corrections depend on several stringent criteria being met should be recognised, and serious attempts should be made to test if these assumptions actually hold. Secondly, while it is important to consider the possible size of the association which could, if certain conditions hold, underlie the observed association, corrected estimates should not be reported as the main findings in abstracts and summaries of papers. Thirdly, equal attention should be given to applying such corrections to confounding factors as well as to main effects, and the fact that there may be missing confounders, factors measured only by proxies, or interactions between exposures should always be considered. Fourthly, sensitivity analyses should be performed, varying the assumptions that are made, and imprecision in the reliability measures should be incorporated in these. The added uncertainty introduced by correction should be reflected in the estimates of precision that are given. Finally, it should be remembered that improving study design by incorporating multiple measurements on all participants and relating outcomes to better measures of exposure will give more reliable estimates in most situations, even when this may lead to a reduction in sample size.⁵

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Commentary: Strength and importance of the relation of dietary salt to blood pressure

Alan R Dyer, Paul Elliott, Michael Marmot, Hugo Kesteloot, Rose Stamler, Jeremiah Stamler, for the Intersalt Steering and Editorial Committee

George Davey Smith and Andrew Phillips raise some issues concerning methods used in correcting for regression dilution in Intersalt but offer no judgment on the crucial issue of the strength and importance of the relation of dietary salt to blood pressure. To assess this matter properly, in the context of Intersalt findings, three judgments must be made: firstly, on the aetiological significance of the salt-blood pressure relation; secondly, on the probable underestimate of the size of this relation in Intersalt analyses of individuals; and thirdly, on the soundness of Intersalt's updated estimates "revisiting" the strength of this relation, both from its within population and its cross population analyses.¹ Davey Smith and Phillips deal not at all with the first and second of these points, and only partially and inconsistently with the third. Much of their commentary deals with generalities, not with specifics of the salt-blood pressure relation and Intersalt results. As a consequence, their commentary neither sheds light on the substantive matter nor contributes positively to public policy.

Aetiological significance of the dietary salt-blood pressure relation

Davey Smith and Phillips state that corrections for regression dilution and other biases "could as well be applied to spurious associations as to causal ones Judgment has to be applied to decide if an association is causal." They make no such judgment on the

salt-blood pressure relation, the subject of their commentary. In fact, the totality of the evidence—the only sound basis for judgment on this matter—supports the conclusion that this association is causal. The significant independent findings on the sodium-blood pressure relation in Intersalt's within population and cross population analyses¹ are components of that total evidence. Independent expert groups, national and international, have repeatedly concluded that the extensive, concordant, strong data from all disciplines—clinical investigation, randomised controlled trials, animal experimentation, epidemiological research (within and cross population), anthropology—constitute a firm foundation for the consensus assessment that habitual high intake of dietary salt is one important cause of the adverse blood pressure levels generally prevailing in the adult population.¹⁻⁶ Therefore, the example of non-aetiological associations such as yellowed fingers and lung cancer, cited by Davey Smith and Phillips, is irrelevant. Rather, the issue is the size of the aetiological association of the salt-blood pressure relation. This entails making corrections for biases, in so far as possible, as Intersalt has done.¹

Why Intersalt has probably underestimated the size of the salt-blood pressure relation in individuals

For seven reasons (see box) previously noted by Intersalt,^{7,8} its original uncorrected regression coefficients

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on the size of the sodium-blood pressure relation in individuals are probably biased underestimates. Updated Intersalt corrections for biases, yielding estimates of systolic/diastolic blood pressure lower by 3/0 mm Hg to 6/3 mm Hg with 100 mmol/day lower sodium intake,¹ relate only to points (1) to (3) and in a limited way to point (4) in the box but do not correct for the other sources of bias. Davey Smith and Phillips pay attention to reasons (1), (2), and (4) but do not address the others.

