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META-ANALYSIS

Therapy area: Other

Total and drinking water intake and risk of all-cause and cardiovascular mortality: A systematic review and dose-response meta-analysis of prospective cohort studies

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Summary

Background: Understanding the association between water consumption and mortality is important for guiding consumers and prioritizing dietary guidelines to reduce the risk. Therefore, in the current study, we conducted a systematic review and dose–response meta-analysis of prospective cohort studies to summarise the association between total water and drinking intake and risk of mortality from all causes and CVD.

Method: A comprehensive search was performed on PubMed/Medline, Scopus, and ISI Web of Science up to February 2020. The random-effects model was used to calculate the pooled effect size (ES) and 95% confidence interval.

Result: Seven prospective cohort studies were included in the systematic review and meta-analysis. During the follow-up period of 6 to 19.1 years, 14 754 deaths (7611 from cardiovascular disease) occurred among 116 816 participants. No significant association was found between drinking water intake and all-cause mortality (ES: 0.82; 95% CI: 0.63-1.08, $I^2 = 77.3\%$, $P = .16$). Total water intake was not associated with all-cause mortality (ES: 0.94; 95% CI: 0.82-1.08, $I^2 = 66.5\%$, $P = .41$). However, a significant inverse association was seen between total water intake and risk of CVD mortality (ES: 0.86; 95% CI: 0.78-0.95, $I^2 = 0\%$, $P = .002$). Linear dose–response meta-analysis revealed a significant inverse association between total water intake and all-cause mortality by an additional one cup per day (pooled ES: 0.98; 95% CI: 0.97-0.99, $P = .001$). Furthermore, each additional cup of total water intake per day was associated with a 3% lower risk of death from CVD (pooled ES: 0.97; 95% CI: 0.96-0.98, $P < .001$).

Conclusion: High consumption of total water is associated with a lower risk of CVD mortality. However, total water intake was not associated with risk of all-cause mortality.

1 | INTRODUCTION

Several modifiable risk factors, such as tobacco use, unhealthy diet, and excessive alcohol intake, are linked to the greater risk of non-communicable diseases, and some evidence suggests that adequate water intake may improve health outcomes.[1] Adequate hydration and water intake are necessary for critical physiological and metabolic processes.[2] Using this framework, dietary guidelines recommended

consumption of 2 and 2.5 L per day of water for females and males, respectively.[3] Low fluid consumption and inadequate water intake may provoke dehydration. This condition can lead to low-grade inflammation in the human body. It should be noted that the low-grade inflammation in long-term was associated with inanition and progression of some chronic diseases,[4] CVD,[5] and mortality.[6] Previous meta-analysis on the association of specific beverages, such as tea,[7,8] coffee,[9,10] and alcohol,[11-13] with mortality reported inverse, positive, or null associations. Findings on the link between water intake and longevity remain an unresolved question. While total water intake from foods and beverages was associated with a lower risk of mortality in some investigations,[14] others failed to find such evidence.[15,16] No information is available about the strength and shape of the dose–response relationship between water intake and risk of mortality. On the other hand, understanding the association between water consumption and mortality is important for guiding consumers and prioritizing dietary guidelines to reduce the risk. Therefore, in the current study, we conducted a systematic review and dose–response meta-analysis of prospective cohort studies to summarise the association between total water intake from foods and beverages and drinking intake and risk of mortality from all causes and CVD.

Review criteria

A systematic search was conducted in PubMed/Medline, Scopus, and ISI Web of Science, from inception up to February 2020.
The systematic review adheres to the PRISMA guidelines, with articles screening and data extraction being performed by two independent reviewers.

Message for the clinic

Total water intake was inversely associated with the risk of CVD mortality.
Total water intake significantly reduces all-cause mortality for an additional cup of total water intake per day.

2 | METHODS

Findings from this systematic review and meta-analysis were reported based on Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) guideline.[17]

2.1 | Search strategy

A systematic literature review was conducted by using the databases PubMed/Medline, Scopus, and ISI Web of Science up to February 2020 with no language or time restriction. Details of the search terms are provided in Table S1. Furthermore, the reference list of the relevant articles was manually searched.

2.2 | Inclusion criteria

Published studies that met the following criteria were included: (i) observational prospective studies conducted on adults; (ii) reported effect sizes (ESs) including hazard ratios (HRs) or relative risks (RRs) or odds ratios (ORs) with corresponding 95% CIs for the association between intakes of total water from all foods and beverages as well as drinking water as the exposure of interest and mortality from all causes and CVD as the outcome of interest.

