Urinary Sodium Excretion and Cardiovascular Disease Mortality

To the Editor: Dr Stolarz-Skzypek and colleagues reported that lower urinary sodium excretion was associated with higher cardiovascular disease (CVD) mortality.[1] These findings contradict a large body of evidence that established elevated sodium consumption as a risk factor for CVD and shed doubts on the worldwide efforts to implement population-based sodium reduction strategies.[2] We have a number of concerns that suggest their findings should be interpreted cautiously.

First, the volume of urine collected during 24 hours was significantly higher among people with high sodium excretion compared with those with low sodium excretion. It is not clear whether these differences reflect true urinary volume output or problematic completeness of urine collections. The authors assessed the accuracy of urine collection based on 24-hour urinary volume and creatinine excretion. However, these parameters are not reliable markers for completeness; instead, the proposed standard criterion in nutritional epidemiology studies is the use of paraaminobenzoic acid.[3]

Furthermore, the lowest levels of 24-hour urinary volume and sodium excretion were found in people with low educational attainment, which is in contradiction with observations from earlier studies that found sodium excretion to be higher in people with low educational level.[4] Lower education level is also associated with unfavorable CVD risk profile and higher risk of developing CVD.[5] Thus, although the authors adjusted for educational attainment, the findings may have been caused by misclassifying people with low socioeconomic status but higher CVD risk as having low 24-hour sodium excretion.

Second, although the authors used the same study protocol, the baseline examinations in the cohorts that were combined for analysis were conducted in different time periods (a gap of roughly 10 years between recruitment) and in geographically and culturally different regions of Europe. In addition, nearly all cardiovascular deaths (81/84) occurred in FLEMENGHO (Flemish Study on Environment, Genes, and Health Outcomes; eTable 6 in the article), and mean sodium excretion was substantially lower in this cohort compared with the other study. Simple pooling of the FLEMENGHO cohort with the cohort in EPOGH (European Project on Genes in Hypertension) raises concern that ecological or temporal differences between cohorts may account for the observed association of sodium excretion with CVD mortality. It would be informative to see stratified results based on study-specific tertiles of sodium excretion.

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To the Editor: Low sodium excretion was associated with higher CVD mortality in a prospective study of 3681 participants from Europe.[1] The results should be interpreted with caution considering several flaws in study design, analysis, and interpretation.

A major source of bias in this study was misclassification of urinary sodium excretion. A single 24-hour urine sample, which was used for all the analyses, cannot represent usual sodium intake at the individual level. Nor can it be used to accurately classify individuals into categories as the authors did in analyzing 3 levels of sodium excretion with CVD mortality. It has been well documented that differential misclassification bias occurs when a continuous exposure variable with random measurement error is classified into multiple categories.[2] Although the authors tried to exclude participants with inaccurate urine collection, it would have been helpful to examine the association of CVD mor-

tality with sodium/creatinine ratio because this ratio is less affected by incompleteness of urine sample collection.

Furthermore, their cohort appears to have included persons who followed a very low sodium diet (50 mmoL/day). Since dietary salt reduction is a common recommendation to patients with hypertension and other conditions that increase the risk of CVD, the inclusion of persons prescribed a low sodium diet in the low sodium excretion tertile could have biased the study findings.

Previous studies have identified an interaction between sodium intake and obesity on the risk of CVD.[3,4] Individuals with obesity or metabolic syndrome are more sensitive to dietary sodium intake.[5] A stratified analysis by obesity or metabolic syndrome would provide additional insight on the sodium-CVD relationship.

Finally, the authors' conclusion of lower sodium excretion with higher CVD mortality was based on an observed weak association ($P = .04$ for hazard ratio and $P = .02$ for trend across tertiles). A large number of statistical tests were conducted in this study, and observed statistical significance could be due to chance alone. If multiple comparisons were taken into account, these observed associations may not remain statistically significant.

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Conflict of Interest Disclosures: Both authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

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To the Editor: The inverse association between urinary sodium excretion and CVD mortality found in a recent prospective cohort study[1] challenges strategies to reduce salt intake in populations that are being implemented in an increasing number of countries.[2] We would like to point out a few issues related to internal validity (potential exposure misclassification, confounding, and survival bias) and external validity (age of the participants in the study).

