Debate   *Drinking water safety revisited*

Drinking water and endemic gastrointestinal illness

We consider that the strength of the evidence linking drinking water to endemic gastrointestinal illness in developed nations has been overstated in the recent paper in this journal by Schwartz *et al*[1] and the accompanying editorial.[2]

As noted in the editorial, there have been a number of documented waterborne outbreaks in countries with good water treatment practices. In such outbreaks the association between drinking water and disease has been supported by a substantial amount of evidence including very large effect size, evidence of water treatment failures, robust epidemiological study designs, or identification of the responsible microorganisms in both water and stool specimens. In contrast, the existing body of evidence linking drinking water to endemic gastrointestinal illness is not of an equivalent standard.

The paper by Schwartz *et al* presents a complex analysis of the relation between variations in drinking water turbidity and hospital admissions for gastrointestinal illness among elderly residents of Philadelphia. The paper is similar in nature to a previous publication by two of the authors,[3] which attracted considerable controversy and was the subject of a detailed peer review by the US EPA that criticised many aspects of the analysis (EPA internal memorandum dated February 19, 1998 from W Diamond and J Wiltse to R Levin on EPA Peer Review of Article Linking Finished Water Turbidity with Gastrointestinal Disease in Philadelphia).

The authors responded to some of the criticisms raised in the EPA review,[4] however they failed to resolve the fundamental problem with the quality of the turbidity dataset upon which their analyses are based. According to information supplied to the EPA by the Philadelphia Water Department, 75% of the turbidity readings were below the lower calibration limit of the turbidity meters (0.20 NTU). We believe it is not acceptable to include these data in the analysis unless suitable statistical techniques are used to account for the lack of reliability in readings below the quantitation limit.

Moreover, the inherent weaknesses of such ecological studies such as their inability to assess individual exposures or control for bias or confounding, prevent their use in hypothesis testing. To provide substantive evidence of a causal association a consistent body of evidence from epidemiological studies of much stronger design is required. Indeed, when the magnitude of the effect is as small as that proposed for endemic waterborne gastroenteritis (around 15%), many would argue that only blinded randomised controlled trials are sufficiently rigorous to answer the question.[5]

Even if the problems with data quality and study design of the study by Schwartz *et al* are ignored, the hypothesis that gastrointestinal disease rates show correlation with minor variations in water turbidity is not biologically plausible. This theory implicitly assumes that physical removal is the major barrier to pathogens in the water supply. However, the Philadelphia water supply is also chlorinated to levels that provide at least 35-log inactivation of viruses and bacteria, and also provide a degree of inactivation for some protozoal pathogens (for example, *Giardia*). Thus one needs to postulate that most gastrointestinal illness is caused by chlorine resistant organisms (for example, *Cryptosporidium*) and that these rise significantly in number when minor variations in turbidity occur. However, this is not supported by the clinical experience that such pathogens are relatively uncommon causes of community gastroenteritis.

The two intervention trials carried out in Canada[6][7] represented a significant improvement over ecological studies but were still subject to a number of methodological concerns. In both trials the participants were not blinded to the interventions and thus bias in the reporting of illness may have influenced the results. In addition, interpretation of the second study was hampered by a very high drop out rate (50%) in one of the intervention groups.

While a number of observations have suggested that drinking water meeting accepted water quality standards may be contributing to endemic gastrointestinal illness, the strength of the epidemiological evidence is not convincing. To overcome the design problems associated with previous studies, randomised controlled trials investigating waterborne disease are being undertaken in a number of countries.[8][9] The outcomes of these studies will provide the strongest possible quality of evidence on the existence of endemic waterborne disease and its magnitude in different water supply systems.

This issue is an important public health concern, but it must be scrutinised critically and judged on the best possible quality of evidence. Otherwise we risk committing large expenditures on changes in water treatment technology that may be unnecessary and produce no tangible benefit to public health.

MARTHA I SINCLAIR
CHRISTOPHER K FAIRLEY
Cooperative Research Centre for Water Quality and Treatment,
Department of Epidemiology and Preventive Medicine, Monash
University Medical School, Alfred Hospital, Prahran VIC 3181, Australia

Correspondence to: Dr Sinclair (martha.sinclair@med.monash.edu.au)

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Water and health: precaution must be guided for the health of the public

We agree with Martha Sinclair and Christopher Fairley[1] that the existing evidence linking drinking water to gastrointestinal illness in developed countries is not comparable to that of waterborne outbreaks. We also agree with most of their comments about potential biases of time series studies on water turbidity and need for better designs.[2] However, we disagree in some other of their comments.

