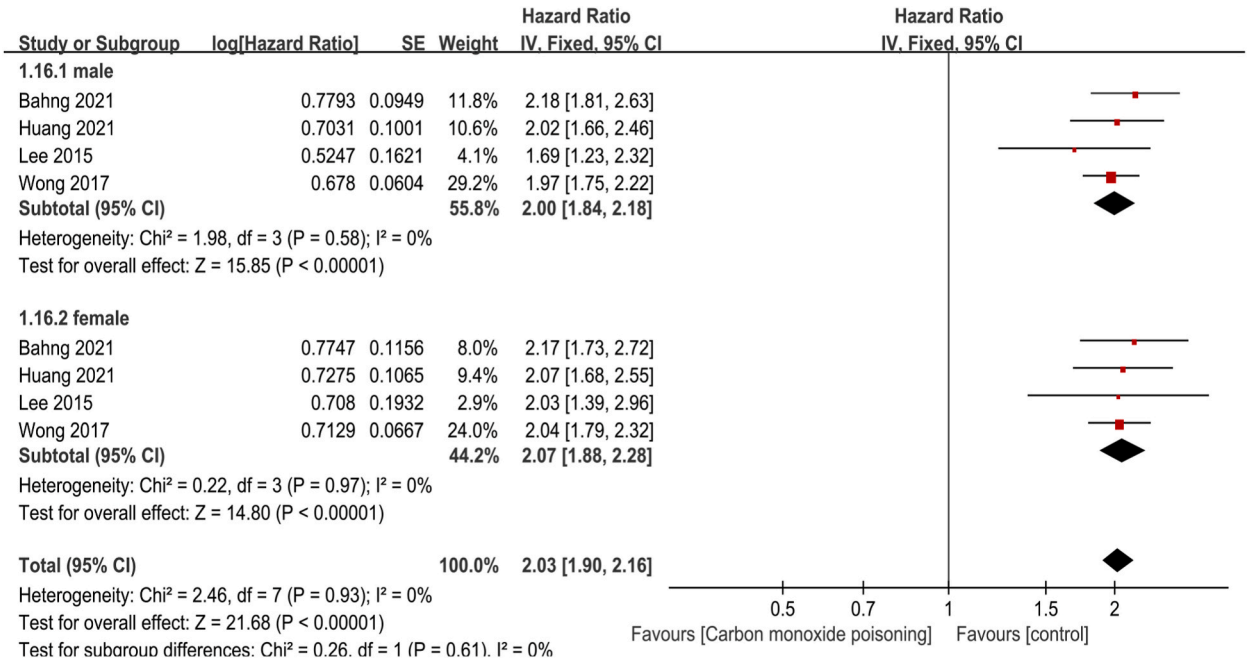

Fig. 9. Forest plots of sex.

