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Limited salt consumption reduces the incidence of chronic kidney disease: a modeling study

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ABSTRACT

Background In addition to blood pressure and cardiovascular disease, high-salt intake has been associated with renal diseases. The aim of this study is to estimate the potential health impact of salt reduction on chronic kidney disease (CKD) and end-stage kidney disease (ESKD) in the Netherlands.

Methods We developed a dynamic population health modeling tool to estimate the health impact of salt reduction on CKD and ESKD. We used data from the PREVEND study and extrapolated that to the Dutch population aged 30–75 years. We estimated the potential health impact of salt reduction comparing the current situation with the health impact of the adherence to the recommended maximum salt intake of 6 g/d.

Results In the recommended maximum intake scenario, a cumulative reduction in CKD of 1.1% (N = 290 000; interquartile range (IQR) = 249 000) and in ESKD of 3.2% (N = 470; IQR = 5080) would occur over a period of 20 years.

Conclusions Our health impact estimation showed that health benefits on CKD might be achieved when salt intake is reduced to the recommended maximum intake of 6 g/d.

Keywords food and nutrition, kidney disease, models

Introduction

A high-salt intake (NaCl) is likely to have a substantial burden of chronic disease worldwide. Health impact assessments show that the burden of high-salt intake is around 6% of the total burden of disease.[1,2] Country-specific estimations also show that salt reduction is a cost-effective measure to reduce the burden of cardiovascular disease.

These health impact assessments are all based on the assumption that lowering salt intake will reduce blood pressure, and thereby the incidence of cardiovascular disease. However, they did not consider additional conditions sensitive to blood pressure such as chronic kidney disease (CKD). A long-standing line of evidence shows beneficial effects of salt reduction, in patients with established CKD as well as in healthy people.[3,4] In the general population, increased blood pressure caused by high-salt intake has an adverse effect on the long-term course of the glomerular filtration rate (GFR) and on the susceptibility to develop proteinuria, both measures of kidney function.[3,4] Clinical trials have shown that in patients with CKD, salt reduction will have additional beneficial renal effects, apparent from reduction of proteinuria, even independent of blood pressure.[5] In addition, it appears that medication prescribed in CKD is more effective when salt intake is low,[6,7] by effects on blood

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pressure as well as effects unrelated to blood pressure. As the renal effects of salt are so far not included in health impact assessment studies, the potential health impact of salt reduction is probably higher than estimated in the above-mentioned quantitative health impact assessment studies.

In the present study, we developed a model to assess the health impact of limiting salt consumption to 6 g/d on CKD. For this, we developed a new Markov-type, chronic disease modeling tool that can estimate such an effect for the population in the Netherlands aged 30–75 years.

Methods

Model development

We developed a Markov-type multistate transition model, which simulates the life course of the Dutch population aged 30–75 years. The figure in the Technical Appendix shows a schematic overview of the model. The input data consisted of the initial distribution of salt intake, systolic blood pressure (SBP), albuminuria and estimated GFR (eGFR, as calculated by the CKD–EPI equation[8]) in the Dutch population, the transitions of the risk factors over age, and the association (in relative risks) between salt intake and SBP, albuminuria and eGFR. Time is modeled in discrete intervals of 1 year and all parameters are age- and sex-specific.

Study population for model input

Baseline distributions of salt intake, SBP, eGFR and albuminuria were obtained from the Prevention of Renal and Vascular End-Stage Disease (PREVEND) study, a prospective population-based cohort study.[9] This study is a prospective investigation of albuminuria, renal and cardiovascular disease in a large cohort drawn from the general population aged 28–75 years in the city of Groningen. In short, baseline measures were performed in 1997–98. All inhabitants of the city of Groningen received a questionnaire, and were asked to collect a first morning void urine sample. Of those, 40 856 subjects responded (47.8%) and returned this sample to a central laboratory for urinary albumin assessment. All subjects with a urinary albumin concentration of >10 mg/l (N = 7768) were invited to participate in the study, and 6 000 subjects participated. A randomly selected control group with a urinary albumin concentration of < 10 mg/l was also invited. From this group, 2592 subjects participated (76.4%). The combined group of 8592 subjects is considered the PREVEND cohort, of which 95.5% were Caucasian, 1.0% Negro, 2.2% Asian and 1.3% of other race. Education levels were 12, 20, 26, 26 and 16% for Level 1 (highest) to Level 5 (lowest), according to the International Standard Classification of Education (ISCED)[10]. At baseline, participants collected two consecutive 24-h urine samples. For follow-up, participants were invited to participate in a medical examination at ~3-year intervals. The PREVEND study was approved by the medical ethical committee of the University Medical Centre of Groningen, and conducted in agreement with the Declaration of Helsinki. All participants gave written informed consent.

