G.1.2. Reference list of studies excluded based on full text screening

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n = 22, co-intervention deemed to affect the outcome of interest

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n = 19, outcome(s) not of interest

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n = 78, 24-h urine sodium excretion not measured

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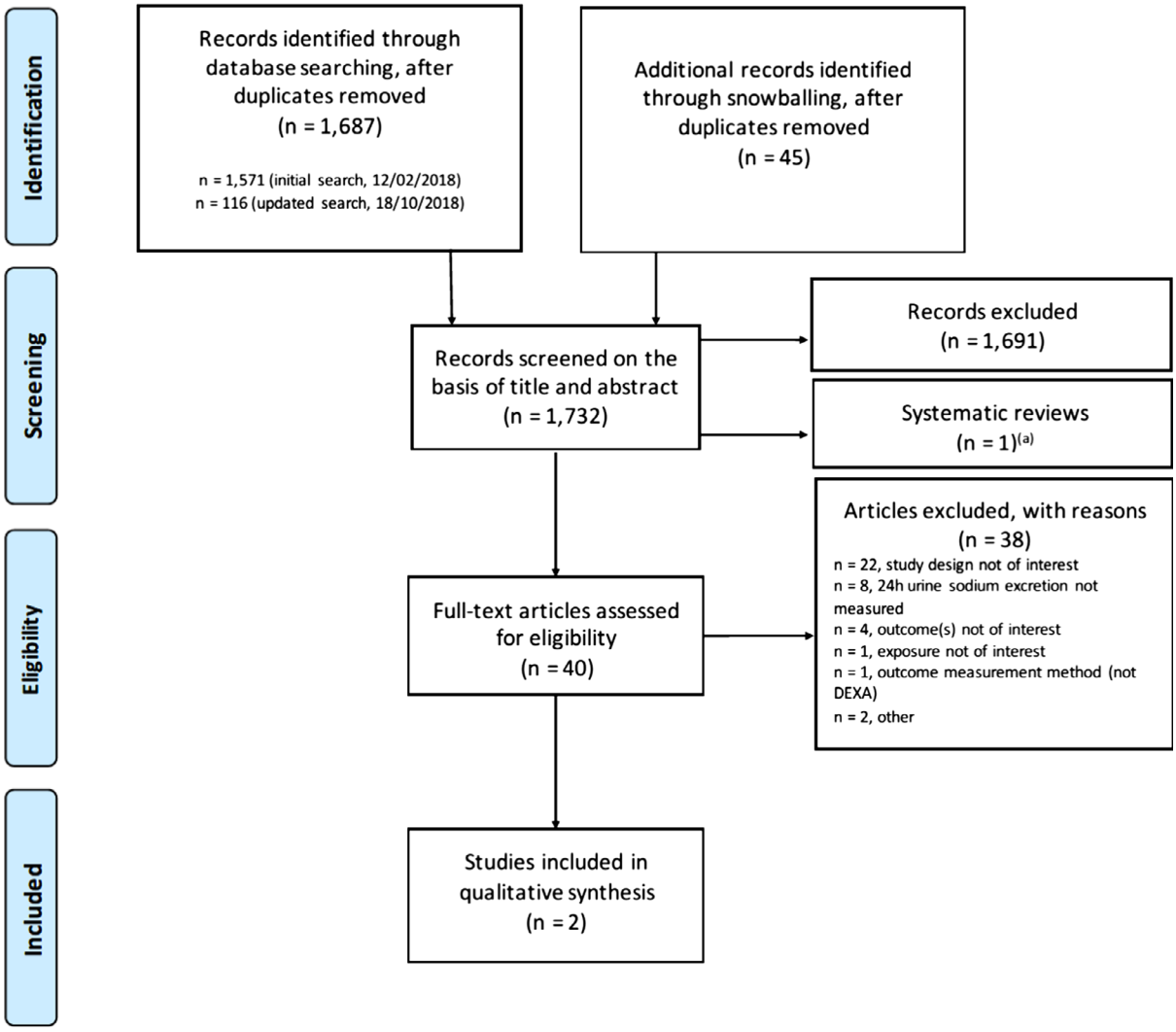
G.2. Bone health

G.2.1. PRISMA chart



(a): No additional eligible study was retrieved from the list of included studies.

G.2.2. Reference list of studies excluded based on full text screening

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Wong MM, Arcand J, Leung AA, Raj TS, Trieu K, Santos JA, Campbell NR, 2016. The Science of Salt: A Regularly Updated Systematic Review of Salt and Health Outcomes (August to November 2015). Journal of clinical hypertension (Greenwich), 18, 1054–1062.

n = 8, 24-h urine sodium excretion not measured

Burger H, De Laet CEDH, Van Daele PLA, Weel AEAM, Witteman JCM, Hofman A, Pols HAP, 1998. Risk factors for increased bone loss in an elderly population: The Rotterdam Study. American Journal of Epidemiology, 147, 871–879.

Carbone L, Johnson KC, Huang Y, Pettinger M, Thomas F, Cauley J, Crandall C, Tinker L, LeBoff MS, Wactawski-Wende J, Bethel M, Li W, Prentice R, 2016. Sodium Intake and Osteoporosis. Findings From the Women's Health Initiative. Journal of Clinical Endocrinology and Metabolism, 101, 1414–1421.

Chan R, Woo J, Leung J, 2011. Effects of food groups and dietary nutrients on bone loss in elderly Chinese population. Journal of Nutrition Health & Aging, 15, 287–294.

Fenton TR, Eliasziw M, Tough SC, Lyon AW, Brown JP, Hanley DA, 2010. Low urine pH and acid excretion do not predict bone fractures or the loss of bone mineral density: a prospective cohort study. BioMed Central Musculoskeletal Disorders, 11, 88.

Gunnes M, Lehmann EH, 1996. Physical activity and dietary constituents as predictors of forearm cortical and trabecular bone gain in healthy children and adolescents: a prospective study. Acta Paediatrica, 85, 19–25.

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Hubert HB, Fries JF, 1994. Predictors of physical disability after age 50. Six-year longitudinal study in a runners club and a university population. Annals of Epidemiology, 4, 285–294.

Reid IR, Ames RW, Evans MC, Sharpe SJ, Gamble GD, 1994. Determinants of the rate of bone loss in normal postmenopausal women. Journal of Clinical Endocrinology and Metabolism, 79, 950–954.

n = 4, outcome(s) not of interest

Breslau NA, Sakhaee K, Pak CY, 1985. Impaired adaptation to salt-induced urinary calcium losses in postmenopausal osteoporosis. Transactions of the Association of American Physicians, 98, 107–115.

Goulding A, Lim PE, 1983. Effects of varying dietary salt intake on the fasting urinary excretion of sodium, calcium and hydroxyproline in young women. New Zealand Medical Journal, 96, 853–854.

McParland BE, Goulding A, Campbell AJ, 1989. Dietary salt affects biochemical markers of resorption and formation of bone in elderly women. British Medical Journal, 299, 834–835.

Wigertz K, Palacios C, Jackman LA, Martin BR, McCabe LD, McCabe GP, Peacock M, Pratt JH, Weaver CM, 2005. Racial differences in calcium retention in response to dietary salt in adolescent girls. American Journal of Clinical Nutrition, 81, 845–850.

n = 1, exposure not of interest

Merrilees MJ, Smart EJ, Gilchrist NL, Frampton C, Turner JG, Hooke E, March RL, Maguire P, 2000. Effects of diary food supplements on bone mineral density in teenage girls. European Journal of Nutrition, 39, 256–262.

n = 1, outcome measurement method (not DEXA)

Nordin BEC, Polley KJ, 1987. Metabolic consequences of the menopause. A cross-sectional, longitudinal, and intervention study on 557 normal postmenopausal women. Calcified Tissue International, 41, S1–S59.

n = 2, other reasons

Chao D, Espeland MA, Farmer D, Register TC, Lenchik L, Applegate WB, Ettinger WH Jr, 2000. Effect of voluntary weight loss on bone mineral density in older overweight women. Journal of the American Geriatrics Society, 48, 753–759.

Frassetto LA, Hardcastle AC, Sebastian A, Aucott L, Fraser WD, Reid DM, Macdonald HM, 2012. No evidence that the skeletal non-response to potassium alkali supplements in healthy postmenopausal women depends on blood pressure or sodium chloride intake. European Journal of Clinical Nutrition, 66, 1315–1322.

G.2.3. Systematic review screened for eligible references

Fatahi S, Namazi N, Larijani B and Azadbakht L, 2018. The association of dietary and urinary sodium with bone mineral density and risk of osteoporosis: a systematic review and meta-analysis. Journal of the American College of Nutrition, 37, 522–532.

G.3. Criteria used to appraise RoB in eligible studies

G.3.1. Randomised controlled trials

Question	Rating	Explanation for expert judgement
1. Was administered dose or exposure level adequately randomised? **Key question**	++	There is direct evidence that subjects (or clusters) were allocated to any study group including controls using a method with a random component. Acceptable methods of randomisation include: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, or drawing of lots (Higgins and Green, 2011). Restricted randomisation (e.g. blocked randomisation) to ensure particular allocation ratios will be considered low risk of bias. Similarly, stratified randomisation and minimisation approaches that attempt to minimise imbalance between groups on significant prognostic factors (e.g. body weight) will be considered acceptable
	+	There is indirect evidence that subjects (or clusters) were allocated to study groups using a method with a random component (i.e. authors state that allocation was random, without description of the method used) **OR** It is deemed that allocation without a clearly random component during the study would not appreciably bias results. For example, approaches such as biased coin or urn randomisation, replacement randomisation, mixed randomisation, and maximal randomisation may require consultation with a statistician to determine risk-of-bias rating (Higgins and Green, 2011)
	NR	There is insufficient information provided about how subjects (or clusters) were allocated to study groups.
	–	There is indirect evidence that subjects (or clusters) were allocated to study groups using a method with a non-random component. *NOTE: Non-random allocation methods may be systematic, but have the potential to allow participants or researchers to anticipate the allocation to study groups. Such 'quasi-random' methods include alternation, assignment based on date of birth, case record number, or date of presentation to study*
	– –	There is direct evidence that subjects (or clusters) were allocated to study groups using a non-random method including judgement of the clinician, preference of the participant, the results of a laboratory test or a series of tests, or availability of the intervention (Higgins and Green, 2011)
2. Was allocation to study groups adequately concealed?	++	There is direct evidence that at the time of recruitment the research personnel and subjects did not know what study group subjects were allocated to, and it is unlikely that they could have broken the blinding of allocation until after assignment was complete and irrevocable. Acceptable methods used to ensure allocation concealment include central allocation (including telephone, web-based and pharmacy-controlled randomisation); sequentially numbered drug containers of identical appearance; sequentially numbered, opaque, sealed envelopes; or equivalent methods
	+	There is indirect evidence that the research personnel and subjects did not know what study group subjects were allocated to and it is unlikely that they could have broken the blinding of allocation until after recruitment was complete and irrevocable **OR** It is deemed that lack of adequate allocation concealment would not appreciably bias results (e.g. some crossover designs)

Question	Rating	Explanation for expert judgement
	NR	There is insufficient information provided about allocation to study groups
	–	There is indirect evidence that at the time of recruitment it was possible for the research personnel and subjects to know what study group subjects were allocated to, or it is likely that they could have broken the blinding of allocation before assignment was complete and irrevocable *NOTE: Inadequate methods include using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number; or any other explicitly unconcealed procedure. For example, if the use of assignment envelopes is described, but it remains unclear whether envelopes were sequentially numbered, opaque and sealed*
	– –	There is direct evidence that at the time of recruitment it was possible for the research personnel and subjects to know what study group subjects were allocated to, or it is likely that they could have broken the blinding of allocation before recruitment was complete and irrevocable
3. Were the research personnel and human subjects blinded to the study group during the study?	++	There is direct evidence that the subjects and research personnel were adequately blinded to study group, **AND** it is unlikely that they could have broken the blinding during the study. Methods used to ensure blinding include central allocation; sequentially numbered drug containers of identical appearance; sequentially numbered, opaque, sealed envelopes; or equivalent methods
	+	There is indirect evidence that the subjects and research personnel were adequately blinded to study group, **AND** it is unlikely that they could have broken the blinding during the study **OR** There is direct evidence for no blinding during the study (including where it was not possible to implement) AND it is deemed that no blinding would appreciably bias results BUT bias minimising measures have been adequately implemented **OR** It is deemed that lack of adequate blinding or no blinding during the study would not appreciably bias results (e.g. controls unlikely to behave differently for factors other than sodium intake) (e.g. cross-over)
	NR	There is insufficient information provided about blinding to study group during the study (including possible breaking and minimising measures)
	–	There is indirect evidence that it was possible for research personnel or subjects to infer the study group **AND** it is deemed that lack of adequate blinding or no blinding during the study would appreciably bias results (e.g. no comparable treatment of controls, including not comparable exposure to factors other than the interventions of interest; differential behaviour) **AND** no bias minimising measures have been adequately implemented
	– –	There is direct evidence for lack of adequate blinding of the study group (including no blinding or incomplete blinding) of research personnel and subjects **AND** it is deemed that lack of adequate blinding or no blinding during the study would appreciably bias results (e.g. no comparable treatment of controls, including not comparable exposure to factors other than the interventions of interest, differential behaviour) **AND** no bias minimising measures have been adequately implemented

Question	Rating	Explanation for expert judgement
4. Were outcome data complete without attrition or exclusion from analysis?	++	There is direct evidence that there was no loss of subjects during the study and outcome data were complete **OR** Loss of subjects (i.e. incomplete outcome data) was adequately addressed and reasons were documented when human subjects were removed from a study or analyses. Review authors should be confident that the participants included in the analysis are exactly those who were randomised into the trial. Acceptable handling of subject attrition includes: very few missing outcome data (e.g. less than 10% in each group (Genaidy et al., 2007)) **AND** reasons for missing subjects unlikely to be related to outcome (for survival data, censoring unlikely to be introducing bias) **AND** missing outcome data balanced in numbers across study groups, with similar reasons for missing data across groups (i.e. unlikely to be related to exposure) **OR** Analyses (such as intention-to-treat analysis) in which missing data have been imputed using appropriate methods (ensuring that the characteristics of subjects lost to follow up or with unavailable records are described in identical way and are not significantly different from those of the study participants) *NOTE: Participants randomised but subsequently found not to be eligible need not always be considered as having missing outcome data) (Higgins and Green, 2011*
	+	There is indirect evidence that loss of subjects (i.e. incomplete outcome data) was adequately addressed and reasons were documented when human subjects were removed from a study **OR** It is deemed that the proportion lost to follow-up would not appreciably bias results (e.g. less than 20% in each group in parallel studies (Genaidy et al., 2007)). This would include reports of no statistical differences in characteristics of subjects lost to follow up or with unavailable records from those of the study participants. Generally, the higher the ratio of participants with missing data to participants with events, the greater potential there is for bias. For studies with a long duration of follow-up, some withdrawals for such reasons are inevitable *NB: For crossover designs, this may be less of an issue*
	NR	There is insufficient information provided about numbers of subjects lost to follow-up
	–	There is indirect evidence that loss of subjects (i.e. incomplete outcome data) was unacceptably large (e.g. greater than 20% in each group in parallel studies (Genaidy et al., 2007)) and not adequately addressed
	– –	There is direct evidence that loss of subjects (i.e. incomplete outcome data) was unacceptably large and not adequately addressed (e.g. greater than 20% in each group in parallel studies (Genaidy et al., 2007)). Unacceptable handling of subject attrition includes: reason for missing outcome data likely to be related to true outcome, with either imbalance in numbers or reasons for missing data across study groups (i.e. likely to be related to the exposure); or potentially inappropriate application of imputation

Question	Rating	Explanation for expert judgement
5. Can we be confident in the exposure characterisation? **Key question**	++	There is direct evidence that the exposure (including compliance with the treatment, if applicable) was independently characterised **AND** that exposure was consistently administered (i.e. with the same method and time-frame) across treatment groups *NOTE: applies to studies that collected single or multiple 24-h urine samples for each time point AND checked the completeness of the samples (by any kind of method)*
	+	There is indirect evidence that the exposure (including compliance with the treatment, if applicable) was independently characterised AND there is indirect evidence that exposure was consistently administered (i.e. with the same method and time-frame) across treatment groups *NOTE: applies to studies that collected single or multiple 24-h urine samples without completeness check of the urine samples (or not reported), but there is no evidence for concern*
	NR	There is insufficient information provided to judge the exposure characterisation
	–	There is indirect evidence that the exposure (including compliance with the treatment, if applicable) was assessed using poorly validated methods (e.g. FFQs, spot urine etc.) **OR** There is indirect evidence that the exposure assessment was probably biased *NOTE: applies to studies for which 24-h urine samples were collected but there is indirect evidence that the proportion of incomplete samples included in the analysis was substantial*
	– –	There is direct evidence that the exposure (including compliance with the treatment, if applicable) was assessed using poorly validated methods (e.g. FFQs, spot urine etc.) **OR** There is direct evidence that the exposure assessment was biased *NOTE: applies to studies for which 24 h urine samples were collected but there is direct evidence that the proportion of incomplete samples included in the analysis was substantial*
6. Can we be confident in the outcome assessment? Key question	++	There is direct evidence that the outcome was assessed using well-established methods (e.g. for office BP: according to a clearly described methodology, including e.g. repeated measurements per visit, trained technician, resting period before each measurement) **AND** There is direct evidence that the outcome assessors were adequately blinded to the study group, and it is unlikely that they could have broken the blinding prior to reporting outcomes

Question	Rating	Explanation for expert judgement
	+	There is indirect evidence that the outcome was assessed using acceptable methods (i.e. deemed valid and reliable but not the gold standard) **OR** it is deemed that the outcome assessment methods used would not appreciably bias results **AND** There is indirect evidence that the outcome assessors were adequately blinded to the study group, and it is unlikely that they could have broken the blinding before reporting outcomes **OR** it is deemed that lack of adequate blinding of outcome assessors would not appreciably bias results
	NR	There is insufficient information provided about blinding of outcome assessors or the method of measurement
	–	There is indirect evidence that the outcome assessment method is an unacceptable method **OR** There is indirect evidence that it was possible for outcome assessors to infer the study group before reporting outcomes
	– –	There is direct evidence that the outcome assessment method is an unacceptable method **OR** There is direct evidence for lack of adequate blinding of outcome assessors (including study subjects if home BP is the outcome), including no blinding or incomplete blinding
7. Were all measured outcomes reported?	++	There is direct evidence that all of the study's measured outcomes (primary and secondary) outlined in the protocol, methods, abstract, and/or introduction (that are relevant for the evaluation) have been reported
	+	There is indirect evidence that all of the study's measured outcomes (primary and secondary) outlined in the methods, abstract, and/or introduction (that are relevant for the evaluation) have been reported **OR** Analyses that had not been planned in advance (i.e. retrospective unplanned subgroup analyses) are clearly indicated as such and it is deemed that the unplanned analyses were appropriate and selective reporting would not appreciably bias results (e.g. appropriate analyses of an unexpected effect). This would include outcomes reported with insufficient detail such as only reporting that results were statistically significant (or not)
	NR	There is insufficient information provided about selective outcome reporting
	–	There is indirect evidence that all of the study's measured outcomes (primary and secondary) outlined in the methods, abstract, and/or introduction (that are relevant for the evaluation) have not been reported **OR** There is indirect evidence that unplanned analyses were included that may appreciably bias result

Question	Rating	Explanation for expert judgement
	– –	There is direct evidence that all of the study's measured outcomes (primary and secondary) outlined in the methods, abstract, and/or introduction (that are relevant for the evaluation) have not been reported. In addition to not reporting outcomes, this would include reporting outcomes based on composite score without individual outcome components or outcomes reported using measurements, analysis methods or subsets of the data (e.g. subscales) that were not pre-specified or reporting outcomes not pre-specified, or that unplanned analyses were included that would appreciably bias results
8. Were there no other potential threats to internal validity? ***NOTE:*** *Baseline characteristics should be appraised* ***only if*** *Q1 (randomisation) was rated with ++/+ and Q2 (allocation concealment) was rated with ++/+/ NR*	++	There is evidence that variables, other than the exposure and outcome, did not differ between groups during the course of the intervention in a way that could bias results AND, in case randomisation is rated 'probably low'/'definitely low' RoB and allocation concealment is rated 'probably low'/'definitely low' RoB or 'not reported': There is no evidence of differences in baseline characteristics **OR** There is no information on both BUT no concern
	+	1. There is evidence that variables, other than the exposure and outcome, differed between groups during the course of the intervention AND it is deemed that these differences would not appreciably bias results (no concern or adequately addressed by analysis) AND, in case randomisation is rated 'probably low'/'definitely low' RoB and allocation concealment is rated 'probably low'/'definitely low' RoB or 'not reported': There is evidence that reported variables differed between groups at baseline AND It is deemed that these differences would not appreciably bias results (no concern or adequately addressed by analysis) ------- OR 2. There is evidence that variables, other than the exposure and outcome, did not differ between groups during the course of the intervention in a way that could bias results AND, in case randomisation is rated 'probably low'/'definitely low' RoB and allocation concealment is rated 'probably low'/'definitely low' RoB or 'not reported':

Question	Rating	Explanation for expert judgement
		There is evidence that reported variables differed between groups at baseline. AND It is deemed that these differences would not appreciably bias results (no concern or adequately addressed by analysis). ------ OR 3. There is evidence that variables, other than the exposure and outcome, differed between groups during the course of the intervention.ANDIt is deemed that these differences would not appreciably bias results (no concern or adequately addressed by analysis) AND, in case randomisation is rated 'probably low'/'definitely low' RoB and allocation concealment is rated 'probably low'/'definitely low' RoB or 'not reported': There is no evidence of differences in baseline characteristics. OR There is no information BUT no concern
	–	There is no information on baseline characteristics AND/OR there is no information about differences between groups during the course of the intervention. AND There is concern
	– –	There is evidence that variables, other than the exposure and outcome, differed between groups during the course of the intervention. AND It is deemed that these differences appreciably biased results (there is concern (e.g. not adequately addressed by analysis)) OR, in case randomisation is rated 'probably low'/'definitely low' RoB and allocation concealment is rated 'probably low'/'definitely low' RoB or 'not reported':

Question	Rating	Explanation for expert judgement
		There is evidence that reported variables differed between groups at baseline. AND It is deemed that these differences appreciably biased results (there is concern (e.g. not adequately addressed by analysis))

++: Definitely low RoB; +: Probably low RoB; NR: Not Reported; –: Probably high RoB; – –: Definitely high RoB.