2.3 | Exclusion criteria

We excluded letters, comments, reviews, meta-analyses, ecologic studies, and studies performed on children or adolescences. Since fluid intake of patients is affected by their disease, we also did not include studies that were conducted among chronic kidney disease or hemodialysis patients, critically ill patients, and those with acute respiratory distress syndrome. We did not include unpublished studies too.[18] All outcomes were classified based on the World Health Organization's international classification of disease criteria.

2.4 | Data extraction

The selection and data extraction process were executed by 2 independent reviewers (MM and FH). We extracted the following data from each study: name of the first author, publication year, study design, location of the study conducted, gender, the sample size of the cohort, the age range at entry, duration of follow-up, exposure, method of assessment of exposure, incidence of death, comparison categories, and relevant effect size along with 95% CIs and list of confounders adjusted in the statistical analysis.

2.5 | Risk of bias assessment

We used the Risk OF Bias IN Non-randomised Studies of Exposures (ROBINS-E) tool to assess the risk of bias. The ROBINS-E tool comprises 7 domains: (i) bias caused by confounding, (ii) bias in selection of participants into the study, (iii) bias in the classification of exposures, (iv) bias caused by departure from intended exposures, (v) bias caused by missing data, (vi) bias in the measurement of outcomes, and (vii) bias in the selection of reported results. Studies were categorised as low risk, moderate risk, serious risk, and critical risk of bias under each domain. The results of risk of bias assessment are presented in Table S2.

2.6 | Statistical analysis

Risk estimates, hazard ratios, and odds ratios (and 95% CIs) were considered the same. As different studies might report different exposure categories, we used the study-specific effect size for

the highest versus lowest category of water intake for the meta-analysis. The analyses were performed with the use of a random-effects model, in which we calculated both Q-statistic and I^2 as indicators of heterogeneity. We interpreted I^2 values according to the Cochrane Handbook thresholds (0%-40%, might not be important; 30%-60%, might represent moderate heterogeneity; 50%-90%, might represent substantial heterogeneity; and 75%-100%, considerable heterogeneity).[18] As random-effects model can account for variation between studies, it can provide more conservative results than a fixed-effects model. For studies that reported effect sizes separately for drinking water and other fluids, we first combined the estimates using the fixed-effects model to obtain an overall estimate, and then, the pooled effect size was included in the meta-analysis. In the study of Wu et al that reported effect size for CKD and non-CKD patients separately, we included only non-CKD patients in the meta-analysis. We considered studies that had done on water intake and CVD mortality as all-cause mortality in this meta-analysis because the CVD is a significant part of all-cause mortality. We conducted a sensitivity analysis, using a fixed-effects model, in which each prospective cohort study was excluded to examine the influence of that study on the overall estimate. In case of finding a significant between-study heterogeneity, we performed subgroup analysis to examine possible sources of heterogeneity. Between-subgroup heterogeneity was examined through the fixed-effects model. Publication bias was examined by visual inspection of funnel plots. Formal statistical assessment of funnel plot asymmetry was also done with the use of Egger's test. In case of significant publication bias, the trim-and-fill method was used to detect the effect of missing studies on the overall effect of meta-analysis.

A method suggested by Greenland and Orsini was used to compute the trend from the ORs/RRs/HRs estimates and their 95% CIs across categories of water intake.[19] In this method, the distribution of cases and the ORs/RRs/HRs with the variance estimates for ≥3 quantitative categories of exposure were required. We assigned the median or mean amount of water intake in each category to the corresponding ES for each study. For studies that reported the water intake as range, we estimated the midpoint in each category by calculating the mean of the lower and upper bound. When the highest and lowest categories were open-ended, the length of these open-ended intervals was assumed to be the same as that of the adjacent intervals. A two-stage random-effects dose–response meta-analysis was applied to examine a possible non-linear association between water intake and mortality. This was done through modelling of water intake and restricted cubic splines with three knots at fixed percentiles of 10%, 50%, and 90% of the distribution. Based on the Orsini method,[19] we calculated restricted cubic spline models using the generalised least-squares trend estimation method, which takes into account the correlation within each set of reported ORs/RRs/HRs. Then, all the study-specific estimates were combined with the use of the restricted maximum likelihood method in a multivariate random-effects meta-analysis. A probability value for non-linearity was estimated using null hypothesis testing in which the coefficient of the second spline was considered equal to 0. A linear dose–response association was investigated using the two-stage generalised least-squares trend estimation method. First, study-specific slope lines were estimated, and then, these lines were combined to obtain an overall average slope. Study-specific slope lines were combined using a random-effects model. Statistical analyses were conducted using STATA version 14.0. $P < .05$ was considered as statistically significant for all tests, including Cochran's Q test.