In PREVEND, salt intake was assessed by 24-h urinary sodium excretion, with urinary sodium concentration measured by indirect potentiometry using a MEGA clinical chemistry analyzer (Merck, Darmstadt, Germany). Urinary sodium excretion is calculated as urinary sodium concentrating times 24-h urine volume and given as the average value of the two 24-h urines that were collected at each screening visit. Blood pressure was measured in sitting position, every minute for 10 min (first visit) and 8 min (second visit) with an automatic device Dinamap XL Model 9300 series device (Johnson-Johnson Medical, Tampa, FL, USA). SBP was defined as the mean of the last two recordings of both visits. Urinary albumin concentration was determined by nephelometry (Dade Behring Diagnostic, Marburg, Germany). Estimated GFR (eGFR) was calculated with the CKD–EPI equation.[8]

To mimic the Dutch population, we adjusted for the complex sample design (i.e. the oversampling of subjects with higher albuminuria) to create a cohort that has a representative albumin excretion. In a following step, this cohort was extrapolated to represent the Dutch population, using a weight factor based on the actual age and sex distribution of the Dutch population aged 30–75 years old in 2015 (Statistics Netherlands). More details can be found in the Technical Appendix.

Transition of the risk factors over age

We used the sequential rounds of PREVEND to estimate the trend of salt intake (two rounds), SBP and albumin excretion (four rounds) over time. Salt intake was constant until the age of 60, and declined with 0.08 (females) to 0.10 (males) g/d per year thereafter (see Technical Appendix). SBP increased with age and salt intake (see Technical Appendix). Albuminuria was divided into three categories (<30, 30–300 and ≥300 mg/d)[11]. Transition between classes was simulated through 1-year transition probabilities that were age- and sex-specific, and that depended on SBP. eGFR was calculated with individual decline rates, and depended on sex, age, SBP and albuminuria category (see Technical Appendix).

More details on the derivation of the trends of the risk factors over age can be found in the Technical Appendix.

Modeling the association of salt intake to blood pressure, albumin excretion and eGFR

We assumed an association of salt intake with SBP and of SBP with eGFR and albumin excretion in the general population. The association of salt intake with SBP was obtained from He and MacGregor[12] and was translated to an exponential association between salt intake and SBP. The details of this dose–response association have previously been described in more detail;[13] see also Technical Appendix.

The association of SBP with albuminuria was derived from Xie *et al.*[3] In this meta-analysis of randomized controlled trials with at least 6 months' follow-up, the effect of intensive blood pressure reduction on albuminuria was estimated. This dose–response association shows that a high SBP will lead to a higher risk of developing albuminuria. More details of this dose–response association can be found in the Technical Appendix.

The association of SBP with eGFR was derived from Garofalo *et al.*[14] This meta-analysis of 16 cohort studies found that a 10 mmHg increase in SBP was associated with a higher risk of decreased eGFR. This association was transformed to create the risk per unit change of SBP (see Technical Appendix). In addition, in the PREVEND data we observed an inverse association of albumin excretion of more than 300 mg/d with eGFR, and this association was also included in the model.

Scenarios and model outputs

We compared the estimated health impact of the scenario based on the current salt intake against a scenario in which the maximum salt intake was 6 g/d, as recommended in the Dutch dietary guidelines developed by the Dutch Health Council. The reference scenario simulates 20 years of unaltered salt intake. Salt consumption in the intervention scenario is identical to intake in the reference scenario except for person-years with consumption above 6 g/d: those were set to 6 g/d. We calculated the estimated cumulative incidence of CKD and ESKD in the cohort initially aged 30–75 over a period of 20 years. We defined CKD as having an eGFR lower than 60 ml/min/1.73 m^2 or as having an albumin excretion of >30 mg/d. ESKD was defined as having an eGFR <15 ml/min/1.73 m^2.[11] Validation of the model was done to compare the outputs of our model with the incidence and prevalence data of eGFR (Stages 3, 4 and 5) in the Dutch population.[15] For more information, see Technical Appendix.