G.3.2. Prospective observational studies

Question	Rating	Explanation for expert judgement
Did the study design or analysis account for important confounding? **Key question**	++	There is direct evidence that appropriate adjustments or explicit considerations were made for the potential confounders in the final analyses through the study design (e.g. matching, restriction) and/or through the use of statistical models to reduce research-specific bias including standardisation, adjustment in multivariate model, stratification, propensity scoring, or other methods that were appropriately justified *NOTE: Acceptable consideration of appropriate adjustment factors includes cases when the factor is not included in the final adjustment model because: i) there was evidence indicating that a factor did not need to be included as a confounders (e.g. the author conducted analyses that indicated it did not need to be included; study restricted to males only) OR ii) it is deemed that not considering the factor would not bias the result* **AND** There is direct evidence that confounders were assessed using reliable methods
	+	There is indirect evidence that appropriate adjustments were made, OR it is deemed that not considering or only considering a partial list confounders in the final analyses would not substantially bias results **AND** There is evidence (direct or indirect) that confounders were assessed using reliable methods, OR it is deemed that the methods used would not appreciably bias results (i.e., the authors justified the validity of the methods from previously published research)
	NR	There is insufficient information provided about the distribution of potential confounders (record 'NR' as basis for answer) **OR** There is insufficient information provided about the methods used to assess confounders (record 'NR' as basis for answer)
	–	There is indirect evidence that the distribution of potential confounders differed between the groups and was not appropriately adjusted for in the final analyses

Question	Rating	Explanation for expert judgement
	– –	There is direct evidence that the distribution of confounders differed between the groups, confounding occurred but was not adjusted for in the final analyses **OR** There is direct evidence that confounders were assessed using non-reliable methods
2. Were outcome data complete without attrition or exclusion from analysis?	++	There is direct evidence that loss of subjects (i.e. incomplete outcome data) was adequately addressed and reasons were documented when human subjects were lost or removed from a study *NOTE: Acceptable handling of subject attrition includes: very few missing outcome data AND reasons for missing subjects unlikely to be related to outcome (for survival data, censoring unlikely to be introducing bias) AND missing outcome data balanced in numbers across study groups, with similar reasons for missing data across groups (i.e. unlikely to be related to exposure)*
	+	There is indirect evidence that loss of subjects (i.e. incomplete outcome data) was adequately addressed and reasons were documented when human subjects were removed from a study **OR** It is deemed that the proportion lost to follow-up would not appreciably bias results, due to the similarity between the characteristics of subjects lost to follow-up and study participants. Generally, the higher the ratio of participants with missing data to participants with events, the greater potential there is for bias. For studies with a long duration of follow-up, some withdrawals for such reasons are inevitable
	–	There is indirect evidence that loss of subjects (i.e. incomplete outcome data) was unacceptably large and not adequately addressed
	– –	There is direct evidence that loss of subjects (i.e. incomplete outcome data) was unacceptably large and not adequately addressed. Unacceptable handling of subject attrition includes: reason for missing outcome data likely to be related to true outcome, with either imbalance in numbers or reasons for missing data across study groups (i.e. likely to be related to the exposure)
3. Can we be confident in the exposure characterisation? **Key question**	++	Sodium intake was assessed through multiple 24-h urinary collection (in a 'reasonably short time-frame') **AND** There is direct evidence that quality assurance measures were in place for the collection of 24-h urine (e.g. first and last void at the clinic; careful instructions of the participants) OR incomplete collections were excluded on the basis of any method (e.g. PABA, creatinine, self-reported, volume…)
	+	Sodium intake was assessed through a single 24-h urinary collection

Question	Rating	Explanation for expert judgement
		AND There is direct evidence that quality assurance measures were in place for the collection of 24-h urine (e.g. first and last void at the clinic; careful instructions of the participants) OR incomplete collections were excluded on the basis of any method (e.g. PABA, creatinine, self-reported, volume. . .)
	NR	There is insufficient information provided about the method of exposure assessment
	–	There is indirect evidence that the exposure (including compliance with the treatment, if applicable) was assessed using poorly validated methods (e.g. FFQs, spot urine etc.) **OR** There is no evidence that quality assurance measures were in place for the collection of 24-h urine (single or multiple) AND no measures were taken to exclude incomplete samples
	– –	There is direct evidence that the exposure (including compliance with the treatment, if applicable) was assessed using poorly validated methods (e.g. FFQs, spot urine etc.) **OR** There is direct evidence for systematic error in the exposure characterisation (exposure misclassification)
4. Can we be confident in the outcome assessment? **Key question**	++	There is direct evidence that the outcome was assessed using well-established methods
	+	There is indirect evidence that the outcome was assessed using acceptable methods (i.e. deemed valid and reliable but not the gold standard) **OR** It is deemed that the outcome assessment methods used would not appreciably bias results
	NR	There is insufficient information provided about the method of measurement
	–	There is indirect evidence that the outcome assessment method is an unacceptable method
	– –	There is direct evidence that the outcome assessment method is an unacceptable method
5. Were all measured outcomes reported?	++	There is direct evidence that all of the study's measured outcomes (primary and secondary) outlined in the protocol, methods, abstract, and/or introduction (that are relevant for the evaluation) have been reported. This would include outcomes reported with sufficient detail to be included in meta-analysis or fully tabulated during data extraction and analyses had been planned in advance
	+	There is indirect evidence that all of the study's measured outcomes (primary and secondary) outlined in the methods, abstract, and/or introduction (that are relevant for the evaluation) have been reported

Question	Rating	Explanation for expert judgement
		OR Analyses that had not been planned in advance (i.e. retrospective unplanned subgroup analyses) are clearly indicated as such and it is deemed that the unplanned analyses were appropriate and selective reporting would not appreciably bias results (e.g. appropriate analyses of an unexpected effect). This would include outcomes reported with insufficient detail such as only reporting that results were statistically significant (or not)
	NR	There is insufficient information provided about selective outcome reporting
	–	There is indirect evidence that all of the study's measured outcomes (primary and secondary) outlined in the methods, abstract, and/or introduction (that are relevant for the evaluation) have been reported **OR** There is indirect evidence that unplanned analyses were included that may appreciably bias results
	– –	There is direct evidence that all of the study's measured outcomes (primary and secondary) outlined in the methods, abstract, and/or introduction (that are relevant for the evaluation) have not been reported. In addition to not reporting outcomes, this would include reporting outcomes based on composite score without individual outcome components or outcomes reported using measurements, analysis methods or subsets of the data (e.g. subscales) that were not pre-specified or reporting outcomes not pre-specified, or that unplanned analyses were included that would appreciably bias results
6. Were the statistical methods applied appropriate?	++	There is direct evidence that the statistical analysis was appropriate
	+	There is indirect evidence that the statistical analysis was appropriate
	NR	There is insufficient information provided about the statistical analysis
	–	There is indirect evidence that the statistical analysis was not appropriate
	– –	There is direct evidence that the statistical analysis was not appropriate

++: Definitely low RoB; +: Probably low RoB; NR: Not Reported; –: probably high RoB; – –: Definitely high RoB.

G.3.3. Algorithm applied to allocate studies to the three tiers of RoB

The judgements to the RoB questions were combined into an overall RoB judgement for each individual study (by outcome). As a result, studies were classified as being at low (tier 1), moderate (tier 2) or high (tier 3) RoB. Following the OHAT/NTP guidance (OHAT/NTP, 2015), key questions were identified for each type of design (see Table below) and the following algorithm was applied to allocate the studies to the tiers:

Tier 1: study rated as 'definitely low' or 'probably low' risk of bias for the key questions AND most other applicable questions answered 'definitely low' or 'probably low' risk of bias.

Tier 2: study met neither the criteria for tiers.

Tier 3: study rated as 'definitely high' or 'probably high' risk of bias for the key questions AND most other applicable questions answered 'definitely high' or 'probably high' risk of bias.

Bias Domains and Questions	RCT	Prospective observational
Selection Bias		
Was administered dose or exposure level adequately randomised?	X*	
Was allocation to study groups adequately concealed?	X	
Confounding Bias		
Did the study design or analysis account for important confounding?		X*
Performance Bias		
Were the research personnel and human subjects blinded to the study group during the study?	X	
Attrition/Exclusion Bias		
Were outcome data complete without attrition or exclusion from analysis?	X	X
Detection Bias		
Can we be confident in the exposure characterisation?	X*	X*
Can we be confident in the outcome assessment?	X*	X*
Selective Reporting Bias		
Were all measured outcomes reported?	X	X
Other Sources of Bias		
Were there no other potential threats to internal validity?	X	
Were the statistical methods applied appropriate?		X

*: Key question.

Appendix H – Outcome of the systematic review on blood pressure levels in children

H.1. Evidence tables

Experimental studies

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
He et al. (2015) **(China)** **School-EduSalt**	Study design: cluster-randomised Run-in type: none Intervention type: counselling N participants randomised/completed: 279/274 Duration: 14 weeks	% boys G1: 47.5 G2: 48.6 Age (mean ± SD, years) G1: 10.0 ± 0.5 G2: 10.2 ± 0.5 BMI (mean ± SD, kg/m^2) G1: 17.1 ± 3.2 G2: 16.7 ± 2.7 Ethnicity: NR BP status: NR 24-h UK (mean ± SE, mmol) G1: 23.5 ± 0.9 G2: 25.4 ± 0.9 Energy intake: NR	Type: multiple 24 h Mean ± SE Beginning of intervention G1: 124.2 ± 5.1 G2: 116.7 ± 5.2 End of intervention G1: 112.2 ± 5.1 G2: 137.2 ± 5.2	Type: point office, sitting Mean ± SE Beginning of intervention G1: 106.2 ± 1/67.0 ± 1.1 G2: 106.2 ± 1/66.8 ± 1.1 End of intervention G1: 110 ± 1/69.4 ± 1.1 G2: 110.6 ± 1/70.2 ± 1.1
Miller et al. (1988) **(USA)**	Study design: parallel Run-in type: low sodium diet Intervention type: feeding N participants randomised/ completed: 88/NR Duration: 4 weeks	% boys: NR Age (years): NR BP status: NT Ethnicity: NR 24-h UK (mean, mmol) G1: 35.3 G2: 36.7 Energy intake: NR	Type: multiple 24 h Mean Beginning of intervention NR End of intervention G1: 44.4 G2: 72.1	Type: point office, sitting Mean Beginning of intervention NR End of intervention G1: 91.7/53.0 G2: 92/52.9

BMI: body mass index; BP: blood pressure; DBP; diastolic blood pressure; G1: group 1 ('low' sodium); G2: group 2 ('high' sodium); mm Hg: millimetre of mercury; N: number; UNa: sodium urinary excretion; NR: not reported; NT: normotensive; SBP: systolic blood pressure; SD: standard deviation; SE: standard error; UK: potassium urinary excretion.

Observational studies

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
Shi et al. (2014) **(Germany) DONALD**	Prospective cohort N = 1,107 DONALD initial cohort N = 435 included in the analyses (included: at least 3 BP measurements with three parallel 24-h UNa and 3-day weighed dietary records; excluded: preterm children, birth weight and breast-feeding data missing; taking BP-lowering treatment; implausible SBP/DBP values) Mean duration: 10 years	Male (%) 51.0 Age (median (p25–p75), years) b 6 (4–8) g 6 (4–7) BMI (median (p25–p75), kg/m^2) b 15.7 (15.0–16.8) g 15.3 (14.7–16.4) SBP (median (p25–p75), mm Hg) b 97.1 (90.8–104.0) g 97.0 (90.0–102.0) DBP (median (p25–p75), mm Hg) b 57.0 (50.0–65.0) g 55.0 (49.6–64.1) 24-h UK (median (p25–p75), mmol) b 39.1 (29.7–47.8) g 32.4 (26.2–38.9) 24-h UK UCr (median (p25–p75), mmol/kg bw) b 0.16 (0.14–0.18) g 0.15 (0.13–0.17) Energy intake (median (p25–p75), MJ/day) b 6.1 (5.3–7.2) g 5.3 (4.8–6.3)	Single 24-h urine sample at yearly intervals	Median (p25–p75) First assessment b 67.4 (50.6–89.9) g 58.7 (45.9–74.5) Last assessment b 131.0 (96.9–176.0) g 108.0 (81.7–133.0)	Longitudinal change in SBP and DBP BP measured every 2 years (2 BP readings averaged at each visit)	Age, age 2, age 3, sex, pubertal group, intraindividual change in Na excretion × pubertal group, person-specific mean-Na excretion × pubertal group, TEI, TEI × pubertal group, BMI-SDS, height-SDS, birth weight, full breast-feeding status, maternal SBP, FVI, FVI × pubertal group	Change in SBP (mm Hg)/ 1 mmol/MJ increase in Na excretion (β (95% CI)): Prepubertal group Between person effect −0.2 (−0.4, 0.04) p = 0.1 Within person effect −0.03 (−0.2, 0.09) p = 0.6 Pubertal group Between person effect 0.1 (−0.1, 0.4) p = 0.3 Within person effect 0.1 (−0.004, 0.2) p = 0.06
						Age, age 2, age 3, sex, pubertal group, intraindividual change in Na excretion × pubertal group, person-specific mean-Na excretion × pubertal group, TEI, TEI × pubertal group, BMI-SDS, height-SDS, growth velocity, full breast-feeding status, maternal SBP, FVI, FVI × pubertal group and Ca intake	Change in DBP (mm Hg)/ 1 mmol/MJ increase in Na excretion (β (95% CI)): Prepubertal group Between person effect −0.1 (−0.4,0.07) p = 0.2 Within person effect −0.1 (−0.2, 0.03) p = 0.1 Pubertal group Between person effect −0.2 (−0.4, 0.04) p = 0.1 Within person effect 0.1 (−0.02, 0.2) p = 0.09

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
Krupp et al. (2015) **(Germany)** **DONALD**	Prospective cohort N = NR N = 206 included in the analyses ('included: reached adult age, had a BP measurement and assessed in parallel anthropometrical data between 18 and 25 years, had ≥ 3 parallel plausible dietary records 24 h UNa and ≥ 3 BP measurements during adolescence (11–16 years); excluded: preterm, missing data on birth weight and gestational age, implausible BP data) Mean duration: 7 years	Male (%) 52.0 Age (median (p25–p75), years) b 12.4 (12.1–13.0) g 12.3 (12.0–13.0) BMI (mean (SD), kg/m^2) b 18.9 (2.3) g 18.7 (2.7) SBP (mean (SD), mm Hg) b 104 (7) g 103 (8) DBP (mean (SD), mm Hg) b 62 (6) g 61 (6) 24-h UK (mean (SD), mmol) b 56 (14) g 51 (13) 24-h UK UCr (mean (SD), mmol/kg bw) b 0.18 (0.02) g 0.17 (0.02) Energy intake (mean (SD), MJ/day) b 9.1 (1.5) g 7.9 (0.9)	Single 24-h urine sample at yearly intervals Samples with UCr < 0.1 mmol/kg BW excluded from the analysis	NaCl (mean (SD)) b 116 (27) g 105 (32) calculated as (Na (mmol/day) + Cl (mmol/day))/2	Longitudinal change in SBP and DBP BP measured every 2 years (2 blood pressure readings averaged at each examination)	Mean pubertal SBP SDS, adult age, standardised energy intake, intake of saturated fat, height-SDS, maternal education, maternal BP, FVI, adult BMI	Change in SBP (mm Hg)/1 mmol increase in NaCl excretion (ß (95% CI)): b 0.1 (0.03, 0.18) p = 0.01 g -0.05 (−0.11, 0.02) p = 0.1
						Mean pubertal SBP SDS, adult age, standardised energy intake, calcium intake, birth weight, smoking in the household, maternal BP, FVI, adult BMI	Change in DBP (mm Hg)/1 mmol increase in NaCl excretion (ß (95% CI)): b 0.02 (−0.08, 0.04) p = 0.6 g 0.02 (−0.03, 0.08) p = 0.4

b: boys; BMI: body mass index; BP: blood pressure; bw: body weight; 95% CI: 95% confidence interval; Cl: chloride; DBP: diastolic blood pressure; DONALD: Dortmund Nutritional and Anthropometrical Longitudinally Designed study FVI: fruit and vegetable intake; g: girls; MJ: megajoule; mm Hg: millimetre of mercury; mmol: millimole; N: number; Na: sodium; NR: not reported; SBP: systolic blood pressure; SD: standard deviation; SDS: standard deviation score; TEI: total energy intake; UCr: creatinine urinary excretion; UK: potassium urinary excretion; UNa: sodium urinary excretion.

H.2. Outcome of the RoB appraisal

Experimental studies

References	Risk of bias domains[a]								Tier[b]
	Randomisation	Allocation concealment	Blinding	Attrition	Exposure	Outcome	Reporting	Other threats to internal validity	
Miller et al. (1988)	+	NR	–	+	++	+	+	+	1
He et al. (2015)	++	++	+	++	++	–	++	+	2

(a): Expert judgement was translated into a rating scale for each question to be answered as follows: (++): definitely low RoB; (+): probably low RoB; (NR): not reported; (–): probably high RoB; (––): definitively high RoB.
(b): The individual rating for each question was combined by an algorithm and translated to an overall tier of reliability for each individual study (RoB tier 1: low RoB; RoB tier 2: moderate RoB; RoB tier 3: high RoB).

Observational studies

References	Risk of bias domains[a]						Tier[b]
	Confounding	Attrition	Exposure	Outcome	Reporting	Statistics	
Krupp et al. (2015)	++	–	+	++	++	++	1
Shi et al. (2014)	++	+	+	++	++	+	1

(a): Expert judgement was translated into a rating scale for each question to be answered as follows: (++): definitely low RoB; (+): probably low RoB; (NR): not reported; (–): probably high RoB; (––): definitively high RoB.
(b): The individual rating for each question was combined by an algorithm and translated to an overall tier of reliability for each individual study (RoB tier 1: low RoB; RoB tier 2: moderate RoB; RoB tier 3: high RoB).

Appendix I – Outcome of the systematic review on blood pressure levels in adults

I.1. Evidence tables

I.1.1. Experimental studies

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
Alli et al. (1992) **(Italy)**	Study design: cluster-randomised Run-in type: normal diet Intervention type: counselling N participants randomised/completed: 77/56 Duration: 12 months	% men G1: 35 G2: 50 Age (mean ± SD, years) G1: 44.3 ± 10.2 G2: 51.7 ± 11 BMI (mean ± SD, kg/m^2) G1: 25 ± 2.5 G2: 24.8 ± 2.8 Ethnicity: NR BP status: HT 24-h UK (mean ± SD, mmol) G1: 61.2 ± 18.1 G2: 59.8 ± 16.5 Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention G1: 178.7 ± 60.5 G2: 168.8 ± 65 End of intervention G1: 186.5 ± 73 G2: 163.4 ± 60.7	Type: point office, supine Mean ± SD Beginning of intervention G1: 148.5 ± 13.5/96.2 ± 4.4 G2: 148.2 ± 10.6/97.9 ± 4.9 End of intervention G1: 144.4 ± 8.1/90.6 ± 8.2 G2: 146.6 ± 15.1/94.5 ± 5.3
ANHMRCDS (1986) **(Australia)**	Study design: parallel Run-in type: normal diet Intervention type: counselling N participants randomised/completed: 107/100 Duration: 12 weeks	% men G1: 83 G2: 89 Age (mean ± SE, years) G1: 52.3 ± 0.8 G2: 52.3 ± 0.8 BP status: mixed Ethnicity: NR 24-h UK (mean, mmol) G1: 66 G2: 71 Energy intake: NR	Type: single 24 h Mean ± SE Beginning of intervention G1: 142 G2: 157 End of intervention G1: 85.8 ± 7.1 G2: 155.6 ± 8.3	Type: point office, sitting Mean Beginning of intervention G1: 150/95 G2: 149/95 End of intervention G1: 144/89 G2: 148/93

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
ANHMRCDS (1989) **(Australia)** **Australian National Health h Medical Research Council Dietary Salt Study**	Study design: parallel Run-in type: normal diet Intervention type: feeding N participants randomised/completed: 111/108 Duration: 8 weeks	% men G1: 80 G2: 87 Age (years) G1: 18 + G2: 18 + Ethnicity: NR BP status: HT 24-h UK (mean ± SE, mmol) G1: 73 ± 3 G2: 74 ± 3 Energy intake: NR	Type: single 24 h Mean ± SE Beginning of intervention G1: 142 ± 6 G2: 134 ± 6 End of intervention G1: 90 ± 6 G2: 153 ± 6	Type: point office, sitting Mean ± SE Beginning of intervention G1: 155.2 ± 2.2/95.1 ± 0.6 G2: 152.8 ± 2.1/95.4 ± 0.6 End of intervention G1: 149.1 ± 1.9/91.4 ± 0.7 G2: 152.2 ± 1.9/94.6 ± 0.9
Benetos et al. (1992) **(France)**	Study design: cross-over Wash out: 1 week Intervention type: feeding N participants randomised/completed: 22/20 Duration: 4 weeks	% men: 45 Age (mean ± SE, years): 41.5 ± 2.4 Ethnicity: NR BP status: HT 24-h UK (mean ± SE, mmol): 70 ± 3 Energy intake: NR	Type: single 24 h Mean ± SE Beginning of intervention 173 ± 13 End of intervention G1: 85 ± 9.6 G2: 163 ± 13.3	Type: point office, supine Mean ± SE Beginning of intervention 154 ± 2.2/96.1 ± 1.5 End of intervention G1: 142.6 ± 2.6/89.5 ± 1.5 G2: 149.1 ± 2.3/93.2 ± 1.3
Cappuccio et al. (1997) **(United Kingdom)**	Study design: cross-over Wash out: none Run-in type: low sodium diet Intervention type: feeding N participants randomised/completed: 48/47 Duration: 4 weeks	% men: 51 Age (mean ± SD, years): 66.8 ± 5.3 Ethnicity: mixed BP status: mixed 24-h UK (mean ± SD, mmol): 66 ± 20 Energy intake: NR	Type: multiple 24 h Mean ± SD Beginning of intervention 72 ± 40 End of intervention G1: 94 ± 50 G2: 177 ± 49	Type: point office, supine Mean ± SD Beginning of intervention 154.9 ± 20.4/87.1 ± 9.5 End of intervention G1: 155.9 ± 21.6/86.9 ± 8.8 G2: 163.2 ± 20.6/90.1 ± 10.5

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
Cobiac et al. (1992) **(Australia)**	Study design: parallel Run-in type: low sodium diet + sodium tablets Intervention type: feeding N participants randomised/completed: 57/54 Duration: 4 weeks	% men G1: 69 G2: 64 Age (mean ± SE, years) G1: 67 ± 1 G2: 67 ± 1 BMI (mean ± SE, kg/m^2) G1: 25 ± 1 G2: 25 ± 1 Ethnicity: NR BP status: mixed 24-h UK (mean ± SE, mmol) G1: 74 ± 4 G2: 68 ± 6 Energy intake: NR	Type: single 24 h Mean ± SE Beginning of intervention G1: 166 ± 10 G2: 166 ± 9 End of intervention G1: 79 ± 7 G2: 152 ± 10	Type: point office, sitting Mean ± SE Beginning of intervention G1: 132 ± 2/77 ± 2 G2: 135 ± 3/78 ± 2 End of intervention G1: 127.8/77.1 G2: 132.5/77.3
Dickinson et al. (2014) **(Australia)**	Study design: cross-over Wash out: none Intervention type: feeding N participants randomised/completed: 50/25 Duration: 6 weeks	% men: 32 Age (years): 18+ BMI (range, kg/m^2) : 27–40 Ethnicity: NR BP status: NT 24-h UK (mean ± SD, mmol): 76 ± 23 Energy intake (mean ± SD, MJ/d): 9.1 ± 2.5	Type: multiple 24 h Mean ± SD Beginning of intervention 154 ± 58 End of intervention G1: 113 ± 45 G2: 155 ± 58	Type: point office, sitting Mean ± SD Beginning of intervention 120 ± 13/77 ± 7 End of intervention G1: 115 ± 10/73 ± 6 G2: 118 ± 16/74 ± 8
Erwteman et al. (1984) **(Netherlands)**	Study design: Parallel Intervention type: counselling N participants randomised/completed: 107/94 Duration: 4 weeks	% men G1: 61 G2: 62 Age (mean ± SD, years) G1: 45 ± 11 G2: 46.5 ± 9.5 Ethnicity: mixed BP status: HT Energy intake: NR	Type: multiple 24 h mean ± SD Beginning of intervention NR End of intervention G1: 72 ± 31 G2: 130 ± 50	Type: point office, supine Mean ± SD Beginning of intervention G1: 157 ± 11.4/101.5 ± 5.4 G2: 156.2 ± 11.8/100.5 ± 3.4 End of intervention G1: 141 ± 15.4/92.9 ± 10.4 G2: 142.9 ± 16.4/94.4 ± 12.0