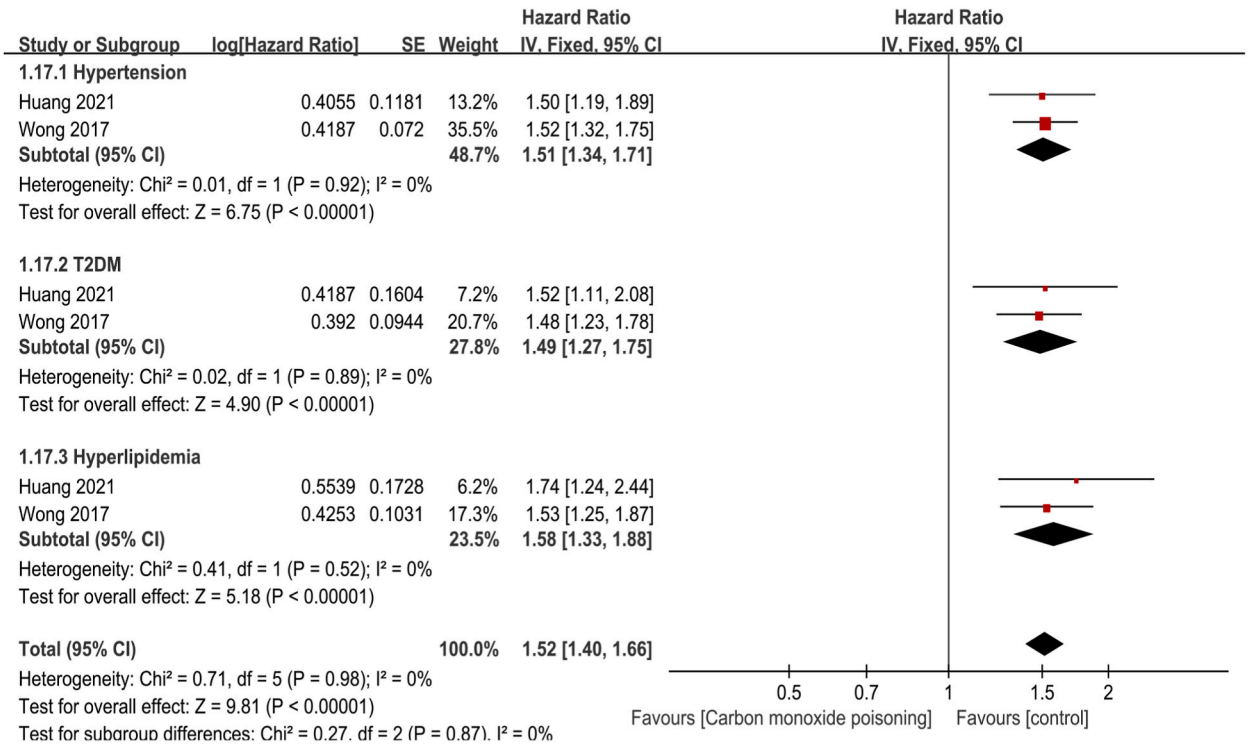

Fig. 10. Forest plots of Hypertension, diabetes, hyperlipidemia.

4. Discussion

COP can affect multiple organs, resulting in diverse complications. Severe manifestations include cerebral edema with impaired consciousness, pulmonary edema leading to respiratory failure, diminished myocardial contractility, arrhythmias, heart failure and