One-way sensitivity analyses

We performed two additional sensitivity analyses. First, we modeled a direct effect of salt reduction on albuminuria in patients with CKD. We obtained a dose–response association from salt intake on albumin from Kwakernaak *et al.*[16] In this double blind, crossover and placebo-controlled trial, patients with type 2 diabetic nephropathy received a sodium-restricted diet in combination with a standardized treatment on albuminuria for 6 weeks. This study showed that sodium restriction resulted in reduced albuminuria. The details are described in the Technical Appendix.

Second, some studies suggest that the increase in blood pressure by age may attenuated with lower dietary salt intake.[17,18] In this sensitivity analysis, we reduced the increase in SBP over age for people with a salt intake <6 g/d. The details are described in the Technical Appendix.

Probabilistic uncertainty analyses

We used Monte Carlo simulations to estimate the upper and lower confidence limits of the dose–response associations to assess the uncertainty of our projections. Additionally, we included the standard error of the parameters that were estimated based on the PREVEND data. For each outcome measure, we report the median and the 95% confidence interval for 100 simulations, which represents the range of the expected effects.

Results

The mean salt intake in the modeled cohort representative of the Dutch population aged 30–75 years was 8.8 (SD = 2.5) g/d in the baseline situation, and the mean SBP was 125.6 (SD = 17.9) mmHg. The albumin excretion was 15.2 (sd = 76.5) mg/d and the mean eGFR was 94.8 (SD = 17.1) ml/min/1.73 m^2. In the intervention scenario, mean salt intake was 5.9 (SD = 0.3) g/d leading to an altered mean blood pressure of 123.6 (SD = 15.0) mmHg. Table 1 shows these results stratified to age and sex. As shown in Fig. 1A, SBP increases at a similar rate after salt reduction in the intervention scenario as in the baseline situation. Figure 1B shows the development of eGFR over time in the baseline situation and in the intervention scenario. At the start of the modeling period, eGFR is identical in the baseline and the intervention scenario. Over the 20-year period, the decline in eGFR is less in the situation when the salt intake is reduced to the recommended maximum intake of 6 g/d. After 20 years, eGFR in the intervention scenario is 0.14 ml/min/1.73 m^2 higher than in the reference scenario.

Table 1 Mean (SD) characteristics of the population included in the health impact model (Dutch men and women aged 30–75 years)

	Age group	*Baseline situation*		*Intervention situation (salt intake 6 g/d)*	
		Men	*Women*	*Men*	*Women*
Salt intake (g/d)	30–45	10.2 (2.6)	8.1 (2.1)	6.0 (0.2)	5.9 (0.3)
	45–60	10.3 (2.6)	8.0 (2.0)	6.0 (0.1)	5.9 (0.4)
	60–75	9.1 (2.4)	7.2 (1.7)	5.9 (0.3)	5.8 (0.5)
Systolic blood pressure (mmHg)	30–45	125.7 (12.6)	114.1 (12.6)	123.0 (8.8)	113.2 (9.7)
	45–60	129.6 (15.0)	121.2 (16.4)	126.4 (11.3)	119.9 (13.6)
	60–75	139.3 (18.8)	138.4 (20.5)	135.8 (15.5)	136.5 (18.2)
Albumin excretion (mg/d)[a]	30–45	7.4 (59.3)	6.5 (48.5)	7.4 (59.3)	6.5 (48.5)
	45–60	7.5 (93.4)	6.5 (69.0)	7.5 (93.4)	6.5 (69.0)
	60–75	9.0 (112.5)	7.0 (88.7)	9.0 (112.5)	7.0 (88.7)
eGFR (ml/min/1.73 m^2)	30–45	104.5 (13.5)	104.0 (13.7)	104.5 (13.5)	104.0 (13.7)
	45–60	94.0 (13.5)	94.1 (14.0)	94.0 (13.5)	94.1 (14.0)
	60–75	78.7 (14.8)	78.0 (14.3)	78.7 (14.8)	78.0 (14.3)

[a]Median value.