First, long-term salt intake may not be fully captured by a single 24-hour urine collection. Second, participants in the low sodium excretion tertile were less educated than the other participants; this suggests that socioeconomic status, a major proximal determinant of mortality,[3] may differ across tertiles of urinary sodium excretion and explain the wide differences in mortality. Third, the small numbers of CVD deaths in urinary sodium excretion tertiles (50 in the low, 24 in the medium, and 10 in the high tertile) are problematic considering that the authors included several etiologically different cardiovascular outcomes (eg, aortic aneurysm, cor pulmonale, pulmonary embolism, stroke, and myocardial infarction). Fourth, the association of low salt intake with cardiovascular mortality appears stronger than the association with combined fatal and nonfatal cardiovascular events, suggesting a survival bias.

Finally, participants were fairly young (mean age, 40 years). Since blood pressure sensitivity to salt markedly increases with age[4] and the large majority of CVD deaths occur in elderly people, data on older persons are also needed.

Because of these limitations, we believe that this study does not provide sufficient evidence to dismiss policies aimed at reducing salt intake in populations. It does, however, strengthen the need for further studies, including experimental ones, to help guide appropriate policy.

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To the Editor: The authors of a recent study[1] concluded that their findings " . . . refute the estimates of . . . lives saved and health care costs reduced with lower salt intake." We believe that this study does not shift the weight of evidence that strongly supports recommendations to reduce population sodium intake as an effective means to reduce heart disease, stroke, and all-cause mortality.[2]

This study has several important methodologic shortcomings. First, there was a high proportion of exclusions from the study population. Of the initial sample of 5655 persons, 1922 (33.9%) declined to participate or were excluded for inadequate urine samples, leading to lack of representativeness and potential bias.

Second, the exposure assessment was unreliable. The study relied on a single baseline 24-hour urine collection, which does not necessarily reflect usual intake. In addition, some of the low sodium group had low urine volumes and cre-

atinine concentrations, suggesting incomplete collections. Such urine collections are likely inadequate to estimate usual sodium intake in individuals who are less healthy, have poorer adherence, or are otherwise more likely to have adverse outcomes.

Third, outcomes were incompletely assessed. The identified cardiovascular events (232) or deaths (84) were far outnumbered by losses to follow-up (494, including 219 deaths), potentially invalidating the outcome analysis. Finally, the young average age of participants (~40 years at study entry, with only 6-8 years median follow-up) limits relevance of the study to age groups at greatest risk for cardiovascular events.

The authors characterized as "indiscriminate" current recommendations to reduce salt intake at the population level. In the United States today, the mean reported sodium intake of adults is more than 3400 mg per day, several times the physiologic requirement.[3,4]

The authors' conclusions contrast sharply with decades of research and scientific consensus showing that from tens of thousands to more than 100 000 deaths per year could be prevented in the United States alone by population-wide reduction in sodium intake.[5]

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To the Editor: A recent study[1] found an inverse association of urinary sodium excretion and subsequent CVD, in contrast to a wide body of evidence showing the opposite.[2] The study has several analytic limitations.

First, the posited mechanism by which sodium affects CVD is through blood pressure. The authors, however, controlled for baseline blood pressure, thus removing the intermediate effect of sodium on CVD. A well-known principle of epidemiology is to avoid controlling for factors in the causal pathway. In their analysis of CVD outcomes, the authors included an additional analysis unadjusted for blood pressure, but this still controlled for antihypertensive medication. They inappropriately controlled for baseline blood pressure in analyses of incident hypertension, reversing the crude positive association.

More subtle is bias from reverse causation. The authors claimed to have avoided this by eliminating those with a history of CVD. However, those without overt CVD but with hypertension are advised to reduce their sodium intake. Thus, those with underlying high blood pressure may have moved to the lowest tertile of sodium intake. In fact, there was a U-shaped relationship between baseline hypertension and sodium in their data, suggesting such a bias. Since hypertension is a multifaceted disorder, this reverse causation could confound the observed effect of sodium on CVD.

Finally, even in analyses of changes in blood pressure and sodium, which showed the opposite positive effect, bias remains. The authors reasonably eliminated those receiving antihypertensive medication at baseline. However, they also eliminated those who began taking such medication during the follow-up, which effectively truncated the y-axis in their regression analysis. These nonignorable missing data are a familiar concept in the statistical literature, and several publications suggest methods to avoid such a bias.[3,4] Their analysis instead effectively reduced the slope of the regression line, biasing the effect to the null.

