EPA'S STAGE 2 DISINFECTION BYPRODUCTS RULES (DBPR) AND NORTHERN KENTUCKY WATER: AN ECONOMIC AND SCIENTIFIC REVIEW

Hugh Henry
Northern Kentucky University

Follow this and additional works at: https://scholarworks.umass.edu/dose_response

Recommended Citation
Implementation of EPA’s Stage 2 Disinfection Byproducts Rules (DBPR) in Northern Kentucky will cause a water rate increase of over 25%. Hence a review was undertaken, considering both economics and science in the context of President Obama’s 2009 scientific integrity directive. The rules purport to avoid up to 0.49% of new bladder cancers by reducing the levels of DBPs in drinking water – a benefit so small that failure to implement will not cause unreasonable risk to health (URTH). It suggests at most one Northern Kentucky death avoided over 17 years for a cost of $136,000,000 ($1700 per household). Even this small benefit is probably overstated. EPA finds no “causal link” between DBPs and bladder cancer, and EPA acknowledges problems with the epidemiological data used in their calculation: the data appear contradictory and inconsistent, may be skewed toward “positive” results, and suggest different cancer sites than animal studies. Two similar international agencies disagree with EPA’s conclusions. The science is based on the Linear No Threshold (LNT) dose response model for DBPs, but this may not be the correct model. 83% of EPA’s epidemiological data show a statistical possibility that low levels of DBPs might be beneficial or have no effect.

Key words: Disinfection byproducts (DBP), Trihalomethane (THM), EPA Water Regulations, Chlorinated Drinking water, LNT model, Hormesis

1. INTRODUCTION

Chlorine and other chemical disinfectants have been widely used by public water systems as a principal barrier to microbial contaminants in drinking water. Disinfection byproducts (DBPs) are formed when certain disinfectants interact with organic and inorganic materials in source waters.

The EPA Stage 2 DBPR (USEPA 2012) seeks to reduce the levels of nine specific DBPs in chlorinated drinking water: four Trihalomethanes (THMs) and five Haloacetic Acids (HAAs). The four THMs are Chloroform (CHCl₃), Bromodichloromethane (CHBrCl₂, aka BDCM), Dibromochloromethane (CHBr₂Cl, aka DBCM), and Bromoform (CHBr₃). The HAAs are Monochloroacetic acid (ClCH₂CO₂H), Dichloroacetic acid (Cl₂CHCO₂H), Bromoacetic acid (BrCH₂CO₂H), and Bromoacetamide (BrCH₂CONH₂), among others. The Rules also address byproducts containing both halogens and nitrogen, such as Nitrogenous Halogenated Acids (NHAs) and Trihaloamines (THAs), and low-levels of DBPs in non-chlorinated waters. The Rules require regular monitoring and reporting of DBPs in water supplies and the development of management plans for those exceeding the allowable levels.

Address correspondence to Hugh Henry, Ph.D., Department of Physics, Northern Kentucky University, Nunn Drive, Highland Heights, KY 41099. Phone: 859.363.0686; Fax: 859.363.1090; henryh1@nku.edu or hugh.henry@insightbb.com

Conflict of interest notification: The author is a customer of Northern Kentucky Water District and implementation of Stage 2 DBPR will cost him personally about $100/year. However, this point is moot, because NKWD is already implementing Stage 2 Disinfection Byproducts Rules and rate increases are going into effect. The author has no other financial interest.
Dichloroacetic acid (CHCl₂CO₂H, aka DCAA), Trichloroacetic acid (CCl₃CO₂H), Bromoacetic acid (BrCH₂CO₂H), and Dibromoacetic acid (CHBr₂CO₂H).

When EPA’s Stage 2 DBPR is fully implemented by Northern Kentucky Water District (NKWD),¹ it is estimated to cost more than 100 times the average amount estimated by EPA. President Obama has called for elimination of government regulations which are a deterrent to economic recovery, so the unexpectedly high cost to the consumer of Stage 2 DBPR make it a candidate for review and elimination under EPA’s program for Retrospective Reviews of Existing Regulations (USEPA 2011).

Furthermore, in a Memorandum of March 9, 2009, on the subject of “Scientific Integrity,” President Obama charged every federal agency “to ensure the integrity of scientific and technological information and processes on which the agency relies in its decisionmaking” (Obama 2009). There are reasons to question whether Stage 2 DBPR is consistent with this directive.

For these reasons, a review of the economic and scientific efficacy of EPA Stage 2 DBPR was undertaken, including EPA’s “Economic Analysis for the Final Stage 2 Disinfectants Byproducts Rule, December 2005” (USEPA 2005), EPA document 815-R-05-010; EPA’s “National Primary Drinking Water Regulations: Stage 2 Disinfectants and Disinfection Byproducts Rule, Final Rule,” (USEPA 2006) published in the Federal Register Volume 71, Issue 2 (January 4, 2006); and other government documents and journal articles. The objective was to review the science underlying EPA’s Stage 2 DBPR, and consider it in the context of President Obama’s directive and of cost vs benefit.

2. COST VS BENEFIT FOR EPA STAGE 2 DBPR

EPA Significantly Underestimated the Cost

EPA justified Stage 2 DBPR based on an estimated total U. S. cost of $77 million annually (range $55-101 million), with an average household cost of less than $1.00 per year (USEPA 2006).

By contrast, bids given to Northern Kentucky Water District (NKWD) show that implementation will cost the average NKWD household an additional $100/year – 100 times as much. This is based on NKWD estimates that the total cost of the Stage 2 rules will be $8 million/year (Harrison 2011), spread across about 80,000 households with approximately 300,000 persons.

¹This rule is required to be implemented for large community water systems (CWSs) such as Northern Kentucky Water District (NKWD) by April 1, 2012. After NKWD had partially implemented this rule, it received a two-year extension from the Commonwealth of Kentucky for completion. A request for a full variance or waiver was denied.
NKWD is the largest community water system (CWS) in Kentucky, and anecdotal evidence suggests people served by smaller CWSs in the state will experience even larger rate increases in 2013 (Lovant 2011).2

Is the Cost Increase Affordable?

The weighted average median household income (MHI) for Kenton and Campbell Counties is $56,500 (Tri-Ed 2012). The average household currently pays 0.67% of MHI for water ($380/yr, Lovan 2012) – consistent with the US average (USEPA 2005). With Stage 2 DBPR, this will increase by 26% to 0.85% of MHI ($480/yr, Lovan 2012) – 21% higher than average. Such a large percentage increase raises the question of affordability – especially for the large number of low income people who fall significantly below average MHI.