Seven reasons why Intersalt has probably underestimated the size of the sodium-blood pressure relation in individuals

- (1) Using a single 24 hour urine collection to quantify habitual sodium intake is a weak method because of large variability from day to day in individuals and would result in misclassification of individuals, with resultant biasing of true associations towards zero: the regression dilution bias problem
- (2) Multiple regression analyses of blood pressure on sodium, a variable measured imprecisely in Intersalt, would be affected by inclusion of confounders measured with very high reliability—age, sex, body mass index
- (3) Multiple regression analyses of blood pressure on sodium controlled for body mass index may be affected by overadjustment, because part of the blood pressure-body mass association may be due to the positive correlation of body mass with sodium
- (4) Urine collection by some participants may be incomplete, varying in degree
- (5) Some participants may have previously reduced salt intake to various degrees, biased toward occurrence in people with higher blood pressures
- (6) Antihypertensive drugs affect blood pressure
- (7) Data on the long term influences of salt on blood pressure are lacking as Intersalt is a cross sectional study

As to these other issues, point (5), prior salt reduction, and point (6), effect of antihypertensive drugs, relate to the probability that biased low estimates were obtained both for the independent variable (sodium) and the dependent variable (blood pressure). This could lead to a diminution, or even reversal, of a true causal effect.⁹⁻¹¹ In regard to the cross sectional nature of the study (point 7), relevant longitudinal data are available elsewhere. In the Rotterdam trial of salt intake and blood pressure in infancy, the group randomised to higher salt intake from birth had significantly higher systolic blood pressure (by about 2 mm Hg) at six months than the group randomised to lower salt intake.¹² At 15 year follow up, the group fed higher salt during the first six months of life had systolic blood pressure (adjusted for confounders) higher by almost 4 mm Hg, even though there had been no intervention since infancy.¹³ These data indicate the possibility of pathophysiological conditioning by high salt intake in infancy, with adverse effects on blood pressure for years. It is also relevant to note the recent report from animal research on the quantitative effect longitudinally of adding salt to the usual low sodium diet of chimpanzees, closest genetic relative of the human species: with 86 mmol/day sodium (5 g salt) added, systolic blood pressure was 12 mm Hg higher on average, and with 259 mmol/day sodium (15 g salt) added, systolic blood pressure was

higher by 26 mm Hg; when salt was no longer added to the diet, the original low-normal blood pressure was restored.¹⁴

Intersalt correction for regression dilution bias

Much of Davey Smith and Phillips' commentary deals with correction for regression dilution bias, which results from the well known large variability within individuals in sodium intake. Intersalt has developed straightforward corrections, in line with the specific provision in the study design to address this problem. In the first major report of findings, a then standard method was used to make a univariate correction for reliability of sodium excretion, estimated as 0.460 from repeat 24 hour urine collections (done by a random 8% of participants on average three weeks later); that is, sodium-blood pressure regression coefficients were divided by 0.460 to correct for the regression dilution bias; it was noted there that this "adjustment may be incomplete."¹⁵ Accordingly, Intersalt then used more sophisticated methods to take account of age and sex and correlations of sodium with confounders such as body mass index and potassium. These yielded revised estimates of reliability of 24 hour sodium excretion and of sodium-blood pressure coefficients corrected for regression dilution bias.^{1 8 16 17}

While Davey Smith and Phillips question the appropriateness of corrections for regression dilution bias, they assert that they "are not intending to imply that the basic Intersalt findings are erroneous." They seem to acknowledge that estimates of the reliability of measurements of sodium, used in making such corrections, are improved when age and sex are taken into account, as was done with Intersalt's updated estimates.

In correcting for regression dilution bias, Davey Smith and Phillips state that sensitivity analyses should be performed. Intersalt did, in fact, carry out such analyses: in our 1994 papers, we examined effects on corrected regression coefficients of different estimates of reliability, the inclusion of measurement error in confounding variables, and the use of bootstrap techniques to obtain estimates of the standard errors of corrected coefficients.^{16 17}

Davey Smith and Phillips also state hypothetically that if day to day "fluctuations in urinary sodium and blood pressure tended to coincide, then the [sodium-blood pressure] association would not have been underestimated to the degree that the correction methods assume." To the best of our knowledge, there are no data showing physiological day to day parallel fluctuations of sodium and blood pressure. Indeed, data from intervention studies show that in trials shorter than four weeks, the response of blood pressure to sodium reduction is small.¹⁸ Studies cited by Davey Smith and Phillips concern repeated measurements seven weeks apart or longer.