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
Fotherby and Potter (1993) **(United Kingdom)**	Study design: cross-over Wash out: none Run-in type: low sodium diet Intervention type: feeding N participants randomised/completed: 18/17 Duration: 5 weeks	% men: 22 Age (mean (range), years): 73 (66–79) Ethnicity: Caucasian BP status: HT 24-h UK (mean ± SD, mmol): 66 ± 25 Energy intake: NR	Type: multiple 24 h Mean ± SD Beginning of intervention 104 ± 59 End of intervention G1: 95 ± 36 G2: 174 ± 40	Type: point office, supine Mean ± Beginning of intervention 176 ± 17/96 ± 11 End of intervention G1: 171 ± 21/96 ± 8 G2: 179 ± 18/96 ± 11
Gates et al. (2004) **(USA)**	Study design: cross-over Wash out: none Intervention type: feeding N participants randomised/completed: 12/12 Duration: 4 weeks	% men: 50 Age (mean ± SE, years): 63 ± 1 BMI (mean ± SE, kg/m^2): 25.1 ± 1 Ethnicity: Caucasian BP status: HT 24-h UK (mean ± SE, mmol): 72 ± 8 Energy intake (mean ± SE, MJ/d): 9.4 ± 0.7	Type: single 24 h Mean ± SE Beginning of intervention 134.5 ± 13.4 End of intervention G1: 60.5 ± 6.8 G2: 150.1 ± 17.9	Type: point office, supine Mean ± SE Beginning of intervention 148.1 ± 2.3/84.0 ± 2.4 End of intervention G1: 137.1 ± 2.8/79.0 ± 2.1 G2: 144.2 ± 3.7/79.9 ± 2.0
Gijsbers et al. (2015) **(Netherlands)**	Study design: cross-over Wash out: none Run-in type: low sodium diet Intervention type: feeding N participants randomised/completed: 37/36 Duration: 4 weeks	% men: 67 Age (mean (range), years): 65.8 (40–80) BMI (mean, kg/m^2): 27.2 Ethnicity: Caucasian BP status: mixed 24-h UK (mean, mmol): 81.8 Energy intake (mean, kcal/d): 2,774	Type: single 24 h mean ± SD Beginning of intervention 90.8 End of intervention G1: 105.1 ± 39.7 G2: 202.9 ± 54.8	Type: point office, supine Mean ± SD Beginning of intervention 133.4/75.7 End of intervention G1: 125.1 ± 15/72.3 ± 7.7 G2: 132.9 ± 17.6/75.7 ± 7.5
Grobbee et al. (1987) **(Netherlands)**	Study design: cross-over Wash out: none Run-in type: normal diet Intervention type: feeding N participants randomised/completed: 42/40 Duration: 6 weeks	% men: 85 Age (mean ± SD, years): 24 ± 3 Ethnicity: NR BP status: HT 24-h UK (mean ± SD, mmol): 71 ± 22 Energy intake: NR	Type: multiple 24 h mean ± SE Beginning of intervention 141 ± 7 End of intervention G1: 57 ± 4 G2: 129 ± 5	Type: point office, supine Mean ± SE Beginning of intervention 143 ± 2.2/78 ± 1.6 End of intervention G1: 135.9 ± 1.8/73.7 ± 1.5 G2: 136.8 ± 1.8/73.5 ± 1.8

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
He et al. (2009) **(United Kingdom)**	Study design: cross-over Wash out: none Run-in type: low sodium diet Intervention type: feeding N participants randomised/completed: 185/169 Duration: 6 weeks	% men: 67 Age (mean ± SD, years): 50 ± 11 BMI (mean ± SD, kg/m^2) : 29 ± 5 Ethnicity: mixed BP status: HT 24-h UK (mean ± SD, mmol): 77 ± 26 Energy intake: NR	Type: multiple 24 h Mean ± SD Beginning of intervention NR End of intervention G1: 110 ± 49 G2: 165 ± 58	Type: point office, sitting Mean ± SD Beginning of intervention NR End of intervention G1: 141 ± 12/88 ± 9 G2: 146 ± 13/91 ± 8
Jablonski et al. (2013) **(USA)**	Study design: cross-over Wash out: none Intervention type: feeding N participants randomised/completed: 11/11 Duration: 5 weeks	% men: 73 Age (mean ± SE, years): 60 ± 2 BMI (mean ± SE, kg/m^2): 27.2 ± 1.3 Ethnicity: mixed BP status: mixed Energy intake: NR	Type: multiple 24 h Mean ± SE Beginning of intervention 159 ± 13 End of intervention G1: 77 ± 9 G2: 144 ± 7	Type: point office, supine mean ± SE Beginning of intervention 139 ± 2/83 ± 2 End of intervention G1: 127 ± 3/77 ± 2 G2: 138 ± 5/81 ± 2
Kumanyika et al. (2005) **(USA)** **TOHP II**	Study design: parallel Intervention type: counselling N participants randomised/completed: 1159/1159 Duration: 36 months	% men G1: 65 G2: 68 Age (mean ± SD, years) G1: 44.2 ± 6.1 G2: 43.2 ± 6.1 BMI (range, kg/m^2) 24.4–37.4 Ethnicity: mixed BP status: NT 24-h UK (mean ± SD, mmol) G1: 66.8 ± 28.1 G2: 65.3 ± 26.6 Energy intake: NR	Type: multiple 24 h Mean ± SD Beginning of intervention G1: 186.1 ± 80.7 G2: 188.0 ± 80.9 End of intervention G1: −50.9 ± 86.3 G2: −10.5 ± 88.5	Type: point office, sitting Mean ± SD Beginning of intervention G1: 127.7 ± 6.6/86.1 ± 1.9 G2: 127.3 ± 6.4/85.8 ± 1.9 End of intervention G1: −0.7 ± 9.2/−3.0 ± 6.5 G2: 0.6 ± 8.5/−2.4 ± 7.0

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
Kumanyika et al. (1993) **(USA)** **TOPH I**	Study design: parallel Intervention type: counselling N participants randomised/completed: 744/744 Duration: 18 months	% men G1: 71 G2: 72 Age (mean ± SD, years) G1: 43.4 ± 6.6 G2: 42.6 ± 6.5 BMI (mean ± SD, kg/m^2) G1: 27.1 ± 3.8 G2: 27.1 ± 3.6 Ethnicity: mixed BP status: NT 24-h UK (mean ± SD, mmol) G1: 61.8 ± 23.4 G2: 62.8 ± 23.8 Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention G1: 154.6 ± 59.9 G2: 156.4 ± 60.5 End of intervention G1: 99.4 ± 60.0 G2: 146.5 ± 79.2	Type: point office, sitting Mean ± SD Beginning of intervention G1: 124.8 ± 8.5/83.7 ± 2.7 G2: 125.1 ± 8.1/83.9 ± 2.8 End of intervention G1: −5.08 ± 7.94/−4.35 ± 5.65 G2: −3.02 ± 8.31/−3.18 ± 5.8
MacGregor et al. (1982) **(United Kingdom)**	Study design: cross-over Wash out: none Run-in type: low sodium diet Intervention type: feeding N participants randomised/completed: 19/19 Duration: 4 weeks	% men: 74 Age (mean (range), years): 49 (30–66) Ethnicity: mixed BP status: HT 24-h UK (mean ± SE, mmol): 59 ± 5 Energy intake: NR	Type: multiple 24 h Mean ± SE Beginning of intervention 83 ± 11 End of intervention G1: 86 ± 9 G2: 162 ± 9	Type: point office, supine Mean ± SE Beginning of intervention 142 ± 3/92 ± 1 End of intervention G1: 144 ± 4/92 ± 1.5 G2: 154 ± 4/97 ± 2.5
MacGregor et al. (1989) **(United Kingdom)**	Study design: cross-over Wash out: none Run-in type: low sodium diet Intervention type: feeding N participants randomised/completed: 20/20 Duration: 4 weeks	% men: 55 Age (mean (range), years): 57 (42–72) Ethnicity: mixed BP status: HT Energy intake: NR	Type: multiple 24 h Mean ± SE Beginning of intervention NR End of intervention G1: 49 ± 8 G2: 108 ± 10 G3: 190 ± 11	Type: point office, supine Mean ± SE Beginning of intervention 164 ± 4 / 101 ± 2 End of intervention G1: 147 ± 4/91 ± 2 G2: 155 ± 3/95 ± 2 G3: 163 ± 4/100 ± 2

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
Meland et al. (1997) **(Norway)**	Study design: cross-over Wash out: none Run-in type: normal diet Intervention type: feeding N participants randomised/completed: 16/16 Duration: 8 weeks	% men: 81 Age (mean (range), years): 50 (20–69) Ethnicity: NR BP status: HT Energy intake: NR	Type: single 24 h Mean (95% CI) Beginning of intervention 177 (149, 204) End of intervention G1: 125 (104, 146) G2: 191 (159, 223)	Type: point office, sitting Mean ± (95% CI) Beginning of intervention 146 (140, 153)/95 (92, 98) End of intervention G1: 141 (135, 147)/92 (89, 94) G2: 145 (137, 153)/94 (90, 97)
Melander et al. (2007) **(Sweden)**	Study design: cross-over Wash out: none Intervention type: feeding N participants randomised/completed: 46/39 Duration: 4 weeks	% men: 51 Age (mean ± SD, years): 53 ± 11 BMI (mean ± SD, kg/m^2): 26.3 ± 3.1 Ethnicity: NR BP status: NT 24-h UK (mean ± SD, mmol): 75 ± 22.9 Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention 165 ± 67.4 End of intervention G1: 50.7 ± 17.3 G2: 140 ± 39.5	Type: point office, supine Mean ± SD Beginning of intervention 136 ± 12.6/78.2 ± 7.8 End of intervention G1: 125 ± 12.4/73 ± 7.3 G2: 132 ± 14.7/75.2 ± 7.5
Nestel et al. (1993) **(Australia)**	Study design: parallel Run-in type: low sodium diet + sodium tablets Intervention type: feeding N participants randomised/completed: 70/66 Duration: 6 weeks	% men G1: 50 G2: 50 Age (mean ± SD, years) G1: 65.5 ± 4 G2: 65.5 ± 4 BMI (mean ± SD, kg/m^2) G1: 24.5 ± 3 G2: 24.5 ± 3 Ethnicity: NR BP status: NT 24-h UK (mean ± SD, mmol) G1: 87 ± 26 G2: 76.5 ± 20 Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention G1: 182.5 ± 48 G2: 162 ± 47 End of intervention G1: 91.5 ± 41 G2: 156 ± 40	Type: point office, sitting Mean ± SD Beginning of intervention G1: 126.5 ± 10/72.5 ± 7 G2: 127.5 ± 13/73.5 ± 9 End of intervention G1: 122.5 ± 9/72 ± 8 G2: 127.5 ± 14/74.5 ± 9

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
Parijs et al. (1973) **(Belgium)**	Study design: cross-over Wash out: none Run-in type: normal diet Intervention type: feeding N participants randomised/completed: 22/17 Duration: 4 weeks	% men: 46 Age (mean ± SD, years): 41.2 ± 8.21 Ethnicity: NR BP status: HT Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention NR End of intervention G1: 92.8 ± 41.8 G2: 191.1 ± 61.2	Type: point office, supine mean ± Beginning of intervention 179.1 ± 17.8/115.2 ± 10.9 End of intervention G1: 167.8 ± 24.3/115.5 ± 12.45 G2: 174.5 ± 20.02/112.3 ± 15.17
Puska et al. (1983) **(Finland)**	Study design: parallel Intervention type: counselling N participants randomised/completed: 76/72 Duration: 6 weeks	% men: NR Age (range, years): 30–50 Ethnicity: NR BP status: mixed 24-h UK (mean ± SE, mmol) G1: 81 ± 4 G2: 73 ± 3 Energy intake (mean ± SE, kcal/d) G1: 2,665 ± 120 G2: 2,609 ± 103	Type: single 24 h Mean ± SE Beginning of intervention G1: 192 ± 11 G2: 165 ± 9 End of intervention G1: 77 ± 5 G2: 167 ± 8	Type: point office, sitting Mean ± SE Beginning of intervention G1: 138.9 ± 2.3/89.6 ± 1.7 G2: 137.8 ± 2/89.3 ± 1.5 End of intervention G1: 137.2 ± 2.7/86.5 ± 1.8 G2: 136 ± 2.1/86.9 ± 1.5
Richards et al. (1984) **(New Zealand)**	Study design: cross-over Wash out: none Intervention type: feeding N participants randomised/completed: 16/12 Duration: 5 weeks	% men: 67 Age (range, years): 19–52 Ethnicity: NR BP status: HT Energy intake: NR	Type: multiple 24 h Mean ± SE Beginning of intervention NR End of intervention G1: 94 ± 7.1 G2: 202.8 ± 14.1	Type: point office, supine mean ± SE Beginning of intervention NR End of intervention G1: 144.7 ± 4/90.6 ± 3.6 G2: 149.9 ± 4.2/92.4 ± 3.5
Ruppert et al. (1993) **(Germany)**	Study design: cross-over Wash out: none Intervention type: feeding N participants randomised/completed: 25/25 Duration: 4 weeks	% men: 40 Age (mean ± SE, years): 47 ± 5 Ethnicity: NR BP status: NT Energy intake: NR	Type: single 24 h Mean ± SE Beginning of intervention NR End of intervention G1: 82 ± 3.4 G2: 199.6 ± 5.3	Type: point office, sitting Mean ± SE Beginning of intervention NR End of intervention G1: 112 ± 2.1/73.3 ± 1.4 G2: 110.3 ± 2.6/72.3 ± 1.8

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
Sacks et al. (2001) **(USA)** **DASH**	Study design: cross-over Wash out: none Run-in type: high sodium diet Intervention type: feeding N participants randomised/completed: 204/192 Duration: 4 weeks	% men: 46 Age (mean ± SD, years): 49 ± 10 BMI (mean ± SD, kg/m^2): 30 ± 5 Ethnicity: mixed BP status: mixed (41% HT) Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention 152 ± 72 End of intervention G1: 64 ± 37 G2: 106 ± 44 G3: 141 ± 55	Type: point office, sitting mean ± Beginning of intervention 134.8 ± 9.5/85.7 ± 4.5 End of intervention G1: 126 ± 10/79.8 ± 6 G2: 130 ± 11.7/82 ± 6.4 G3: 132.7 ± 11.9/83.2 ± 6.9
Santos et al. (2010) **(Portugal)**	Study design: cross-over Wash out: 6 weeks Intervention type: feeding N participants randomised/completed: 17/17 Duration: 7 weeks	% men: 47 Age (range, years): 24–53 Ethnicity: NR BP status: NT Energy intake (mean (range) kcal/d): 2,290 (1,461–3,646)	Type: single 24 h Median (IQR) Beginning of intervention NR End of intervention G1: 115 (87, 162) G2: 138 (123, 170)	Type: point office, supine Median (IQR) Beginning of intervention NR End of intervention G1: 118.3 (89, 124.3)/68 (51.7, 97.7) G2: 116.7 (92.3, 125)/68.3 (50.3, 81.3)
Schorr et al. (1996) **(Germany)**	Study design: cross-over Wash out: 2 weeks Intervention type: feeding N participants randomised/completed: 21/16 Duration: 4 weeks	% men: 44 Age (mean ± SD, years): 64.1 ± 3.6 BMI (mean ± SD, kg/m^2): 26.1 ± 3.6 Ethnicity: NR BP status: NT Energy intake: NR	Type: multiple 24 h Mean ± SD Beginning of intervention 141.8 ± 33.6 End of intervention G1: 104.6 ± 21.7 G2: 175.2 ± 29.6	Type: point office, NR mean ± Beginning of intervention 132.5 ± 22.2/73.6 ± 12.5 End of intervention G1: 125.9 ± 17.3/69.5 ± 10.1 G2: 133.6 ± 23.7/71.2 ± 12.2
Swift et al. (2005) **(United Kingdom)**	Study design: cross-over Wash out: none Run-in type: low sodium diet Intervention type: feeding N participants randomised/completed: 46/40 Duration: 4 weeks	% men: 43 Age (mean ± SD, years): 50 ± 10 BMI (mean ± SD, kg/m^2): 28 ± 4 Ethnicity: African including African Americans BP status: HT 24-h UK (mean ± SD, mmol): 63 ± 16 Energy intake: NR	Type: NR Mean ± SD Beginning of intervention NR End of intervention G1: 89 ± 52 G2: 167 ± 73	Type: point office, supine Mean ± SD Beginning of intervention NR End of intervention G1: 151 ± 13/98 ± 8 G2: 159 ± 13/101 ± 8

References (country)	Design	Subject characteristics at baseline	UNa (mmol/24 h)	BP measurement SBP/DBP (mm Hg)
van Berge-Landry and James (2004) **(USA)**	Study design: cross-over Wash out: none Run-in type: normal diet Intervention type: counselling N participants randomised/completed: 48/48 Duration: 4 weeks	% men: 79 Age: middle-aged adults Ethnicity: mixed BP status: HT Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention NR End of intervention G1: 24 ± 13 G2: 309 ± 88	Type: point office, NR Mean ± SD Beginning of intervention NR End of intervention G1: 128 ± 10/85 ± 7 G2: 144 ± 14/93 ± 8
Watt et al. (1983) **(United Kingdom)**	Study design: cross-over Wash out: none Run-in type: low sodium diet Intervention type: feeding N participants randomised/completed: 20/18 Duration: 4 weeks	% men: 33 Age (mean, range, years): 52 (31–64) Ethnicity: NR BP status: HT 24-h UK (mean, mmol): 59.2 Energy intake: NR	Type: single 24 h Mean Beginning of intervention NR End of intervention G1: 87.2 G2: 142.8	Type: point office, sitting Mean Beginning of intervention NR End of intervention G1: 136/82.3 G2: 136.5/82.6
Watt et al. (1985) **(United Kingdom)** **High/high**	Study design: cross-over Wash out: none Intervention type: feeding N participants randomised/completed: 75/66 Duration: 4 weeks	% men: 37 Age (mean, years): 22.3 Ethnicity: NR BP status: mixed 24-h UK (mean ± SD, mmol): 53.8 ± 18.9 Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention 129.7 ± 55.3 End of intervention G1: 56.3 G2: 130.6	Type: point office, sitting mean ± Beginning of intervention 113.1 ± 13.3/65.2 ± 11.2 End of intervention G1: 112.2/64.5 G2: 113.6/63.3
Watt et al. (1985) **(United Kingdom)** **Low/low**	Study design: cross-over Wash out: none Intervention type: feeding N participants randomised/completed: 75/66 Duration: 4 weeks	% men: 45 Age (mean, years): 22.7 Ethnicity: NR BP status: mixed 24-h UK (mean ± SD, mmol): 60 ± 18.3 Energy intake: NR	Type: single 24 h Mean ± SD Beginning of intervention 151.1 ± 63.6 End of intervention G1: 68.4 G2: 128.4	Type: point office, sitting mean ± SD Beginning of intervention 108.8 ± 11.5/61.9 ± 9.5 End of intervention G1: 110.2/65 G2: 110.7/63.6

BMI: body mass index; BP: blood pressure; 95% CI: 95% confidence interval; DBP: diastolic blood pressure; G1: group 1 ('low' sodium); G2: group 2 ('high' sodium); G3: group 3 ('highest' sodium, for studies with three sodium intake levels); HT: hypertensive; IQR: interquartile range; mm Hg: millimetre of mercury; N: number; Na: sodium; NR: not reported; NT: normotensive; SBP: systolic blood pressure; SD: standard deviation; SE: standard error; UK: potassium urinary excretion; UNa: sodium urinary excretion.

I.1.2. Observational studies

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
(Stolarz-Skrzypek et al., 2011) **(Belgium, Czech Republic, Italy, Poland, Russian Federation)** **FLEMENGHO/ EPOGH**	Prospective cohort N = 3,360 FLEMENGHO initial cohort; N = 1,187 EPOGH initial cohort N = 1,499 included in the analyses ('blood pressure cohort') (excluded: participants with treated hypertension, history of CVD, missing/inaccurate 24-h UNa at baseline) Median duration: 6.1 years	Male (%) 47.6 Hypertensive (untreated) (%) 9.9 Diabetes mellitus (%) 1.9 Age (mean (SD), years) 38.3 (14.2) BMI (mean (SD), kg/m^2) 24.6 (4.0) Smokers (%) 30.4 SBP (mean (SD), mm Hg) 120.9 (12.8) DBP (mean (SD), mm Hg) 74.6 (8.9) 24-h UK (mean (SD), mmol) 66.3 (22.4) 24-h UCr (mean (SD), mmol) 11.9 (3.7)	Single 24-h urine sample at baseline and at last follow-up examination Inaccurate urine collections were defined as a volume < 300 mL/ 24-h, a 24-h UCr < 4 mmol or > 25 mmol in women and < 6 mmol or > 30 mmol in men	Mean (SD) 172.7 (62.5)	Longitudinal change in SBP and DBP BP measured at baseline and follow-up examinations (5 blood pressure readings averaged at each examination)	Study population, sex, baseline values of and changes in: age, BMI, and alcohol intake, 24-h UK, use of female sex steroids, and use of non-steroidal anti-inflammatory drugs	Change in SBP (mm Hg)/ 100 mmol increase in Na excretion (mean (95% CI)): FLEMENGHO: 2.373 (1.154 to 3.392), $p < 0.001$ EPOGH: 0.196 (−1.409 to 1.801), $p = 0.81$ All: 1.711 (0.786 to 2.637), $p < 0.001$ Change in DBP (mm Hg)/ 100 mmol increase in Na excretion (mean (95% CI)): FLEMENGHO: 0.576 (−0.246 to 1.398) $p = 0.17$ EPOGH: −0.052 (−1.317 to 1.212), $p = 0.94$ All: 0.379 (−0.313 to 1.070), $p = 0.28$

BMI: body mass index; BP: blood pressure; 95% CI: 95% confidence interval; CVD: cardiovascular disease; DBP: diastolic blood pressure; EPOGH: European Project on Genes in Hypertension; FLEMENGHO: Flemish Study on Genes and Health Outcomes; m: men; mm Hg: millimetre of mercury; N: number; Na: sodium; SBP: systolic blood pressure; SD: standard deviation; UCr: creatinine urinary excretion; UK: potassium urinary excretion; UNa: sodium urinary excretion; w: women.