EPA claims this is not a problem. Based on their “National-Level Affordability” report (USEPA 1998b), EPA estimates US households can afford to spend 3.6 times more for water (2.5% of MHI). However, this estimate is based on the questionable criteria of purchasing bottled water for direct ingestion (2 liters/person/day) and using tap water for everything else. U. S. water costs at the time of the report were midrange with other developed countries, but 2.5% of MHI would make U. S. water costs almost twice as much as the most expensive country surveyed (USEPA 1998b). For NKWD households, 2.5% of MHI represents about $1412/year: an increase of over $1000/year. “EPA is currently re-evaluating its national-level affordability criteria” (USEPA 2006).

EPA offers Minimal or No Benefit for this Large Cost

EPA’s Economic Analysis estimates that for the first 25 years following implementation, the benefits of the Stage 2 DBPR will be avoidance of about 279 of 56,506 new bladder cases nationwide every year (USEPA 2005). This represents 0.49% of the total bladder cancer incidence.

However, EPA states in the Final Rule that this estimate is an upper limit maximum (USEPA 2006):

“EPA considers these estimates to be an upper bound on the annual reduction in bladder cancer cases due to the rule.”

2CWSs serving fewer than 100,000 persons have a delayed implementation schedule for Stage 2 DBPR (USEPA 2012).
According to National Cancer Institute data (NCI 2012), the population and bladder cancer (BlCa) incidence in the NKWD area is:

<table>
<thead>
<tr>
<th>County</th>
<th>2009 Population</th>
<th>BlCa Incidence*</th>
<th>Annual BlCa Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Campbell</td>
<td>88,423</td>
<td>16.9</td>
<td>14.9</td>
</tr>
<tr>
<td>Kenton</td>
<td>158,729</td>
<td>21.8</td>
<td>34.6</td>
</tr>
<tr>
<td>Other Areas (est)</td>
<td>52,848</td>
<td>22.2 (est)</td>
<td>11.7</td>
</tr>
<tr>
<td>Total (est)</td>
<td>300,000</td>
<td></td>
<td>61.2</td>
</tr>
</tbody>
</table>

Bladder Cancer Cases Avoided by EPA Stage 2 DBRP:

0.49% of 61.2 annual bladder cancer cases 0.30

*Cancer incidence as defined as annual cases per 100,000 of population

This represents approximately three bladder cancer cases avoided every ten years. The American Cancer Society calculates bladder cancer of “all stages” is 80% curable, based on 5-year survival rates (ACS 2010). Hence the maximum benefit estimated by EPA is to avoid four curable bladder cancer cases and one bladder cancer death every 17 years – for which 80,000 NKWD households are asked to pay $136,000,000, or $1700 per household. Based on US averages, the cost to treat five bladder cancer cases ranges $500-900,000 (Botteman 2003); and whereas one must be sympathetic to the possibility of five cancer patients and their families, one must also be cognizant of the costs involved.

Such high cost for a minutely small benefit shows how the law of diminishing marginal utility applies to removing contaminants from water: the last unit of contaminant is much more difficult and costly to remove than the first unit. Furthermore, since EPA’s estimated benefit is an upper limit; the $136,000,000 expense may not avoid even one bladder cancer case.

And the purported health benefit is too small to measure; no one will ever know if it was achieved. The statistical uncertainty for bladder cancer incidence in Campbell and Kenton Counties (with a 95% confidence level) is greater than ± 20% and ± 15%, respectively; for Kentucky as a whole, the uncertainty is ± 2.7% (NCI 2012) – more than 5 times the maximum estimated bladder cancer avoidance due to Stage 2 DBPR of 0.49%.

Unreasonable Risk to Health (URTH) as a Criteria

EPA’s purported maximum benefit from Stage 2 DBPR in Northern Kentucky (0.3 bladder cancer cases avoided per year for a 300,000 population) represents a reduced cancer risk of only 10^-6. Since Unreasonable Risk to Health (URTH) is a criterion for a waiver or exemption from EPA rules, and since Stage 2 DBPR has such a high cost, it seems reasonable
to ask: will failure to implement Stage 2 DBPR cause URTH? EPA’s criteria suggest the answer is “No.”

Under EPA’s “criteria from 1979, an URTH level was generally defined as any concentration of a contaminant that was greater than two times [Maximum Contaminant Level] MCL” (USEPA 1992). By this criterion, the current state of NKWD poses no URTH.

EPA’s most recent criteria for unreasonable risk to health make a recommendation for “Category I nonthreshold contaminants” for which there is “strong evidence of carcinogenicity from drinking water” (USEPA 1992):

“Where the MCL is set at a concentration less than the $10^{-4}$ cancer risk level, the $10^{-4}$ cancer risk may be used as the short-term acceptable risk level for URTH.”

Since disinfection byproducts (DBPs) are less toxic than Category I (as discussed below), that criterion is considered more stringent than required for DBPs – so if it is met, there should be no question of unreasonable risk to health.

And that is indeed the case. According to NCI data, total bladder cancer incidence in the NKWD counties ranges $1.7-2.2 \times 10^{-4}$ (NCI 2012). Most bladder cancer is caused by smoking, diabetes, parasitic infections, and chemicals other than DBPs. However, using a 1993 estimate “that chlorination accounts for 9% of annual U. S. bladder cancer cases” (USEPA SAB 1993) as an upper limit relative source contribution (RSC), the chlorinated drinking water contribution is $2 \times 10^{-5}$, and hence the MCL is below EPA’s acceptable risk level of $10^{-4}$ by at least a factor of 5.

The Safe Drinking Water Act (SDWA) directs the EPA to take cost into consideration in setting permissible contaminant levels (USEPA 2005). In the case of NKWD, the increased cost to subscribers of over 25% far exceeds EPA’s estimated health benefits: $10^6$ decreased bladder cancer risk is 100 times below the acceptable risk level for URTH for contaminants more toxic than DBPs.

Moreover, data uncertainty is another valid reason for determining no URTH (Strawson et al. 2003). EPA candidly acknowledges that their estimate of a $10^6$ benefit is an upper limit and that the benefits “could be zero due to uncertainties in the scientific evidence” (USEPA 2005).

