

# Improved Exposure Assessment on Existing Cancer Studies

Subject Area:  
High-Quality Water



# **Improved Exposure Assessment on Existing Cancer Studies**



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# Improved Exposure Assessment on Existing Cancer Studies

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## FOREWORD

The Awwa Research Foundation is a nonprofit corporation that is dedicated to the implementation of a research effort to help utilities respond to regulatory requirements and traditional high-priority concerns of the industry. The research agenda is developed through a process of consultation with subscribers and drinking water professionals. Under the umbrella of a Strategic Research Plan, the Research Advisory Council prioritizes the suggested projects based upon current and future needs, applicability, and past work; the recommendations are forwarded to the Board of Trustees for final selection. The foundation also sponsors research projects through an unsolicited proposal process; the Collaborative Research, Research Applications, and Tailored Collaboration programs; and various joint research efforts with organizations such as the U.S. Environmental Protection Agency, the U.S. Bureau of Reclamation, and the Association of California Water Agencies.

This publication is a result of one of these sponsored studies, and it is hoped that its findings will be applied in communities throughout the world. The following report serves not only as a means of communicating the results of the water industry's centralized research program but also as a tool to enlist the further support of the nonmember utilities and individuals.

Projects are managed closely from their inception to the final report by the foundation's staff and large cadre of volunteers who willingly contribute their time and expertise. The foundation serves a planning and management function and awards contracts to other institutions such as water utilities, universities, and engineering firms. The funding for this research effort comes primarily from the Subscription Program, through which water utilities subscribe to the research program and make an annual payment proportionate to the volume of water they deliver and consultants and manufacturers subscribe based on their annual billings. The program offers a cost-effective and fair method for funding research in the public interest.

A broad spectrum of water supply issues is addressed by the foundation's research agenda: resources, treatment and operations, distribution and storage, water quality and analysis, toxicology, economics, and management. The ultimate purpose of the coordinated effort is to assist water suppliers to provide the highest possible quality of water economically and reliably. The true benefits are realized when the results are implemented at the utility level. The foundation's trustees are pleased to offer this publication as a contribution toward that end.

The focus of this study was the reanalysis of two well-conducted, peer-reviewed epidemiology studies in Iowa and Ontario that have reported an increased risk of bladder cancer associated with chlorinated drinking water and trihalomethanes; in the reanalysis, other cancer sites (i.e., colon and rectal cancers) were also considered. The motivation for this reanalysis was the application of an improved exposure assessment embodying more complete information on classes and species of disinfection by-products (DBPs) formed during chlorination and chloramination. Given the long latency period for cancer, historical databases contain only limited DBP data, while more recent monitoring efforts (e.g., the U.S. Information Collection Rule program) are more comprehensive. Through modeling/correlation analysis, both in the form of central tendency models and a new case study modeling approach, past DBP levels were estimated based on

present trends and knowledge about changes in source water(s) and treatment practices, both present and past. Such estimates permitted an improved DBP exposure assessment that supported some original results and further elucidated and revealed other trends.

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## EXECUTIVE SUMMARY

The objective of this study was to reanalyze two well-conducted, peer-reviewed epidemiology studies in Iowa and Ontario that reported an increased risk of bladder cancer associated with chlorinated drinking water and trihalomethanes (THMs). In this current study, other cancer sites (i.e., colon and rectal cancers) were also considered. The motivation for such a reanalysis is the application of an improved exposure assessment embodying more complete information on classes and species of disinfection by-products (DBPs) formed during chlorination and chloramination. Given the long latency period for cancer, historical databases contain only limited DBP data, whereas more recent monitoring efforts (e.g., the U.S. Information Collection Rule [ICR] program) are more comprehensive. Through modeling and correlation analysis, it is possible to estimate past DBP levels based on present trends and knowledge about changes in source water(s) and treatment practices, both present and past. Such estimates can permit an improved DBP exposure assessment.

### DBP PREDICTION MODELS FOR AN IMPROVED EXPOSURE ASSESSMENT

A key aspect of the improved exposure assessment was the development of DBP prediction models. This effort began with the acquisition and assembly of national (U.S. and Canadian) DBP databases and was augmented by site-specific databases for the province of Ontario and the state of Iowa. These DBP databases were analyzed and compared, with the goal of using them as a basis for national and site-specific DBP prediction models.

#### Databases

##### *National DBP Databases for the United States and Canada*

In the United States, DBPs were first detected in drinking water systems in the early 1970s and have been regulated and monitored since. National U.S. databases have been developed since 1975 and include the National Organics Reconnaissance Survey, National Organics Monitoring Survey, the Awwa Research Foundation THM Survey, 35-Utility Study, American Water Works Association (AWWA) WATER:STATS, AWWA Water Industry Data Base, and ICR databases. Canadian regulations concerning DBPs were first enacted in 1993 as a guideline. Over the last decade, there have been two national Canadian databases: Health Canada's 53-Utility Survey and One-Year Survey of Halogenated Disinfection By-Products in the Distribution System of Treatment Plants Using Three Different Disinfection Processes.

A comparison of the databases reveals that Canadian drinking waters tend to differ from U.S. drinking waters in two ways. Canadian waters generally have lower levels of influent bromide, resulting in lower levels of brominated DBPs, and reflect natural organic matter properties that promote the formation of haloacetic acids (HAAs) and species over total THMs (TTHM) and species. Both Canadian databases showed significantly less TTHM formation than reported in the U.S. surveys. However, the Canadian databases also indicate similar amounts of HAAs and TTHM, a result not observed in the U.S. databases.

Some of the earlier surveys only reported THMs as TTHM, with later surveys reporting THM species (chloroform, bromodichloromethane [BDCM], dibromochloromethane, and

bromoform). Only more recent surveys have reported HAAs as including HAA5 (sum of the five species regulated in the United States: trichloroacetic acid [TCAA], dichloroacetic acid [DCAA], monochloroacetic acid, dibromoacetic acid, and monobromoacetic acid), HAA6 (HAA5 plus bromochloroacetic acid [BCAA]), HAA9 (HAA6 plus bromodichloroacetic acid, dibromochloroacetic acid, and tribromoacetic acid). Some of the surveys contain DBP precursor information (e.g., total or dissolved organic carbon and bromide ion [Br<sup>-</sup>]), water quality information (e.g., pH and temperature), and disinfection information (e.g., chlorine dose).

### ***Site-Specific Databases for Ontario and Iowa***

The only Ontario database available was the Drinking Water Surveillance Program, a very robust database whose only deficiency is a lack of Br<sup>-</sup> data. A comparison between Ontario and Canada DBP trends is difficult to make because the only extensive national Canadian database was the Health Canada survey conducted in 1993. However, from these data it is apparent that Ontario DBP formation is very similar to national Canadian trends. Some relevant ICR data were also assembled corresponding to U.S. utilities using Lake Ontario water, the major drinking water source in Ontario.