Gastroenteritis caused by chlorine resistant organisms is not so uncommon. In the United States, the estimated incidence of waterborne infection by *Cryptosporidium parvum* is the second highest pathogen identified, after viruses.[3] In England, cryptosporidium infection was identified in 2% of the patients with presumed infective diarrhoea,[4] whereas infectious intestinal disease occurs in one in five people each year in England, but only a small proportion of cases is recorded by national laboratory surveillance.[5]

Secondly, their major criticism to the papers by Schwartz *et al*[6 7] is about the rather poor correlation between water turbidity and levels of infective agents. If there is a correlation, although poor, the results of this information bias, if any, could be the dilution of the association, instead of a spurious association.

In our opinion, the papers by Schwartz appear as a warning of a new potential public health problem, and although we agree with the final comment of Sinclair and Fairley that "...we risk commiting large expenditures on changes in water treatment technology that may be unnecessary and produce no tangible benefit to public health", these results suggest that we risk to produce some health damage if we do not improve the technology. Results from well conducted individual studies besides data from adequate surveillance systems will provide a great amount of evidence, but, quite frequently, decisions in public health must be taken without complete knowledge. The ball is in the court of public health decision makers who have to manage weak evidences of a potential association with a precautionary view.

FERRAN BALLESTER
Valencian School of Health Studies (EVES), Valencia, Spain

JORDI SUNYER
Institut Municipal d'Investigació Mèdica (IMIM), Barcelona, Spain

Correspondence to: Dr Ballester (ballester_fer@gva.es)

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Drs Sinclair and Fairley raise two issues about our paper[1] in their article, which they allege we did not resolve in our response[2] to prior critiques, including one by EPA (unpublished data). They raise the same critiques about the accompanying editorial.[3] Firstly, they raise the hackneyed bugaboos about ecological studies. These are issues in cross sectional comparisons of different populations with different exposures. But in our studies, the population of Philadelphia is compared not with another population, but with itself at different points of time. Hence, the population is its own control, as described in Schwartz and Levin.[2]

Their second argument is that there is too much measurement error in our exposure index, and therefore our reported association cannot be real. But there is an observed association between hospital admissions for gastrointestinal illness and daily turbidity measures (indeed measured with error) in Philadelphia. There are then three possibilities: the association is with the part of turbidity variation that is random measurement error, the association is with the part of turbidity variation that is not measurement error, or that the association is attributable to confounding.

The first possibility seems implausible to us, which leaves either confounding or a real association. The authors' argument that the variation in turbidity is all measurement error is inconsistent with the association being attributable to confounding. A confounder, by definition, must be correlated with the exposure. How could that be true if the exposure is essentially random noise? Even in multivariate measurement error models, if the measurement error is very large compared with the correlation with confounders, the bias in the estimate effect is invariably toward zero. Their critique is simply inconsistent with the data.

However, the authors' contention that the measurement error in our study was large is wrong. Firstly, the article they cite about the uncertainty in turbidity measurements refers to measurement error in single measurements. As we pointed out previously,[2] our analysis does not use individual measurements. We use the mean of 24 daily measurements. Because random noise cancels when averaged, the measurement error in these daily means is much lower than in the individual measurements. Indeed, if day to day variations in the measured turbidity in Philadelphia were primarily measurement error, we would expect little or no correlation between today's daily mean and yesterday's daily mean. In fact, the correlation between these two measures is 0.85, which proves that most of the variation cannot be random noise.

They have also misinterpreted the data on measurement error in individual measurements. Hart *et al*,[4] studying measurement error in individual low level turbidity measurements, showed that the principal factors relating to measurement variability were the type of instrument used and the calibration standard. That is, much of the variations among individual measurements were attributable to variations in equipment or calibration. Variation using the same equipment and standard, as was the case for the Philadelphia data we used, was substantially less.

Moreover, low level turbidity measurements are routinely presented without qualification in water engineering,[5 6] epidemiology (for example, Fox and Lytle[7](incidentally, an official EPA study)), and microbiology.[8] Indeed, many of these studies were published in the same journal that published the Hart article.

Finally, EPA's final Interim Enhanced Surface Water Treatment Rules (EPA 1998b) requires that 95% of all turbidity measurements be below 0.3 NTU, and justifies that such levels are reasonable by defending the precision

and accuracy of turbidity measurements in this range, based on performance studies. EPA cannot simultaneously argue that those studies justify their standard and invalidate our study.

In short, if the arguments about measurement error were valid, they would suggest that the true effects of exposure were larger than we reported (because of the induced downward bias). The real issue is not how well turbidity is measured, but how well it serves as a surrogate for exposure to microbial contamination. We discussed this issue extensively,[2] and concluded, again, that the probable result was a downward bias. We believe there is little news, or relevance, in our Australian colleagues' critique.

JOEL SCHWARTZ
RONNIE LEVIN
Department of Environmental Health, Harvard School of Publich Health, 665 Huntington Avenue, Boston, MA 02115, USA

Correspondence to: Dr Schwartz

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