Therefore, although EPA considers $10^{-4}$ only an acceptable short-term risk level (seven years or less), something that is at least 100 times lower might be considered an acceptable risk for a longer term – especially if there is significant uncertainty about the data and if the cost is extraordinarily high.

Thus it seems reasonable to suggest that failure to implement Stage 2 DBPR for NKWD poses no long term unreasonable risk to health.
EPA Admits: No Cancers May be Avoided

EPA’s Economic Analysis acknowledges that no cancer at all may be avoided:

“EPA recognizes that the benefits may be as low as zero since causality has not yet been established between exposure to chlorinated water and bladder, colon, or rectal cancer” (USEPA 2005).

This is confirmed in the Final Rule:

“EPA cannot conclude there is a causal link between exposure to chlorinated surface water and cancer” (USEPA 2006).

EPA Increases Cost to Consumers by Setting Maximum Contaminant Levels (MCLs) below Maximum Contaminant Level Goals (MCLGs) for Individual DBPs

Even though they are required to take cost into consideration, EPA adds to the cost to consumers by setting allowable levels for the nine DBPs in Stage 2 DBPR below the cumulative safe levels for the individual DBPs. They did this by constructing two larger groupings – total Trihalomethanes (THMs) and total Haloacetic Acids (HAAs) – and by setting lower allowable levels in terms of THMs and HAAs (USEPA 2012).

The Safe Drinking Water Act (SDWA) directs the EPA to set standards for the regulation of covered drinking water contaminants: “EPA sets an MCLG [Maximum Contaminant Level Goal] at a level at which no known or anticipated adverse health effects occur. MCLGs are established solely on the basis of protecting public health and are not enforceable. EPA simultaneously sets an enforceable Maximum Contaminant Level (MCL) as close as technologically feasible to the MCLG, while taking costs into consideration” (italics added) (USEPA 2005). Since the MCL is EPA’s enforcement level and is required to consider costs to the public, the MCL for a contaminant is typically set at or above the MCLG. However:

1. The MCL for total THMs is set at 80 ppb (parts per billion) – 38% lower than the cumulative MCLG for the individual THMs (130 ppb).
2. The MCL for total HAAs is set at 60 ppb – 33% lower than the cumulative MCLG for the individual HAAs (90 ppb).

EPA justifies setting cumulative MCLs below the sum of individual MCLGs by a concern that two or more DBPs together might be more toxic than any single DBP.
“Studies of individual byproducts cannot characterize the entire mixture of disinfection byproducts in drinking water” (USEPA1998a).

EPA does not appear to have updated this 1998 statement, yet current analysis of “several excellent reviews [published 2006-2011] of the mixture toxicity literature… [suggest that] although additivity, independent action, synergism, and antagonism are possible when addressing a specific endpoint response, additivity and independent action dominate the toxicity interactions” (italics added) (Landrum et al. 2012).

The most reasonable MCL for a mixture of DBPs thus may be the sum of the MCLs of the individual DBPs, and EPA ought to supply supporting data and/or mode-of-action details to justify a claim that DBPs together cause greater toxicity than individual DBPs alone. This is especially important because of the magnitude of the impact: if a community water system (CWS) supplies water containing THMs and HAAs at levels which meet EPA’s published MCLGs for those contaminants, the CWS would be out of compliance because MCLs would be 57% higher than EPA allows.

However, EPA provides no such justification; no supporting data or mode-of-action discussion is included in the Stage 2 DBPR documents (USEPA 2005, USEPA 2006).

EPA thus increases the cost to consumers without justification, and the result may be to set MCLs much lower than indicated by current scientific data on mixture toxicity. This brings into question whether Stage 2 DBPR is consistent with President Obama’s requirement for “the integrity of scientific and technological information and processes on which the agency relies in its decisionmaking” (Obama 2009).

3. HAS EPA DOCUMENTED A LINK BETWEEN CHLORINATED DRINKING WATER AND CANCER?

In order to establish a linking between contaminant exposure and an adverse effect – in this case, bladder cancer – there must be a clear link between dose and response. Inference is not enough; causation must be established. “Experimental designs for evaluating complex mixture toxicity in aquatic environments can be highly variable and, if not appropriate, can produce and have produced data that are difficult or impossible to interpret accurately… Determining causation requires that the dose-response be established relative to not just the total mixture but to the compounds in the mixture that are likely contributing to the observed toxicity… If causation has not been established, all that can be concluded is that some compound and/or compounds in the mixture and/or modifying factor and/or factors resulted in the observed toxicity. Without establishing causation, it is inappropriate to arbitrarily assume that all or a specific set of the compounds in the mixture are contributing equally to the observed toxicity” (Landrum et al. 2012).
This need to establish a clear link between dose and response is especially important for a regulation which results in a rate increase to consumers of more than 25% for an admittedly minute reduction in contaminant concentration.

**EPA Demonstrates a Bias to Find DBP/Cancer Association**

EPA’s Economic Analysis and Final Rule demonstrate a bias to find a DBP/cancer link – whether or not it exists. EPA acknowledges no “causal link” has been established, but EPA seeks to regulate DBPs because of a health “concern” and a “potential association”: “EPA concluded that although causality has not been established, the data support a weak association that is worthy of concern” (USEPA 2005). “While EPA cannot conclude there is a causal link between exposure to chlorinated surface water and cancer, EPA believes that the available research indicates a potential association between bladder cancer and exposure to chlorinated drinking water or DBPs” (USEPA 2006).

EPA acknowledges that this “weak [potential] association” could actually be no association (USEPA2005):

> “EPA recognizes that actual risks and PAR [Population Attributable Fraction] values could be zero due to uncertainties in the scientific evidence.”

> “EPA recognizes that the benefits may be as low as zero since causality has not yet been established between exposure to chlorinated water and bladder, colon, or rectal cancer.”

EPA’s bias is shown by using the word “yet” in the above quotation. This suggests they expect research will eventually show chlorinated drinking water causes bladder cancer – even though that is not the verdict of science up to this time.

In summary, “EPA concludes that the epidemiological and toxicological studies support a weight-of-evidence conclusion that there may be an association between DBPs and cancer... [even though] causality has not been established” (USEPA 2005).