The Iowa databases available included a 1987 database in Cantor et al. (1998) used in the original Iowa epidemiology study, the Center for Health Effects of Environmental Contamination, the Iowa Department of Natural Resources, Iowa entries in WATER\STATS, Iowa entries in the ICR database, and an expanded ICR database that included utilities proximate to Iowa and situated in similar watersheds. Iowa waters tend to form less DBPs than the U.S. national levels. The TTHM and HAA5 for Iowa fall slightly below the median values for all of the national databases.

When comparing Iowa with Ontario, it is evident that the Ontario waters tended to form less TTHM than the Iowa waters. The Ontario waters also tended to form higher levels of HAAs and HAA species, presumably because the Ontario waters contain natural organic matter that promotes HAA formation over TTHM formation. The Ontario waters also contained less bromide (inferred from an analysis of THM and HAA species data), resulting in lower formation of brominated DBPs and thus higher formation of chlorinated DBPs. This is evident in the lower BDCM, DBCM, and bromoform concentrations observed in the overall Ontario databases. The trend of Ontario waters in forming HAAs preferentially over THMs may also be attributed to factors other than natural organic matter, including pH and temperature.

### **DBP Prediction Models**

Statistically based models were developed through multiple regression analysis using the various databases. In applying these models to making predictions in Ontario and Iowa, it became apparent that these *central tendency* models work well for utilities with near-median levels of DBP precursors, water quality conditions, and disinfection conditions that lead to the formation of near-median DBP levels. However, they are poor in predicting more extreme (e.g., 10th or 90th percentile) conditions. A decision was made, midway through the project, to develop a second, complementary modeling approach, involving *case study* models based on “an expert system” in the form of the project’s DBP expert, Stuart Krasner. In the case of Ontario, the two modeling approaches were truly complementary, and both approaches were used. However, with Iowa, the case study approach was used almost exclusively because of data limitations in developing robust central tendency models.

The role of the models was to assist in the construction and reconstruction of exposure to chlorination DBPs over an approximately 50-year period in both Ontario and Iowa. The national databases were useful in defining regulatory “transitions” backward through time, resulting in a historical and chronological overview of DBP formation and disinfection practices. Once this historical time line was created, models were utilized to “fill in” any missing gaps of data (e.g., projecting past HAA occurrence). Data were missing because the historical data tended to be extremely fragmented or did not exist at all, especially regarding HAAs, due to the lack of regulations or monitoring requirements at that time. It is noteworthy that THM occurrence in chlorinated drinking water was only discovered in the early 1970s.

### ***Central Tendency Models***

Both national and site-specific multiple regression models were developed to predict individual DBPs from information on DBP precursors, water quality, and disinfection conditions, or to predict certain DBPs (e.g., HAA species) from other DBPs (e.g., THM species). In some cases, simple adjustment factors were applied to predictions where data were unavailable for model development (e.g., approximating  $\text{Br}^-$  from DBP-Br, the sum of the  $\text{Br}^-$  present in THM and HAA species).

### ***Case Study Models***

The case study approach involved careful examination and analysis of data associated with each utility and ascertaining simple site-specific correlations and relationships, taking into account chronological changes in treatment and disinfection practices. All such DBP predictions have been well-documented, along with requisite assumptions. A secondary modeling effort was done for groups of plants that treated water from the same or similar watersheds and whose current treatment and disinfection scheme was the same as another plant’s historical treatment and disinfection scheme, permitting predictions of historical DBP exposure for the latter plant.

## **REANALYSIS OF EPIDEMIOLOGIC STUDIES**

The basic exposure matrices in the Ontario and Iowa case-control studies were recalculated in order to provide more precise estimates of TTHM exposure and to provide estimates of exposure to specific THMs and total and specific HAAs. This report presents the reanalysis of bladder, colon, and rectal cancer risks in the Ontario and Iowa case-control studies and comparison to previous results.

### **Reanalysis of Ontario Case-Control Study**

The reestimation of chlorination by-product levels in Ontario and linkage to the Ontario case-control study resulted in a more robust data set. The assignment of TTHM exposure is similar to the original study, and therefore the risk estimates associated with TTHM exposure are similar for bladder, colon, and rectal cancers. A smaller number of subjects was assigned high exposure in the reanalysis, resulting in wide confidence intervals in some analyses. Perhaps the most important contribution of the new exposure database is the opportunity to explore relationships with other by-product representations, including BDCM and total and specific HAAs that were not available in the original study.

One of the objectives of this study was to reduce misclassification of subject exposure. Although the correlation between new and original predictions at the plant level was only moderate, the correlation was higher among large treatment plants, resulting in a high correlation between TTHM exposure and subject residence. In addition, the correlation between new and original exposure assignment is similar for cases and controls, indicating that misclassification in the original study was likely nondifferential. Nondifferential misclassification of exposure tends to make cases and controls more similar with respect to exposure assignment and therefore to bias risk estimates toward observing no effect.

The Ontario monitoring data for the period 1986–1993, used in the original study, reported peak TTHM values. The peak value is on average 80% of the yearly mean value for each water plant. Analyses presented here, using the original (peak) and new (mean) exposure assignment, consider TTHM categories reflecting this contrast. However, the ratio of TTHM estimates by plant in the new (mean) and original (peak) estimates is only 55%. This resulted in fewer exposed subjects in the analysis using new estimates compared to the original estimates. Categories in the analysis of cumulative TTHM-years were determined by the quartiles of the distribution among controls and are therefore not influenced by systematic differences between peak and mean estimates.

The relative risks associated with TTHM exposure obtained in the original study were compared with similar metrics based on the new exposure estimates. In general, results from the reanalysis were consistent with the original study and do not alter the conclusions or interpretation of the original papers.

The new by-product estimates were used for the analysis of several by-product parameters over a 50-year exposure window for the three cancer sites. The analysis of average exposure is the most robust for ensuring adequate sample size in each exposure category. Relative risks for bladder cancer above 1.4 are associated with high average exposure to TTHM, BDCM, HAA5, TCAA, and BCAA. Of note, the largest bladder cancer risks are found for the brominated by-product exposures (BDCM and BCAA). For colon cancer, excess risk is only observed among males. Relative risks for colon cancer approaching 2.0 are observed for TTHM and all of the HAA5 exposures. Risk estimates are lower and not statistically significant for high BDCM exposure. Risk estimates for rectal cancer are consistently close to the null value.

In addition to the results presented here, the new exposure estimates provided by this study result in numerous other opportunities for analysis. Other analyses that could potentially be explored include risk among subgroups (i.e., bladder risk among smokers and nonsmokers), simultaneous modeling of risk associated with different by-products, examination of different exposure windows, analysis restricted to those with homogeneous exposures, examination of colon and rectal risk according to cancer location (i.e., distal colon, proximal colon, sigmoid junction, rectum), and pooling of the Ontario and Iowa data.

This analysis based on new exposure estimates confirms our previously reported results and provides some additional insights with respect to a relationship between specific by-products and cancer risk. In particular, the relationship between bladder cancer and brominated by-products and the relationship between male colon cancer with TTHM and HAAs are noteworthy.