I.2. Outcome of the RoB appraisal

I.2.1. Experimental studies

References	Risk of bias domains[a]								Tier[b]
	Randomisation	Allocation concealment	Blinding	Attrition	Exposure	Outcome	Reporting	Other threats to internal validity	
Alli et al. (1992)	+	– –	– –	– –	++	– –	++	–	2
ANHMRCDS (1989)	+	NR	+ +	+	+	+ +	+ +	+	1
Benetos et al. (1992)	+	NR	+	+ +	+	+	++	+	1
Cappuccio et al. (1997)	++	NR	+ +	++	+	++	++	++	1
ANHMRCDS (1986)	+	NR	–	+ +	+	+	+	+	1
Cobiac et al. (1992)	NR	NR	++	+	++	+	++	+	2
Dickinson et al. (2014)	++	NR	+	–	+	+	++	++	1
Erwteman et al. (1984)	+	NR	+	+	+	+ +	+ +	–	1
Fotherby and Potter (1993)	+	NR	+	+ +	+	+	++	++	1
Gates et al. (2004)	+	+	+ +	+ +	+	++	+ +	++	1
Gijsbers et al. (2015)	+ +	+ +	+ +	+ +	+	+ +	+ +	+ +	1
Grobbee et al. (1987)	+	NR	+	++	+	++	–	++	1
He et al. (2009)	++	+	++	+	+	++	++	++	1
Jablonski et al. (2013)	++	+	+ +	+	+	+ +	+ +	++	1
Kumanyika et al. (2005)	+	NR	+ +	+ +	+	+ +	+ +	++	1
Kumanyika et al. (1993)	++	++	+	++	+	++	++	+	1
MacGregor et al. (1982)	++	++	+	++	+	++	++	+	1
MacGregor et al. (1989)	+	NR	+	+ +	+	+ +	+ +	+ +	1
Meland et al. (1997)	+	NR	+	+ +	+	+ +	+ +	+ +	1
Melander et al. (2007)	+	NR	+	+ +	+	+	+ +	++	1
Nestel et al. (1993)	+	NR	+ +	+	+	+ +	+ +	+ +	1
Parijs et al. (1973)	NR	NR	+	+ +	+ +	+	+ +	+	2
Puska et al. (1983)	+	NR	–	+	+	–	+ +	+	1
Richards et al. (1984)	+	NR	+	+ +	+ +	+	+ +	–	1
Ruppert et al. (1993)	–	–	–	+	+	+	+	+	2
Sacks et al. (2001)	+	NR	+	+	+	+	++	++	1
Santos et al. (2010)	+	+	+	+ +	+	+ +	+ +	++	1
Schorr et al. (1996)	+	NR	–	+ +	+	+	+ +	+	1
Swift et al. (2005)	+	NR	+	+	+	+	++	+	1
van Berge-Landry and James (2004)	+	NR	+ +	+ +	+	+	++	+ +	1
Watt et al. (1983)	+	NR	–	+ +	+	NR	+ +	+	2
Watt et al. (1985)	+	NR	+	+ +	+	+	+ +	+ +	1

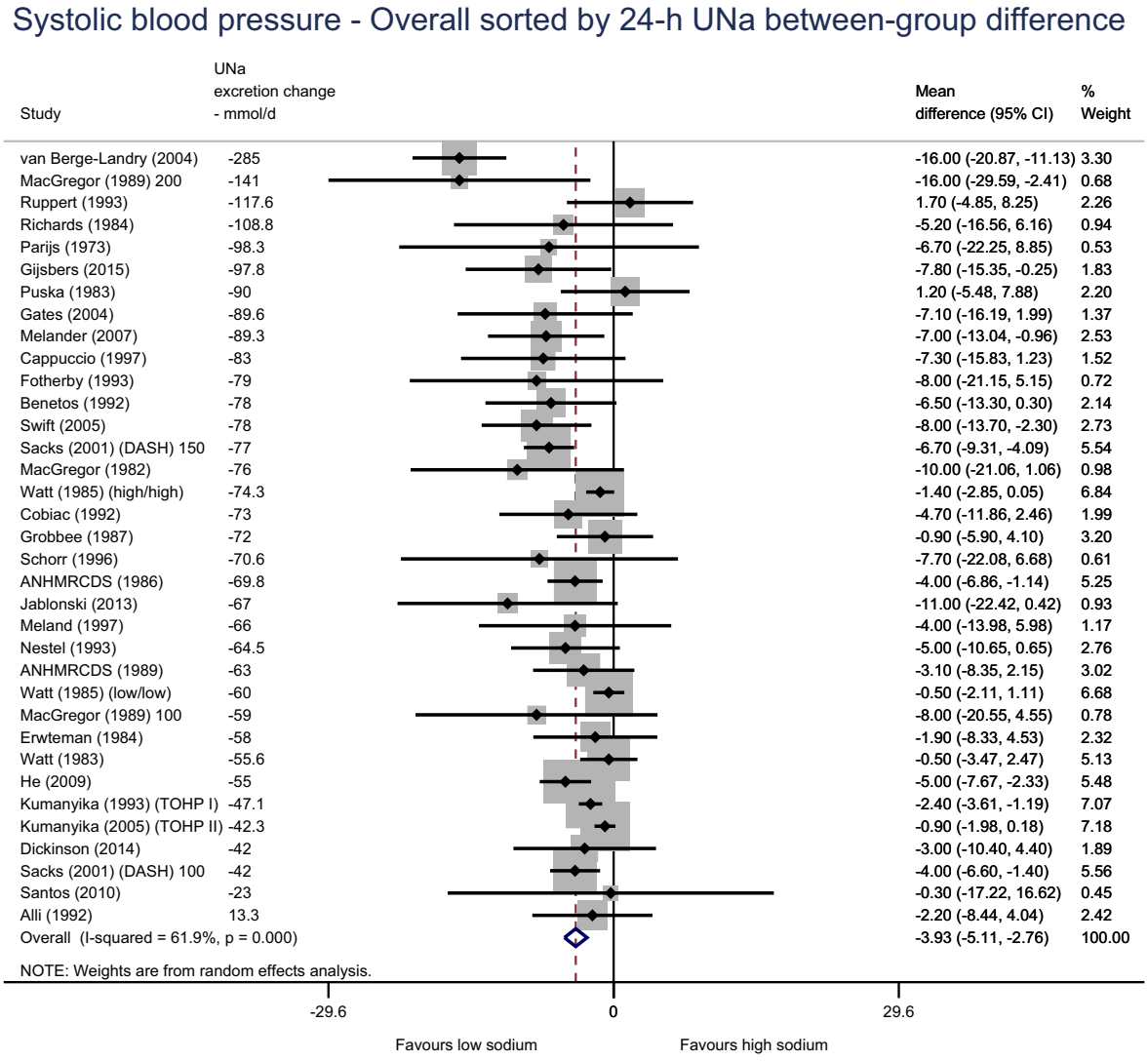
(a): Expert judgement was translated into a rating scale for each question to be answered as follows: (++): definitely low RoB; (+): probably low RoB; (NR): not reported; (–): probably high RoB; (––): definitively high RoB.
(b): The individual rating for each question was combined by an algorithm and translated to an overall tier of reliability for each individual study (RoB tier 1: low RoB; RoB tier 2: moderate RoB; RoB tier 3: high RoB).

I.2.2. Observational studies

References	Risk of bias domains[a]						Tier[b]
	Confounding(a)	Attrition	Exposure	Outcome	Reporting	Statistics	
Stolarz-Skrzypek et al. (2011)	–	–	–	++	++	++	2

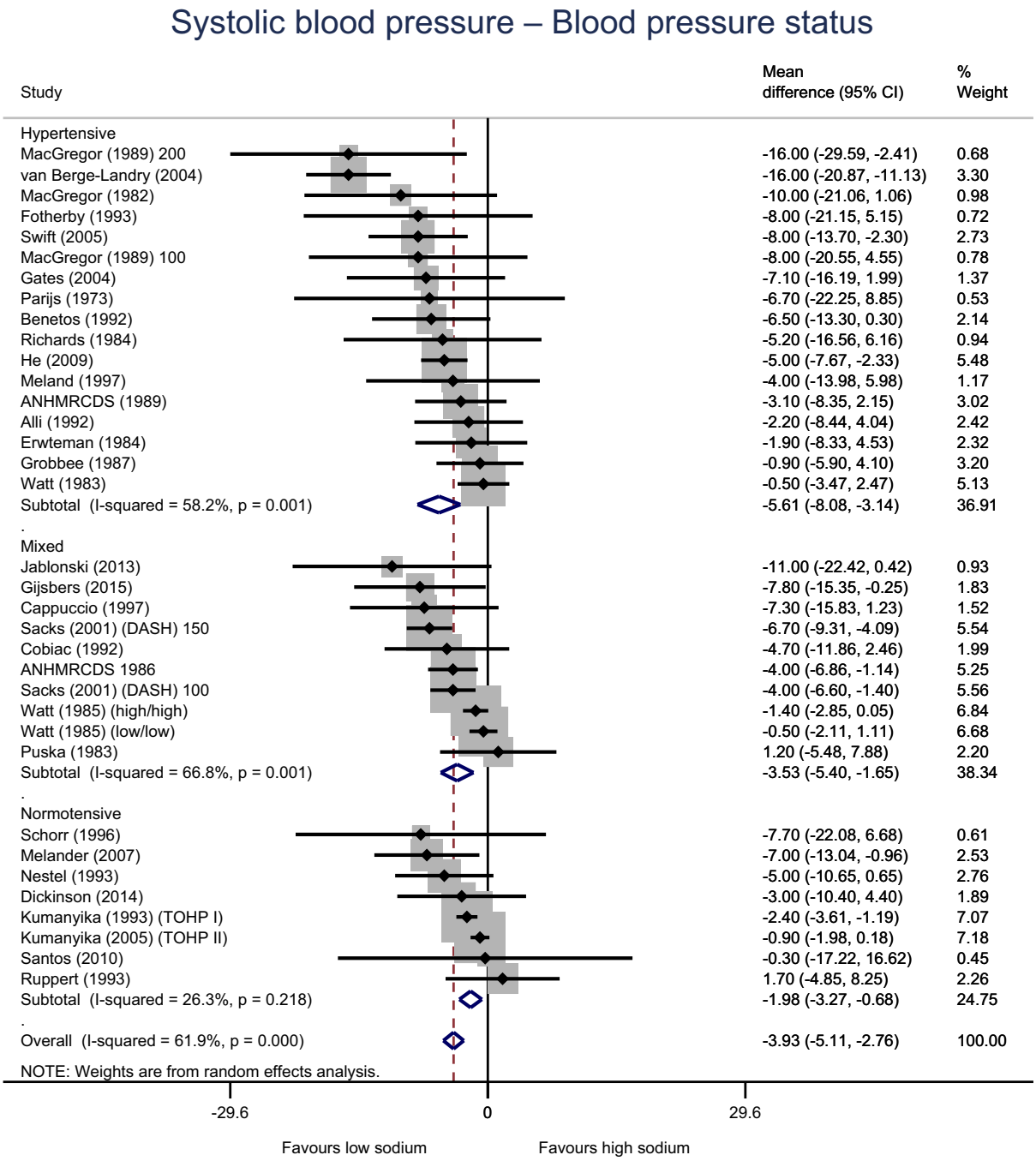
(a): Expert judgement was translated into a rating scale for each question to be answered as follows: (++): definitely low RoB; (+): probably low RoB; (NR): not reported; (−): probably high RoB; (−−): definitively high RoB.
(b): The individual rating for each question was combined by an algorithm and translated to an overall tier of reliability for each individual study (RoB tier 1: low RoB; RoB tier 2: moderate RoB; RoB tier 3: high RoB).

I.3. Random-effects meta-analyses of trials of effects of sodium reduction on SBP and DBP



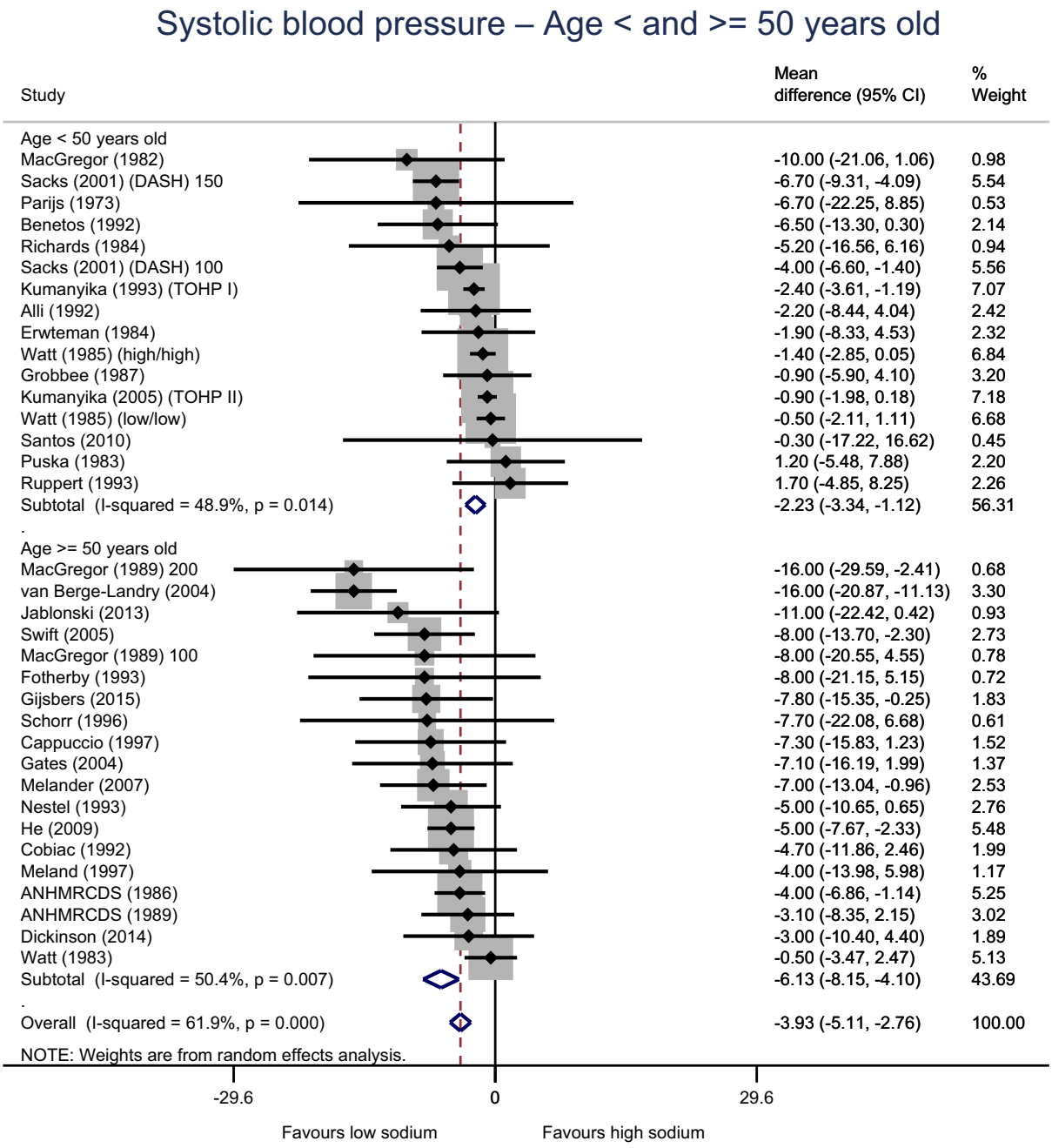
95% CI: 95% confidence interval; UNa: sodium urinary excretion.

Figure I.1: sbp – all adults, sorted by 24-h UNa between-group difference



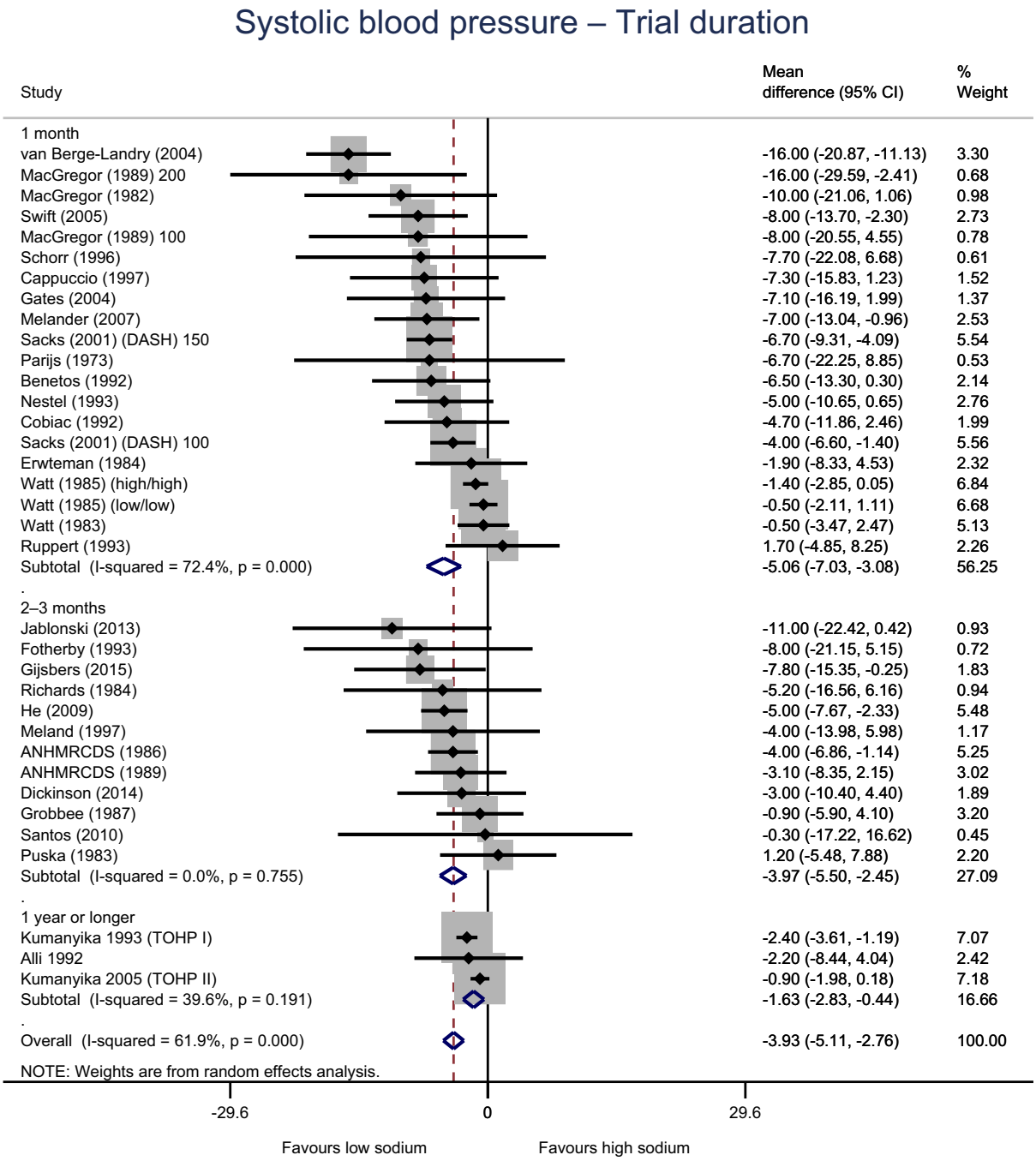
95% CI: 95% confidence interval.

Figure I.2: SBP – all adults, subgroups by blood pressure status



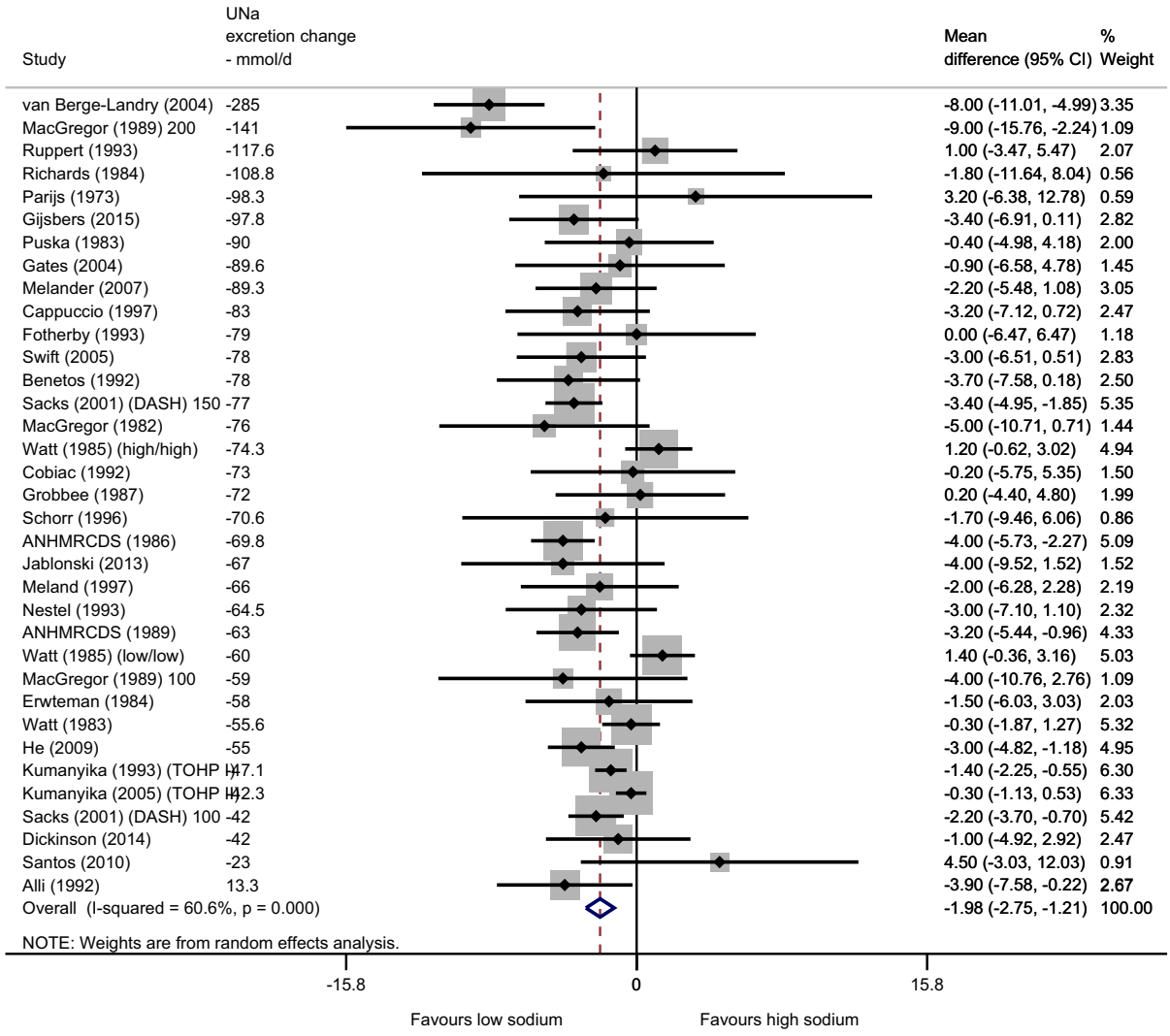
95% CI: 95% confidence interval.