EPA defines the “weight-of-evidence” as a subjective review “of the quality and adequacy of data and consistency of responses” (USEPA 2006). In scientific terms, this is barely more than a hypothesis. Furthermore, since EPA has shown a bias toward the belief that chlorinated drinking water causes cancer, it would not be surprising if that affects their subjective “weight-of-evidence” determination.
Two Similar International Organizations Differ with EPA

In the context of “weight-of-evidence,” it is interesting to contrast the EPA position with that of the International Programme on Chemical Safety (IPCS) and the International Agency for Research on Cancer (IARC).

IPCS is sponsored by the United Nations Environmental Programme, the International Labour Organization, and the World Health Organization. The IPCS Environmental Health Criteria 216 report, updated online 30 November 2004 (Amy 2000), states:

“None of the chlorination by-products studied to date is a potent carcinogen at concentrations normally found in drinking-water.

“There is insufficient epidemiological evidence to support a causal relationship between bladder cancer and exposures to chlorinated drinking-water, THMs, chloroform or other THM species.”

“Owing to the weight of evidence indicating that chloroform can induce cancer in animals only after chronic exposure to cytotoxic doses, it is clear that exposures to low concentrations of chloroform in drinking-water do not pose carcinogenic risks.”

The latest position of the World Health Organization’s IARC – in the IARC Monograph on the Evaluation of Carcinogenic Risks to Humans, Volume 52 (IARC 1997) and Volume 73 (IARC 1999) – also differs from the EPA position. Although IARC has not updated these volumes since 1997 and 1999, respectively, their November 2012 “List of Classifications by cancer sites with sufficient or limited evidence in humans” (IARC 2102) suggests their opinion has not changed based on more recent data.

IARC monograph Volume 52 (IARC 1997) states as follows regarding chlorinated drinking water and the four THMs regulated by EPA Stage 2 DBPR:

“There is inadequate evidence for the carcinogenicity of chlorinated drinking water in humans… [or] experimental animals” (italics in original).

“There is inadequate evidence for the carcinogenicity of bromodichloromethane… chlorodibromomethane… [or] bromoform… in humans (italics in original).

“There is inadequate evidence in humans for the carcinogenicity of chloroform (italics in original).
IARC defines *inadequate evidence* as meaning that “the available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding the presence or absence of a causal association between exposure and cancer” (IARC 1999).

Furthermore, the ToxFAQs summaries of the four regulated Trihalomethanes (THMs) published by the U. S. Government Agency for Toxic Substances and Disease Registry (ATSDR) do not mention human bladder cancer, but do discuss liver, kidney, and (for bromodichloromethane) intestinal cancers in animal studies; and only two of the four (including chloroform – see below) are listed as “reasonably anticipated to be a human carcinogen” (ATSDR 1997, ATSDR 1999, ATSDR 2005). Although some of this information is dated 1997 and 1999, it is believed to be current because ATSDR ToxFAQs are reviewed and updated “no less often than once every three years” (ATSDR 1993).

These animal studies, however, may not be relevant to the issue of DBPs and human bladder cancer – both because the animal studies involve different cancer sites and because DBPs are at low levels in chlorinated drinking water, whereas animal studies are typically done at high doses. Both of these considerations are discussed below.

**The Epidemiological Basis for the EPA Stage 2 DBPR**


EPA also used a meta-analysis, Villanueva *et al.* (2003), which included four of the above five studies (along with four others), and a pooled data analysis, Villanueva *et al.* (2004). These seven studies were used to calculate the Odds Ratios (ORs) and Population Attributable Fractions (PARs) from which EPA estimated the number of bladder cancer cases that might be caused by drinking chlorinated water.

**Contradictions and Inconsistencies in EPA’s Epidemiological Data**

A strong degree of consistency should have been expected with EPA’s epidemiological data because Kenneth P. Cantor (NCI 2009) of the National Cancer Institute (NCI) was first or second author in four of the seven studies. But to the contrary, Dr. Cantor and his colleagues identified several noteworthy contradictions and inconsistencies within the five studies used by EPA (Cantor *et al.* 1998):

“Our observation [Cantor *et al.* (1998)] that risk increased with duration of chlorinated surface water among ever-smokers, but not never-smokers, and among men, but not women, raises questions of inter-
nal consistency, as well as consistency with other findings. In contrast to the current investigation [Cantor et al. (1998)], the National Bladder Cancer Study [Cantor et al. (1987)] found associations for both sexes, primarily among never-smokers. In Ontario, King and Marrett [King and Marrett (1996)] noted somewhat higher risk estimates for never-smokers associated with duration of chlorinated surface water. In Colorado, McGeehin et al. [McGeehin et al. (1993)] reported similar patterns of risk among smokers and never-smokers, and among men and women. Finally, in a case-control study from Washington County, MD, Freedman et al. [Freedman et al. (1997)] reported results that parallel the current findings, namely that the risk associated with chlorinated surface water was primarily observed among men and among smokers. Reasons for differences among these observations and differences with results from our study are unclear. A possible explanation for the apparent discrepancies in findings for smokers and never-smokers among studies may reside in water quality and water treatment differences in the respective study areas, with resulting variations in the chemical composition of byproduct mixtures. Nevertheless, results should not differ by sex.”

The Villanueva et al. (2004) pooled data analysis contains similar inconsistencies. It finds increased bladder cancer risk due to Trihalomethanes (THM) exposure only among men; “among women, no association was observed with any of the exposure indices we used.” In fact, the data for women actually suggest that exposure to THMs and/or chlorinated surface water decrease the risk of bladder cancer in many cases.

The International Agency for Research on Cancer (IARC) likewise noted inconsistencies and contradictions in various studies regarding chlorinated drinking water and cancer. The IARC Monograph, Volume 52 (IARC 1997), echoes several of the same problems identified by Cantor et al. (1998), and it adds others:

“The studies that were considered informative, and therefore included in this summary, were nevertheless difficult to interpret in an evaluation of the carcinogenicity of chlorinated drinking-water. The water variables studied – whether surface or groundwater and others – were generally imperfect surrogates for the subject of this monograph. There is cause for some scepticism about the estimates of exposure to chlorinated drinking-water in all of these studies. Furthermore, very few attempted to document exposure over long periods of the subjects’ lives. Chlorination by-products differ according to local conditions and practices of chlorination, and the health effects found in one place may not be found elsewhere. Many variables, such as smoking habits, dietary practices and environmental conditions, influence the risks for cancer, and they may differ between populations served.
by chlorinated and unchlorinated water supplies. Such factors should ideally be taken into account in an epidemiological study; however, in most of the studies evaluated, there was little if any information available about them.”