3 | RESULTS

3.1 | Literature search

We identified 1327 articles in our initial search. After exclusion of duplicate papers and those that did not meet the inclusion criteria, we identified 11 full-text articles of potentially relevant studies. After full-text review, we excluded an additional 4 articles for the following reasons: studies that enrolled patients with chronic renal diseases or hemodialysis patients, papers that were conducted on critically ill patients, one document that was conducted on children. Finally, seven cohort studies were included in the current systematic review and meta-analysis.[5,6,14-16,20,21] Four studies had reported effect sizes for all-cause mortality[6,15,20,21] and 6 studies for CVD mortality.[5,6,14-16,21] Of these publications, seven had reported effect sizes for total water[5,6,14-16,20,21] and four for drinking water.[5,16,20,21] A flow diagram of study selection is shown in Figure 1.

3.2 | Characteristics of included studies

Characteristics of included prospective cohort studies are presented in Table 1. All included studies were published between 2002 and 2018. Participants in these studies ranged from 1055 to 35 362 people, with an age range between 18 and over 70 years. The follow-up duration of the included cohort was between 6 and 19.1 years. In total 116 816 participants were enrolled in the current study. Among the included studies, 3 studies were done in the United States,[5,6,20] 2 in Australia,[15,21] and the rest in Japan[14] and Netherland.[16] The total number of deaths from all causes was 14 754 and from CVD was 7611. One study included only women[21] and 4 studies[5,14,16,20] reported effect size for men and women separately. Out of the remaining studies, two[6,15] reported effect sizes for both genders in combination. To assay fluid/water intake, all studies had used a food frequency questionnaire and 24h recall. All included studies applied record linkage for assessment of mortality as the outcome. All studies adjusted the associations for age. Most cohorts controlled for some conventional risk factors, including body mass index (n = 5), smoking (n = 5), and energy intake (n = 3). Some others had also adjusted for alcohol consumption (n = 2) and other dietary variables

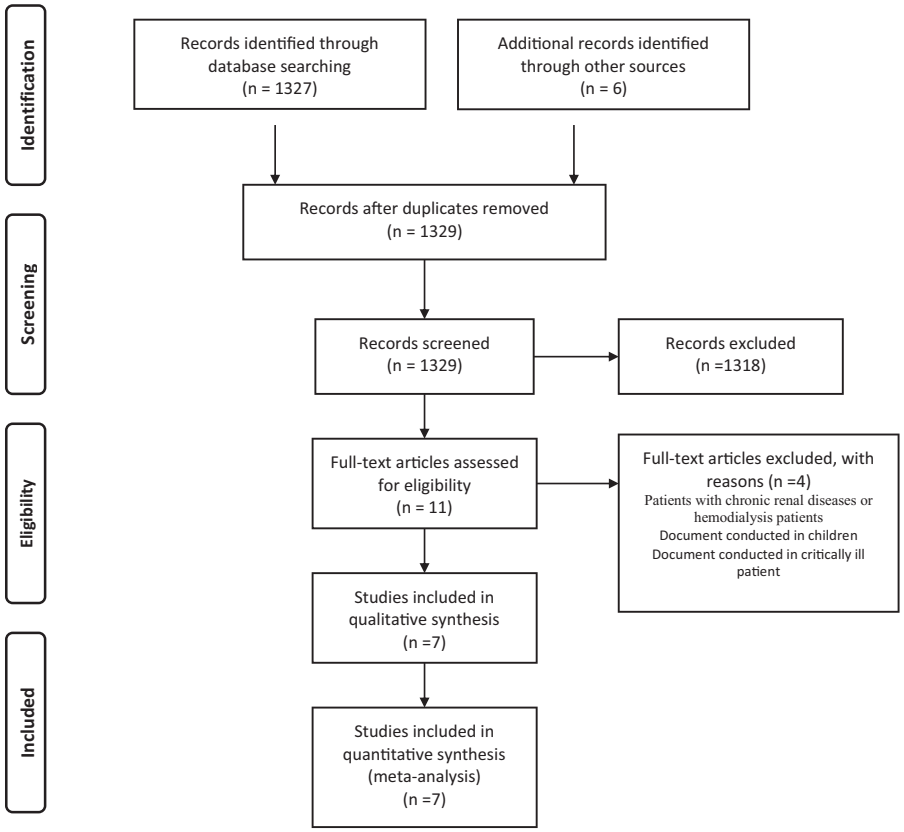

FIGURE 1 Flow diagram of study selection

(n = 2). Based on the ROBINS-E tool, two studies had a low-risk of bias in all components (Table S2).