Figure I.3: SBP – all adults, subgroups by age < or ≥ 50 years



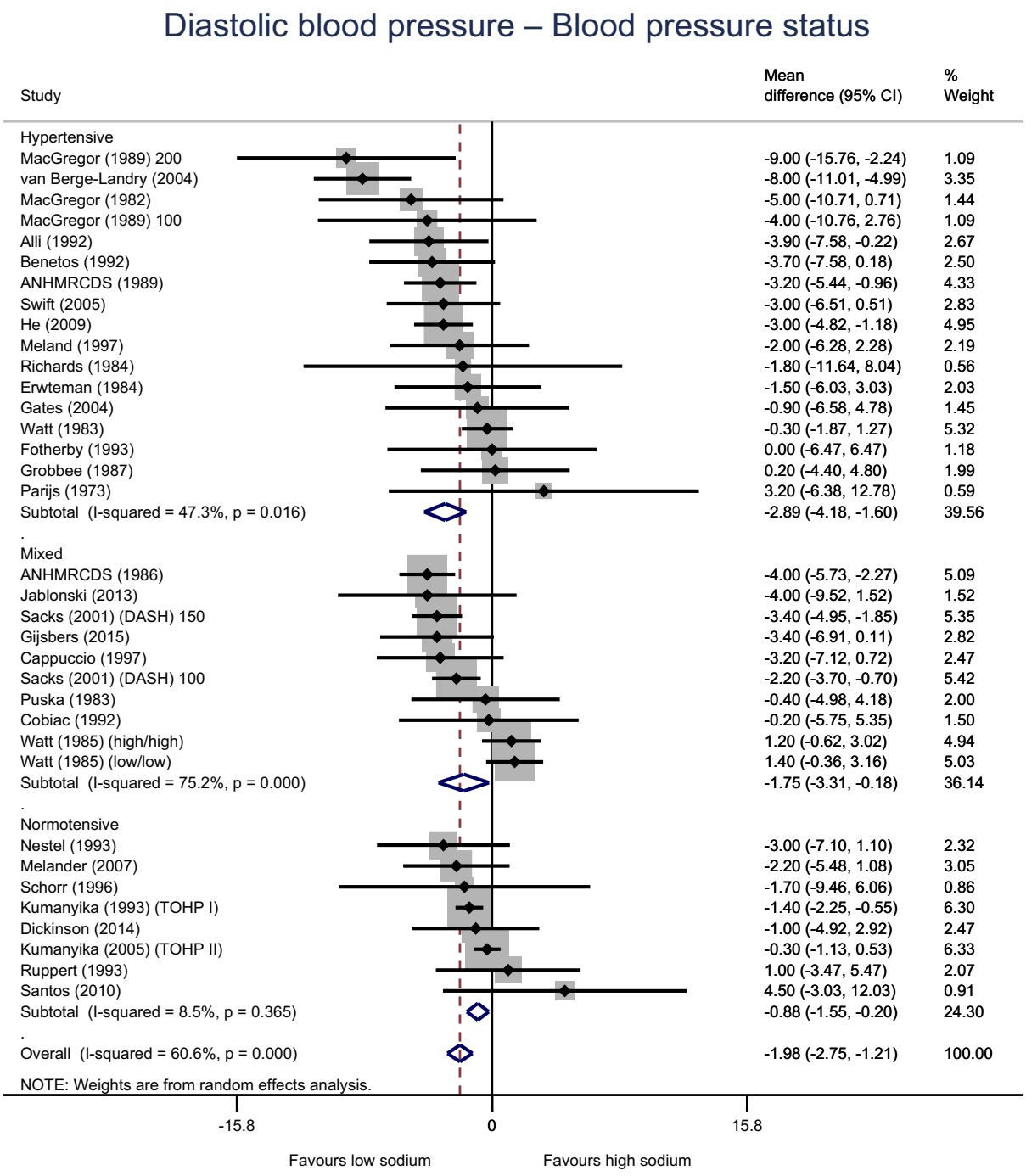
95% CI: 95% confidence interval.

Figure I.4: SBP – all adults, subgroups by trial duration



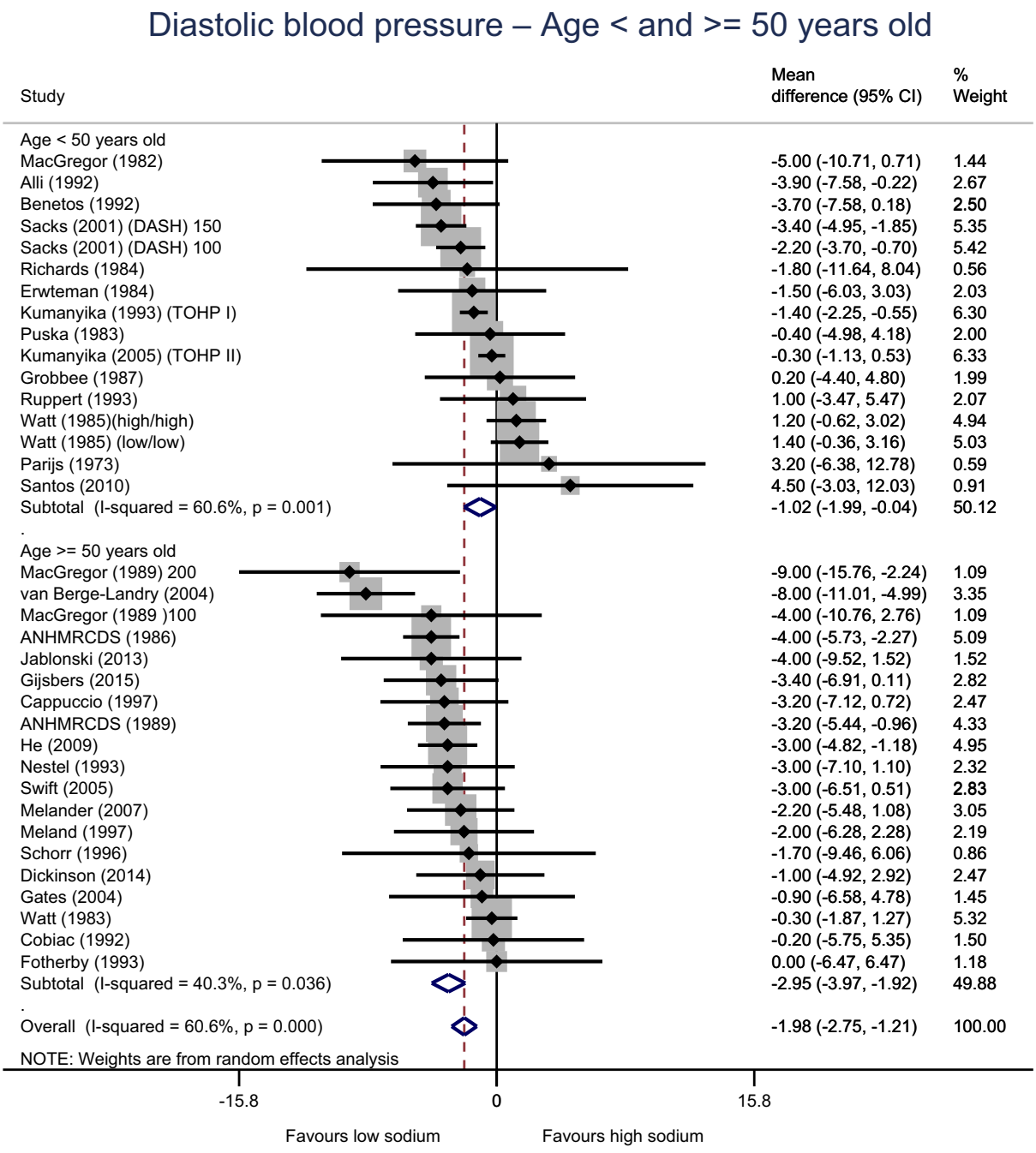
95% CI: 95% confidence interval; UNa: sodium urinary excretion.

Figure I.5: DBP – all adults, sorted by 24-h UNa between-group difference



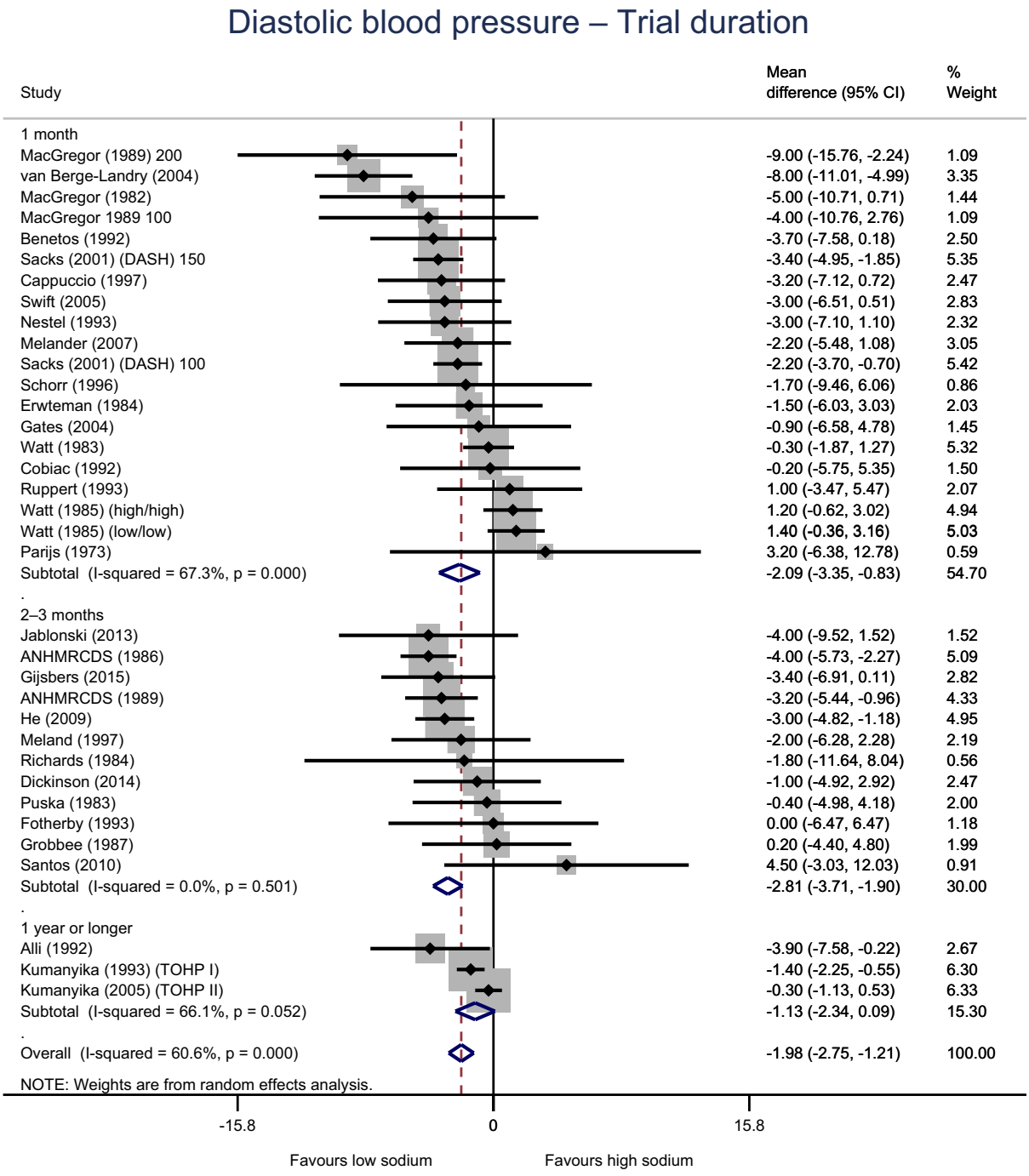
95% CI: 95% confidence interval.

Figure I.6: DBP – all adults, subgroups by blood pressure status



95% CI: 95% confidence interval.

Figure I.7: DBP – all adults, subgroups by age < or ≥ 50 years



95% CI: 95% confidence interval.

Figure I.8: DBP – all adults, subgroups by trial duration

Table I.1: Pooled estimates of the effect of sodium reduction on SBP (mean difference expressed in mm Hg)

		N studies	N particip.	Mean diff.	95% CI		I^2	p
All	**Adults**	35	3,407	−3.9	−5.1	−2.8	62%	< 0.001
BP	**Hypertensive**	17	721	−5.6	−8.1	−3.1	58%	0.001
	Mixed	10	770	−3.5	−5.4	−1.7	67%	0.001
	Normotensive	8	1,916	−2.0	−3.3	−0.7	26%	0.218
Age	**Adults < 50 years**	16	632	−2.2	−3.3	−1.1	49%	0.014
	Adults ≥ 50 years	19	2,775	−6.1	−8.2	−4.1	50%	0.007
Sex	**> 55% men**	14	2,430	−4.5	−6.5	−2.6	74%	< 0.001
	45–55% both genders	14	761	−4.7	−7.0	−2.5	55%	0.007
	> 55% women	7	216	−1.8	−3.6	0.0	20%	0.277
BMI	**< 25**	1	66	−5.0	−10.6	0.6	–	–
	25–29	10	1,132	−4.6	−6.3	−2.8	26%	0.207
	≥ 30	2	384	−5.3	−8.0	−2.7	52%	0.151
	NR	22	1,825	−3.1	−4.7	−1.6	62%	< 0.001
Ethnicity	**Caucasian**	3	65	−7.6	−12.9	−2.3	0%	0.991
	African (including AA)	1	40	−8.0	−13.7	−2.3	–	–
	Mixed	12	2,540	−5.7	−8.0	−3.4	82%	< 0.001
	NR	19	762	−1.7	−2.5	−0.8	0%	0.578
Potassium	**≤ 60 mmol/day**	4	103	−1.0	−2.1	0.1	9%	0.346
	> 60–≤ 70 mmol/day	8	2,008	−3.0	−4.6	−1.4	52%	0.043
	> 70–≤ 80 mmol/day	7	442	−4.3	−6.2	−2.5	0%	0.743
	> 80 mmol/day	3	174	−3.8	−8.7	1.2	41%	0.183
	NR	13	680	−6.4	−9.5	−3.3	59%	0.004
Design	**Parallel**	8	2,217	−2.0	−3.1	−1.0	20%	0.272
	Crossover	26	1,134	−5.0	−6.8	−3.3	67%	< 0.001
	Cluster-randomised	1	56	−2.2	−8.4	4.0	–	–
Specific design	**Run-in – normal diet**	7	380	−5.2	−9.6	−0.9	75%	0.001
	Run-in – low Na diet	9	386	−5.8	−8.6	−2.9	43%	0.083
	Run-in – high Na diet	4	504	−5.3	−7.0	−3.6	0%	0.552
	No run-in	15	2,137	−1.6	−2.5	−0.8	18%	0.254
Trial duration	**1 month**	20	1,005	−5.1	−7.0	−3.1	72%	< 0.001
	2–3 months	12	618	−4.0	−5.5	−2.5	0%	0.755
	≥ 1 year	3	1784	−1.6	−2.8	−0.4	40%	0.191
Intervention type	**Feeding**	28	1,309	−4.0	−5.3	−2.7	44%	0.007
	Counselling	7	2,098	−3.7	−6.2	−1.1	85%	< 0.001
Position	**Supine**	17	517	−5.6	−7.5	−3.6	0%	0.762
	Seated	16	2,826	−2.5	−3.6	−1.5	57%	0.003
	NR	2	64	−14.7	−20.6	−8.8	13%	0.284
UNa difference	**≤ 50 mmol**	6	2,018	−2.0	−3.0	−0.9	23%	0.26
	51–75 mmol	14	773	−2.4	−3.6	−1.2	27%	0.169
	76–100 mmol	11	511	−6.5	−8.3	−4.7	0%	0.814
	> 100 mmol	4	105	−8.6	−19.0	1.7	85%	< 0.001
Tier	**Tier 1**	30	3,171	−3.3	−4.4	−2.2	50%	0.001
	Tier 2	5	236	−6.9	−12.8	−1.1	75%	0.003

BP: blood pressure; 95% CI: 95% confidence interval; diff.: difference; N: number; Na: sodium; NR: not reported; particip.: participants; SBP: systolic blood pressure; UNa: sodium urinary excretion.

Table I.2: Pooled estimates of the effect of sodium reduction on DBP (mean difference expressed in mm Hg)

		N studies	N particip.	Mean diff.	95% CI		I^2	p
All	**Adults**	35	3,407	−2.0	−2.8	−1.2	61%	< 0.001
BP	**Hypertensive**	17	721	−2.9	−4.2	−1.6	47%	0.016
	Mixed	10	770	−1.7	−3.3	−0.2	75%	< 0.001
	Normotensive	8	1,916	−0.9	−1.6	−0.2	9%	0.365
Age	**Adults < 50 years**	16	632	−1.0	−2.0	0.0	61%	0.001
	Adults ≥ 50 years	19	2,775	−2.9	−4.0	−1.9	40%	0.036
Sex	**> 55% men**	14	2,430	−2.7	−3.9	−1.5	70%	< 0.001
	45–55% both genders	14	761	−1.9	−3.3	−0.5	53%	0.01
	> 55% women	7	216	−0.6	−2.0	0.8	33%	0.173
BMI	**< 25**	1	66	−3.0	−7.1	1.1	–	–
	25–29	10	1,132	−1.9	−2.6	−1.2	0%	0.739
	≥ 30	2	384	−2.8	−4.0	−1.6	16%	0.276
	NR	22	1,825	−1.7	−2.8	−0.5	69%	< 0.001
Ethnicity	**Caucasian**	3	65	−2.2	−4.9	0.5	0%	0.579
	African (including AA)	1	40	−3.0	−6.5	0.5	–	–
	Mixed	12	2,540	−3.0	−4.2	−1.7	74%	< 0.001
	NR	19	762	−1.2	−2.3	0.0	54%	0.003
Potassium	**≤ 60 mmol/day**	4	103	0.4	−1.1	1.9	51%	0.105
	> 60–≤ 70 mmol/day	8	2,008	−2.2	−3.4	−0.9	66%	0.005
	> 70–≤ 80 mmol/day	7	442	−2.4	−3.5	−1.3	0%	0.742
	> 80 mmol/day	3	174	−2.5	−4.8	−0.2	0%	0.573
	NR	13	680	−2.9	−4.5	−1.2	51%	0.017
Design	**Parallel**	8	2,217	−1.8	−3.0	−0.7	63%	0.009
	Crossover	26	1,134	−2.0	−3.1	−0.9	61%	< 0.001
	Cluster-randomised	1	56	−3.9	−7.6	−0.2	–	–
Specific design	**Run-in – normal diet**	7	380	−3.6	−5.4	−1.7	55%	0.036
	Run-in – low Na diet	9	386	−2.7	−4.1	−1.2	38%	0.119
	Run-in – high Na diet	4	504	−2.7	−3.7	−1.7	0%	0.57
	No run-in	15	2137	−0.5	−1.3	0.3	30%	0.131
Duration	**1 month**	20	1,005	−2.1	−3.4	−0.8	67%	< 0.001
	2–3 months	12	618	−2.8	−3.7	−1.9	0%	0.501
	≥ 1 year	3	1,784	−1.1	−2.3	0.1	66%	0.052
Intervention type	**Feeding**	28	1,309	−1.7	−2.6	−0.9	46%	0.004
	Counselling	7	2,098	−2.7	−4.4	−1.0	83%	< 0.001
Position	**Supine**	17	517	−2.7	−3.8	−1.5	0%	0.66
	Seated	16	2,826	−1.4	−2.3	−0.5	70%	< 0.001
	NR	2	64	−5.9	−11.7	−0.1	55%	0.138
UNa difference	**≤ 50 mmol**	6	2,018	−1.3	−2.3	−0.2	52%	0.063
	51–75 mmol	14	773	−1.5	−2.8	−0.2	64%	0.001
	76–100 mmol	11	511	−2.9	−3.9	−1.9	0%	0.869
	> 100 mmol	4	105	−4.6	−10.1	0.8	76%	0.006
Tier	**Tier 1**	30	3,171	−1.7	−2.4	−0.9	56%	< 0.001
	Tier 2	5	236	−4.1	−7.0	−1.2	52%	0.081

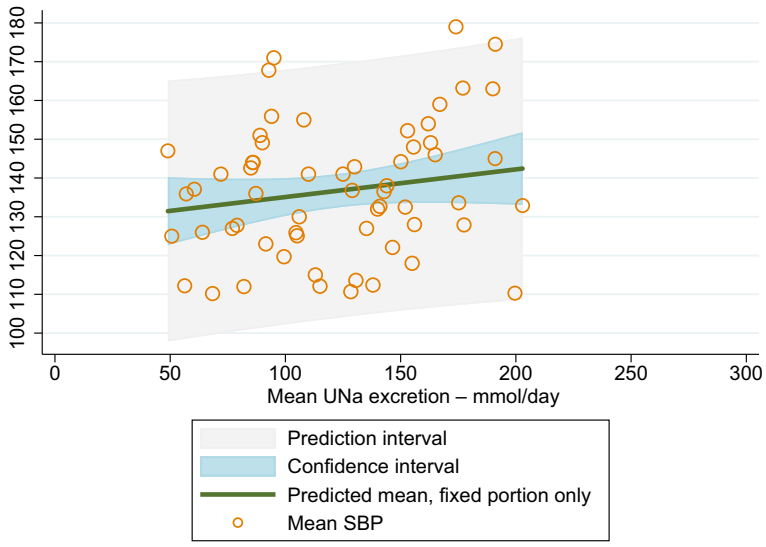
BP: blood pressure; 95% CI: 95% confidence interval; diff.: difference; N: number; Na: sodium; NR: not reported; particip.: participants; DBP: diastolic blood pressure; UNa: sodium urinary excretion.

I.4. Dose–response modelling

Table I.3: Multivariable mixed-effects meta-regression model on SBP, only fixed effects reported

Covariate	β coefficient	Std. Err.	p > |z|	95% CI
Mean UNa – per 100 mmol/day	**5.3**	**0.8**	**< 0.001**	**(3.6 to 6.9)**
Age at baseline				
Age < 40 years old[(a)]	0			
Age 40–49 years old	18.2	4.4	< 0.001	(9.6 to 26.9)
Age 50–59 years old	11.6	4.7	0.013	(2.4 to 20.7)
Age ≥ 60 years old	12.4	4.2	0.003	(4.3 to 20.6)
Blood pressure status				
Normotensive[(a)]	0			
Hypertensive	11.4	4.5	0.011	(2.6 to 20.1)
UNa at baseline[(b)]				
< 100 mmol/day[(a)]	0			
100–149 mmol/day	25.2	6.3	< 0.001	(12.7 to 37.6)
≥150 mmol/day	18.7	6.4	0.004	(6.0 to 31.3)
Not reported	13.4	5.3	0.012	(3.0 to 23.8)
BP measurement method				
Point office, supine[(a)]	0			
Point office, sitting	−13.2	3.2	< 0.001	(−19.6 to −6.9)
Specific trial design				
No Run-in[(a)]		0		
Run-in, Normal diet	11.9	3.4	< 0.001	(5.3 to 18.5)
Run-in, Low Na diet	19.7	5.1	< 0.001	(9.7 to 29.8)
Constant	95.5	6.8	< 0.001	(82.2 to 108.8)

95% CI: 95% confidence interval; Std. Err.: standard error; UNa: sodium urinary excretion.
(a): Reference category.
(b): UNa at baseline: corresponds to UNa at the start of the intervention (i.e. after run-in, where applicable).
Model centred at 49 mmol/day sodium excretion (minimum mean UNa observed in the data set); total heterogeneity (random effects on trial) estimated from null model = 275.2 (95% CI: 161.4–468.9), residual heterogeneity from full model = 33.4 (95% CI: 13.3–53.5).



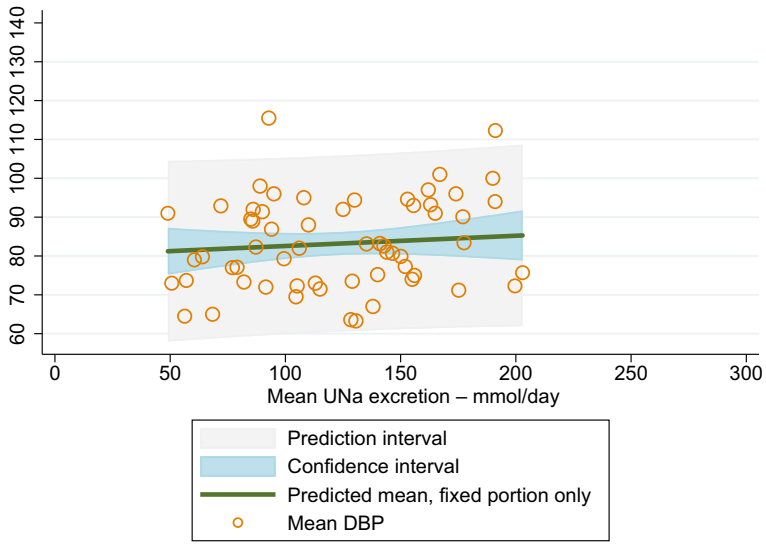
Circles represents mean SBP by arm and their size is proportional to weights from the mixed-effects model. The slope from the full model with moderators did not differ substantially (UNa unadjusted coefficient: 5.2 mm Hg per 100 mmol/day, 95% CI: 3.6–6.9).