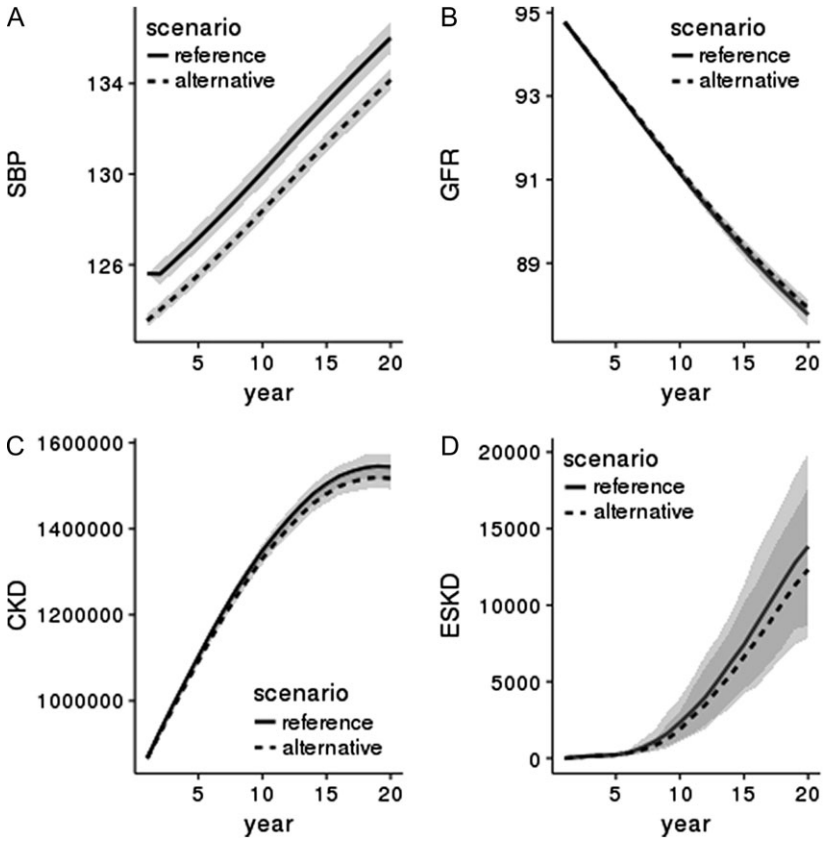

Fig. 1 Mean systolic blood pressure (**A**), estimated glomerular filtration rate (**B**), chronic kidney disease (**C**) and end-stage kidney disease (**D**) over the 20-year simulation period. Solid line: reference scenario, dashed line: max 6 g salt scenario.

If salt intake is reduced to the recommended maximum salt intake of 6 g/d, 1.1% (N = 290 000; interquartile range (IQR) = 249 000) of the cases of CKD and 3.2% (N = 470; IQR = 5080) of the cases of ESKD could be prevented in 20 years (Table 2). In all outcome measures, men benefit more than women do. Especially, the difference in ESKD prevalence has large IQRs, spanning both positive and negative differences. Positive differences are due to better survival rates. Note that numbers for men and women do not exactly add up to total numbers because the table displays median values. Figure 1C and D shows the difference in prevalence of CKD and ESKD between the intervention scenario and the baseline situation. The difference in prevalence of CKD and ESKD is more pronounced at the end of the 20-year modeling period. However, there is an overlap of the 95% uncertainty intervals between the baseline situation and the intervention scenario.

One-way sensitivity analyses

The sensitivity analyses show that a direct effect of salt reduction on albumin excretion in subjects with CKD would lead to a reduction of 2.0% in cases of CKD (N = 535 000; IQR = 257 000) and 3.2% in cases of ESKD (N = 470; IQR 5100) in 20 years (Table 3). A lower increase of SBP with age after salt reduction results to a reduction of 1.1% (N = 287 000; IQR = 247 000) in cases of CKD and 3.1% (N = 550; IQR = 5360) in cases of ESKD.

Discussion

Main finding of this study

Our modeling study demonstrated that if salt intake in the Dutch population would be reduced to the recommended maximum intake of 6 g/d, eGFR would be 0.14 ml/min/1.73 m^2 higher after 20 years in a cohort aged 30–75 years representative for the Dutch population. This accounts for a cumulative reduction in CKD of 1.1% (N = 290 000; IQR = 249 000) and in ESKD of 3.2% (N = 466; IQR = 5080) over a period of 20 years.

What is already known on this topic

Several studies have modeled the health impact of salt reduction on cardiovascular disease,[19] DALYs[20] and all-cause mortality.[13] The general conclusion of these studies is that salt reduction will lead to a substantial reduction in the incidence of CVD and mortality. To our knowledge, the present study is the first to estimate the population health impact of reduced salt consumption on CKD and ESKD.