3.3 | Finding from the meta-analysis on total water intake and all-cause mortality

Six cohort studies investigated the association between total water intake and risk of all-cause mortality.[5,6,14-16,20] These studies included a total of 115 815 participants, among them 14 250 deaths occurred. The summary effect size for all-cause mortality comparing the highest and lowest intakes of total water was 0.94 (95% CI: 0.82-1.08, $P = .41$), indicating no clear significant association between total water intake and risk of all-cause mortality (Figure 2). However, significant between-study heterogeneity was seen ($I^2 = 66.5\%$; $P = .001$).

3.4 | Finding from the meta-analysis on drinking water intake and all-cause mortality

Examining the association between drinking water consumption and risk of all-cause mortality in three studies,[5,16,20] which involved a total of 51 474 participants with 9279 deaths, we found no significant association (pooled ES comparing the highest and lowest intakes: 0.82; 95% CI: 0.63-1.08, $P = .65$), with high heterogeneity among the studies ($I^2 = 77.3\%$; $P = .001$) (Figure 3).

3.5 | Finding from the meta-analysis on total water intake and CVD mortality

Five cohort studies examined the association between total water intake and risk of CVD mortality.[5,6,14-16] These studies included a total of 91 051 participants among them 7107 mortality cases were found. The summary effect size for CVD mortality, comparing the highest and lowest total water intakes, was 0.86 (95% CI: 0.78-0.95, $P = .002$), indicating a significant inverse association between total water intake and risk of CVD mortality (Figure 4). No significant heterogeneity among the studies was observed ($I^2 = 0\%$; $P = .63$).

3.6 | Linear and non-linear dose–response analysis

Four of six studies on the association between total water intake and all-cause mortality were included in the dose–response analysis[6,14-16] (Figure 5). There was evidence of a non-linear association (P-nonlinearity = 0.006). Furthermore, linear dose–response meta-analysis revealed a significant inverse association between total water intake and all-cause mortality by an additional one cup per day (Pooled ES: 0.98; 95% CI: 0.97-0.99, $P = .001$) (Figure S1).

In the dose–response analysis of total water intake and CVD mortality, based on four studies, out of six studies, we found a non-linear

TABLE 1 Characteristics of included cohort studies on the association between fluid intake and mortality in adults aged >18 years

Author	Country	Age, y[a]	n	Follow-up, y	Cases, n	Exposure	Exposure assessment	Outcome	Comparison	ES (95% CI)[b]	Adjustment[c]
Lim et al 2017	Australia	>70	F: 1055	10	142	Total fluid Plain water	beverage intake questionnaire	CVD mortality	Per one cup	HR: 0.98 (0.93-1.03) HR: 1.02 (0.95-1.10)	1,5
Lim et al 2017	Australia	>70	F: 1055	10	362	Total fluid Plain water	life style questionnaire	All-cause mortality	Per one cup	HR: 0.98 (0.95-1.01) HR: 0.99 (0.95-1.04)	1,5
Chan et al 2002	USA	>38	F: 12 017	6	118	Plain water Fluids other than water	life style questionnaire	CVD mortality	T3 vs. T1	RR: 0.52 (0.27-1.03) RR:3.32 (1.18-9.30)	1,5,6,7,9,10,11,13
Chan et al 2002	USA	38	M: 8280	6	128	Plain water Fluids other than water	life style questionnaire	CVD mortality	T3 vs. T1	RR: 0.39 (0.22-0.67) RR: 1.34 (0.59-3.04)	1,5,6,7,9,10,11,13
Cui et al 2018	Japan	40-79	F: 35 362	19.1	1707	Total water	FFQ, self-reported	CVD mortality	Q5 vs. Q1	HR: 0.79 (0.66-0.95)	1,5,7,8,9,10,11,
Cui et al 2018	Japan	40-79	M: 22 939	19.1	1637	Total water	FFQ, self-reported	CVD mortality	Q5 vs. Q1	HR: 0.88 (0.72-1.07)	1.5.7.8.9.10.11
Palmer et al 2014	Australia	>49	M/F: 3858	13.1	547	Total fluid	FFQ, self-reported	CVD mortality	Q4 vs. Q1	HR: 0.91 (0.70-1.19)	1,6,10
Palmer et al 2014	Australia	>49	M/F: 3858	13.1	547	Total fluid	FFQ, self-reported	All-cause mortality	Q4 vs. Q1	HR: 0.91 (0.68-1.21)	1,6,10
Wu et al 2016	USA	>20	2128	15.4	473	Total fluid	24-h recall, interview	CVD mortality (non-CKD)	Q4 vs. Q1	HR: 0.98 (0.75-1.28)	1,2,3,4,5,11
Wu et al 2016	USA	>20	2182	15.4	473	Total fluid	24-h recall	CVD mortality (non-CKD)	Q4 vs. Q1	HR: 1.00 (0.76-1.30)	1,2,3,4,5,11
Leurs et al 2010	Netherland	55-69	F: 2958	10	537	Total fluid Plain water	FFQ, self-reported	IHD mortality	Q4 vs. Q1	HR: 1.04 (0.67-1.61) HR: 0.70 (0.37-1.34)	1,7,11
Leurs et al 2010	Netherland	55-69	M: 3509	10	1252	Total fluid Plain water	FFQ, self-reported	IHD mortality	Q4 vs. Q1	HR: 1.03 (0.73-1.47) HR: 1.33 (0.76-2.36)	1,7,11
Leurs et al 2010	Netherland	55-69	F: 2958	10	291	Total fluid Plain water	FFQ, self-reported	Stroke mortality	Q4 vs. Q1	HR: 0.60 (0.31-1.15) HR: 0.49 (0.19-1.24)	1,7,11
Leurs et al 2010	Netherland	55-69	M: 3509	10	417	Total fluid. Plain water	FFQ, self-reported	Stroke mortality	Q4 vs. Q1	HR: 0.77 (0.45-1.30) HR: 0.85 (0.31-2.29)	1,7,11
Kant et al 2017	USA	>25	F: 12 660	10	3032	Total water Plain water	24-h recall- interview	All-cause mortality	Q4 vs. Q1	HR: 1.15 (0.96-1.39) HR: 0.98 (0.83, 1.14)	3,4,5,8,11
Kant et al 2017	USA	>25	M: 12 050	10	3504	Total water Plain water	24-h recall- interview	All-cause mortality	Q4 vs. Q1	HR: 1.24 (1.09-1.42) HR: 1.15 (1.00, 1.33)	3,4,5,8,11