Figure I.9: Linear dose–response relationship between mean urinary sodium excretion and mean SBP (mm Hg) from meta-regression modelling of trials' arms (crude model)

Table I.4: Multivariable mixed-effects meta-regression model on DBP, only fixed effects reported

Covariate	β coefficient	Std. Err	p > |z|	95% CI
Mean UNa – per 100 mmol/day	2.6	0.5	< 0.001	(1.6 to 3.7)
Age at baseline				
Age < 40 years old[(a)]	0			
Age 40–49 years old	18.2	3.2	< 0.001	(11.9 to 24.6)
Age 50–59 years old	11.8	3.5	0.001	(5.1 to 18.6)
Age ≥ 60 years old	7.8	3.1	0.011	(1.8 to 13.8)
Blood pressure status				
Normotensive[(a)]	0			
Hypertensive	8.7	3.3	0.008	(2.3 to 15.1)
UNa at baseline[(b)]				
< 100 mmol/day[(a)]	0			
100–149 mmol/day	10.4	4.5	0.022	(1.5 to 19.3)
≥ 150 mmol/day	10.2	4.7	0.028	(1.1 to 19.4)
Not reported	10.4	3.8	0.007	(2.9 to 17.9)
BP measurement method				
Point office, supine[(a)]	0			
Point office, sitting	−6.6	2.4	0.005	(−11.3 to −2.0)
Specific trial design				
No Run-in[(a)]		0		
Run-in, Normal diet	7.9	2.5	0.002	(3.0 to 12.7)
Run-in, Low Na diet	7.7	3.7	0.037	(0.5 to 15.0)
Constant	55.4	4.8	< 0.001	(45.9 to 64.8)

BP: blood pressure; 95% CI: 95% confidence interval; Std. Err: standard error; UNa: sodium urinary excretion.
(a): Reference category.
(b): UNa at baseline: corresponds to UNa at the start of the intervention (i.e. after run-in, where applicable).
Model centred at 49 mmol/day sodium excretion (minimum mean UNa observed in the data set); total heterogeneity (random effects on trial) estimated from null model = 129.1 (95% CI: 76.1–218.8), residual heterogeneity from full model = 18.8 (95% CI: 8.1–29.4).



Circles represents mean DBP by arm and their size is proportional to weights from the mixed-effects model. The slope from the full model with moderators did not differ substantially (UNa unadjusted coefficient: 2.6 mm Hg per 100 mmol/day, 95% CI: 1.6–3.7).

Figure I.10: Linear dose–response relationship between mean urinary sodium excretion and mean DBP (mm Hg) from meta-regression modelling of trials' arms (crude model)

Appendix J – Outcome of the systematic review on incidence of hypertension

J.1. Evidence tables

J.1.1. Experimental studies

References (country)	Design	Subjects characteristics at baseline	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Results
Whelton et al. (1997) **(USA)** **TOPH I**	RCT (parallel) G1: sodium reduction counselling (goal < 80 mmol/24 h) (N = 326) G2: usual care (N = 417) Recruitment criteria: aged 35–54 years, with high-normal DBP Duration: 18 months	Sex (male,%) G1: 70.9 G2: 71.7 Age (years, mean ± SD) G1: 43.4 ± 6.6 G2: 42.6 ± 6.0 Ethnicity (White,%) G1: 78.0 G2: 76.5 bw (kg, mean ± SD) G1: 82.7 ± 14.3 G2: 82.8 ± 14.0 SBP (mm Hg, mean ± SD) G1: 124.8 ± 8.5 G2: 125.1 ± 8.1 DBP (mm Hg, mean ± SD) G1: 83.7 ± 2.7 G2: 83.9 ± 2.8	Average of two 24-h urine collection at baseline single 24-h urine collection at the 6-, 12- and 18-month follow-up visits	Mean ± SD Baseline G1: 154.6 ± 77.9 G2: 156.4 ± 60.5 Diff: –1.8 At 18 month G1: 99.4 ± 60.0 G2: 146.5 ± 79.2 Diff: –47.2	Incident hypertension Hypertension diagnosed when mean of 9 DBP measurements ≥ 90 mm Hg or antihypertensive drug therapy prescribed	Incidence of hypertension during follow-up G1: 8.6% G2: 11.3% RR = 0.76 (95% CI 0.49, 1.18)
The Trials of Hypertension Collaborative Research Group (1997) **(USA)** **TOPH II**	RCT (parallel) G1: sodium reduction counselling (goal < 80 mmol/24 h) (N = 594) G2: usual care (N = 596) Recruitment criteria: aged 30–54 years, with high-normal DBP, overweight	Sex (male,%) G1: 64.8 G2: 68.3 Age (years, mean ± SD) G1: 44.2 ± 6.1 G2: 43.2 ± 6.1 Ethnicity (White, %) G1: 81.1 G2: 79.5 bw (kg, mean ± SD) G1: 94.0 ± 14.3 G2: 93.6 ± 13.5	Average of two 24-h urine collection at baseline single 24-h urine collection at the 18-, 36-month follow-up visits	Mean ± SD Baseline G1: 186.1 ± 80.7 G2: 188.0 ± 80.9 Diff: –1.9 At 36 month (change from baseline) G1: –50.9 ± 86.3 G2: –10.5 ± 88.5 Diff: –40.4	Incident hypertension Hypertension diagnosed when mean of 9 DBP measurements ≥ 90 mm Hg or mean of 9 SBP measurements ≥ 140 mm Hg or antihypertensive drug therapy prescribed	Incidence of hypertension At 18 month (cases, %) G1: 108 (18.6) G2: 124 (21.1) RR = 0.88 (p = 0.28) At 36 month (cases, %) G1: 198 (34.4) G2: 229 (39.2) RR = 0.88 (p = 0.09) At 48 month (cases, %) G1: 211 (38.1) G2: 248 (44.4)

References (country)	Design	Subjects characteristics at baseline	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Results
	Duration: 36 to 48 months	SBP (mm Hg, mean ± SD) G1: 127.7 ± 6.6 G2: 127.3 ± 6.4 DBP (mm Hg, mean ± SD) G1: 86.1 ± 1.9 G2: 85.8 ± 1.9				RR = 0.86 (p = 0.04)

bw: body weight; 95% CI: 95% confidence interval; DBP: diastolic blood pressure; Diff: difference; G1: group 1 ('low' sodium); G2: group 2 ('high' sodium); mm Hg: millimetre of mercury; N: number; Na: sodium; RCT: randomised controlled trial; RR: relative risk; SBP: systolic blood pressure; SD: standard deviation; TOPH: Trials of Hypertension Prevention; UNa: sodium urinary excretion.

J.1.2. Observational studies

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
Forman et al. (2012) **(Netherlands)** **PREVEND**	Prospective cohort N = 8,592 initial cohort; IMDM and pregnant women excluded; oversampling of subjects with elevated albumin excretion (> 10 mg/L) N = 5,556 included (excluded: participants with hypertension, missing 24-h UNa at baseline) Median duration: 6.4 years	Male (%) Q1 32.5 Q2 43.5 Q3 48.1 Q4 58.4 Age (median (IQR), years) Q1 43 (36–52) Q2 43 (36–52) Q3 43 (36–51) Q4 44 (37–52) BMI (median (IQR), kg/m^2) Q1 23.7 (21.7–26.2) Q2 24.2 (22.2–26.7) Q3 24.9 (22.6–27.3) Q4 25.7 (23.5–28.4) Smoking status (never, %) Q1 31.5 Q2 30.5 Q3 31.5 Q4 29.4 SBP (median (IQR), mm Hg) Q1 116 (108–126) Q2 118 (110–127) Q3 119 (111–128) Q4 121 (112–129) DBP (median (IQR), mm Hg)	Two 24-h urine specimens collected at baseline (1997–1998), two 24-h urine specimens collected during follow-up first examination (2001–2003) and two 24-h urine specimens collected during follow-up second examination (2003–2006)	Median (IQR) Overall 137 (106–171) Q1 97 (79–110) (n = 1,389) Q2 142 (132–153) (n = 1,389) Q3 188 (176–203) (n = 1,389) Q4 271 (242–316) (n = 1,389)	Incident hypertension BP measured at baseline and at follow-up Hypertension was defined as a SBP > 140 mm Hg, a DBP > 90 mm Hg, or both or the use of antihypertensive medications	Age, BMI, sex, alcohol intake, smoking status, family history of hypertension, estimated GFR, serum levels of glucose ad cholesterol, and 24-h UK, 24-h UCa and 24-h UCr	HR for incident hypertension By 1 g (43 mmol) increase in UNa HR (95% CI) = 1.05 (1.00–1.10) (878 cases) By quartile of UNa: Q1 (reference) (incidence 13.9%) Q4 HR (95% CI) = 1.21 (0.98–1.51) (incidence 19.7%) Effect modification by serum uric acid and UAlbumin excretion

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
		Q1 69 (64–74) Q2 70 (65–75) Q3 70 (65–75) Q4 71 (66–76) UK (median (IQR), mmol/24 h) Q1 56 (43–72) Q2 73 (59–91) Q3 87 (71–109) Q4 114 (87–144) 24-h UCr (median (IQR), g) Q1 1.1 (0.9–1.3) Q2 1.3 (1.1–1.5) Q3 1.4 (1.2–1.7) Q4 1.6 (1.3–1.9) 24-h UAlbumin (median (IQR), mg) Q1 7.3 (5.3–11.7) Q2 7.9 (5.8–12.5) Q3 8.4 (6.1–13.6) Q4 8.7 (6.4–13.9)					
Stolarz-Skrzypek et al. (2011) **(Belgium, Czech Republic, Italy, Poland, Russian Federation)** **FLEMENGHO/ EPOGH**	Prospective cohort N = 3,360 FLEMENGHO initial cohort; N = 1,187 initial EPOGH initial cohort N = 2,096 included in the analyses ('hypertension cohort') (excluded: participants with treated/untreated hypertension at baseline, history of CVD, missing/ inaccurate 24-h UNa at baseline)	Male (%) T1 45.8 T2 46.0 T3 45.9 Hypertensive (%) none Diabetes mellitus w T1 1.9 T2 1.6 T3 1.9 m T1 1.9 T2 2.4 T3 1.9 Age (mean (SD), years) w T1 38.9 (15.3) T2 38.7 (14.1) T3 36.7 (13.1) m T1 39.3 (16.0) T2 40.1 (15.4) T3 38.2 (13.4) BMI (mean (SD), kg/m^2) w T1 23.6 (4.2) T2 24.1 (4.1) T3 25.0 (4.6) m T1 24.6 (3.4) T2 24.9 (3.3) T3 25.2 (3.6) Smokers (%) w T1 32.3 T2 23.6 T3 23.9 m T1 37.7 T2 39.0 T3 32.8	Single 24-h urine sample at baseline and at last follow-up examination Inaccurate urine collections were defined as a volume < 300 mL/ 24-h, a 24-h UCr < 4 mmol or > 25 mmol in women and < 6 mmol or > 30 mmol in men	Mean (SD) Overall 174.2 (74.1) Women T1 94.4 (21.5) (n = 375) T2 147.4 (14.3) (n = 385) T3 222.1 (47.2) (n = 373) Men T1 121.3 (27.9) (n = 318) T2 185.3 (16.1) (n = 328) T3 282.2	Incident hypertension BP measured at baseline and follow-up Hypertension was defined as SBP ≥ 140 mm Hg or DBP ≥ 90 mm Hg use of antihypertensive drugs.	Study population, sex, age, BMI, SBP, 24-h UK, drinking alcohol and educational attainment	HR for incident hypertension By tertile of UNa (whole population as reference) T1 HR (95% CI) = 1.00 (0.87, 1.16) (incidence 27.0%, 187 cases) T2 HR (95% CI) = 1.02 (0.89, 1.16) (incidence 26.6%, 190 cases) T3 HR (95% CI) = 0.98 (0.86, 1.12) (incidence 25.4%, 175 cases)

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
	Median duration: 6.5 years	Alcohol intake (5 g/day,%) w T1 8.8 T2 9.9 T3 12.9 m T1 35.2 T2 32.9 T3 39.8 Higher education (%) w T1 10.7 T2 14.3 T3 16.9 m T1 13.8 T2 12.5 T3 17.7 SBP (mean (SD), mm Hg) w T1 115.6 (11.2) T2 115.6 (10.7) T3 116.8 (10.0) m T1 121.8 (9.7) T2 121.8 (8.7) T3 122.0 (9.3) DBP (mean (SD), mm Hg) w T1 71.3 (8.0) T2 72.1 (7.6) T3 73.0 (8.0) m T1 74.8 (8.2) T2 74.0 (7.9) T3 75.5 (7.7) 24-h UK (mean (SD), mmol) w T1 51.2 (17.2) T2 62.9 (21.1) T3 69.4 (23.4) m T1 62.5 (26.3) T2 73.5 (23.4) T3 84.1 (28.8) 24-h UCr (mean (SD), mmol) w T1 8.6 (2.2) T2 9.6 (2.0) T3 10.8 (2.5) m T1 12.2 (3.1) T2 14.1 (3.2) T3 16.1 (3.4)		(56.4) (n = 317)			

BMI: body mass index; BP: blood pressure; 95% CI: 95% confidence interval; CVD: cardiovascular disease; DBP: diastolic blood pressure; EPOGH: European Project on Genes in Hypertension; FLEMENGHO: Flemish Study on Genes and Health Outcomes; GFR: glomerular filtration rate; HR: hazard ratio; IMDM: insulin-mediated diabetes mellitus; IQR: interquartile range; m: men; mm Hg: millimetre of mercury; N: number; Na: sodium; SBP: systolic blood pressure; PREVEND: Prevention of Renal and Vascular End-stage Disease; SD: standard deviation; UAlbumin: albumin urinary excretion; UCa: calcium urinary excretion; UCr: creatinine urinary excretion; UK: potassium urinary excretion; UNa: sodium urinary excretion; w: women.

J.2. Outcome of the RoB appraisal

J.2.1. Experimental studies

References	Risk of bias domains[a]								Tier[b]
	Randomisation	Allocation concealment	Blinding	Attrition	Exposure	Outcome	Reporting	Other threats to internal validity	
Whelton et al. (1997) (TOPH I)	++	++	+	++	+	++	++	+	1
The Trials of Hypertension Collaborative Research Group (1997) (TOPH II)	++	++	+	++	+	++	++	+	1

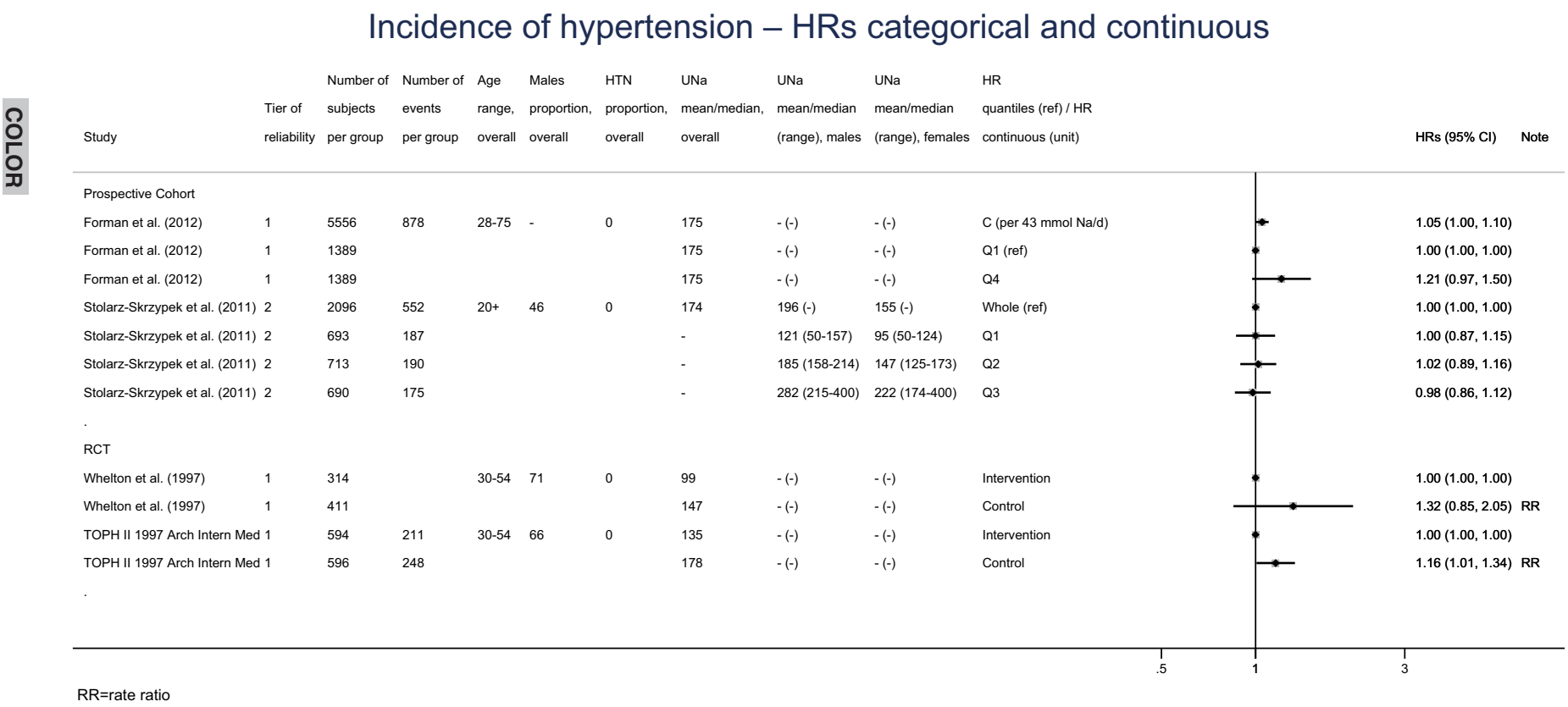
(a): Expert judgement was translated into a rating scale for each question to be answered as follows: (++): definitely low RoB; (+): probably low RoB; (NR): not reported; (−): probably high RoB; (−−): definitively high RoB.
(b): The individual rating for each question was combined by an algorithm and translated to an overall tier of reliability for each individual study (RoB tier 1: low RoB; RoB tier 2: moderate RoB; RoB tier 3: high RoB).

J.2.2. Observational studies

References	Risk of bias domains[a]						Tier[b]
	Confounding	Attrition	Exposure	Outcome	Reporting	Statistics	
Forman et al. (2012)	+	−	++	++	+	++	1
Stolarz-Skrzypek et al. (2011)	−	−	−	++	++	+	2

(a): Expert judgement was translated into a rating scale for each question to be answered as follows: (++): definitely low RoB; (+): probably low RoB; (NR): not reported; (−): probably high RoB; (−−): definitively high RoB.
(b): The individual rating for each question was combined by an algorithm and translated to an overall tier of reliability for each individual study (RoB tier 1: low RoB; RoB tier 2: moderate RoB; RoB tier 3: high RoB).

J.3. Descriptive forest plot



95% CI: 95% confidence interval; HR: hazard ratio.
A hazard ratio > 1 indicates an increased risk of outcome with higher sodium intake.

Figure J.1: Descriptive forest plot of the eligible observational and experimental studies on the incidence of hypertension

Appendix K – Outcome of the systematic review on risk of CVD

K.1. Evidence tables

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
Cook et al. (2014) **(USA)** **TOPH I/TOPH II**	Prospective cohort N = 2,974 TOPH I/TOPH II participants not in active Na intervention eligible for follow-up N = 2,275 included in the analyses (excluded: CVD event during trial period, missing 24-h UNa) Duration: 10 years (TOPH I)/ 15 years (TOPH II)	Men (%) TOPH I 71, TOPH II 67 Hypertensive (treated/ untreated): none Age (mean, years) TOPH I w Q1 44.6 Q2 44.7 Q3 43.0 Q4 42.7 m Q1 42.1 Q2 42.8 Q3 43.3 Q4 42.7 TOPH II w Q1 44.4 Q2 43.8 Q3 43.1 Q4 44.0 m Q1 42.7 Q2 43.6 Q3 43.6 Q4 42.5 bw (mean, lb) TOPH I w Q1 151.3 Q2 162.4 Q3 174.1 Q4 183.7 m Q1 175.8 Q2 185.4 Q3 198.1 Q4 211.4 TOPH II w Q1 172.7 Q2 181.4 Q3 192.0 Q4 203.1 m Q1 202.4 Q2 207.5 Q3 213.8 Q4 229.2 Ethnicity (%, black) w Q1 19.8 Q2 22.6 Q3 28.8 Q4 30.4 m Q1 14.8 Q2 6.5 Q3 8.8 Q4 7.7 TOPH II	3 to 7 24-h collection scheduled over 18 months in TOPH I and over 3 to 4 years in TOPH II To adjust for potential inaccuracy in collection, controlled for UCr/bw in multivariate model or excluded subjects with CVs of UCr/ bw ≥ 20% or 30% (sensitivity analyses)	Men and women Q1 < 100 Q2 100–156 Q3 157–208 Q4 ≥ 209	CVD death or event, including MI, stroke, coronary artery bypass graft, percutaneous transluminal coronary angioplasty. Standardised questionnaires sent at 2-year intervals; medical records reviewed by study physician (non-fatal events); search for National Death Index (fatal events)	Age, sex, race/ ethnicity, clinic and treatment assignment, education status, baseline weight, alcohol se, smoking, exercise, UK, family history of CVD, change in bw, change in smoking and change in exercise during trial periods	HR (95% CI) for fatal and non-fatal CVD events By continuous UNa (by 1 g (43-mmol)/day increase): 1.17 (1.00–1.36) (193 cases, n = 2,275) By quantiles of UNa: Q1 0.68 (0.34–1.37) (TOPH I 15 events/189 total, TOPH II 2 events/47 total) Q2 0.75 (0.50–1.11) (TOPH I 48 events/590 total, TOPH II 13 events/303 total) Q3 1.00 (reference) (TOPH I 40 events/427 total, TOPH II 34 events/341 total) Q4 1.05 (0.68–1.62) (TOPH I 23 events/191 total, TOPH II 18 events/224 total) No significant deviation from linearity (restricted cubic spline). Sensitivity analyses: exclusion of subjects

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
		w Q1 34.5 Q2 30.4 Q3 28.1 Q4 18.2 m Q1 11.1 Q2 10.6 Q3 9.5 Q4 7.9 Smokers (%, current) TOPH I w Q1 14.8 Q2 10.6 Q3 11.0 Q4 21.7 m Q1 10.2 Q2 10.7 Q3 10.5 Q4 10.1 TOPH II w Q1 10.3 Q2 8.7 Q3 13.5 Q4 9.1 m Q1 22.2 Q2 7.0 Q3 8.3 Q4 8.9 Alcohol intake (≥ 1 drink/wk,%) TOPH I w Q1 41.6 Q2 28.8 Q3 23.3 Q4 39.1 m Q1 63.6 Q2 55.2 Q3 52.5 Q4 42.9 TOPH II w Q1 13.8 Q2 19.9 Q3 14.6 Q4 27.3 m Q1 55.6 Q2 42.2 Q3 44.0 Q4 40.1 College degree (%) TOPH I w Q1 46.5 Q2 46.2 Q3 42.5 Q4 39.1 m Q1 84.1 Q2 71.2 Q3 68.8 Q4 66.7 TOPH II w Q1 62.1 Q2 50.3 Q3 44.9 Q4 45.4 m Q1 100.0 Q2 72.5					based on the CV for UCr/bw: little change in estimated coefficients