## Reanalysis of Iowa Case-Control Study

In the late 1980s a case control study was conducted in Iowa to analyze the association between DBPs and cancer risk. In order to develop individual indices of lifetime exposure to DBPs, lifetime residential histories were collected from study subjects and linked with historical source and treatment data available for Iowa water treatment utilities. The study evaluated risk with duration of time spent on chlorinated surface water supplies. In addition, average TTHM exposure was calculated, based on crude estimates of past THM levels in Iowa drinking water supplies. In an effort to reduce misclassification in the original TTHM exposure estimates, the current study has been undertaken to improve estimates of past DBP levels in Iowa waters and to reevaluate their associations with cancer risk. Results from the reanalysis, using the current estimates of exposure, are presented in this report.

In the earlier analysis, increased risks for bladder and rectal cancer were reported, but no significant increases were observed for colon cancer. Risk estimates were generally stronger for time spent on chlorinated surface water than for average TTHM levels. When we reproduced our original analysis of average TTHM using the newly estimated average TTHM and the original categories for classification of exposures, risk estimates were obtained for all three sites comparable to those presented originally. The new exposure data permitted establishing strata of more highly exposed persons. In addition, the new estimates of exposure afforded an opportunity to examine risks associated with HAAs as well as with THMs. The strongest and most consistent increases in cancer risk were observed among males for all three sites for the top exposure categories for the nonbrominated THMs and HAAs. Compared to subjects exposed to TTHM  $\leq 0.5$   $\mu\text{g/L}$ , males exposed to TTHM  $> 96.1$   $\mu\text{g/L}$  were at an 80% increased risk of bladder cancer, a 64% increased risk of colon cancer, and a 166% increased risk of rectal cancer. Comparable or higher risks were observed for average HAA6 levels of  $> 52.6$   $\mu\text{g/L}$  compared to very low levels. Risk estimates were not elevated among females for cancers of the colon and bladder. Some increases, although inconsistent, were observed for rectal cancer among females. Increases in risk were observed during the duration of exposure at high DBP levels as well as at average DBP exposure and seem to be the most prominent for subjects exposed at the very highest levels ( $\geq 100$   $\mu\text{g/L}$  TTHM or  $\geq 60$   $\mu\text{g/L}$  HAA6).

The current estimate of historical DBP levels allowed better examination of the very highest-exposed subjects in the study by increasing the ability to detect the known variability in surface water DBP levels. In the earlier analysis, surface water treatment utilities were assigned one of two possible THM levels depending on the point of chlorination in the treatment chain. The reassessment of historical exposure for the current reanalysis involved the consideration of each utility receiving surface or mixed surface and groundwater on a case-by-case basis. Some utilities that used shallow groundwater were also considered individually. Public water systems with deeper groundwater sources were assigned contaminant levels using a central tendency approach, except for those with elevated levels of brominated DBPs. Multiple treatments and water quality parameters were considered in conjunction with actual DBP measurements to develop estimates of past levels. The highest 10% of exposed controls had average TTHM levels ranging from 30 to 154  $\mu\text{g/L}$  for the recalculated lifetime average variable compared to 33 to 74  $\mu\text{g/L}$  for the original measurement of average TTHM. This greater variability in values allowed us to examine subjects exposed at much higher levels than was previously possible, and it is in these upper strata where the most interesting results were observed.



The improvement of exposure assessment among subjects and the resulting reduction in exposure misclassification was one of the primary goals of the current undertaking. The estimation of TTHM and HAA levels on a utility-by-utility basis, using a wide range of information, is an important step forward in advancing our knowledge of historical exposures to DBPs. Using these methods, the variability in DBP levels in surface and mixed surface and groundwater sources was better able to be captured. Although the correlation between old and new average TTHM levels is quite high when the control group is considered as a whole, the correlation between the two measures decreases at higher TTHM levels. Subjects formerly classified at relatively high TTHM levels ( $>40\text{ }\mu\text{g/L}$ ) are less likely to have a comparable exposure in the current study than subjects exposed at lower TTHM levels. Correlations between the old and new TTHM exposures were similar for cases and for controls, providing evidence that misclassification of the original exposure measure was likely nondifferential. Nondifferential misclassification almost always results in a risk estimate that is biased toward the null, and evidence for this type of bias in the current analysis is in the risks observed among the most highly exposed subjects. The previous TTHM estimates did not allow the estimation of risks among subgroups of subjects with similar high TTHM exposures, and a direct comparison of these risks could not be presented. When the previous exposure categories for lifetime average TTHM estimates are used to present a direct comparison of the cancer risks in the previous and current analyses, the effects of the misclassification of exposure are not apparent, as the cancer risks are generally similar among subjects with similar estimated exposures. This indicates little improvement in exposure definition when as a group all subjects above about  $40\text{ }\mu\text{g/L}$  TTHM are considered. However, the increases in risk that were observed when higher ranges of exposure were considered suggests that our refined estimates of exposure at the high end of the exposure range did result in more precise classifications. There is no doubt that a substantial level of misclassification still remains, implying that risks at the high end of the exposure range are likely greater than those that have been calculated.

One of the issues confronting the authors in the reanalysis of the Iowa data was the choice of analysis subset. Since the earlier analyses of these data examined lifetime exposure, exposure was likewise assessed over each subject's lifetime for comparisons with earlier results. To facilitate comparisons with results from the Ontario reanalysis, exposures that occurred during the 50-year period up to 2 years before study entry were considered. The 50-year window yielded a slightly higher number of eligible subjects than those entering the lifetime analyses, since more subjects had identifiable exposure for at least 70% (35 years in this case) of the exposure period under consideration. Fewer subjects met this criterion for the analyses of lifetime exposures due at least partially to a greater level of missing residential histories and more limited information about water sources and treatments in the distant past. This uncertainty about exposure occurring in the distant past may yield worse estimates of DBP levels than those based on more recent time periods. When risk estimates associated with lifetime and 50-year exposures were compared, slightly higher odds ratios associated with DBP exposures occurring during the 50-year time period were found. A more extensive time-window analysis can be undertaken in the future.

Another topic for future analysis would be the examination of several by-products in a single multiple-regression logistic model. In contrast to the original analysis, where past levels of TTHM (the sum of the four THMs) were only estimated, the current study highlighted the estimation of three HAAs (DCAA, TCAA, and BCAA) and two HAA groups (HAA5 and HAA6) in addition to two of the THMs (chloroform and BDCM) and TTHM. Analyses of each by-product or by-product group separately yielded consistent increases in risk for the highest-exposure categories of the nonbrominated THMs and HAA exposure measurements for bladder, colon, and



rectal cancers among males. Risk increases were moderately higher for HAA compounds than for THMs for cancers of the bladder and colon but not for rectal cancer, making it difficult to comment on the relative importance of either chemical group. No consistent risk increases were noted for either of the evaluated brominated compounds: BDCM or BCAA. It would be of interest to examine the correlations between the individual DBPs and to explore the possibilities of examining multiple by-products in one statistical model.