Table 2 Median (IQR) cumulative absolute number and difference between baseline situation and the intervention situation in disease prevalence of CKD and ESKD and life years over 20 years for the Dutch population aged 30–75 years old

	Baseline situation (×1000)			*Intervention scenario (×1000)*			*Absolute difference (×1000)*			*Relative difference (%)*		
	Men	*Women*	*Total*	*Men*	*Women*	*Total*	*Men*	*Women*	*Total*	*Men*	*Women*	*Total*
CKD prevalence	15 670 (802)	10 900 (1.4,7)	26 740 (1 980)	15 470 (717)	10 820 (1 326)	26 430 (1 940)	−217 (188)	−79.5 (105)	−290 (249)	−1.4 (1.1)	−0.74 (0.83)	−1.1 (0.9)
ESKD prevalence	7.18 (20.12)	9.46 (38.40)	17.20 (59.70)	7.24 (17.19)	9.39 (35.40)	16.90 (55.80)	−0.26 (2.22)	−0.21 (3.30)	−0.47 (5.08)	−3.2 (9.7)	−2.4 (8.0)	−3.2 (8.7)
Life years	89 900 (427)	93 290 (220)	183 200 (608)	90 070 (255)	93 330 (189)	183 400 (435)	171 (147)	38.1 (30.0)	208 (176)	0.19 (0.16)	0.04 (0.03)	0.11 (0.10)

Table 3 Median (IQR) cumulative absolute number and difference between baseline situation and the intervention situation in disease prevalence of CKD and ESKD and life years over 20 years for the Dutch population aged 30–75 years old in two one-way sensitivity analyses

	Baseline situation (×1000)			*Intervention scenario (×1000)*			*Absolute difference (×1000)*			*Relative difference (%)*		
	Men	*Women*	*Total*	*Men*	*Women*	*Total*	*Men*	*Women*	*Total*	*Men*	*Women*	*Total*
Direct effect of salt reduction in CKD patients												
CKD prevalence	15 670 (802)	10 900 (1 417)	26 740 (1 980)	15 250 (708)	10 800 (1 331)	26 190 (1 920)	−435 (200)	−125 (101)	−535 (257)	−2.7 (1.1)	−1.1 (0.8)	−2.0 (0.8)
ESKD prevalence	7.18 (20.10)	9.46 (38.40)	17.20 (59.70)	7.23 (17.20)	9.39 (35.50)	16.90 (55.80)	−0.28 (2.20)	−0.23 (3.30)	−0.47 (5.10)	−4.0 (9.7)	−2.6 (8.0)	−3.2 (8.8)
Life years	89 900 (427)	93 290 (220)	183,200 (608)	90 070 (255)	93 330 (189)	183,400 (435)	171 (147)	38 (30)	208 (176)	0.19 (0.16)	0.04 (0.03)	0.11 (0.10)
Lower increase in SBP over age after salt reduction												
CKD prevalence	15 660 (793)	10 860 (1 324)	26 690 (1 920)	15 460 (705)	10 790 (1 245)	26 380 (1 860)	−216 (187)	−79 (101)	−287 (247)	−1.4 (1.1)	−0.7 (0.8)	−1.1 (0.9)
ESKD prevalence	7.19 (19.70)	9.02 (32.90)	16.90 (54.00)	7.27 (16.80)	8.74 (29.40)	16.50 (49.20)	−0.23 (2.30)	−0.26 (3.40)	−0.55 (5.36)	−3.9 (9.7)	−2.6 (8.8)	−3.1 (9.5)
Life years	89 900 (425)	93 360 (207)	183 300 (599)	90 100 (253)	93 400 (177)	183 500 (420)	171 (147)	38 (30)	208 (176)	0.19 (0.16)	0.04 (0.03)	0.11 (0.10)

Currently, there is sufficient evidence available that salt reduction has also beneficial renal effects[6] and thus that a health impact estimation of salt reduction should include an assessment on renal disease. The impact estimates of the present study are in the same order of magnitude as compared with the impact estimates of the studies that estimated the effect on CVD, although slightly lower. This can be explained by the effect that the relative risks of SBP on albuminuria and eGFR are lower as compared to the relative risks of SBP to CVD. In addition, in the present study we included only subjects aged 30–75 years, which may have led to an underestimation of the effects that would occur in a population aged 30 years and older. As impaired kidney function and albuminuria increases the risk of CVD by two to four times, prevention of CKD will ultimately also result in a lower incidence of CVD.[21] In the present study, we did not estimate this effect, as it would not result in an additional effect in the incidence of CVD. Instead, there could even be adverse effect of reduced salt consumption on kidney disease via reduced CVD when CVD would reduce CKD incidence. However, there is no unequivocal evidence to support such a relation.