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
		Q3 67.1 Q4 62.9 SBP (mm Hg, mean) TOPHI W Q1 125.2 Q2 125.7 Q3 125.3 Q4 123.9 M Q1 124.3 Q2 124.2 Q3 125.2 Q4 125.3 TOPH II W Q1 128.2 Q2 128.4 Q3 128.2 Q4 130.0 M Q1 127.7 Q2 127.5 Q3 126.7 Q4 127.6 DBP (mm Hg, mean) TOPH I W Q1 83.7 Q2 83.7 Q3 84.5 Q4 83.6 M Q1 84.0 Q2 83.7 Q3 84.0 Q4 83.7 TOPH II W Q1 85.7 Q2 85.7 Q3 85.8 Q4 86.2 M Q1 85.8 Q2 86.1 Q3 85.9 Q4 86.2 24-h UK (mean, mmol) TOPH I w Q1 42 Q2 49 Q3 58 Q4 67 m Q1 51 Q2 60 Q3 68 Q4 78 TOPH II w Q1 39 Q2 50 Q3 56 Q4 68 m Q1 56 Q2 60 Q3 69 Q4 75 24-h UCr (mean, g) TOPH I W Q1 0.9 Q2 1.1 Q3 1.3					

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
		Q4 1.5 M Q1 1.3 Q2 1.5 Q3 1.7 Q4 2.0 TOPH II W Q1 1.0 Q2 1.2 Q3 1.4 Q4 1.5 M Q1 1.5 Q2 1.7 Q3 1.9 Q4 2.2					
Stolarz-Skrzypek et al. (2011) **(Belgium, Czech Republic, Italy, Poland, Russian Federation)** **FLEMENGHO/ EPOGH**	Prospective cohort N = 3,360 FLEMENGHO initial cohort; N = 1,187 initial EPOGH initial cohort N = 3,681 included in the analyses ('outcome cohort') (excluded: participants with history of CVD, missing/ inaccurate 24-h UNa at baseline) Median duration: 7.9 years	Men (%) T1 47.1 T2 47.4 T3 47.3 Hypertensive (treated) (%) w T1 14.9 T2 12.5 T3 16.6 m T1 11.1 T2 7.4 T3 8.9 Diabetes mellitus w T1 3.3 T2 2.9 T3 4.9 m T1 5.0 T2 4.4 T3 4.5 Age (mean (SD), years) w T1 42.5 (17.6) T2 41.0 (16.0) T3 39.2 (14.7) m T1 41.8 (18.1) T2 41.3 (16.4) T3 39.5 (14.4) BMI (mean (SD), kg/m^2) w T1 24.6 (5.1) T2 24.9 (4.6) T3 25.9 (5.3) m T1 24.7 (3.8) T2 25.2 (3.8) T3 26.1 (4.3) Smokers (%) w T1 25.9 T2 22.2 T3 22.1 m T1 35.1 T2 37.3 T3 29.1 Alcohol intake (5 g/day, %)	Single 24-h urine sample at baseline and at last follow-up examination Inaccurate urine collections were defined as a volume < 300 mL/ 24-h, a 24-h UCr < 4 mmol or > 25 mmol in women and < 6 mmol or > 30 mmol in men	Mean (SD) Women T1 95.1 (22.0) (n = 645) T2 150.2 (15.0) (n = 658) T3 231.7 (50.9) (n = 638) Men T1 120.1 (28.4) (n = 575) T2 188.8 (17.6) (n = 592) T3 290.5 (56.2) (n = 573)	CHD events included fatal and non-fatal MI and coronary revascularisation. Fatal and non-fatal CVD events comprised CHD events, stroke, fatal and non-fatal left ventricular HF, aortic aneurysm, cor pulmonale and pulmonary or arterial embolism. Hospitalisations for unstable angina coded as IHD. Standardised questionnaire at follow-up visits. Physicians ascertained the diseases reported on the death certificates or by the questionnaires against medical records	Study population, sex, and baseline variables: age, BMI, 24-h UK, antihypertensive drug treatment, smoking and drinking alcohol, diabetes, total cholesterol and educational attainment	HRs expressed the risk in each tertile of UNa compared with the overall risk in whole group HR (95% CI) for fatal CVD events T1 1.41 (0.94, 2.12) (50 events, n = 1,220) T2 0.98 (0.69, 1.40) (24 events, n = 1,250) T3 1.02 (0.71, 1.45) (10 events, n = 1,211) HR (95% CI) for non-fatal CVD events T1 1.12 (0.90, 1.41) (100 events, n = 1,220) T2 1.09 (0.89, 1.34) (79 events, n = 1,250) T3 0.92 (0.74, 1.13) (53 events, n = 1,211) HR (95% CI) for non-fatal CHD events T1 1.41 (0.99, 2.01)

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
		w T1 10.7 T2 10.3 T3 14.0 m T1 34.3 T2 35.6 T3 44.0 Higher education (%) w T1 11.6 T2 15.1 T3 19.3 m T1 15.3 T2 13.0 T3 19.7 SBP (mean (SD), mm Hg) w T1 123.3 (19.4) T2 121.0 (16.8) T3 121.7 (15.8) m T1 128.7 (17.9) T2 126.6 (14.9) T3 128.1 (15.3) DBP (mean (SD), mm Hg) w T1 74.7 (11.0) T2 74.6 (9.7) T3 75.4 (10.2) m T1 77.8 (11.0) T2 76.6 (10.4) T3 79.0 (10.9) 24-h UK (mean (SD), mmol) w T1 51.6 (17.8) T2 61.0 (20.5) T3 69.1 (24.0) m T1 61.6 (26.3) T2 71.6 (27.1) T3 84.5 (29.2) 24-h UCr (mean (SD), mmol)					(45 events, n = 1,220) T2 1.15 (0.87, 1.52) (34 events, n = 1,250) T3 0.87 (0.66, 1.15) (19 events, n = 1,211)

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
		w T1 8.4 (2.2) T2 9.5 (2.0) T3 10.6 (2.5) m T1 12.1 (3.2) T2 13.9 (3.4) T3 16.1 (3.8)					HR (95% CI) for non-fatal stroke events T1 1.05 (0.56, 1.96) (13 events, n = 1,220) T2 1.28 (0.75, 2.17) (13 events, n = 1,250) T3 0.78 (0.46, 1.33) (7 events, n = 1,211)
Joosten et al. (2014) **(Netherlands)** **PREVEND**	Prospective cohort N = 8,592 initial cohort; IMDM and pregnant women excluded; oversampling of subjects with elevated albumin excretion (> 10 mg/L) N = 7,543 included (excluded: participants with CVD, renal disease requiring dialysis, malignancies, missing values of covariates at baseline)	Men (%) Q1 48.7 Q2 48.7 Q3 48.7 Q4 48.7 Age (mean ± SD, years) Q1 50 ± 13 Q2 49 ± 13 Q3 48 ± 12 Q4 47 ± 11 BMI (mean ± SD, kg/m^2) Q1 25.0 ± 3.7 Q2 25.5 ± 3.7 Q3 26.1 ± 4.1 Q4 27.5 ± 4.8 Smoking status (never, %) Q1 30.1 Q2 32.1 Q3 28.6 Q4 29.9 SBP (mean ± SD, mm Hg) Q1 129 ± 22 Q2 128 ± 20 Q3 128 ± 20 Q4 129 ± 20 DBP (mean ± SD, mm Hg) Q1 74 ± 10 Q2 74 ± 10 Q3 74 ± 10 Q4 74 ± 9	Two 24-h urine specimens collected at baseline (1997–1998) Sensitivity analyses excluding 24-h urine samples with possible over- or undercollections (i.e. samples upper and lower 2.5% of the difference between the estimated and measured volume of a subject's 24-h urine sample.)	Men Q1 < 122 Q2 122–154 Q3 155–190 Q4 > 190 Women Q1 < 95 Q2 95–121 Q3 122–151 Q4 > 151	CHD defined as MI, acute and subacute IHD and coronary artery bypass grafting or percutaneous transluminal coronary angioplasty. Data from Dutch bureau of statistics (fatal events) and Dutch national registry of hospital discharge diagnoses (non-fatal events)	Age, sex, body mass index, smoking status, alcohol intake, parental history of coronary heart disease, type 2 diabetes mellitus, total to HDL cholesterol ratio, and 24-h UK, magnesium and creatinine excretion	HR (95% CI) for fatal and non-fatal CHD By continuous UNa (by 1 g (43-mmol)/day increase): 1.07 (0.98–1.18) p = 0.15 (452 cases; 71,491 person-years) By sex-specific quintiles of UNa: Q1 (reference) (123 cases, 17,738 person-years) Q2 0.99 (0.76–1.29) (111 cases, 17,975 person-years) Q3 1.09 (0.83–1.44) (112 cases, 17,878 person-years) Q4 1.19 (0.88–1.62) (106 cases, 18,000 person-years) No significant deviation from linearity (restricted cubic spline).

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
	Median duration: 10.5 years (IQR 9.9–10.8)	Antihypertensive drugs (%) Q1 14.3 Q2 12.6 Q3 12.4 Q4 12.7 24-h UK (median (IQR), mmol) Q1 59 (47–73) Q2 67 (56–81) Q3 73 (61–86) Q4 80 (66–95) 24-h UCr (median (IQR), mmol) Q1 10.3 (8.4–12.7) Q2 11.5 (9.5–13.8) Q3 12.3 (10.1–15.1) Q4 13.6 (11.1–16.8) 24-h UAlbumin (median (IQR), mmol) Q1 8.1 (5.5–14.7) Q2 9.1 (6.3–16.4) Q3 9.4 (6.4–17.1) Q4 10.3 (7.0–19.3)					Evidence for effect modification by mean arterial pressure ($P_{interaction}$ = 0.08) and plasma NT-proBNP concentration ($P_{interaction}$ = 0.002). Sensitivity analyses: exclusion of subjects with potential under- or overcollections in 24-h urine samples: results unchanged restricted to subjects with UAlbumin > 10 mg/L: results unchanged
Kieneker et al. (2018) **(Netherlands)** **PREVEND**	Prospective cohort N = 8,592 initial cohort N = 7,330 included (excluded: participants with CVD, renal disease requiring dialysis, missing values of covariates at baseline)	Men (%) Q1 48.6 Q2 48.6 Q3 48.6 Q4 48.6 Q5 48.6 Age (mean ± SD, years) Q1 50.5 ± 13.0 Q2 50.1 ± 12.9 Q3 49.2 ± 12.5 Q4 48.2 ± 12.0 Q5 47.0 ± 11.1 BMI (mean ± SD, kg/m^2) Q1 25.0 ± 3.8 Q2 25.3 ± 3.6 Q3 25.7 ± 4.0 Q4 26.3 ± 4.0 Q5 27.8 ± 4.9 Ethnicity (% white)	Two 24-h urine specimens collected at baseline (1997–1998) and two 24-h urine specimens collected during follow-up (2001–2003)	Men Q1 < 116 (712) Q2 116–142 (713) Q3 143–167 (713) Q4 168–201 (713) Q5 > 201 (713) Women Q1 < 89 (753) Q2 89–110 (754) Q3 111–132 (753) Q4 133–160 (753)	Haemorrhagic, ischaemic, unspecified stroke, fatal and non-fatal events Data from municipal registers (fatal events) and Dutch national registry of hospital discharge diagnoses (non-fatal events)	Age, sex, height, weight, race/ethnicity, smoking status, alcohol consumption, education, type 2 diabetes, and total to HDL cholesterol ratio, 24-h UK, magnesium, creatinine and albumin and estimated GFR	HR (95% CI) for fatal and non-fatal stroke By continuous UNa (by 51 mmol/24 h decrease): 1.44 (1.14–1.82) (183 cases; 83,189 person-years) By sex-specific quintiles of UNa: Q1 1.45 (0.92–2.29) (57 cases, 16,272 person-years) Q2 1.13 (0.71–1.79) (49 cases, 16,515

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
	Oversampling of subjects with elevated albumin excretion (> 10 mg/L) Median duration: 12.5 years (IQR 11.9–12.9)	Q1 93.9 Q2 95.2 Q3 96.5 Q4 96.1 Q5 96.0 Smoking status (current, %) Q1 39.0 Q2 33.1 Q3 33.9 Q4 31.6 Q5 32.3 Education (higher vocational education or university, %) Q1 31.1 Q2 32.9 Q3 32.2 Q4 31.4 Q5 26.5 SBP (mean ± SD, mm Hg) Q1 129 ± 21 Q2 129 ± 20 Q3 128 ± 20 Q4 128 ± 20 Q5 129 ± 18 DBP (mean ± SD, mm Hg) Q1 74 ± 10 Q2 74 ± 10 Q3 74 ± 10 Q4 73 ± 10 Q5 74 ± 9 Antihypertensive drugs (%) Q1 14.7% Q2 12.0% Q3 12.2% Q4 12.3% Q5 13.0% 24-h UK (median (IQR), mmol) Q1 57 (46–72) Q2 66 (55–79) Q3 70 (58–83) Q4 74 (62–88) Q5 81 (68–96) 24-h UCr (median (IQR), mmol) Q1 10.0 (8.3–12.6) Q2 11.2 (9.3–13.6) Q3 11.8		Q5 > 160 (753)			person-years) Q3 (reference) (33 cases, 16,774 person-years) Q4 1.04 (0.64–1.71) (25 cases, 16,720 person-years) Q5 0.81 (0.46–1.41) (19 cases, 16,908 person-years) Adjustment for potential mediators (SBP and antihypertensive medication, plasma renin, aldosterone, and sodium levels) did not change the result. No evidence for effect modification by age, sex, BMI, hypertension, and UK (all $P_{interaction} > 0.10$). Sensitivity analyses: excluding individuals who were taking antihypertensive drugs at baseline (n = 6,388, 126 cases): results unchanged. excluding individuals with malignancies, type 2 diabetes, or chronic kidney disease at baseline (n = 6,054, 112 cases): results unchanged.

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
		(9.8–14.3) Q4 12.5 (10.3–15.6) Q5 13.8 (11.3–16.8) 24-h UAlbumin (median (IQR), mmol) Q1 8.0 (5.5–14.4) Q2 8.9 (6.1–16.0) Q3 9.1 (6.3–16.2) Q4 9.5 (6.5–18.1) Q5 10.5 (7.0–19.9)					
Lelli et al. (2018) **(Italy)** **InCHIANTI**	Prospective cohort N = 1,170 initial cohort N = 514 included (excluded: younger than 65 years, participants with CVD at baseline) Median duration: 9 years	NR for the subcohort	Single 24-h urine specimen collected at baseline (1998–2000)	NR for the subcohort	Non-fatal CVD event, including angina pectoris, MI, heart failure, stroke Standardised questionnaire at follow-up visits; clinical documentation reviewed	Age, sex, education, CKD-EPI, SBP, pack/years, hypertension, diabetes, BMI, caloric intake/bw, and antihypertensive drugs and diuretics)	RR (95% CI) for non-fatal CVD events By continuous UNa (By NR mmol/day increase): 0.96 (0.90–1.02) (169 cases, n = 514)
Tuomilehto et al. (2001) **(Finland)**	Prospective cohort N = 3,607 initial cohort Non-fatal CHD analysis	Age (mean ± SD, years) w Q1 45.7 ± 11.6 Q2 45.4 ± 11.8 Q3 44.8 ± 11.1 Q4 45.6 ± 11.3 m Q1 45.4 ± 11.6 Q2 45.3 ± 11.0 Q3 46.2 ± 10.4 Q4	Single 24-h urine specimens collected at baseline Participants reporting incomplete collection excluded from the analysis	Median (min–max) Male 205 (25–552) Female 154 (12–512)	Coronary deaths, non-fatal coronary events, stroke events, cardiovascular deaths Statistics Finland (fatal events) and	Age, study year, smoking, serum total and HDL cholesterol, SBP and BMI	HR (95% CI) for non-fatal CHD events By 100 mmol/24-h increase in UNa Men 1.34 (1.06–1.70) (98 cases, n = 1145)

References (country)	Design	Baseline characteristics	Na intake assessment method	UNa (mmol/24 h)	Outcomes assessed	Confounders adjusted for	Results
	N = 2,402 (prevalent cases of CHD also excluded) Non-fatal stroke analysis N = 2,420 (prevalent cases of stroke also excluded) Duration: up to 14 years	45.4 ± 10.6 BMI (mean ± SD, kg/m^2) w Q1 24.6 ± 4.2 Q2 25.1 ± 4.0 Q3 26.3 ± 4.6 Q4 27.8 ± 5.4 m Q1 25.5 ± 2.4 Q2 26.4 ± 3.3 Q3 26.9 ± 3.3 Q4 28.1 ± 4.2 Smoking (current,%) w Q1 15% Q2 20% Q3 18% Q4 16% m Q1 31% Q2 40% Q3 33% Q4 44% SBP (mean ± SD, mm Hg) w Q1 141 ± 22 Q2 140 ± 22 Q3 141 ± 22 Q4 142 ± 22 m Q1 144 ± 22 Q2 145 ± 19 Q3 148 ± 20 Q4 147 ± 19 DBP (mean ± SD, mm Hg) w Q1 83 ± 12 Q2 83 ± 12 Q3 83 ± 12 Q4 85 ± 22 m Q1 86 ± 11 Q2 86 ± 12 Q3 89 ± 13 Q4 90 ± 13			national hospital discharge register (non-fatal events)		Women 1.35 (0.77–2.35) (30 cases, n = 1257) Men + Women 1.34 (1.08–1.67) (128 cases, n = 2,402) HR for non-fatal stroke events By 100 mmol/24-h increase in UNa Men 1.00 (0.68–1.47) (43 cases, n = 1161) Women 1.34 (0.87–2.07) (41 cases, n = 1259) Men + Women 1.13 (0.84–1.51) (84 cases, n = 2,420)

BMI: body mass index; BP: blood pressure; bw: body weight; 95% CI: 95% confidence interval; CKD-EPI: estimated creatinine clearance; CHD: coronary heart disease; CVD: cardiovascular disease; DBP: diastolic blood pressure; EPOGH: European Project on Genes in Hypertension; FLEMENGHO: Flemish Study on Genes and Health Outcomes; GFR: glomerular filtration rate; HDL: high density lipoprotein; HF: heart failure; HR: hazard ratio; IHD: ischaemic heart disease; IMDM: insulin-mediated diabetes mellitus; IQR: interquartile range; m: men; mm Hg: millimetre of mercury; MI: myocardial infarction; N: number; Na: sodium; NR: not reported; NT-proBNP: N-terminal pro-B-type natriuretic peptide; RR: relative risk; SBP: systolic blood pressure; SD: standard deviation; TOPH: Trials of Hypertension Prevention; UAlbumin: urinary albumin; UCr: creatinine urinary excretion; UK: potassium urinary excretion; UNa: sodium urinary excretion; w: women.

K.2. Outcome of the RoB appraisal

K.2.1. Observational studies

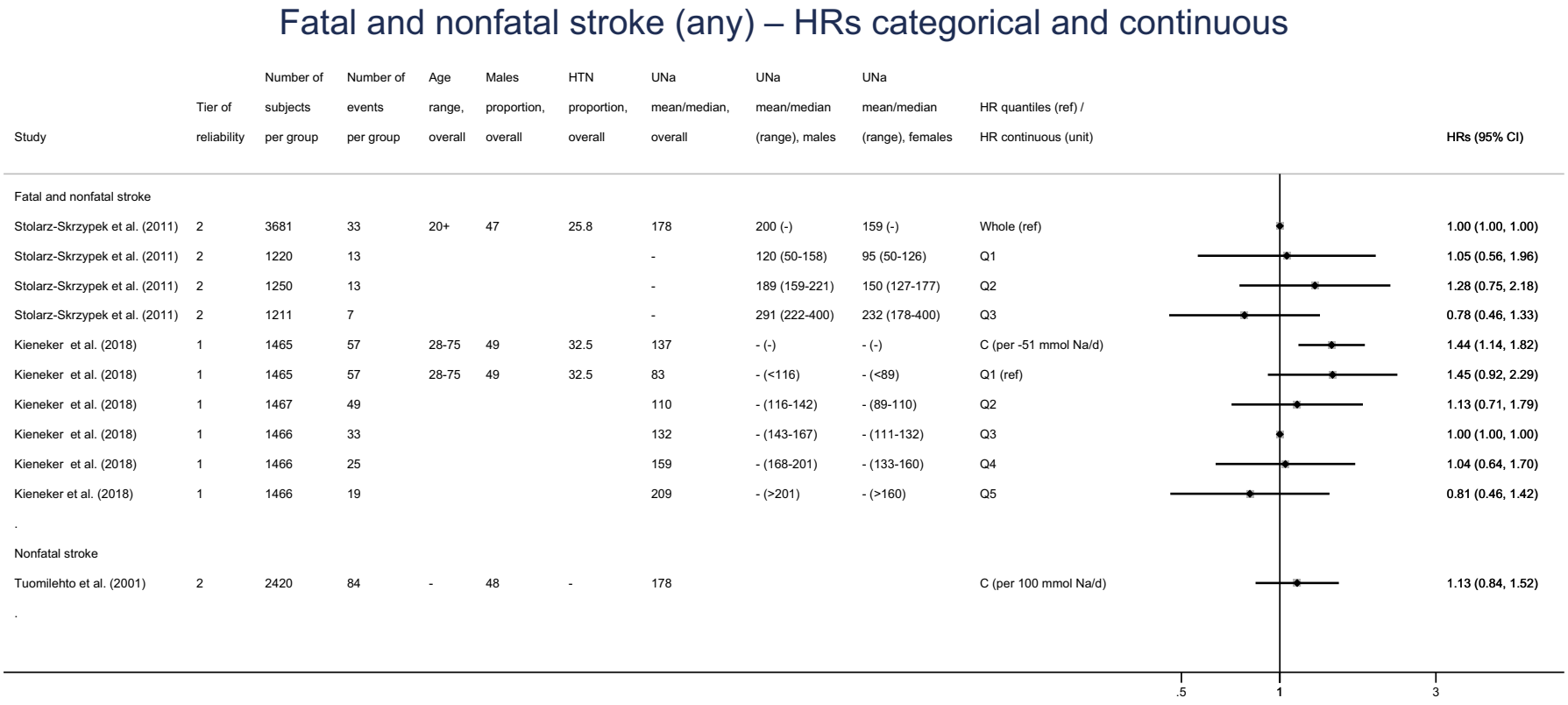
References	Outcomes	Risk of bias domains[a]						Tier[b]
		Confounding	Attrition	Exposure	Outcome	Reporting	Statistics	
Lelli et al. (2018)	Non-fatal CVD (any)	–	++	+	–	++	–	2
Joosten et al. (2014)	Fatal and non-fatal CHD	+	NR	++	++	++	++	1
Kieneker et al. (2018)	Fatal and non-fatal stroke	+	NR	+	+	++	+	1
Stolarz-Skrzypek et al. (2011)	Fatal CVD Fatal and non-fatal CVD Fatal and non-fatal CHD Fatal and non-fatal stroke	+	–	–	+	++	–	2
Cook et al. (2014)	Fatal and non-fatal CVD (any)	++	+	++	++	++	++	1
Tuomilehto et al. (2001)	Non-fatal stroke Non-fatal CHD	–	++	+	+	++	–	2

CHD: coronary heart disease; CVD: cardiovascular disease

(a): Expert judgement was translated into a rating scale for each question to be answered as follows: (++): definitely low RoB; (+): probably low RoB; (NR): not reported; (–): probably high RoB; (––): definitively high RoB.

(b): The individual rating for each question was combined by an algorithm and translated to an overall tier of reliability for each individual study (RoB tier 1: low RoB; RoB tier 2: moderate RoB; RoB tier 3: high RoB).