The International Programme on Chemical Safety (IPCS) is even more explicit about the contradictions and inconsistencies found in the various chlorinated drinking water studies. Their report (Amy 2000) states:

“The epidemiological evidence for an increased relative risk of bladder cancer is not consistent – different risks are reported for smokers and non-smokers, for men and women, and for high and low water consumption. Risks may differ among various geographic areas because the DBP mix may be different or because other water contaminants are also present. More comprehensive water quality data must be collected or simulated to improve exposure assessments for epidemiological studies.”

EPA acknowledges these problems in the Final Rule (USEPA 2006): “While the results of [recent studies on human epidemiology and animal toxicology] have been mixed, EPA believes they support a potential hazard concern” (italics added). EPA defines “hazard” as “the possibility that a health effect may be attributed to a certain exposure.” Hence the strongest statement EPA can make is that DBPs in drinking water pose a “potential possibility” of a concern.

The epidemiological studies used in EPA’s Economic Analysis (USEPA 2005) were retrospective case-control studies, which require careful adjustment of “confounding factors” such as smoking and other lifestyle factors which independently affect the risk of disease. These problems were pointed out by IARC and IPCS; failure to adequately adjust for these confounding factors and for local water conditions could easily lead to these kinds of inconsistencies and contradictions. As Cantor et al. (1998) observe, the consistent pattern which suggests a statistically significant difference between men and women with regard to bladder cancer risk from drinking chlorinated water is difficult to explain.

Hence even if the studies taken together predict an increased bladder cancer risk from chlorinated drinking water, the reliability of this prediction can be questioned. In fact, as noted below, EPA data suggest decreased cancer risk is a statistical possibility.
Morris et al. (1992): A Case Study for the Problems Obtaining Accurate Epidemiological Data

The problems obtaining accurate and reliable data seem illustrated by a 1992 meta-analysis (Morris et al. 1992). This study of chlorination, chlorination by-products and cancer found “a 21% increase in risk for bladder cancer and a 38% increase in the risk for rectal cancer,” and it also found quantifiable risks of brain, breast, colon, colorectal, esophageal, kidney, liver, lung, and pancreatic cancers – eleven of the eighteen principle cancers listed in Cancer Facts and Figures 2010 (ACS 2010). These extraordinarily broad results led EPA’s Scientific Advisory Board to speculate if “chlorinated drinking water [might be] a major source of human cancer” (USEPA SAB 1993).

A 2002 publication quotes an EPA epidemiologist about Morris et al. (1992): “There was a vested interest in having that meta-analysis because it appeared as if it were an open and shut case” (Driedge 2002). In 1993, EPA’s SAB cited Morris et al. (1992) as the basis for stating that “human data indicate that chlorination accounts for 9% of annual U. S. bladder cancer cases… and 15% of rectal cancer cases” (USEPA SAB 1993), and NIEHS scientists cited Morris et al. (1992) as a reason to reject the International Agency for Research on Cancer (IARC) position that there is no link between DBPs and cancer (Dunnick and Melnick 1993).

On the other hand, these broad results met with professional skepticism such as illustrated in the Journal of Clinical Epidemiology: “I know of no chemical agent that has been found, by means of appropriate studies, to induce cancer in every organ that has been examined” (Bailar 1995). As a result, in 1997 EPA commissioned a review of Morris et al. (1992) (“the Poole Report”) which found numerous problems, including “evidence of publication bias within the body of literature,” meaning that the sample of studies used was “not representative of all the research that has been done on [the] topic” (Poole 1997). The Poole Report stressed that the studies evaluated in the Morris et al. (1992) publication were highly inconsistent, undermining the utility of a meta-analysis for developing a single estimate of risk. Peer review concurred with this conclusion, and “EPA concluded that Poole presented reasonable and supportable evidence to suggest that the work of Morris et al. (1992) should not be used for risk assessment purposes” (USEPA 1998a).

Why Do Inconsistencies and Contradictions Persist in EPA’s Data, Despite Lessons from Morris et al. (1992)?

The controversy regarding Morris et al. (1992) illustrates fundamental difficulties with epidemiological studies of chlorinated drinking water and cancer.

EPA states that “higher quality studies have adequately controlled for confounding and have limited the potential for exposure misclassifica-
tion” (USEPA 2006) – implying that the studies supporting Stage 2 DBPR avoid the deficiencies of Morris et al. (1992). Yet as noted above, these studies still contain noteworthy contradictions and inconsistencies – especially in the areas of sex, smoking habits, and water variables. This may reflect what EPA acknowledges are inherent problems in obtaining useful data about chlorinated drinking water and cancer (USEPA 1998a):

“The assessment of public health risks from chlorination of drinking water currently relies on inherently difficult and incomplete empirical analysis. On one hand, epidemiologic studies of the general population are hampered by difficulties of design, scope, and sensitivity. On the other hand, uncertainty is involved in using the results of high dose animal toxicological studies of a few of the numerous byproducts that occur in disinfected drinking water to estimate the risk to humans from chronic exposure to low doses of these and other byproducts.”

NIEHS scientists have also expressed concern about inherent difficulties with epidemiological studies of by-products of water chlorination due to poor characterization of DBP exposures and failure to adequately account for other confounding risk factors (Melnick et al. 1994). Another possible source of error is the “wide range of synthetic chemicals other than by-products of chlorination… [which make it] difficult or impossible to distinguish a chlorination effect from a surface-water effect” (Poole and Greenland 1999).

It thus appears that fundamental issues remain even if the questions of publication bias and heterogeneity raised by the Poole Report and by EPA are overcome.

**Discrepancies Between Animal Studies and Epidemiological Studies**

As noted above, experimental animal studies with DBPs suggest only liver and kidney cancers (ATSDR 1999), yet EPA’s analysis of epidemiological data suggests an association with bladder cancer. In 1993, EPA’s Scientific Advisory Board (SAB) noted this discrepancy with grave concern (USEPA SAB 1993):

“There are substantive qualitative and quantitative discrepancies, however, between the human and animal data. First, human epidemiology suggests that the major target areas are the bladder and rectum,… while by-products studied in the usual animal models suggest that the major targets should be liver and kidney… This lack of correspondence in tumor sites has been disregarded in Agency regulatory activities in the past, for policy reasons. However, it is dangerous to ignore it in the present circumstance.”
The SAB was unable to explain this disparity but emphasized that:

“These discrepancies must be resolved if the agency is to develop a scientific basis for a disinfection rule” (italics added).