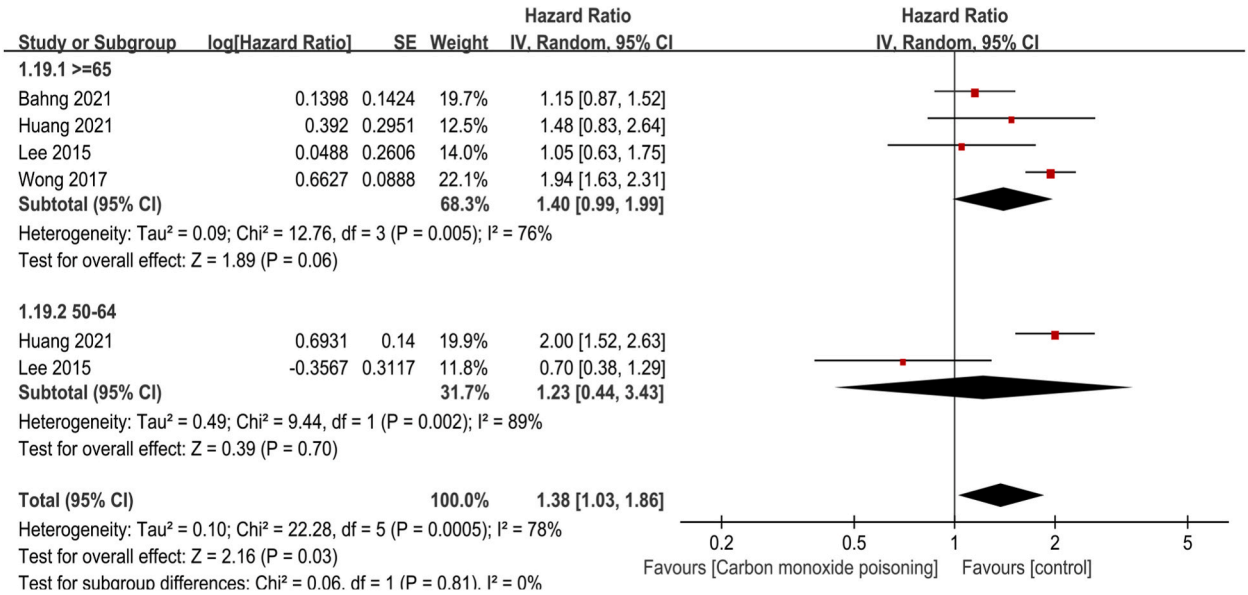

Fig. 11. Forest plots of age.

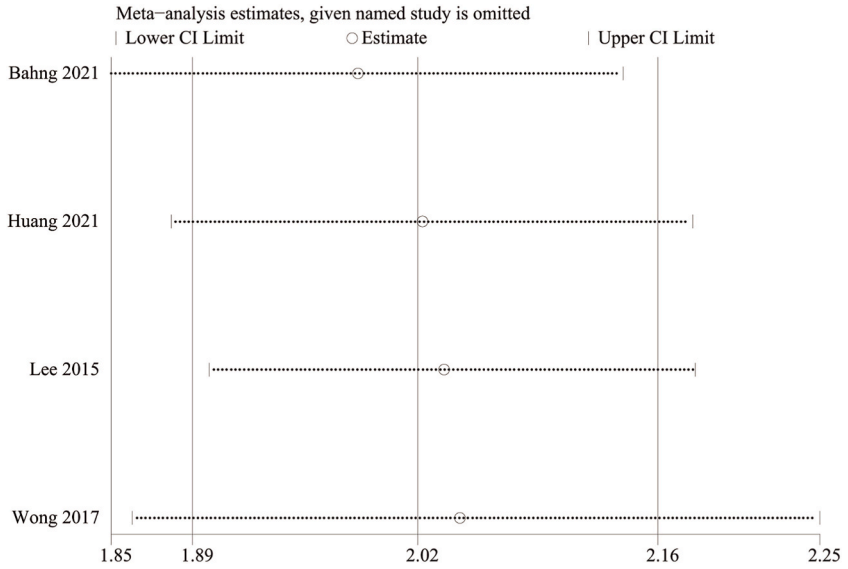

Fig. 12. Sensitivity analysis of MACE.

renal failure [11,29]. Due to the heart's heightened oxygen demand, COP significantly jeopardizes the cardiovascular system, contributing to myocardial damage in approximately one-third of patients with moderate to severe. This cardiac involvement increases long-term mortality rates [16,30,31]. Although neurologic interventions are well-established in clinical protocols, cardiovascular considerations are frequently overlooked. Timely identification and targeted cardiovascular management may improve the prognosis of patients with COP-related cardiovascular complications.

To strengthen the theoretical basis for clinical practice, this study systematically reviewed and analyzed the relationship between COP and the development of cardiovascular diseases. The findings indicate no statistically significant difference in the incidence of coronary heart disease and heart failure between COP patients and the control group when analyzed separately. However, these conclusions are based on a limited dataset comprising only two studies and two cohorts, warranting further investigations due to the small sample size. In contrast, the occurrence of MACE in COP patients exceeded that in the control group, aligning with previous research findings. Multiple mechanisms underlie the influence of COP on MACE incidence, primarily: direct binding of CO with myoglobin, resulting in cardiac dysfunction [11]; CO-induced inhibition of oxidative phosphorylation, leading to cardiac dysfunction [16,32]; hypoxia prompting increased cardiac output for compensation, culminating in prolonged compensation and eventual heart

failure [33]; cardiomyocyte apoptosis following poisoning [34]; inflammatory responses triggered by nitric oxide and reactive oxygen species causing cardiac damage [1]. The mechanisms contributing to coronary heart disease encompass: COP-induced reduction in oxygen supply and increased myocardial contractility, potentially leading to myocardial infarction in individuals with underlying coronary artery disease [1]; CO-induced endothelial dysfunction, increased free radical production, augmented coronary artery constriction, and subsequent myocardial damage [16]; elevated thrombosis risk due to increased CO binding to fibrinogen-bound heme and platelet aggregation [31].