Abbreviation: CI, confidence interval; F, female; FFQ, food frequency questionnaire; HR, hazard ratio; M, male; RR, risk ratio; US, United States.

[a]Presented as mean or range.

[b]Effect size.

[c]Adjustments: age (1), gender (2), race (3), ethnicity (4), BMI (5), Fluid intake (6), total energy (7), alcohol consumption (8), education (9), family history of CVD (10), smoking (11), physical activity (12), and hypertension (13).

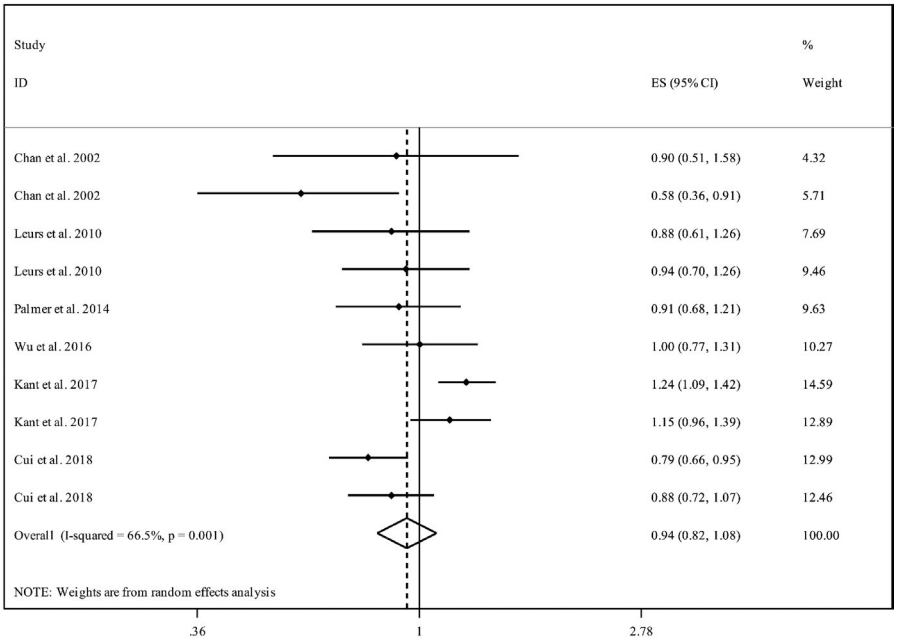

FIGURE 2 Forest plot for the association between total water intake and risk of all-cause mortality in adults aged >18 years, expressed as the comparison between the highest and lowest categories of total water intake. Horizontal lines represent 95% CIs. Diamonds represent the pooled estimates from the random-effects analysis. CI, confidence interval; ES, effect size