This analysis is susceptible to biases due to factors in the causal pathway, reverse causation, and nonignorable missing data. Proper analytic methods can control for the first and last of these. However, given the known effects of sodium among persons with hypertension, it is difficult to avoid the possibility of reverse causation, including among those with high normal blood pressure. The strongest evidence for etiologic effects comes from randomized trials. The Trials of Hypertension Prevention Follow-up Study,[5] which showed a reduction in CVD among those randomized to a sodium reduction intervention, is one such piece of evidence.

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To the Editor: The study by Dr Stolarz-Skrzypek and colleagues[1] showed that systolic blood pressure changes over time were associated with changes in sodium excretion but not with a higher risk of hypertension or CVD complications. Lower sodium excretion was associated with higher CVD mortality. In our view, the study has some limitations.

First, patients with established CVD at baseline were excluded. However, the authors did not report whether patients with other risk factors, such as family history of CVD,[2] high levels of low-density lipoprotein cholesterol, abdominal obesity, or inflammatory markers,[3] were also excluded.

Second, no food frequency questionnaire was administered to participants even though other dietary factors beyond sodium and potassium can play an important role in prevention of hypertension and high blood pressure.[4]

Third, no information about physical activity or use of statins, protective factors for CVD and mortality, was provided nor were they included as potential confounders in the multivariable analyses.

Fourth, the study population was relatively young and predominantly white and lacked information about sodium sensitivity, although it is known that among normotensive young adults with a family history of hypertension, higher sodium intake might contribute to the increased risk for hypertension.[5]

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Conflict of Interest Disclosures: Both authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

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In Reply: Many letters questioned our use of 24-hour urine collections. Volume and levels of potassium and creatinine were approximately 20% lower in the low-sodium than in the high-sodium tertile, whereas sodium was 60% lower. All measurements should have shown a similar gradient from the high to the low tertile if urine collections were incomplete. Administration of para-aminobenzoic acid to assess completeness, as suggested by Dr Aleksandrova and colleagues, could introduce error[1] and is impractical in population studies. As Drs Rebholz and He request, we classified participants by sodium-to-creatinine ratio, including those with incomplete urine collections. The adjusted hazard ratios (HRs) for CVD mortality in the low, medium, and high tertiles were 1.54, 1.04, and 0.63 ($P=.01$).

Dr Bochud and colleagues and Drs Labarthe and Briss note that a single 24-hour urine collection may not characterize the usual salt intake of an individual. However, it does reflect the mean of groups, as in our study or the Intersalt study.[2]

The letters raised many issues about study design, but additional analyses provide consistent results. Aleksandrova et al questioned whether differences between the 2 cohorts could have influenced the results. The HRs for cardiovascular mortality in the tertiles with low, medium, and high sodium excretion were 1.40, 0.79, and 1.27 in 2674 FLEMENGHO participants ($P=.14$), indicating that our findings cannot be explained by pooling participants from different countries. Additional adjustment for enrollment time did not alter the results.

Many were concerned about the young age or educational attainment of participants. The results were similar when only looking at the 487 participants 60 years or older (HRs for low, medium, and high tertiles: 1.52, 1.06, and 0.94; $P=.056$); or the 1210 participants with low educational attainment (1.51, 0.75, and 0.89, respectively; $P=.09$).

Dr Cook suggested that hypertensive patients at high risk of cardiovascular death might reduce their salt intake, leading to reverse causation. The HRs were 1.37, 0.81, and 0.90 in the low, medium, and high tertiles in 949 hypertensive patients ($P=.17$), with the trend consistent with that in the whole cohort. Cook was also concerned that we eliminated the presumed association between CVD mortality and sodium excretion by accounting for baseline blood pressure and antihypertensive treatment. The HRs in 3681 participants without such adjustment were 1.42, 0.98, and 1.02 in the low, medium, and high tertiles ($P=.03$). We also repeated our analysis of the blood pressure cohort including hypertensive patients who started receiving drug treatment during follow-up, extending the y-axis by adding 10/5 mm Hg^3 to follow-up blood pressure. In 1743 participants, the partial regression coefficients relating change in blood pressure to change in sodium excretion were 1.779 mm Hg per 100 mmoL systolic ($P<.001$) and 0.195 mm Hg per 100 mmoL diastolic ($P=.57$), confirming our results.

In response to criticism from Drs Oliveira de Abreu-Silva and Marcadenti that we did not account for risk factors other than blood pressure, we repeated the analysis with adjustment for antiplatelet drugs, lipid-lowering drugs, and energy expenditure in physical activity (HRs for low, medium, and high tertiles: 1.54, 1.04, and 0.96; $P=.02$). To address the suggestion from Dr Bochud and colleagues to diminish heterogeneity in end points, we excluded 4 deaths due to embolism or aortic aneurysm and found HRs of 1.50, 1.03, and 0.97 in the lowest to highest tertiles ($P=.03$).