Regarding ECG parameters, Tp-e (milliseconds) and Tp-e/QTc indices in COP patients differed siginicantly from the control group. The arrhythmia mechanisms in COP involve diminished ATP due to disruoted oxidative gradient on the action potential, leading to arrhythmia [35]. CO-mediated elevation of late sodium current is another factor contributing to arrhythmogenesis [36].

The findings revealed significantly higher levels of COHb% in COP patients compared to the control group. The diagnosis of COP relies on a clinical triad: (1) consistent symptoms, (2) recent CO exposure history, and (3) elevated COHb levels with COHb% serveing as a pivotal diagnostic marker [1]. Studies indicate that the risk of cardiovascular events is heightened at CO-Hb levels of 5–10 %, lowering the threshold for angina, arrhythmia, and myocardial infarction [37]. A more severe risk of atherosclerosis and sudden cardiac death emerges at CO-Hb levels of 20–30 % [37,38]. The pH value in blood gas analysis, an indicator of blood pH, did not differ significantly between COP patients and the control group, suggesting that COP has little influence on blood pH.

In subgroup analysis, the incidence of MACE in COP patients from South Korea was slightly higher than in China, though not statistically significant. This suggests similar physical conditions and corresponding degrees of cardiovascular disease after COP in Chinese and Korean populations. Limited Korean data necessitates further investigation. While female COP patients exhibited a slightly higher incidence of major adverse cardiovascular events (MACE) compared to males, the difference lacked statistical significance. It suggested comparable cardiovascular risk between genders post-COP. COP demonstrated consistent MACE incidence across diverse underlying diseases, emphasizing a similar impact on cardiovascular outcomes. MACE incidence did not significantly differ between COP patients aged $\geq$65 and 50–64 years and controls across different age groups. These results indicate a similar effect of COP on MACE incidence in individuals over 50 years old, while further research is needed in those under 50 years of age.

This paper has notable limitations. Firstly, ethical constraints preclude prospective clinical studies on poisoning, necessitating the inclusion of retrospective analyses. Additionally, the study predominantly involves participants from China and South Korea, lacking representation from Europe and Africa, introducing regional limitations. Lastly, concerning demographic characteristics, a statistical disparity exists in the baseline balance of diabetes and hyperlipidemia.

This paper offers several merits. Firstly, it pioneers as the inaugural meta-analysis exploring the relationship between COP and cardiovascular diseases, a notably innovative subject. Secondly, it meticulously examines numerous research aspects across multiple subgroups, offering diverse avenues for future exploration. Thirdly, the inclusion of a sizable sample enhances the reliability of the conclusions drawn. Notably, these findings align with previous studies, providing a theoretical foundation for the prevention and treatment of complications, thus holding practical significance.

5. Conclusion

Comprehensive analysis revealed an increased incidence of MACE in COP. COP affected certain ECG parameters. The incidence of MACE after COP showed no significant difference between Korean and Chinese populations.The incidence of MACE after COP did not significantly differ by gender or underlying disease. The incidence of MACE after COP was comparable in individuals aged 50 years and older. Given heterogeneity and potential bias, emergency physicians should consider the likelihood of cardiovascular events in COP patients.

Data availability Statement

Data included in article/supp. material/referenced in article.

Ethics Statement

Ethical review and approval were not required for the study on human participants in accordance with the local legislation and institutional requirements.

Funding

This study was funded by the Wu Jieping Medical Foundation Project (Project No. 320.6750.2022-26-12).

CRediT authorship contribution statement

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e34062.

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