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

We agree with Martha Sinclair and Christopher Fairley 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. 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. In England, cryptosporidium infection was identified in 2% of the patients with presumed infective diarrhoea, 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.

Secondly, their major criticism to the papers by Schwartz et al 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 committing 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.

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Drs Sinclair and Fairley raise two issues about our paper in their article, which they allege we did not resolve in our response to prior critiques, including one by EPA (unpublished data). They raise the same critiques about the accompanying editorial. 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.

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, 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 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, epidemiology (for example, Fox and Lytle (incidentally, an official EPA study)), and microbiology. 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, and concluded, again, that the probable result was a downward bias. We believe there is little news, or relevance, in our Australian colleagues’ critique.

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