These discrepancies still have not been resolved. This raises a question about “the integrity of scientific and technological information” used by EPA as the basis for Stage 2 DBPR, as required by President Obama’s 2009 memorandum on the subject of “Scientific Integrity” in the decision making processes of federal agencies (Obama 2009).

Publication Bias and the Overstatement of Positive Results

Another concern raised in the Poole Report is publication bias, defined as follows (Poole 1997):

“Publication bias occurs when the literature search and inclusion criteria produce a sample of studies that is not representative of all the research that has been done on a topic. Because of obvious disincentives in the social systems of science, medicine and public health, results that point away from a direction that is considered plausible are less likely to be published.”

RD Morris, first author of Morris et al. (1992), suggests this is a general problem with epidemiology (Morris 1994):

“The potential for [publication] bias in cancer epidemiology is arguably high. Case-control studies may investigate a wide range of hypotheses. The lack of a strong incentive to publish negative results from these studies may lead to publication bias.”

The Poole Report author elaborates on publication bias in a 1999 follow-up article: “The reluctance of investigators to publish results close to the null value and their extreme reluctance to publish implausible results are well documented” (Poole and Greenland 1999). Articles from a diverse range of disciplines (Begg and Berlin 1989, Davidson 1986, Dickersin and Berlin 1992, Dickersin et al. 1992, Easterbrook et al. 1991, Mahoney 1977, Shapiro 1985) suggest this is a general and all-pervasive phenomenon including “a preponderance of false-positive results in the literature,” “an overestimate of positive results and an underestimate of negative ones,” and the need for a cautious interpretation of observational studies because “a meaningful proportion of studies remain forever unpublished.” Comprehensive studies published in 2000 and 2010 to update the state of publication bias confirm this as an ongoing phenom-
enon: “studies with significant or positive results are more likely to be published than those with non-significant or negative results” (Song et al. 2000, Song et al. 2010).

Hence because of publication bias, even the “weak association” EPA finds between DBPs and bladder cancer might be an overstatement: a “false positive.”

EPA Bias May Overstate Odds Ratios (ORs) and Population Attributable Fractions (PARs); 83% of Studies Show Possible Decreased Cancer Risk from DBPs

Arguably the most problematic form of publication bias is that: “Some authorities explicitly advocate withholding implausible results from the published record” (Poole and Greenland 1999). This is precisely what EPA did in Stage 2 DBPR. In summarizing 95% Confidence Intervals for their PAR calculations, EPA arbitrarily threw out negative PARs (which imply decreased cancer risk) and set the lowest level at zero cancer risk. This is justified with the following footnote (USEPA 2005):

“Confidence levels truncated to zero to reflect biological plausibility. The actual lower confidence level is often negative.”

In other words, EPA rejects data showing that chlorinated drinking water decreases cancer risk; they claim such data is a biologically implausible statistical aberration. To the contrary, as detailed below, publications over the last fifteen-twenty years consistently show that many – arguably most – substances which are harmful at high doses are beneficial at low doses. In other words, there is a sound scientific basis to assume this data is both valid and biologically plausible.

This is important in the context of Stage 2 DBPR. Even though EPA’s data may be skewed toward positive results because of publication bias, five of the six studies cited – 83% – include a negative PAR within the 95% Confidence Interval (USEPA 2005). That is, 83% of the EPA studies have a statistical possibility of decreased cancer risk from chlorinated drinking water.

EPA May Have Substantially Overstated Stage 2 DBPR Benefits

In summary, there is sound basis to suggest EPA may have substantially overestimated cancers avoided by the Stage 2 DBPR:

1. The well documented phenomenon of publication bias shows that studies which produce negative and/or “implausible” results are often not published, and hence the epidemiological studies used by EPA may be skewed toward positive results.
2. EPA rejected data from 83% of the studies in their Economic Analysis which show a statistical possibility that DBPs might decrease cancer risk.

The real result may be an OR closer to one – maybe even less than 1, indicating reduction of the risk of cancer. This may be part of the reason the International Agency for Research on Cancer (IARC) has concluded there is “inadequate evidence for the carcinogenicity of chlorinated drinking water in humans” (italics in original) (IARC 1997) and why the International Programme on Chemical Safety (IPCS) concurs.

4. DOES EPA ESTIMATE RISK USING AN APPROPRIATE DOSE-RESPONSE MODEL?

Choice of the correct dose-response model is critical in fulfilling EPA’s mandate to protect the environment while taking cost into consideration (USEPA 2005) and to comply with President Obama’s directive for “Scientific Integrity” in the decision making processes of federal agencies (Obama 2009). As discussed above, choice of the wrong model can drive up the cost substantially with no health benefit because the last unit of contaminant is much more costly to remove than the first unit. Moreover, choice of the wrong dose-response model can even take away a health benefit.

**EPA Assumes the LNT Dose-Response Model**

Regulations such as Stage 2 DBPR are due to EPA’s adherence to the Linear No Threshold (LNT) dose-response model. LNT suggests that if something presents a risk at high doses, it also presents a risk at low doses – even minutely low doses that approach zero as a lower limit:

“EPA assumes there is a linear relationship between average DBP concentration and relative risk of bladder cancer… [and] that there is no threshold below which there is no risk” (USEPA 2005).

This LNT model assumes that the effects of a substance vary linearly with dose from high doses to zero doses, and that some degree of harm occurs at even the lowest non-zero dose. It is surely the reason EPA rejected as biologically implausible their data which showed low level DBPs might decrease cancer risk.

But is it reasonable to apply the LNT model to DBPs? Or is another dose-response model more appropriate? Stage 2 DBPR deals with minute quantities of DBPs: parts-per-billion (ppb). Chloroform, for example, is regulated to 70 ppb; EPA believes quantities this low present a risk. And EPA applies the LNT model as a default to “chemicals for which the MOA
[mode-of-action] is not known” (Dourson and Haber 2010). For example, EPA has set MCLGs at zero in Stage 2 DBPR for three DBPs: Bromodichloromethane (CHBrCl₂, aka BDCM), Bromoform (CHBr₃), and Dichloroacetic acid (CHCl₂CO₂H, aka DCAA) (USEPA 2012). This claims one molecule of these substances is harmful; in the case of bromoform at least, this is based on lack of data (ATSDR 2005). That is the central point of this section. EPA believes they take a conservative approach by setting an extremely low MCLG for DBPs. This is true, however, only if the LNT model applies. If DBPs follow the threshold (TM) or hormetic dose-response models (discussed below), even the minuscule health benefit EPA estimated for Stage 2 DBPR is impossible to justify; the rules are economically costly for no benefit – and possibly even harmful to health.