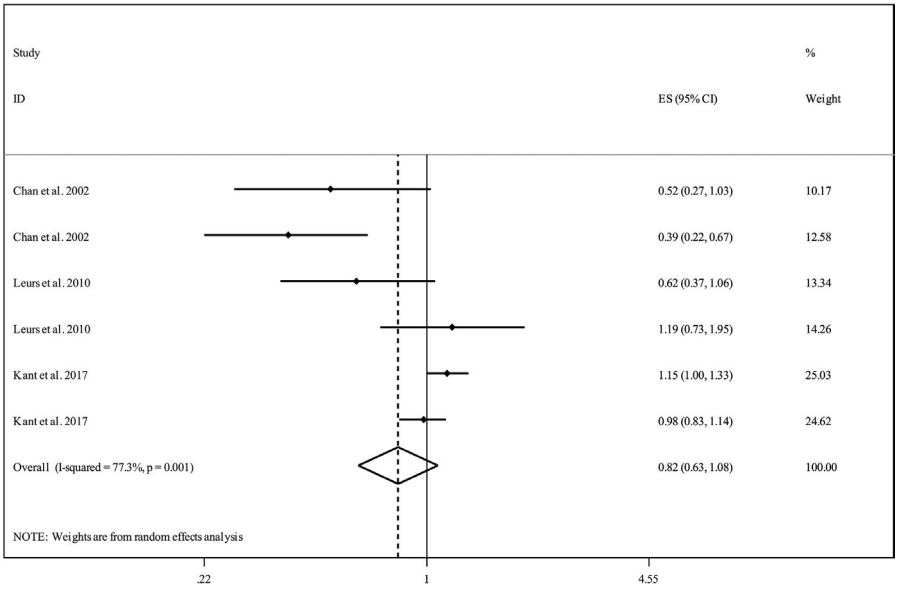

FIGURE 3 Forest plot for the association between drinking water intake and risk of all-cause mortality in adults aged >18 years, expressed as the comparison between the highest and lowest categories of total water intake. Horizontal lines represent 95% CIs. Diamonds represent the pooled estimates from the random-effects analysis. CI, confidence interval; ES, effect size

association (P-non-linearity = 0.004) (Figure 6). Based on linear dose–response analysis, each additional cup of total water intake per day was associated with a 3% lower risk of death from CVD (pooled ES: 0.97; 95% CI: 0.96-0.98, $P < .001$) (Figure S2).

3.7 | Subgroup, sensitivity analyses, and publication bias

To test the robustness of the results and investigate the between-study heterogeneity, we conducted subgroup analyses. Table S3 presents findings for the different subgroups. In terms of total water intake and risk of all-cause mortality, a significant inverse association was obtained in studies conducted in the United States. However, a significant positive association was seen in studies conducted in the United States and those that had applied food recall for total water intake assessment. In terms of total water intake and risk of CVD mortality a significant inverse association was seen in studies that conducted in US and non-US countries, those that performed in females and males, studies that had used FFQ for total water intake assessment, studies with a >10 years follow-up duration and those that controlled for BMI and energy intake in their analysis. In terms

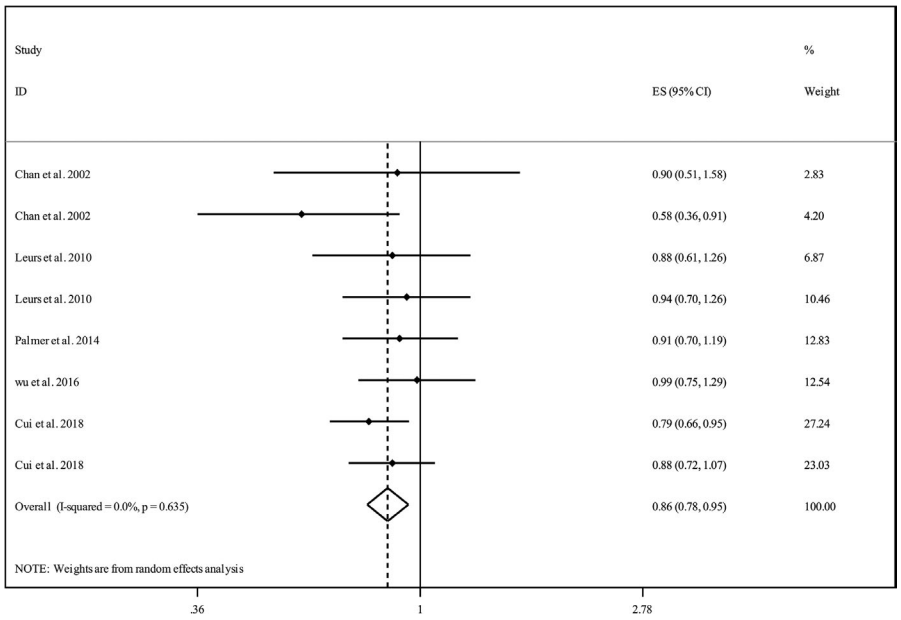

FIGURE 4 Forest plot for the association between total water intake and risk of CVD mortality in adults aged >18 years, expressed as the comparison between the highest and lowest categories of total water intake. Horizontal lines represent 95% CIs. Diamonds represent the pooled estimates from the random-effects analysis. CI, confidence interval; CVD, cardiovascular disease; ES, effect size