Accurate assessment of exposure is essential in estimating cancer risk that may be linked with occupational and environmental carcinogens. The current results confirm the authors' past findings and suggest some specificity for effects linked with particular classes of by-products or their correlates in the mixture. In particular, when compared with the earlier findings, the higher estimates of risk here for the highest-exposure categories of TTHM and HAAs for male bladder, colon, and rectal cancers are of interest. In addition, the risks seen for the 50-year window are comparable to, and in many instances higher than, risks based on lifetime exposures, suggesting the adequacy of the 50-year estimates.



# CHAPTER 1

## INTRODUCTION

### BACKGROUND

Although the importance of ample drinking water *quantity* for drinking and other purposes has long been apparent, an understanding of drinking water *quality* from the perspective of organic compounds was not well known or documented until recently (U.S. Environmental Protection Agency [USEPA] 2000a, b). Although records throughout history note aesthetic problems associated with drinking water, such as taste or odor, it has taken a considerable time period for people to recognize that their senses alone were not accurate judges of water quality (USEPA 2000a, b).

In earlier times, water was treated to address aesthetic water quality problems. However, as time progressed, scientists discovered drinking water contaminants, especially those not detected with the human eye. Scientists in the 1850s were able to conclude that microbial contamination could be directly correlated with influent water's turbidity. Treatment systems, in the United States and elsewhere, used filtration to reduce turbidity levels and treat microbial contamination.

Although filtration was a fairly effective treatment method for reducing turbidity levels, it was the utilization of disinfectants such as chlorine ( $\text{Cl}_2$ ) that played the largest role in reducing the number of waterborne disease outbreaks in the early 1900s (USEPA 2000a, b). Other disinfectants were also used, such as ozone, chlorine dioxide, and ultraviolet (UV). Although alternative disinfectants were first used in Europe, the United States has begun to implement these into their drinking water plant process trains.

Although water treatment has been shown to significantly reduce the risk of infectious disease outbreaks, other potential health risks may have been introduced. In the mid-1970s, the presence of chloroform ( $\text{CHCl}_3$ ), trihalomethanes (THMs), and other organic chemicals in drinking water was directly linked to chlorination. Scientists were able to demonstrate that disinfectants such as chlorine reacted with natural organic matter present in the source water to form a variety of disinfection by-products (DBPs).

### Epidemiologic Studies of DBPs and Cancer Risks

Epidemiologic studies have examined the association of chlorination by-products and a number of cancer sites. Cancer of the bladder has received the most attention, although cancers of the colon and rectum have also been the subject of multiple studies. The sum of evidence from epidemiologic studies conducted to date supports a modest increase in the risk of bladder cancer associated with exposure to chlorination by-products (Cantor et al. 1998). Studies of colon and rectal cancer are less consistent (Hildesheim et al. 1998; King, Marrett, and Woolcott 2000).

In studies of bladder cancer and chlorination by-products, increases in risk were apparent only after many years of exposure. Stronger risk estimates and more consistent results have been found in studies with the most detailed exposure assessment methods (Lynch et al. 1989; Morris et al. 1992; Cantor 1997; King, Marrett, and Woolcott 2000). These studies point to the importance of taking into account residential and water source histories and volume of water consumed. Even in the most recent studies, measures of exposure to chlorination by-products have been primarily based on surrogate indicators such as chlorinated surface water (King, Marrett, and Woolcott 2000). Variations in chlorinated surface water characteristics contribute to the misclassification inherent in such

assessment methods and do not permit examination of risk associated with varying concentrations of chlorination by-products or cumulative lifetime exposure.

Recent expert panel reviews in Canada and the United States and by the World Health Organization have identified specific research needs, with improved exposure assessment a priority for epidemiologic studies (Mills et al. 1998; World Health Organization 2000). Examination of specific by-products and dose-response patterns are also areas where information is lacking for understanding this relationship (Mills et al. 1998).

## **The Importance of Exposure Assessment**

Because of the changing nature of water quality over time and the complexities associated with assessing individual exposures, epidemiologic studies of cancer risks and drinking water exposures are fraught with uncertainty. For example, exposure misclassification can occur due to study participants erroneously recalling water consumption over long periods or to investigators' inadequate characterizations of historical levels of DBPs over the study period. Studies may under- or overestimate the cancer risks due to either differential or nondifferential exposure misclassification. Nondifferential or randomly distributed misclassification bias almost always leads study results in the direction of not observing an effect (or observing a smaller change in risk than may actually be present). Differential misclassification bias can result in either higher or lower estimates of risk, depending on how the misclassification is distributed. The investigator may acknowledge exposure misclassification bias, but it may be difficult to assess whether the bias was differential or nondifferential. Many investigators assume that exposure misclassification in environmental epidemiologic studies is nondifferential and resulted in the observation of a risk smaller than the "actual" risk. Few studies have evaluated the effect of exposure misclassification in water disinfection studies. Lynch et al. (1989) examined the effects of misclassification of exposure using empirical data from an interview-based case-control study of bladder cancer in Iowa. Bladder cancer risk estimates were found to be higher when more information was known about the study participants' residential histories and their possible exposure to chlorinated water sources. This suggests misclassification bias in epidemiologic studies of chlorinated water may be nondifferential, underestimating the risk.

Poor or incomplete exposure assessment in epidemiologic studies is not the only source of uncertainty. The observed association may be confounded because exposure to chlorinated water and its by-products also results in exposure to other water contaminants. Surface and groundwater sources may be contaminated by naturally occurring and synthetic water-soluble, nonvolatile chemicals, which also may be associated with increased cancer risks.

## **RESEARCH OBJECTIVES**

The primary objective of this project is to provide an improved exposure assessment for the reanalysis of two well-conducted, peer-reviewed epidemiology studies conducted in Iowa and Ontario. These case control studies assessed bladder, colon, and rectal cancer risks that may be associated with chlorinated drinking water and THMs. An improved exposure assessment would embody more complete information on DBP classes and species formed under chlorination and chloramination processes. Cancer has a long latency period, up to several decades. Historical databases typically contain very limited DBP data, and there are no data prior to the 1970s, when DBPs were first discovered. Therefore, this research proposes that, through models and correlations, it

may be possible to estimate past DBP levels based on present trends and knowledge about changes in source water and treatment practices, both in the present and past. Such estimates will provide an improved exposure assessment for the reanalysis of the epidemiology studies.