The LNT Model Has Been Proven Wrong with the DBP Chloroform

Since EPA assumes the LNT model with DBPs, it is noteworthy that this model has been proven wrong with one DBP, chloroform. The International Programme on Chemical Safety (IPCS) Environmental Health Criteria 216 report (Amy 2000) states:

“It is clear that exposures to low concentrations of chloroform in drinking-water do not pose carcinogenic risks.”

In March 2000 the D. C. Circuit Court enjoined EPA from using the LNT model with chloroform because it failed to use the “best available science.” In Chlorine Chemistry Council v. E.P.A., 206 F.3d 1286 (D.C.Cir. 2000), the court ruled the Maximum Contaminant Level Goal (MCLG) for chloroform should be 300 ppb, based on the recommendations of a panel of experts (LSU 2000).

Yet EPA set the chloroform MCLG at 70 ppb – 77% lower than the court’s ruling – based on the assumption of “a relative source contribution (RSC) of 20%” (USEPA 2006). Such a low RSC assumption might be disputed by the California Air Resources Board (CARB 1990). Furthermore, this reduction of the chloroform MCLG is an important element in the high cost of Stage 2 DBPR.

An Alternative: the Hormetic Dose-Response Model

EPA’s use of the LNT dose-response model for chemicals believed to be carcinogenic was based on recommendations of the first National Academy of Sciences (NAS) Safe Drinking Water Committee in 1977, which in turn were based on the belief that the LNT model applied to ionizing radiation (Calabrese 2009, Dourson and Haber 2010). However, this latter assumption may have been based on “blatant dishonesty within
a framework of ideological science” (Calabrese 2013), and it is increas-
ingly being questioned as new data becomes available (Higson 2004). This in turn raises questions about application of the LNT to chemicals. An editorial in Environmental Toxicology and Chemistry suggests the LNT model has become “The New Homeopathy” (Calabrese et al. 2012).

One alternate dose-response model is the threshold model, which assumes a chemical presents a risk only above a threshold value. TM is generally assumed with non-carcinogens (Rhomberg et al. 2011), and it appears to be the model most applicable in the court case, Chlorine Chemistry Council v. E.P.A. Chloroform is generally believed to follow the threshold model (TM) – even though, as noted below, some animal studies suggest it might follow the hormetic model.

The hormetic dose-response model could be considered a variation of the threshold model, because it assumes that a substance which is harmful at high doses is beneficial at doses below a threshold. This concept is fundamental to the pharmaceutical industry. Dose is all-important; the effect of many drugs is therapeutic or toxic depending on the dose. Furthermore in comprehensive studies over the past fifteen years comparing the three dose-response models – LNT, TM, and hormesis – “only the hormetic (biphasic) dose-response made consistently accurate predictions” (Calabrese 2013).

The hormetic dose-response model is defined as a “low dose beneficial response to a stressor agent” (Calabrese 2010) and as “a quantitative manifestation of a reparative process that is adaptive in nature” (Calabrese 2008). The general idea is that a disruption in homeostasis (ie, toxicity) is followed by an overcompensatory response that is seen as stimulation (Calabrese 2010, Stebbing 1998). In other words, the hormetic dose response quantifies how the system allocates resources, such that low doses stimulate the body’s natural protective mechanisms, allowing the body to combat ill effects from higher doses. Hormetic dose responses have also been observed “as a result of direct stimulation, with no initial disruption in homeostasis” (Calabrese 2010).

Live virus vaccinations are an example: by injecting a small amount of live virus, the body is stimulated to produce antibodies which fight off larger amounts of the virus. Hence the small amount of toxin is beneficial – not necessarily because it is beneficial per se, but because the net result is an overcompensatory response which is beneficial.

**Hormetic Model is the Most Common Dose-Response Model, Demonstrated with Chloroform and with Bladder Cancer, and Consistent with EPA's Economic Analysis**

This section now focuses on the central question: can a weight-of-evidence argument be constructed that DBPs might follow the hormetic dose response model?
Chloroform, one of the nine DBPs regulated by EPA’s Stage 2 DBPR, has been extensively studied. Although it is generally considered to follow the threshold model, multiple experimental animal studies some thirty-forty years ago showed that “while very high exposures of chloroform caused cancer in laboratory animals, low levels actually improved the survival of rats, mice, and dogs” (Calabrese et al. 1987, Druckrey 1968, Heywood et al. 1979, Jorgenson et al. 1985, Palmer et al. 1979, Roe and van Abbe 1980, Roe et al. 1979). These data suggest that chloroform exhibits hormesis.

Just because chloroform might follow the hormetic model does not mean that is true of the other eight DPBs regulated by EPA’s Stage 2 DBPR; that depends on the mode-of-action on a chemical-by-chemical basis.

However, as noted earlier, the epidemiological studies used in EPA’s Economic Analysis suggest chlorinated drinking water as a whole might follow TM or the hormetic model: even though EPA’s epidemiological data may be skewed toward positive results, 83% of the studies used by EPA showed a probability within the 95% confidence interval of a decrease in cancer risk from chlorinated drinking water (USEPA 2005). EPA rejected this data as not reflecting “biological plausibility” – yet EPA’s Economic Analysis for Stage 2 DBPR acknowledges the possibility that a hormetic model or threshold model might apply in the list of “Uncertainties and Possible Effect on Estimate of Benefits” which might result in an “overestimate” of the “benefit estimates” (USEPA 2005). This attitude follows what one toxicologist believes is a pattern: “the U. S. EPA position is remarkable in that it acknowledges not only the possibility and indeed the likelihood of adaptive responses, but also its clear intention not to consider such responses in their evaluation” (Calabrese 2008).