What this study adds

Our modeling study demonstrated that salt reduction could reduce the CKD prevalence in the Dutch population, which is an additional gain in health as compared to the studies that have estimated its effect on CVD. The uncertainty analysis showed that the range of the potential effect between the baseline situation (current salt intake) and the intervention scenario (6 g salt) might overlap; meaning that in certain simulations, the potential effect of salt reduction on CKD and ESKD was limited. Such an event could occur when values that would lead to smaller effects of salt reduction are assigned to a couple of model parameters simultaneously in the random draw, as this happened only rarely. At this time, the few long-term intervention studies to reduce salt consumption have insufficient power to detect effects on disease incidence, but the present model calculations show that it could yield positive results if a large intervention study were to be undertaken in the future. An intervention study would also clarify any direct relation between salt consumption and kidney diseases.

Several individual studies show that salt reduction in people with CKD has an independent effect on blood pressure and proteinuria. However, there is a large variation in study designs and a recent meta-analysis could not pool the data from these studies.[22] The sensitivity analysis where we included this effect was therefore based on a single trial of Kwakernaak *et al.*,[16] where we could derive a dose–response

association. This sensitivity analysis showed increased favorable effects in the prevalence of CKD, but not on ESKD. The sensitivity analysis modeling lower increase in SBP over age for people consuming <6 g/d did not have an influence on the results of the main analysis. Both sensitivity analyses barely affected life years. It seems that the results from the main analysis are quite robust.

Limitations of this study

We developed a new Markov-type model specifically designed for the Dutch population. The output from any model is limited by the data that is entered in the model. Therefore, we presented a range of effects based on uncertainty analyses, the uncertainty in the estimated parameters, and the range of the relative risks obtained from literature.

Our study population is based on the PREVEND cohort. The advantage of this study population is that it contains data for salt intake, blood pressure, albumin and eGFR for everyone over sequential rounds. Limitation is that it is not a representative sample from the Dutch population; it is a random sample of the northern city of Groningen, and with an oversampling of some subjects with a higher albumin excretion. We extrapolated the number of individuals in PREVEND to the Dutch population. On key input parameters, such as salt intake and SBP, the extrapolation seemed to be sufficient. However, the comparison with the incidence and prevalence of CKD proved to be difficult as in the Netherlands no official data registry for CKD exists. Van Blijderveen *et al.*[15] reported on the incidence rates of all stages of CKD for the entire Dutch population, stratified by sex, 5-year age groups and diabetes. However, this data may be an underestimation of the 'true' prevalence and incidence of CKD as the participants were sampled in day-to-day patient care and blood samples were measured only once. Compared with Van Blijderveen *et al.*, the increase in incidence and prevalence of CKD in our model started at an older age. This may indicate that we have underestimated the prevalence and incidence of CKD in our model calculations, especially at older age. This study only included subjects aged 30–75 years. Probably, the results presented here may be an underestimation of the actual impact on CKD that can be obtained by salt reduction, as the prevalence of CKD increases by age.

Another limitation is related to the categorization of albumin in three non-linear categories. Because of the small changes that could be established due to SBP lowering, our model may not have been sensitive enough to detect all changes over the albumin categories. Additionally, the prevalence and incidence of ESKD are rare, which could have affected our calculations.

In the general population, a maximum salt intake of 6 g/d will be a challenge, as it requires a reduction of almost a third of the current salt consumption. Most likely, a combination of food reformulation, as well as changes in food consumption patterns will be the most effective interventions to reduce salt intake. We did not simulate this indirect effect of altered dietary patterns on kidney disease incidence because quantitative scientific evidence is lacking. Similarly, we do not expect any direct negative effects on public health by limited salt consumption (such as possibly increased incidence of DM), also because the Dutch Health Council's recommendation is only to limit the intake to a maximum of 6 /d rather than to minimize salt consumption.

To conclude, this is the first study that showed that limiting maximum salt consumption to 6 g/d would reduce the prevalence of CKD by 1.1% (N = 290 000; IQR = 249 000) and of ESKD by 3.2% (N = 466; IQR = 5080) in the Dutch population over a period of 20 years. These health gains are additional to the expected health gains in cardiovascular diseases.

Supplementary data

Supplementary data are available at the *Journal of Public Health* online.

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