In regard to estimates of the size of the sodium-blood pressure relation in individuals, it is relevant to note results from Intersalt cross population analyses, indicative of effects of long term exposure to sodium. For example, with the average sodium excretion in the population sample lower by 100 mmol/day, the average difference in blood pressure (age 55 compared with age 25) was less by 10-11 mm Hg systolic and 6 mm Hg diastolic.¹

Effects of body size

Davey Smith and Phillips give no attention to the possibility that inclusion of body mass index in multiple linear regression analyses in Intersalt produced underestimation of sodium-blood pressure relations (see points (2) and (3) in box).^{1 8} Instead they discuss four issues concerning body size: the use of body mass index rather than height and weight considered

separately; the use of body mass index rather than some other measure of obesity; values used for reliability correction for body mass index; and possible interactions. We deal with each of these in turn.

USE OF BODY MASS INDEX RATHER THAN HEIGHT AND WEIGHT SEPARATELY

Davey-Smith and Phillips cite a paper to imply that use of height and weight rather than body mass index would have resulted in "considerably more" attenuation of the sodium-blood pressure relation. This citation is inappropriate, since the reference is to a study in children for whom height has a much greater correlation with blood pressure than in adults.^{19 20} In fact, Intersalt data show that regression coefficients for the sodium-blood pressure relation were virtually identical with body mass index or height-weight included as confounders.

USE OF BODY MASS INDEX RATHER THAN SOME OTHER MEASURE OF OBESITY

Davey Smith and Phillips also criticise the use of body mass index as a measure for the aspect of body size and obesity that is related to blood pressure. Data are available indicating that body mass index is a valuable index of body fatness in adults, at least as good as other variables cited by Davey Smith and Phillips. The inter-correlation of all these variables is high; body mass index is in general measured more reliably than other variables; and the body mass index seems to be as strongly related to blood pressure as other measures of obesity.²¹⁻²⁵

RELIABILITY CORRECTION FOR BODY MASS INDEX

Since height and weight were not measured twice in Intersalt, direct calculation of the reliability of body mass index was not possible. Davey Smith and Phillips assert that the value of 0.98 for body mass index reliability used by Intersalt in its sensitivity analyses "seems unlikely." In fact, data from several population studies showing very high correlations with repeat measurements 4, 8, 12, or 24 months apart support use of 0.98 with measurements a few weeks apart.^{24 26 27}

POSSIBLE INTERACTIONS

Davey Smith and Phillips further state that in the presence of interactions between sodium and body mass index in relation to blood pressure "multivariate correction methods could produce very unreliable results," again referring inappropriately to a study in children. They also state that Intersalt data suggest a greater association between sodium excretion and blood pressure among individuals with higher than lower body mass index. Intersalt carried out two types of analyses on this question.²⁸ One divided Intersalt participants into two groups (mean body mass index 21.7 and 27.2 based on a cut point of 24.1). The other involved individuals from 26 samples with lower average body mass index (mean 22.9) and 26 samples with higher body mass index (mean 25.7). In the first of these analyses, a non-significantly larger coefficient for sodium-systolic blood pressure was found in the individuals with higher body mass; in the second, the coefficient was non-significantly larger for people from samples with lower body mass index. These findings do not support the hypothesis of a sodium-body mass index interaction in relation to blood pressure in Intersalt, nor the related hypothesis that Intersalt's multivariate corrections for regression dilution bias are flawed because of such interactions. Nor do the findings lend support to the erroneous notion that dietary salt has little or no adverse influence on blood pressure in non-obese people.

Estimates based on sodium difference of 100 mmol per day

Davey Smith and Phillips implicitly fault Intersalt's use of 100 mmol/day difference in 24 hour sodium excretion (intake) to estimate size of the sodium-blood pressure relation and consequent impact on major adult cardiovascular diseases. Use of the 100 mmol/day difference has important scientific foundations: mean sodium intake of the three British Intersalt samples—around 150 mmol/day—is about 15–19 times the basal sodium requirement for growing and adult humans (8–10 mmol/day; possibly less).² This reflects the fact that during most of human evolution, diet was low in salt, hence the human species is adapted to low salt, not high salt.