The hormetic model is acknowledged by sister government agencies (although the author is not aware that any government agency has incorporated hormesis into any risk management analysis, except for essential nutrients). The Federal Agency for Toxic Substances and Disease Registry (ATSDR), for example, “has long been aware of... substances... toxic at high exposure levels but... beneficial at much lower exposure levels” (De Rosa et al. 1998). The Senior Scientific Advisor to the Director of NIEHS suggested in 2003 (Fouts 2003) that hormesis could be quite important in environmental policy because “most environmental pollution will give most populations low exposures, and therefore hormesis could be of great importance to the evaluations of human dangers.” He asks rhetorically: “What might be the impact of hormesis on such widely occurring problems as... water purification. Are very low doses of halogenated hydrocarbons OK in water?”

As a second point in this weight-of-evidence argument: since the EPA Stage 2 DBPR are directed toward avoidance of bladder cancer, it is note-
worthy that one of the largest animal studies ever undertaken identified hormetic effects with bladder cancer for a known carcinogen (Bruce et al. 1981, Calabrese 2010):

“The mega-mouse testing of the carcinogen 2-AAF [2-Acetylamino Fluorene]... revealed an unequivocal hormetic dose response for bladder cancer, with risks decreasing below the control group at low doses.”

Although this information cannot be generalized to other chemicals believed to cause bladder cancer, it raises the question of the statistical probability of the various dose-response models.

That is the third point in this weight-of-evidence argument: based on studies of the effects of various chemicals, the hormetic dose-response model is much more likely to be followed than the LNT or TM models.

Throughout the twentieth century, neither the TM nor LNT models were ever validated by the regulatory and scientific communities below the toxicological threshold. However, a group at the Department of Public Health at the University of Massachusetts Amherst undertook such a comprehensive study over the past fifteen years. This group “put the threshold, hormesis, and Linear No-Threshold (LNT) models to the test (actually, three substantial validation tests). In each of these tests the threshold and LNT models made poor predictions of responses in the low-dose zone. Only the hormetic (biphasic) dose-response made consistently accurate predictions” (Calabrese 2013).

In a study which assessed “the responses of [nearly 1800] doses below the toxicological NOAEL (no observed adverse effect level)… from 664 dose response relationships derived from a previously published database,” hormetic dose-response curves were 2.5 times more prevalent than the no effect condition predicted by the threshold model (TM) (Calabrese and Baldwin 2003a).

In a study of the “U. S. National Cancer Institute Yeast Anti-Cancer Drug Screen database, which contains 2,189 chemical agents that were tested on 13 strains of yeast over five concentrations within a replicated study framework... All 12,000 dose responses demonstrated evidence consistent with the hormetic dose response” (Calabrese et al. 2006, Calabrese 2008).

Based on these and other studies, hormesis has been described as “a universal or near-universal phenomenon... [because] hormesis serves a series of strong survival interests” (Calabrese 2008).

**Weight-of-Evidence May Really Point to the Hormetic Model**

In summary, there is no definitive indication that DBPs follow the LNT model, and there is limited evidence that they do not. In contrast, it
might be reasonable to assume DBPs follow the hormetic dose-response model and are beneficial at low levels because:

1. Statistically, the hormetic model appears much more likely than TM or LNT.
2. Experimental animal studies have shown hormetic effects with bladder cancer and with at least one DBP regulated by the EPA Stage 2 DBPR.
3. EPA’s Economic Analysis suggests the statistical probability that low levels of DBPs in chlorinated drinking water might reduce cancer risk.

Hence the real “weight-of-evidence” argument may point to a hormetic response to parts-per-billion contamination of drinking water with DBPs.

5. CONCLUSION

Implementation of EPA’s Stage 2 DBPR carries an extremely high cost to the consumer – yet promises negligible or no health benefit – and failure to implement Stage 2 DBPR appears to pose no unreasonable risk to health (URTH).

EPA fails to prove that these rules will avoid even one case of cancer, because they fail to demonstrate that chlorinated drinking water is a cause of cancer; the most they claim is a “hazard concern” (USEPA 1998a). Epidemiological data supporting the Stage 2 rules are inconclusive and contain significant contradictions and inconsistencies; publication bias suggests the data may be skewed toward finding a cancer link which does not exist.

EPA does not dispute these findings and acknowledges inherent difficulties of design, scope, and sensitivity, poor characterization of DBP exposures, and failure to adequately account for other confounding risk factors in the data (Melnick et al. 1994); and as a result they feel required to base “quantitative risk estimates on less than comprehensive information” (USEPA 1998a). The result is a subjective conclusion: “EPA believes that the weight-of-evidence... [supports] a hazard concern and a protective public health approach to regulation” (USEPA 1998a).

President Obama has directed every federal agency “to ensure the integrity of scientific and technological information and processes on which the agency relies in its decisionmaking” (Obama 2009). Stage 2 DBPR seems inconsistent with this directive in three areas in particular:

1. EPA’s SAB emphasized that the unexplained discrepancies between the cancer sites suggested by experimental animal studies and by EPA’s epidemiological studies “must be resolved if [EPA] is to develop
a scientific basis for a disinfection rule” (USEPA SAB 1993). This discrepancy remains unresolved.

2. EPA set allowable levels for the nine DBPs below cumulative safe levels for the individual DBPs. EPA did this out of concern that DBPs together might be more toxic than the sum of individual DBPs, but this approach seems contrary to a review of current scientific data which suggests “additivity and independent action dominate the toxicity interactions” of mixtures (Landrum et al. 2012).

3. EPA may be using the wrong dose-response model to generate Stage 2 DBPR. EPA’s LNT (Linear No Threshold) model is not as accurately predictive in the low-dose zone as other dose response models, and it has been proven wrong with one of the eight regulated DBP’s. Furthermore, EPA’s epidemiological data even suggest chlorinated drinking water may reduce or not affect the risk of bladder cancer – meaning that DBPs in aggregate might follow the threshold (TM) or hormetic dose-response models.

In summary, EPA’s Stage 2 DBPR should be revised or eliminated. Since they impose a large unfunded mandate on the public with no demonstrated benefit, they are costly and/or unnecessary federal regulations which fall under EPA’s program for Retrospective Reviews of Existing Regulations. Also, since the science is questionable in at least three areas, they might be inconsistent with President Obama’s 2009 directive about scientific integrity for federal regulations.

ACKNOWLEDGEMENT

The author would like to thank an anonymous referee for some especially useful review comments.

REFERENCES


Lovan C.R., President/CEO of Northern Kentucky Water District (NKWD) 2012. Private communication.


Strawson J, Haber LT, Dourson ML, and Barkhurst M. 2003. Approaches to Determining "Unreasonable Risk to Health." National Rural Water Association, Duncan, OK.