Rebholz and He were concerned about the higher salt sensitivity of obese patients. Among 1736 overweight or obese participants, the HRs for cardiovascular mortality were 1.82, 0.76, and 0.72 in the low, medium, and high tertiles ($P=.02$). In contrast to their suggestion, our analysis does not raise an issue of multiple testing. Cardiovascular outcomes are correlated, so each test does not represent an independent opportunity for type I error.

Labarthe and Briss apply double standards to the evaluation of quality of salt studies. Sample size, number of events, and participation and drop-out rates in our study are similar to those in most previous reports. As highlighted by Cook, the strongest evidence comes from randomized clinical trials rather than observational studies. In the Trials of Hypertension Prevention,[4] participants collected up to 7 urine samples over 3 years, but the risk of a cardiovascular event across quartiles was unrelated to sodium excretion over time. A Cochrane Review[5] demonstrated that the relative risk of all-cause mortality associated with salt reduction was 0.90 (95% CI, 0.58-1.40) in normotensive participants and 0.96 (95% CI, 0.83-1.11) in hypertensive patients. Thus, currently available randomized clinical trials do not contradict our longer-term follow-up data.

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Conflict of Interest Disclosures: The author has completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none were reported.

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Information Disclosure and the Physician Payments Sunshine Act

To the Editor: Three key elements of a fair information-disclosure process are specificity and clarity about what information will be requested and posted, review by individuals of draft data about themselves, and a clear and timely appeal and correction process. In areas as diverse as personal income taxes and ethics disclosures for government officials, these principles are typically followed.

Unfortunately, in the development of Web sites disclosing financial relationships of physicians, physicians' rights have been ignored and remained unaddressed by Drs Carpenter and Joffe.[1] In an era in which personal information is posted publicly and, given Internet caches and search engines, is sometimes irrevocable, physicians should be entitled to some safeguards. They should be informed of disclosure and what will be included before posting, especially personal information such as addresses or other identifying information. This is important so physicians can choose ahead of time whether they want to participate in given activities.

Physicians should also be notified of the amounts of the posting before release to review for accuracy. Because this information is already being gathered, it should be made available to the physician. One option would be to make a password-protected version of the Web site for the physician to review before public launch. Finally, processes for correction should be clear and easy to find and have a time frame in which the disclosure can be evaluated and corrected.

In a review of pharmaceutical company Web sites identified by ProPublica as currently disclosing payments to physicians (Pfizer, Viiv, Eli Lilly, Johnson and Johnson [Centocor, Tibotec, Ortho-McNeil-Janssen], GlaxoSmithKline, Merck, AstraZeneca, Cephalon, and Allergan) performed in June 2011, none of these principles could be identified.[2] There was no specific posted information about correction processes or contacts for physicians. Only Novartis listed a mechanism to review disclosure data for error, which starts with a request in written form. It commits to a response within 30 days.

Public figures, such as celebrities and politicians, knowingly give up privacy in exchange for their public roles.[3] Physicians are not public figures and therefore should not be subject to invasions of privacy on any Web site containing disclosure information, including on the federal Web site proposed under the Physician Payments Sunshine Act, without some assurances. Since the posted information on such Web sites may be subject to media and public scrutiny, I believe the people whose reputations are being evaluated in a public forum should be protected by a fair and accurate process.

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Conflict of Interest Disclosures: The author has completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Dr Kimball reported serving on the Partners Health Care Committee on Conflicts of Interest; being an investigator or steering committee member for Pfizer, Merck, GlaxoSmithKline, and Centocor; and being a consultant for Novartis. She also reported having been a speaker for Johnson and Johnson, having received fellowship funding from Centocor, and previously having been a consultant for Lilly.

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In Reply: Dr Kimball's argument that physicians deserve some mechanism for correcting factual errors in disclosed information about their financial ties is a reasonable one. In the context of the Physician Payments Sunshine Act, such mechanisms should presumably involve the companies that are responsible for providing payment data to the Department of Health and Human Services. We take issue, however, with the assumption that requirements for disclosure of physicians' financial relationships imply a negative normative judgment about the legitimacy of those relationships. Disclosure mandates are not motivated by a desire to "out" physicians who have financial relationships with industry.