The general study approach involved the following:

- Assembling historical and present DBP data for Iowa and Ontario from the Center for Health Effects of Environmental Contamination (CHEEC 1998) at the University of Iowa; the Iowa Department of Natural Resources (DNR 1998); and the Ministry of the Environment in Ontario that maintains the Drinking Water Surveillance Program (DWSP) database (Ministry of the Environment 1986–1998)
- Soliciting DBP data and information on source water(s) and treatment practices (present and past) from participating individual utilities throughout Iowa and Ontario
- Assembling and evaluating other historical databases, such as the National Organics Reconnaissance Survey (NORS; Symons et al. 1975), National Organics Monitoring Survey (NOMS; Pojasek 1997), WATER\STATS (AWWA 1996), Water Industry Data Base (WIDB; AWWA 1992b), 35-Utility Study (Krasner et al. 1989), and the Information Collection Rule (ICR) database (McGuire, McLain, and Obolensky 2002)
- Using existing and developing new models and correlations to estimate missing data on historical DBP occurrence
- Using results from the more rigorous modeling of total trihalomethanes (TTHM) exposures, several reanalyses were conducted for two previously reported case-control epidemiologic studies of bladder, colon, and rectal cancer risks in Iowa and Ontario to compare the previously and newly reported cancer risks and assess the effect of exposure misclassification on the previously reported study results
- Considering exposure to specific THM species and other species of DBPs of health concern (i.e., haloacetic acids), in addition to conducting an improved exposure assessment of TTHM exposure
- Conducting a separate analysis of the cancer risks for populations exposed only to surface water in Ontario, where significant populations were exposed to surface waters with relatively low levels of DBPs

## **Significance of Research**

The two well-conducted and peer-reviewed epidemiology studies (Cantor et al. 1998; King and Marrett 1996), summarized in the next chapter, were based only upon TTHM formation. This study is the first to attempt to perform an improved exposure assessment and should be beneficial in assisting the water industry in reanalyzing other epidemiology studies—both cancer (chronic) and reproductive and developmental (acute). A clear identification of carcinogenic compounds and the water quality and treatment conditions under which they are formed would provide insight into control and prevention strategies. More importantly, this study will address the fundamental issue of how strongly the water industry relies on chlorine for disinfection, and whether estimates of cancer risk associated with consumption of chlorinated drinking water are greater or less than currently thought.

## **METHODS**

This study highlights the four individual chlorinated and brominated THM species (regulated in the United States as the sum of the four species) and the nine chlorinated and brominated

haloacetic acid (HAA) species (regulated in the United States as the sum of five of the species). State and provincial as well as national DBP databases were assembled.

Basic statistics, such as median, average, and percentile values, were calculated using Microsoft Excel (1997, Microsoft Corp., Redmond, Wash.), a spreadsheet and data analysis program. To obtain data from the ICR database, Microsoft Access (1997, Microsoft Corp., Redmond, Wash.), a database management tool, was used. For the modeling efforts, the statistical program Statistica (Version 5.5, StatSoft, Tulsa, Okla.) was utilized. This is a comprehensive, integrated statistical data analysis, graphics, database management, and custom application development system. Statistica was used to develop both linear and nonlinear and simple and multiple regression models as a basis for the improved exposure assessment. This approach led to the development of central tendency models. As a complement to the central tendency models, a unique case study modeling approach was employed to provide better DBP predictions for higher and lower DBP levels.

## STUDY OUTLINE

The goal of this study was to provide an improved exposure assessment and the results of a preliminary reanalysis of two well-conducted epidemiology studies investigating the risk of bladder, colon, and rectal cancer associated with drinking water. The study went through several iterative steps to reach the improved exposure assessment.

First, as detailed in chapter 2, an extensive literature review was performed. The goal of the review was to provide information about DBP regulatory history and development, the health effects associated with DBP formation and exposure, disinfection practices and their effects on DBP formation, DBP control strategies, and DBP modeling. Although the literature review provided information about these topics, the most important role was providing DBP occurrence levels in the United States and Canada, illustrating both current and past trends.

After the literature review was compiled, several national (primarily U.S.) databases were assembled. The national databases, used to provide insight into both present and historical DBP occurrence, tended to be large and contained significant amounts of data, especially concerning DBPs, DBP precursors, and disinfection practices. The national databases can be used to evaluate trends in regulatory compliance.

However, for developing robust models and correlations for linking DBPs and DBP species to water quality and treatment, the use of national (U.S. and/or Canadian) central tendency models may not always be appropriate for improving exposure assessment in specific regions of North America, especially since the two epidemiology studies that were to undergo the improved exposure assessment were site-specific studies. This national database “bias” led to the assembling of site-specific database for Iowa and Ontario. These site-specific databases were employed to augment the national databases and helped discern past and present trends in DBPs and disinfection and treatment practices in Ontario and Iowa.

Once the national and site-specific databases were assembled and analyzed, models were created to predict DBP formation under a variety of conditions. The models were then used to construct a DBP exposure time line for both Iowa and Ontario that spanned a 50-year period up to 2 years prior to diagnosis of cases or completion of questionnaires by controls. A 50-year time period was selected because cancer induction takes several decades to occur and develop. Models employed take two forms: central tendency and case study (utility-specific). With the improved exposure assessment that evolved in this research, a basis was provided for the reanalysis of the two subject epidemiology studies.

## **CHAPTER 2**

### **LITERATURE REVIEW**

#### **INTRODUCTION**

As stated earlier, drinking water has undergone many different treatment methods throughout time. Methods have advanced from earlier times, when ancient Sanskrit and Greek writings recommended methods such as filtering water through charcoal, exposing it to sunlight, and boiling and straining the water, to modern technologies that include membranes and alternative disinfectants such as ozone and UV light. Currently, instead of undergoing treatment for primarily aesthetic problems, water is now being treated for microbial contamination. However, such treatment has its drawbacks, including the formation of potentially harmful DBPs. As stated earlier, DBPs form when disinfectants react with naturally organic matter found in the source water. Several classes of disinfectants can form, such as THMs, HAAs, ketones, chlorate, chlorite, bromate, cyanogen chloride (CNCl), and a variety of others. In addition to natural organic matter, bromide ( $\text{Br}^-$ ) can also react with the disinfectant (e.g., chlorine), serving as an inorganic DBP precursor. DBP formation, however, can be controlled somewhat through treatment plant operations by limiting DBP precursors. Alternative disinfectants and treatment methods can also be employed to limit DBP production.

This literature review includes information for the following:

- DBP regulatory history and development
- DBP occurrence
- Health effects associated with DBP formation
- Disinfection practices
- DBP control strategies
- DBP modeling
- Epidemiology reanalysis

#### **DBP REGULATORY HISTORY AND DEVELOPMENT**

In the United States, federal regulations for drinking water were first enacted in 1914. The U.S. Public Health Service set standards for several known disease-causing microbes. These standards were revised and expanded, and eventually, with minor modifications, all 50 states adopted the final 1962 Public Health Service standards as either regulations or guidelines (USEPA 2000c). However, during the 1960s, human-made chemicals were found in public drinking water. This phenomenon caused both local and federal officials to investigate the extent of drinking water contamination throughout the nation. Two main monitoring studies were conducted—NORS in 1975 and NOMS in 1976–1977—that found chlorination by-products in drinking water sources across the nation. This eventually led to the passage of several federal laws in the 1970s, the first of which was the Safe Drinking Water Act (SDWA) of 1974 that was administered by the USEPA. The main goal of the SDWA is to ensure that public water supplies meet national standards that protect consumers from harmful contaminants.