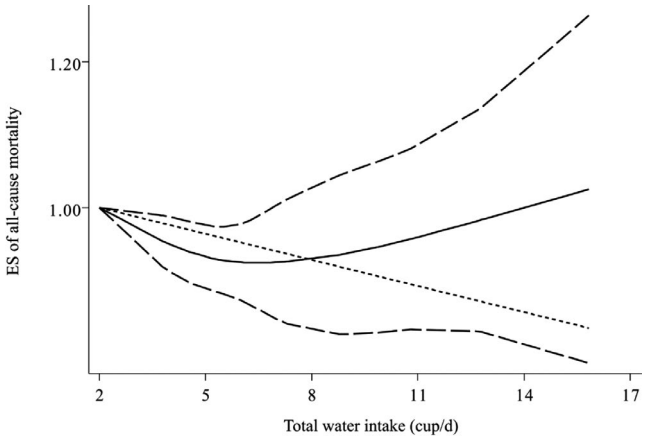

FIGURE 5 Non-linear dose–response association of total water intake (based on cup/day) with risk of mortality from all-cause in adults aged ≥18 years. Total water intake was modeled with restricted cubic splines in a multivariate random-effects dose–response model. Dotted line indicates the linear model. Solid line indicates the spline model. Dashed line presents the 95% CI. CI, confidence interval; CVD, cardiovascular disease; ES, effect size

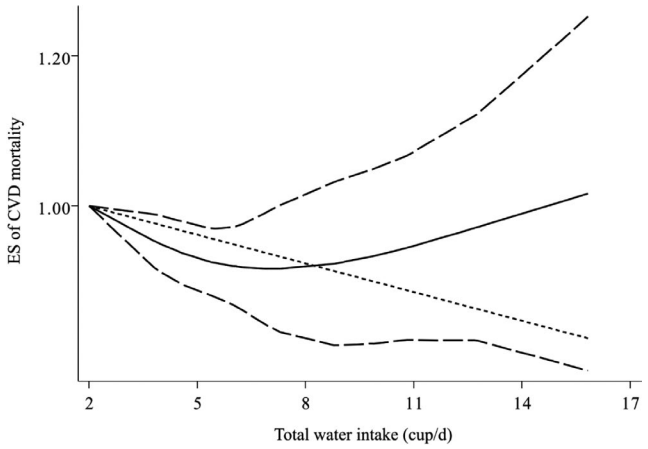

FIGURE 6 Non-linear dose–response association of total water intake (based on cup/day) with risk of mortality from CVD mortality in adults aged ≥18 years. Total water intake was modeled with restricted cubic splines in a multivariate random-effects dose–response model. Dotted line indicates the linear model. Solid line indicates the spline model. Dashed line presents the 95% CI. CI, confidence interval; CVD, cardiovascular disease; ES, effect size

of drinking water intake and all-cause mortality, a significant inverse association was reached in studies that had applied FFQ for drinking water assessment.

Findings from the sensitivity analysis using a fixed-effects model revealed that the exclusion of any single study from the analysis did not appreciably alter the pooled effect sizes. There were no missing studies imputed in regions of the contour enhanced funnel plots. No publication bias was found based on Egger's linear regression test. The application of the trim and fill method did not change the average effect size, further suggesting that results were not affected by publication.

4 | DISCUSSION

This systematic review and meta-analysis support a significant inverse association between total water intake and risk of CVD

mortality. Moreover, total water intake was inversely linked with all-cause and CVD mortality in a linear and non-linear dose–response manner.