K.3. Descriptive forest plots

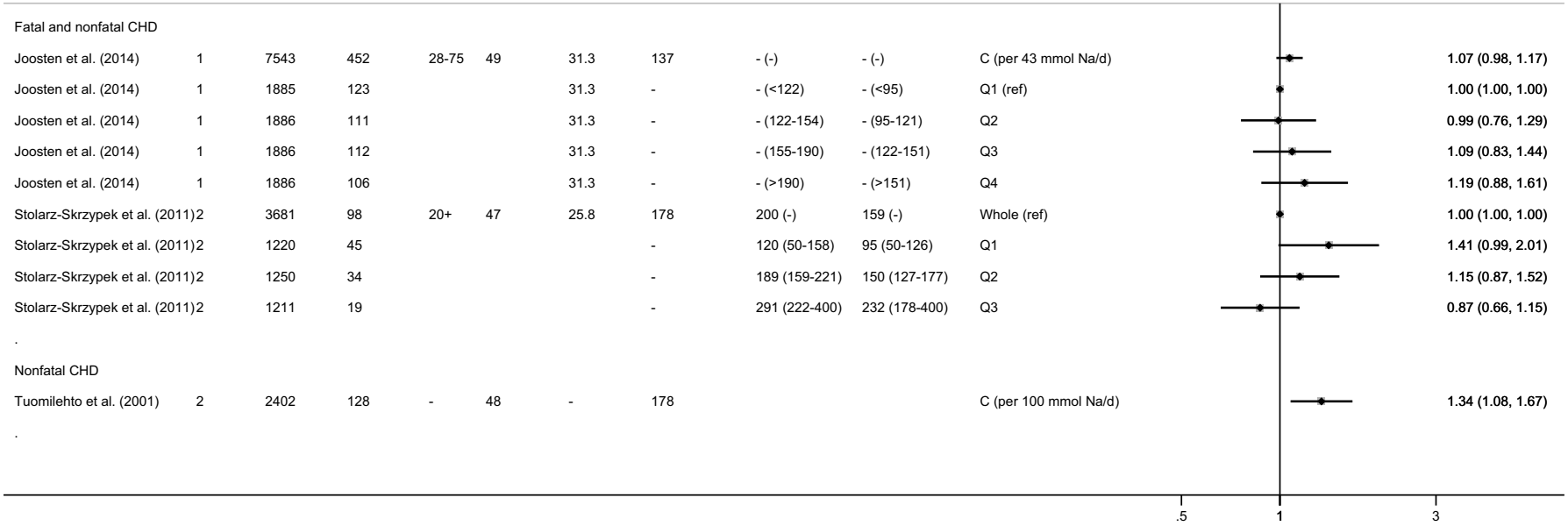


95% CI: 95% confidence interval; HR: hazard ratio; HTN: hypertensive; UNa: sodium urinary excretion.
A hazard ratio > 1 indicates an increased risk of outcome with higher sodium intake.

Figure K.1: Descriptive forest plot of the eligible observational studies on stroke

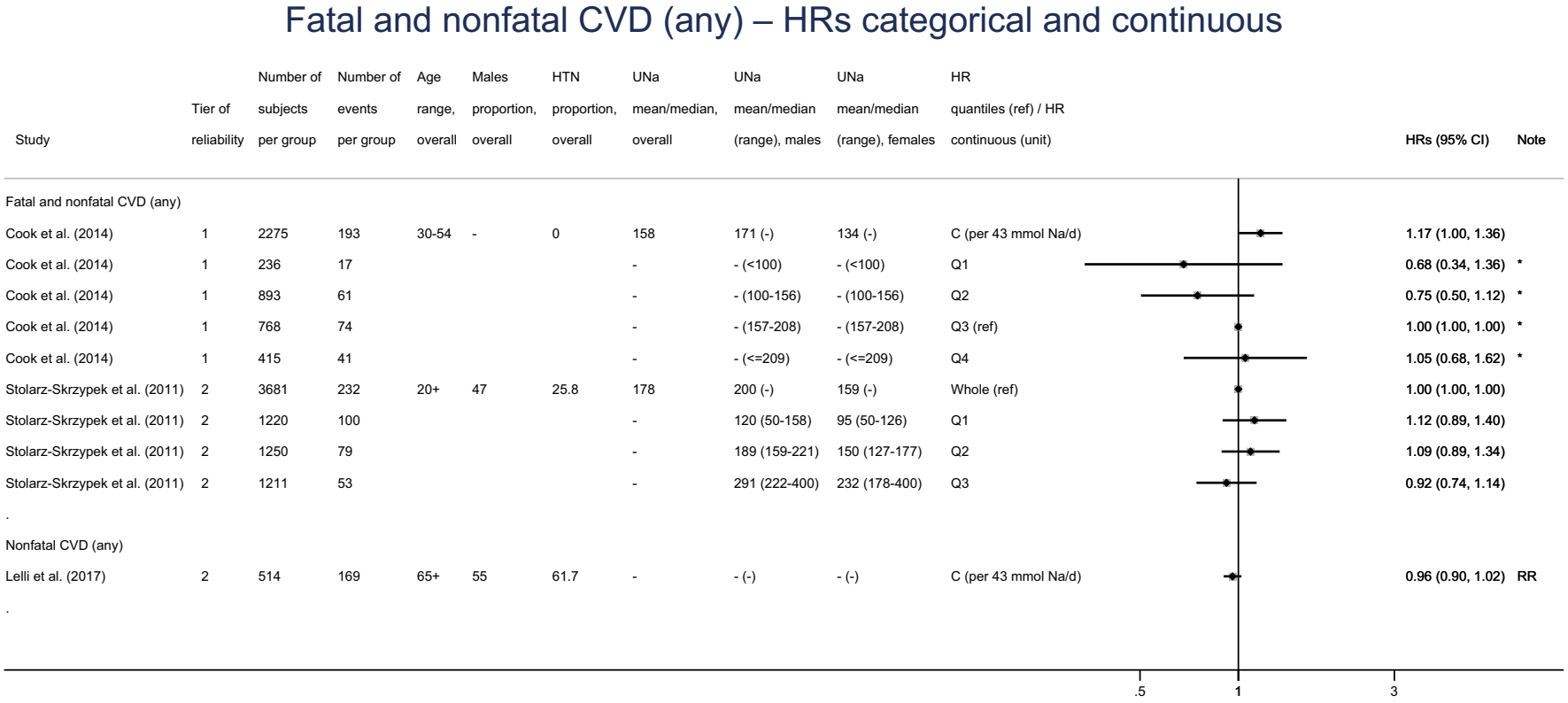
Fatal and nonfatal CHD (any) – HRs categorical and continuous

Study	Tier of reliability	Number of subjects per group	Number of events per group	Age range, overall	Males proportion, overall	HTN proportion, overall	UNa mean/median, overall	UNa mean/median (range), males	UNa mean/median (range), females	HR quantiles (ref) / HR continuous (unit)	HRs (95% CI)
Fatal and nonfatal CHD											
Joosten et al. (2014)	1	7543	452	28-75	49	31.3	137	- (-)	- (-)	C (per 43 mmol Na/d)	1.07 (0.98, 1.17)
Joosten et al. (2014)	1	1885	123			31.3	-	- (<122)	- (<95)	Q1 (ref)	1.00 (1.00, 1.00)
Joosten et al. (2014)	1	1886	111			31.3	-	- (122-154)	- (95-121)	Q2	0.99 (0.76, 1.29)
Joosten et al. (2014)	1	1886	112			31.3	-	- (155-190)	- (122-151)	Q3	1.09 (0.83, 1.44)
Joosten et al. (2014)	1	1886	106			31.3	-	- (>190)	- (>151)	Q4	1.19 (0.88, 1.61)
Stolarz-Skrzypek et al. (2011)	2	3681	98	20+	47	25.8	178	200 (-)	159 (-)	Whole (ref)	1.00 (1.00, 1.00)
Stolarz-Skrzypek et al. (2011)	2	1220	45				-	120 (50-158)	95 (50-126)	Q1	1.41 (0.99, 2.01)
Stolarz-Skrzypek et al. (2011)	2	1250	34				-	189 (159-221)	150 (127-177)	Q2	1.15 (0.87, 1.52)
Stolarz-Skrzypek et al. (2011)	2	1211	19				-	291 (222-400)	232 (178-400)	Q3	0.87 (0.66, 1.15)
.											
Nonfatal CHD											
Tuomilehto et al. (2001)	2	2402	128	-	48	-	178			C (per 100 mmol Na/d)	1.34 (1.08, 1.67)
.											



CHD: coronary heart disease; 95% CI: 95% confidence interval; HR: hazard ratio; HTN: hypertensive; UNa: sodium urinary excretion.
A hazard ratio > 1 indicates an increased risk of outcome with higher sodium intake.

Figure K.2: Descriptive forest plot of the eligible observational studies on coronary heart disease



CVD: cardiovascular disease; 95% CI: 95% confidence interval; HR: hazard ratio; HTN: hypertensive; UNa: sodium urinary excretion.
A hazard ratio > 1 indicates an increased risk of outcome with higher sodium intake.

Figure K.3: Descriptive forest plot of the eligible observational studies on cardiovascular disease

Appendix L – Outcome of the systematic review on bone health

L.1. Evidence table

References (country)	Design	Baseline characteristics mean ± SD (N)	Na intake assessment method	UNa (mg/day) Mean ± SD (N)	Outcomes assessed	Confounders adjusted for	Key findings
Devine et al. (1995); Prince et al. (1995) **(Australia)**	RCT in postmenopausal women (≥ 10 years after menopause) Group 1: Ca suppl. (1 g/day) Group 2: Ca suppl. (1 g/day) + exercise programme Group 3: Placebo Group 4: ~200 mL milk/day (1 g Ca/day) N = 196 screened N = 168 randomised (42/group) Duration: 2 years	Age: 63 (168) bw: 66 ± 10 (191) Nutrient intakes based on 4-day weighed diet records (190): Protein: 76 ± 16 g/day Ca (food only): 805 ± 320 mg/day P: 1,269 ± 311 mg/day Energy: 1,632 ± 348 kcal/day	Single 24-h urine samples collected at baseline, year 1 and year 2; excretions averaged over the 2-year period. No information on instructions to participants or on measures to check completeness of collections.	Baseline UNa: 2,783 ± 1,081 (196) Average year 1 and 2 UNa: 3,049 ± 808 (127)	BMD (total hip, intertrochanter, femoral neck, ultradistal ankle, lumbar spine) By DEXA at baseline, 1 year and 2 years	Body weight, change in METs/day over the 2-year period, average daily Ca intake	Multiple regression analysis: Negative associations between average UNa and 2-year change in BMD for total hip (SRC = −0.20, p = 0.002) and ultradistal ankle (SRC = −0.18, p = 0.016)
Ilich et al. (2010) **(USA)**	RCT in postmenopausal women (≥ 5 years after menopause); all supplemented with Ca (630 mg/day) and vitamin D (~ 400 IU/day) Group 1: instructed to reduce Na intake to 1,500 mg/day	Age: 68.6 ± 7.1 (136) bw: 68.0 ± 11.3 (136) BMI: 26.0 ± 3.8 (136) Nutrient intakes based on 4-day weighed diet records (136): Protein: 70.6 ± 18.6 g/day Ca (food only): 872 ± 365 mg/day P: 1,077 ± 351 mg/day	Single 24-h urine samples collected every 6 months. Cumulative Na/Cr used as variable. Careful instructions to participants and use of Cr to screen for errors	Baseline UNa: 2,404 ± 963 (136) Baseline UNa/g Cr: 2,465 ± 903 (136)	BMD (composite femur, forearm, lumbar spine, total body) By DEXA at baseline and every 6 months	Age, height, cumulative lean and/or fat tissue, cumulative total calcium intake and cumulative modes of physical activity	Multiple regression analysis (ITT): Positive associations between cumulative UNa/Cr and BMD at 36 months at LS (Coeff. = 5.85^{e-5}, p = 0.056), at forearm (Coeff. = 8.12^{e-5}, p = 0.076) and total body BMD

References (country)	Design	Baseline characteristics mean ± SD (N)	Na intake assessment method	UNa (mg/day) Mean ± SD (N)	Outcomes assessed	Confounders adjusted for	Key findings
	Group 2: usual Na intake (~ 3,000 mg/day) N = 136 randomised (68/group) N = 97 completed Duration: 3 years	Energy: 1,691 ± 382 kcal/day	or incomplete collection				(Coeff. = 2.81^{e-5}, p = 0.041)
							Random-effects regression analysis (accounting for repeated measures and missing data): Main effect of UNa in the forearm: higher UNa associated with higher BMD at baseline and subsequent time points (t = 2.63, p = 0.0089). Similar observation in the spine. No effect on total body or femoral BMD

BMD: bone mineral density; BMI: body mass index; bw: body weight; Ca: calcium; Cr: creatinine; DEXA: dual-energy X-ray technology; MET: metabolic equivalent activity; IU: international unit; N: number; Na: sodium; NR: not reported; P: phosphorus; RCT: randomised controlled trial; SD: standard deviation; SRC: standardised regression coefficient; UNa: urinary sodium excretion.

L.2. Outcome of the RoB

L.2.1. Observational evidence

References	Risk of bias domains[a]						Tier[b]
	Confounding(a)	Attrition	Exposure	Outcome	Reporting	Statistics	
Devine et al. (1995)	+	–	–	++	++	++	2
Ilich et al. (2010)	++	+	+	++	+	+	1

(a): Expert judgement was translated into a rating scale for each question to be answered as follows: (++): definitely low RoB; (+): probably low RoB; (NR): not reported; (–): probably high RoB; (––): definitively high RoB.
(b): The individual rating for each question was combined by an algorithm and translated to an overall tier of reliability for each individual study (RoB tier 1: low RoB; RoB tier 2: moderate RoB; RoB tier 3: high RoB).

Appendix M – Expert knowledge elicitation

In 2014, EFSA published a guidance on expert knowledge elicitation (EKE) in food and feed safety risk assessment (EFSA, 2014). The reader is referred to the guidance for general information about the objectives, procedures and methods of EKE methods. Appendix M details the specific process used for the two EKEs that were conducted to inform the setting of DRVs for sodium.

M.1. Principle

EKE refers to the drawing out of knowledge from one or more experts, following a transparent, structured and comprehensive method based on rigorous probabilistic judgement techniques (EFSA, 2014).

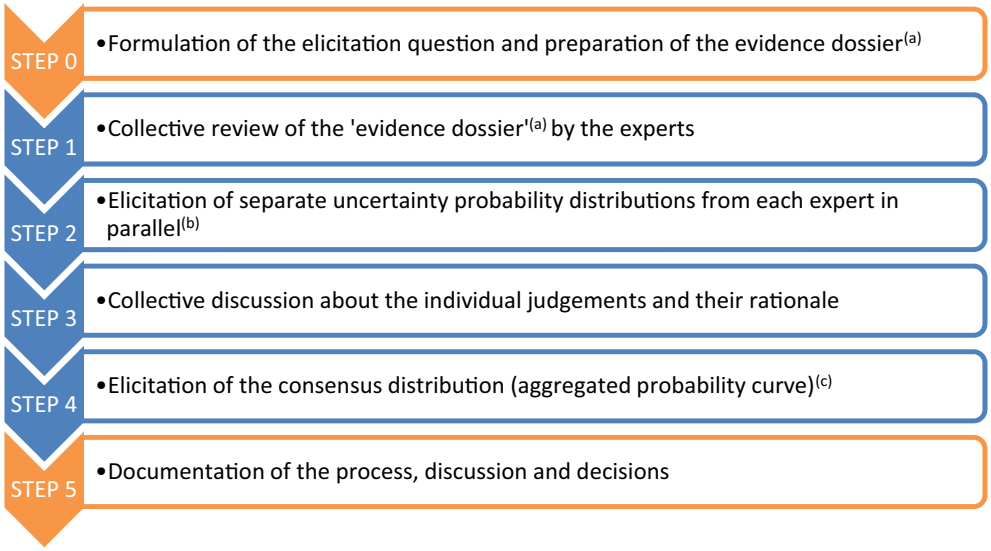
Through the EKE, experts can be asked for judgements about an uncertain quantity of interest. The method aims at obtaining from the experts not only their 'best judgement' of the quantity of interest based on the available evidence, but also a representation of the uncertainty surrounding it.

M.2. The Sheffield protocol

The Sheffield protocol is an EKE method that is based on behavioural aggregation, i.e. judgements of several experts are combined via a moderated discussion (EFSA, 2014).[14]

The main steps of the protocol are illustrated in Figure M.1. It is characterised by two rounds of judgements from the experts. In the first round, experts answer the elicitation question privately, by deriving their individual uncertainty probability distribution. The individual judgements, together with their rationale (see Sections M.3 and M.4 below), are then discussed by the group. In the second round, the group agrees on a consensus judgement through a moderated group discussion. To that end, the group is requested to express the reasonable judgement of an impartial expert informed by the range of judgements and rationales expressed in the first round. The final outcome of the EKE is an uncertainty probability distribution that represents the experts aggregated judgement about the quantity of interest.

The protocol requires an experienced facilitator to manage the experts, and to address possible sources of bias in group interactions.



(a): The evidence dossier documents all the evidence and related uncertainties relevant to the formulation of the expert judgement (see Section M.3 below).

(b): In this step, each expert answered the EKE questions individually. To that end, experts had to express the probability that each of the proposed ranges of sodium intake/excretion would include the 'true' level of interest (roulette method, see Section M.4 below). As a result, each expert provides his/her own uncertainty probability distribution.

(c): Following the collective discussion, a consensus uncertainty probability distribution is elicited from the group of experts, which reflect the aggregated view of the experts on the parameter of interest and related uncertainty.

Figure M.1: Main steps of the EKE process following the Sheffield method

[14] The Sheffield Elicitation Framework was created by Tony O'Hagan and Jeremy Oakley at the University of Sheffield, UK (http://www.tonyohagan.co.uk/shelf/)

M.3. Preparatory phase: elicitation questions and evidence dossier

Based on the review of the available evidence, it was considered that (i) data on the relationship between sodium intake and level of blood pressure or CVD risk could inform about the levels of sodium intake associated to a reduced risk of chronic diseases; and (ii) balance studies could inform about the levels of sodium intake that are adequate to maintain a null sodium balance. Because of the limited evidence available and of the associated uncertainties, it was not possible to identify such levels of sodium intake with certainty. Two questions were formulated to elicit evidence-based judgement of the experts about the levels of interest:

EKE question 1: *What is the lowest level of sodium intake at which the risk of chronic disease (i.e. stroke, CHD) is minimised in the majority (≥ 97.5%) of the general population of adults?*

EKE question 2: *What is the lowest level of sodium intake which is adequate (i.e. amount which allows to maintain sodium balance) for the majority (≥ 97.5%) of the general population of adults?*

Through the EKE, experts are required to formulate evidence-based judgements, providing a rationale. An evidence dossier was prepared before the EKE meeting, which assembled all the relevant evidence into a single document, in the form of structured tables and graphical representations (BOX 2). During the process, the experts are requested to provide their rationales behind their probability assessments by referring to the dossier.

The two EKEs were conducted with the seven experts of the EFSA working group on DRVs for minerals,[15] who had been in charge of selecting, reviewing and appraising the available evidence. All experts received a training on the method before starting the elicitation process.

BOX 2 – Content of the evidence dossier

EKE question 1:

a) All relevant evidence on the relationship between sodium and blood pressure, including:

- The outcome of the meta-regression modelling of sodium and blood pressure
- The data from the eligible randomised studies on the incidence of hypertension
- The data from the eligible prospective cohort study on blood pressure
- The data from the eligible prospective cohort studies on the incidence of hypertension
- Considerations about the reliability, external validity and consistency of each source of evidence

b) All relevant evidence on the relationship between sodium and risk of CVD, including:

- The data from the eligible prospective cohort studies on risk of stroke
- The data from the eligible prospective cohort studies on risk of coronary heart disease
- The data from the eligible prospective cohort studies on risk of CVD
- Considerations about the reliability, external validity and consistency of each source of evidence
- The outcome of the risk of bias assessment for each individual study
- Graphical representations of the dose-responses from the eligible observational studies on coronary heart disease and stroke
- A summary of relevant mechanistic data

EKE question 2:

- The data from the eligible balance studies
- Considerations about the reliability, external validity and consistency of the available evidence
- A summary of relevant mechanistic data

[15] https://www.efsa.europa.eu/en/nutrition/working-groups

M.4. Elicitation of the uncertainty probability distributions: the roulette method

For illustrative purposes, the uncertain quantity of interest is denoted *X*. The objective of the EKE is to elicit a probability distribution for *X* from the experts. The roulette method was used for the EKEs on sodium (Johnson et al., 2010; EFSA, 2014). The range of possible values for *X* was divided into fixed intervals (bins) (Figure M.2). In answering the elicitation questions, experts were asked to distribute 20 chips into the bins, with the probability of *X* lying in a particular bin interpreted as the proportion of chips allocated into that bin. As the total number of chips was 20, each chip represented 5% probability.

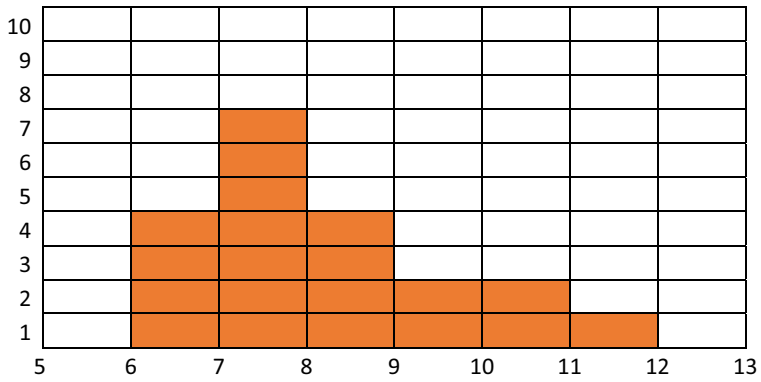


Figure M.2: Illustration of the roulette method. In this example, a range of possible values for *X* has been set between 5 and 13. Each orange rectangle represents one chip. For instance, by placing four of their 20 chips in the bin (6,7) the expert expresses a probability of 4/20 (20%) that the 'true value' of *X* lies in that bin. The probability that the range 6 to 12 includes the 'true value' of *X* is 100%.

In the first round of judgements, one histogram was obtained from each expert, representing his/her uncertainty probability distribution. In the second round of judgements, the experts built a group histogram, i.e. consensus uncertainty probability distribution, through discussion. The group consensus uncertainty probability distributions elicited in response to the two EKE questions are provided in Figures 1 and 2. The rationales for the distributions were documented and are reported in Tables 9 and 10.

M.5. Derivation of parametric distributions from the group consensus uncertainty probability distributions

Parametric distributions were fitted to the group consensus uncertainty probability distributions to derive probability density functions. This allows the derivation of probabilities for values within the range of each bin used for the elicitation. Several parametric distributions (e.g. normal, log-normal, truncated normal, gamma, Weibull and logistic) have been considered as possible candidates to estimate the probability density functions that better fit the centiles obtained through the EKE process. Log-normal distributions were finally chosen based on indicators of goodness of fit (Akaike Information Criterion and Bayesian Information Criterion), with the following parameters: 0.72 (mean) and 0.23 (standard deviation) for the question on sodium intake at which CVD risk is reduced; 0.5 (mean) and 0.3 (standard deviation) for the question on sodium balance.

Based on the two probability density functions, cumulative uncertainty density functions were derived (Figure 3) and served as the basis to inform the setting of the reference value for sodium.

Annex A – Protocol for sections 5.5 and 6 of the scientific opinion on DRVs for sodium: Assessment of the relationship between sodium intake and pre-specified health outcomes, including dose–response relationships, and integration of different lines of evidence for setting DRVs for sodium

Annex A can be found in the online version of this output ('Supporting information' section): https://doi.org/10.2903/j.efsa.2019.5778

Annex B – Analysis of evidence from published scientific literature as preparatory work for the setting of Dietary Reference Values for Sodium

Annex B can be found in the online version of this output ('Supporting information' section): https://doi.org/10.2903/j.efsa.2019.5778