USEPA was given authority to regulate drinking water through two primary methods. The first step involved the creation of national interim primary drinking water regulations



which, under congressional direction, were based largely on the 28 standards of the 1962 Public Health Service (USEPA 2000c). The second step included the revision of these standards. In 1975, 18 interim standards were enacted, and in 1976, standards for radionuclides were set. Finally, in 1979, a standard for TTHM was enacted and designated as the 1979 THM Rule. USEPA also enacted guidelines for secondary drinking water contaminants such as chloride, color, copper, corrosivity, foaming agents, iron, manganese, odor, pH, sulfate, total dissolved solids (TDS), and zinc (USEPA 2000c).

The SDWA underwent another significant change in 1986, when amendments required the USEPA to set maximum contaminant levels (MCLs) and MCL goals for 83 named contaminants. At the same time, the standards created in the original SDWA would be enforced as the final drinking water standards. In 1988, the SDWA was amended to include the Lead Contamination Control Act, created to eliminate lead-containing drinking water coolers in American schools (USEPA 2000c). The SDWA underwent its last amendment in 1996. The amendments emphasized comprehensive public health protection through risk-based standard setting, increased funding, reliance on the best available science, prevention tools and programs, strengthened enforcement authority for the USEPA, and public participation in drinking water issues (USEPA 2000c).

In the late 1990s, USEPA collected data from 296 public water systems that each served more than 100,000 people from July 1997 to December 1998 and compiled the data into a robust database designated the ICR database. The goal of the database was to provide USEPA with pertinent information regarding chemical by-products (DBPs) that form when disinfectants used for microbial control react with chemicals already present in source water (natural organic matter and bromide). From this database, Stage 1 of the Disinfectants/Disinfection By-Products (D/DBP) Rule and the Interim Enhanced Surface Water Treatment Rule were promulgated. Stage 1 of the D/DBP Rule, which was proposed in 1994 and became effective in 1998, dictated that utilities use treatment technologies to reduce the formation of DBPs. The utilities were required to meet the following standards:

- TTHM:  $\leq 80$  parts per billion (ppb)
- HAA5 (the sum of five HAA species: trichloroacetic acid [TCAA], dichloroacetic acid [DCAA], monochloroacetic acid [MCAA], dibromoacetic acid [DBAA], and monobromoacetic acid [MBAA]):  $\leq 60$  ppb
- Bromate:  $\leq 10$  ppb
- Chlorite:  $\leq 1.0$  parts per million (ppm)

The standards for TTHM, HAA5, and bromate are annual averages. A system is considered to be in violation if the running annual average of any consecutive four-quarter period exceeds the set MCL. Stage 1 of the D/DBP Rule also sets maximum residual levels for disinfectants, including chlorine  $\leq 4$  mg/L as free chlorine, chloramines  $\leq 4$  mg/L as chlorine, and chlorine dioxide  $\leq 0.8$  mg/L. Large surface water plants had to comply with these standards by the year 2002, whereas smaller surface water plants and all groundwater plants had to meet the standards by 2004. Data from the ICR database were used to consider further DBP control under the Stage 2 D/DBP Rule. The Stage 2 MCLs for TTHM and HAA5 have remained at 80 ppb and 60 ppb, respectively, but are now based on a more specific compliance location (a distribution system maximum).



## DBP OCCURRENCE

In 1974, the USEPA coordinated a nationwide survey to determine the quality of drinking water across the nation for organic chemicals and the potential health effects they may incur. Eighty cities were selected and monitored during the NORS, which had three main objectives:

1. To determine the occurrence of the four THM species ( $\text{CHCl}_3$ , bromodichloromethane [BDCM], dibromochloromethane [DBCM], and bromoform [ $\text{CHBr}_3$ ]), 1,2-dichloroethane, and carbon tetrachloride in finished water
2. To determine the effect of raw water sources and water treatment practices upon the formation of DBPs
3. To characterize the organic content of finished drinking water

Upon completion of the monitoring and sampling analyses, it was first discovered that THMs were not evident in a majority of the raw untreated waters sampled. However, all of the finished waters contained  $\text{CHCl}_3 > 0.1 \text{ mg/L}$ . Although a number of finished waters did not contain BDCM, DBCM, or  $\text{CHBr}_3$ , their presence was frequent enough to be considered widespread throughout the finished waters of the United States (Symons et al. 1975). For 1,2-dichloroethane and carbon tetrachloride, the species were not found in approximately 68% and 88%, respectively, of the sampled waters. In about one third of the cases where these compounds were present in the finished water, they were also present in the raw water, indicating they were environmental contaminants and were not formed during water treatment (Symons et al. 1975).

For the second objective, an initial examination of the data indicated that the dominant factor influencing the creation of chlorination by-products was the general organic matter in the water, provided sufficient chlorine was added to produce a chlorine residual at the time of sampling (Symons et al. 1975). It was also observed that other factors influenced DBP formation, such as the following:

- *Source influence:* Groundwater sources formed less DBPs than surface waters.
- *Treatment influence:* Raw water chlorination plants tended to form higher levels of DBPs, which was associated with the chlorine dose. Higher THM levels were also found in plants that employed precipitative softening.

From this study, it was concluded that THMs are widespread contaminants that can be found in drinking water sources around the nation and are the result of disinfection. In general, the TTHM concentrations were related to the organic matter content of the water, as measured by nonvolatile total organic carbon (TOC), when sufficient chlorine was added to create a chlorine residual (Symons et al. 1975). It was also determined that for surface waters that were prechlorinated and formed a sufficient chlorine residual, THMs were formed at higher concentrations.

After the NORS was completed, USEPA conducted the NOMS from 1976 to 1977 to identify contaminant sources, to determine the frequency of occurrence of specific contaminants in drinking water supplies, and to provide data for the possible establishment of additional maximum contaminant levels of organic compounds in drinking water (Pojasek 1977). The study surveyed 113 public water supplies that encompassed a variety of water sources and treatment practices across the nation over an 18-month sampling period. Samples were analyzed for naturally occurring organic matter, contaminants formed through treatment (THMs), and synthetic

chemicals from point and nonpoint sources. Compounds that were analyzed in the study, specific to this research's interest, were  $\text{CHCl}_3$ , BDCM, DBCM, and  $\text{CHBr}_3$ . The NOMS study was able to conclude that THMs occur on a regular basis in finished drinking waters and that a substantial effort was still required to precisely evaluate the THM concentrations that can reach the tap of a consumer in a given water system (Pojasek 1977). As with the NORS, this study was also able to conclude that surface waters tended to form higher amounts than groundwater systems of THMs.

As a result of the SDWA 1986 amendments, the USEPA was required to develop a priority list of chemicals of potential health concern that were present in drinking water. THMs were included on this list. The 35-Utility Study, conducted for USEPA and the California Department of Health Services, focused on 25 water utilities located across the United States and 10 utilities in California (Krasner et al. 1989).