Salting of food, for preservation, developed only about 6000–8000 years ago with the invention of agriculture. Modern technology makes this use of salt largely unnecessary. Nevertheless, salt is still a major food additive in food processing, accounting for about 75% of total salt intake.^{2 8} The focus on 100 mmol less sodium per day is to help the human species move toward a physiological condition. In this regard, expert groups have repeatedly recommended that the high salt intake by the population be lowered to 6 g/day or less—that is, about 100 mmol/day or less—and as one report noted, "a greater reduction in salt intake (i.e. to 4.5 g or less [about 78 mmol/day sodium or less]) would probably confer greater health benefits...but...6 g [is] an initial goal that can be achieved more readily."²² Reducing sodium intake by 100 mmol/day, over time, is scientifically justifiable.

Impact of lower salt intake on cardiovascular disease

In discussing possible impacts on mortality of reduction in sodium intakes, Davey Smith and Phillips ignore the crucial issue of which Intersalt coefficients give the most sound estimates of the size of the sodium-blood pressure relation. In our judgment, this estimate for individuals is, with 100 mmol/day lower habitual sodium excretion (intake), systolic blood pressure lower by between 3 and 6 mm Hg. This translates into death rates from all cardiovascular diseases lower by about 7% to 14%.⁸ These substantial estimates of potential for reducing mortality may—it is appropriate to re-emphasise—be underestimates. For example, results for the cross population analyses of difference in systolic blood pressure with age translate into death rates in middle age estimated to be lower by about 21% to 23% from all cardiovascular diseases. A 1991 review on salt and blood pressure concluded with this evaluation of population-wide reduction in sodium intake by 100 mmol/day: "Few measures in preventive medicine are as simple and economical and yet can achieve so much."¹⁸

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The Ljubljana Charter on Reforming Health Care

See editorial by Richards

Rising inequity in the quality and distribution of health care have led WHO's European member states to draw up a charter on reforming health care at their meeting in Ljubljana last week. We reproduce the text of the final draft (18 June)

PREAMBLE

1. The purpose of this Charter is to articulate a set of principles which are an integral part of current health care systems or which could improve health care systems in all Member States of the World Health Organisation in the European Region. These principles emerge from the experience of countries implementing health care reforms and from the European Health for All targets, especially those related to health care systems.

2. This Charter addresses health care reforms in the specific context of Europe and is centred on the principle that health care should first and foremost lead to better health and quality of life for people.

3. The improvements in the health status of the population are an indicator of development in the society. Health services are important, but they are not the only sector influencing peoples' wellbeing; other sectors also have a contribution to make and responsibility to bear in health, and intersectorality must therefore be an essential feature of health care reform.

4. In cognizance of this, we the Ministers of Health or their representatives from the European Member States of WHO (participants in the Ljubljana Conference) hereby pledge ourselves to promote the following principles and call upon all citizens and urge all governments, institutions and communities to join us in this endeavour. We further request the WHO Regional Office for Europe to take the necessary action to support Member States in giving effect to these principles.

FUNDAMENTAL PRINCIPLES

5. Within the European context, health care systems need to be:

5.1 Driven by values

Health care reforms must be governed by principles of human dignity, equity, solidarity and professional ethics.

5.2 Targeted on health

Any major health care reform should relate to clear targets for health gain. The protection and promotion of health must be a prime concern of all society.

5.3 Centred on people

Health care reforms must address citizens' needs, taking into account their expectations about health and health care. They should ensure that the citizen's voice and choice decisively influence the way in which health services are designed and operate. Citizens must also share responsibility for their own health.

5.4 Focused on quality

Any health care reform must have as its aim—and include a clear strategy for—continuous improvement in the quality of the health care delivered, including its cost-effectiveness.

5.5 Based on sound financing

The financing of health care systems should enable such care to be delivered to all citizens in a sustainable way. This entails universal coverage and equitable

Fig 1—Meeting in Ljubljana, the 49 member states of WHO's European region have drawn up a charter which underlines the fundamental principle that health care should be to improve people's health, not contain costs