Water affects numerous physiological processes, and, therefore, its net effects on health outcomes is important. There is substantial evidence that mild dehydration may account for many morbidities. The previous meta-analyses have focused mainly on individual fluid intakes with risk of mortality and total water intake has received less attention.[22] To the best of our knowledge, this is the first meta-analysis of prospective cohort studies that examined the association between water intake and risk of mortality. In the current study, total water intake was associated with a lower risk of CVD mortality. In line with our, a review revealed that good hydration reduces the risk of hypertension, fatal coronary heart disease, venous thromboembolism, and cerebral infarct. Further study indicated serum sodium as a risk factor for CVDs and give additional support to recommendations for dietary salt restriction and adequate water intake as preventives of CVD. Moreover, finding from earlier meta-analyses have documented that coffee and tea consumption as total fluid subtypes were associated with a lower risk of CVD mortality.[7,8,22,23] However, it must be kept in mind that tea and coffee are mild diuretics and thus may raise blood viscosity, which could increase the risk of mortality. Therefore, these inverse associations might be attributed to biologically active factors in tea and coffee. The inverse association between total fluid intake and cardiovascular mortality might be explained by reduced risk of inflammation and coagulation which are considered as major risk factors for the development of cardiovascular diseases. In our study, total and drinking water was not associated with all-cause mortality. This might be attributed to the consumption of fluids other than water such as caffeinated beverages or high energy drinks, which can cause a rapid elevation in blood viscosity after consumption.

In the context of our findings, it must be kept in mind that individual fluids contain diverse biologically active components, which may also account for the association between water intake and mortality. Also, other conditions such as medications and higher salt intake should be taken into account, which can affect the fluid balance and in turn alter the risk of mortality.

Exact mechanisms underlying the relation between water intake and mortality risk are not completely understood. Concerns about recommendations for high water intake have been based on the assumption that low fluid intake is associated with dehydration, which can induce inflammation and physiological dysfunction in the body. Evidence has shown that chronic dehydration is associated with a higher risk of certain diseases, such as cardiovascular diseases.[5] Besides, chronic dehydration may be related to increasing levels of hemorrhagic factors.[24,25] Elevation coagulation factors along with high blood viscosity, fibrinogen, and hematocrit levels are correlated with coronary heart disease.[26-29] They respond to circadian changes in hydration, daily activity, and medication such as diuretics.[30-33] Moreover, raising of serum sodium within the physiological range as a result of dehydration can lead to vascular changes by stimulating inflammatory signaling in endothelial cells and promote atherosclerosis. Another theory concerns arginine vasopressin (AVP), which regulates the water balance in the body. AVP also has vasoconstrictive effects, and there is evidence that elevated plasma levels have adverse effects on blood pressure and ventricular function.[34,35] Increased water intake suppresses plasma AVP and exerts other hemodynamic effects.[36-38]

4.1 | Strengths and weaknesses of the study

The present meta-analysis has several strengths. First, the relatively large number of participants and deaths included allowed us to quantitatively assess the association of water intake and risk of mortality, thus making it more powerful than any single study. Second, a dose–response analysis was conducted to evaluate the linear and non-linear relationship. Third, because all included studies had a prospective design, the influence of recall and selection bias is minimised that are common in case–control studies. Fourth, in the significant relations of our study low heterogeneity among studies was seen, which further confirms our results. Finally, we evaluated the associations separately for total and drinking water intake as the exposure of interest. These data provide the most comprehensive insight into the association between water intake and risk of mortality based on the current evidence. Our findings also need to be interpreted in the context of some limitations, most of them are common to observational studies and meta-analyses. Residual or unmeasured confounding factors may have affected the magnitude of the association between water intake and mortality. Although most studies had controlled for potential confounders, some did not control the analyses for dietary intake of other nutrients and some did not consider total energy intake and BMI as covariates. Lack of control for such factors might affect the independent association of water intake with mortality. Also, some studies in this review did not report sufficient information to be included in the dose–response meta-analysis. Also, different methods were used for water intake assessment including FFQ and dietary recall in the included cohorts. Measurement errors in dietary assessment are inevitable and would have alter the associations with water intake. The overlap in the categories of water intake across different studies was observed; however, we handled this problem by performing dose–response analysis. As we considered studies on apparently healthy populations, our conclusions about water intake cannot be generalised to those with specific diseases, such as hemodialysis, CKD, and ARDS, in which fluid imbalance is more prevalent among them.

5 | CONCLUSION

This current systematic review and meta-analysis of prospective cohort studies provide evidence that higher total water intake

is associated with a lower risk of CVD mortality. However, total water intake was not associated with an increased risk of all-cause mortality. Further studies are needed to investigate the associations of water intake with all-cause and other causes of mortality as well.

Further studies with a prospective design are required to confirm these findings.

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DISCLOSURES

The authors declare no conflict of interest.

AUTHOR CONTRIBUTIONS

MM and FH contributed to literature search, data extraction, and, data analysis. SS-B contributed to study conception and data analysis. SN and MM contributed to study conception, manuscript drafting, and data analysis. SS-B critically revised the manuscript. All authors acknowledge the full responsibility for the analyses and interpretation of the report.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of the article at the publisher's website.

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