During the first year of the project, 1988, baseline data were gathered for the utilities. Samples were analyzed for DBPs such as THMs, haloacetonitriles (HANs), haloketones (HKs), HAAs, chloropicrin (CP), chloral hydrate (CH),  $\text{CNCl}$ , and several others. The median THM (TTHM) values for the four sampling quarters were found to be 34, 44, 40, and 30  $\mu\text{g/L}$ , respectively. These data were compared with the THM values obtained in a survey of 727 utilities nationwide conducted for the Awwa Research Foundation (AwwaRF) in 1987 of data from 1984 to 1986 (McGuire and Meadow 1988). The 35-Utility Study also concluded that seasonal temperatures affected THM formation, and the highest levels occurred during the warmer months. Further examination of the data showed that, on a weight basis, THMs were the largest class of DBPs detected in the study (Krasner et al. 1989). The second largest class was HAAs. After high levels of brominated species were observed, bromide and chloride analyses of each plant's influent water source were made. For the 35 utilities, bromide levels ranged from  $<0.01$  to 3.00 mg/L. When high bromide contents were observed, THM formation shifted from the chlorinated species to the brominated species. This also occurred with the other DBP classes, specifically HAAs and HANs.

In the late 1980s, AwwaRF conducted a survey to determine the extent and costs of compliance with the existing MCL for TTHM, which was set at  $\leq 0.10$  mg/L and promulgated with the 1979 THM Rule. A questionnaire was sent to all utilities that served populations greater than 10,000 and were required to monitor THMs as stated by the SDWA. It was found that, on average, the MCL of 0.10 mg/L had reduced concentrations of THMs in larger utility systems by 40% to 50% (McGuire and Meadow 1988).

Overall, the 35-Utility Study was able to determine a median TTHM value of 39  $\mu\text{g/L}$  in all of the waters sampled. The THMs were the largest fraction of DBPs formed, with HAA5 being the second largest with an overall median of 19  $\mu\text{g/L}$ . When the DBP data were evaluated for correlations among various parameters, TTHM were found to have a high correlation with the sum of halogenated DBPs (Krasner et al. 1989). Although chloramination use by utilities was observed to limit the formation of THMs and other DBPs,  $\text{CNCl}$  was found to significantly increase in these systems. Increased influent bromide content was shown to shift DBP formation from chlorinated DBPs to brominated DBPs.

According to the D/DBP Rule, most utilities are required to reduce influent TOC concentrations prior to disinfection. Currently, the best available technology to meet the Stage 1 requirements for THM and HAA5 MCLs includes enhanced coagulation (or enhanced softening) or granular activated carbon (GAC) adsorption. To meet the chlorite and bromate requirements, the best available technologies would include controlled application of chlorine dioxide and ozonation (Arora, LeChevallier, and Dixon 1997). In 1994, the American Water System conducted a

national survey to evaluate DBP occurrence with respect to the D/DBP Rule. The survey also evaluated the impact of source supply, treatment practices, and the effect of seasonal variation on DBP formation. GAC was reviewed to determine if it could be effectively used to control DBP formation. Raw water and filtered water samples were collected and analyzed from approximately 60 plants. From the analysis, it was first determined that water temperature, raw water TOC values, and distribution system THM and HAA5 concentrations were highest in summer, somewhat lower in the fall, and lowest in the winter, verifying the long-established relationship that temperature and precursor concentrations (measured by TOC) directly affect DBP formation (Arora, LeChevallier, and Dixon 1997). Therefore, treatment strategies can be changed seasonally to minimize DBP formation. Although surface water systems tended to show higher THM and HAA5 concentrations, groundwater systems tended to have minimal DBP formation. It was also concluded that GAC systems did not effectively reduce THM formation, because the GAC possessed a low affinity for THM species. Therefore, it was concluded that approximately 20% and 66% of the American Water System plants would exceed the Stage 1 MCLs and potentially more restrictive MCLs of 80 µg/L THM and 40 µg/L HAA5, respectively (Arora, LeChevallier, and Dixon 1997).

## **HEALTH EFFECTS ASSOCIATED WITH DBP FORMATION**

Chlorination by-products can be found in the vast majority of drinking water systems around the nation. Chloroform, a THM species, is a known carcinogen. This raises the possibility that the time-tested benefits of using chlorine to control infectious disease may be, in part, offset by increased cancer risk in continuously exposed populations (Cantor et al. 1985). In 1981, Wilkins and Comstock (1981) performed a study investigating the source of drinking water and site-specific cancer incidence in Maryland. Vital records and nonofficial census data for more than 31,000 people were used to compute selected sex- and site-specific cancer incidence rates in a well-defined county population. Incidence rates for cancer of the bladder among men and for cancer of the liver among women were nearly twofold higher in the drinking water cohort that had been supplied chlorinated surface water at home when compared to the cohort with a history of consumption of unchlorinated groundwater.

In 1985, results were published from another case-control study of bladder cancer in Iowa performed by Cantor et al. (1985). The researchers studied eligible candidates who were diagnosed with bladder cancer in 1978. A year-by-year record of water source and treatment was created for each study respondent. After conducting their analysis, the researchers were unable to detect an increase in bladder risk among persons exposed to chlorinated drinking water sources compared to those exposed to nonchlorinated drinking water sources. The results also showed that smokers exposed to nonchlorinated drinking water had a higher bladder cancer incidence compared to smokers exposed to chlorinated drinking water. However, the researchers observed that the risk of bladder cancer increased with the duration of exposure to chlorinated surface water among persons otherwise at lowest risk and that this was consistent across the sexes.

Cantor et al. (1998) performed another population-based study of bladder cancer in Iowa from 1986 to 1989 to evaluate the risk posed by tap water containing chlorinated DBPs. The study consisted of more than 1,100 cases and 1,900 controls who had spent 70% of their lifetime at the same drinking water source. The researchers found bladder cancer risk increased with duration to chlorinated groundwater use, as well as with total duration of chlorinated drinking water use (surface plus ground). This proved that the duration of exposure to DBP is more important than

the actual DBP level. It was also found that, among nonsmokers in both men and women, an association between drinking water and bladder cancer did not exist. However, among men, smoking and exposure to chlorinated DBPs mutually enhanced the risk of bladder cancer.

While the previous studies investigated the risk of cancer associated with long-term exposure to DBPs in chlorinated drinking water, Savitz, Andrews, and Pastore (1995) evaluated the risk associated with THM levels in drinking water and miscarriage, preterm delivery, and low birth rate. While the researchers were unable to characterize changes in water consumption during pregnancy, the researchers were able to consider THM changes over time. The dates of pregnancy were used to assign the reported quarterly average THM value from the appropriate utility supplier as the subjects' exposure classification. From their results, it was observed that women who consumed bottled water were at higher risk than women who consumed tap water. Women who also reported that they did not drink any water were at the highest risk, and those drinking the largest amounts of water were at a slightly decreased risk. For THM-associated risk, preterm delivery showed virtually no association with water source, THM concentration, or THM dose. It was concluded that, overall, drinking water was not related to the risk of adverse pregnancy outcome.

Although past research tended to focus on long-term cancer and acute health effects such as spontaneous abortion, Itoh and Matsuoka, in 1996, investigated whether DBPs in drinking water contributed to activity-inducing chromosomal aberrations. The first goal of the study was to measure nitro and carbonyl groups in waters treated with disinfectants such as chlorine, chlorine dioxide, chloramines, and ozone. The second goal was to investigate the relationship between mutagenic activity and DBPs, focusing on nitro and carbonyl groups. The contribution of carbonyl group to activity-inducing aberrations was found to be large, while the contribution of the nitro group was found to be small. It was also found that activity-inducing aberrations of chlorinated water, including the same amount of carbonyl groups, were demonstrated to reach approximately 10 times those of waters treated with the other three types of disinfectants.

Waller et al. (1998) examined exposure to THMs and the risk of spontaneous abortion in 1998. More than 5,000 women were included in the study. THM levels were back-calculated to the first trimester of each study subject. A weak association was discovered between drinking tap water and the risk of spontaneous abortions. The researchers believed that limitations of the study might have led to some misclassification of a woman's exposure. Average TTHM exposure was measured using several sampling sites within a utility. THM levels can vary over short time periods during the day, and most of the samples in the study were based on a single day's analysis. This largest misclassification may have occurred when the study could not fully characterize the exposure to THMs via routes other than ingestion, such as washing dishes and clothes and bathing.

Although all of the aforementioned studies examined DBP exposure and its correlation with adverse health risks, Black et al. (1996) investigated the reduction of cancer risks by improving organic carbon removal. As mentioned earlier, organic carbon present in water as natural organic matter is a precursor for DBP formation. Therefore, controlling natural organic matter prior to treatment is an important strategy for DBP control. More than 3,800 water utilities (approximately 60%) could use this strategy to comply with the Stage 1 regulation (Black et al. 1996). A computer simulation was used on a case-specific basis to evaluate the effect of water quality on the formation of THMs and their associated cancer risks. The modeling results indicated that the concentrations of brominated DBPs formed during chlorination increased, and the organic carbon concentrations decreased. However, the overall theoretical cancer risk corresponded with a decrease in organic carbon. Therefore, reduced risk can be achieved by utilities switching their current coagulation practices to enhanced coagulation for optimal organic carbon removal.



It is a well-known fact that, although disinfection of drinking water significantly reduces the risk of microbial infection, it results in the formation of DBPs that may pose health risks to the public consumers. Some disinfectants are believed to have toxicological effects, both long-term (cancer) and acute (adverse reproductive effects). To balance this effect, utilities need to minimize the levels of potentially toxic disinfectants and DBPs in treated water while maintaining adequate protection of the distribution system against microbiological contamination (Glaze et al. 1993). Therefore, the microbial risks have to be carefully balanced and weighed against disinfection risks to achieve minimal adverse health risks.

## DISINFECTION PRACTICES

For the last several decades, chlorine has been the primary disinfectant used for treating drinking water. As stated earlier, chlorine readily reacts with natural organic matter present in the source water to form DBPs. Therefore, risk trade-offs have to be considered between disinfection (inactivation of microorganisms) and the production of DBPs (Lykins, Koffskey, and Patterson 1994). To address this problem, alternative disinfectants are being utilized. Primary disinfection is utilized in the treatment plant to achieve complete inactivation of target microbial contaminants such as *Cryptosporidium* and *Giardia*. Primary disinfectants include chlorine, chloramines, chlorine dioxide, ozone, and UV light. Secondary disinfection is utilized to maintain a disinfectant in the distribution system to inhibit any additional microbial growth. Secondary disinfectants include chlorine and chloramines.

Lykins, Koffskey, and Patterson (1994) evaluated chlorine and the alternative disinfectants ozone and chlorine dioxide over a 1-year time period in terms of THMs, HAAs, HANs, HKs, CH, and CP. The results of this study showed that the combination of pre- and postdisinfectants that produced the lowest levels of halogenated DBPs were preozonation and postchloramination. However, the researchers noted that, although ozone may be a useful primary disinfection strategy, the assimilable organic carbon (AOC) must be removed prior to reaching the distribution system to control microbial growth. Ozone may also produce other DBPs, such as aldehydes, ketones, and carboxylic acids. If bromide is present, ozone may also promote the formation of bromate, significantly limiting ozone's use, because bromate is stringently regulated. Therefore, many drinking water plants are using alternatives to chlorine in disinfection treatment, and many others are contemplating switching to an alternative.

Norton and LeChevallier (1997) conducted a study on chloramination and its effect on distribution system water quality. Two treatment facilities were extensively monitored as the utilities switched from free chlorination to chloramination in the distribution system. From laboratory results, free chlorine and monochloramine were found to be equally effective for bacterial inactivation. However, the results for coliform occurrence were different for the two utilities. For the week prior to the switchover, 25% of one utility's distribution system samples were positive for coliforms whereas, after one week, the utility was coliform free. At the other utility, however, coliform bacteria were not completely eliminated. Although coliform occurrence decreased, bacteria were still detected. This was explained by the fact that this utility had a higher AOC concentration. While chloramination was effective for microbial control, the implementation of chloramines resulted in an overall decrease in DBP concentrations at both utilities. Therefore, chloramines can result in substantial decreases in coliform occurrences and DBP formation compared to similar treatment using chlorination.

## DBP CONTROL STRATEGIES

Although it is well-known that DBPs form in chemically disinfected drinking water, little is known about the DBP species, specifically regarding toxicity, both chronic and acute. Therefore, there are three primary approaches to solving the problem of DBP formation. One is to eliminate DBP formation through precursor removal. The second is to allow the DBPs to form and then remove the DBPs through additional processes (Marhaba 2000). The third approach considers the implementation of alternative disinfectants to reduce DBP formation. However, it is important to note that alternative disinfectants may have some disadvantages. For example, some can produce additional DBPs, such as bromate, chlorite and chlorate, and CNCl (Chen and Rest 1996).

In 1998, Carlson and Hardy investigated DBP formation under several varying water qualities. The study investigated how common treatment variables influence the formation of THMs and HAAs. From the analysis, several conclusions were drawn:

- As chlorine contact time increased, total THM and HAA concentrations increased.
- For reaction times less than 30 minutes and a pH that ranged from 5.5 to 7.0, THM formation appeared to increase; pH levels >7.5 led to a less significant increase in THM formation.
- Contrary to previous work, pH had a variable effect on HAAs.
- THM and HAA formation varied linearly with temperature.
- As chlorine dosages were increased, THM formation increased.
- HAA concentrations also corresponded to the chlorine dose increase.
- A positive linear relationship between TOC and THM concentration was found.

## DBP MODELING

Modeling DBPs has been approached in two ways: using empirical models or kinetic models (Koch et al. 1991; Harrington, Chowdhury, and Owen 1992; Garcia-Villanova et al. 1997; Clark 1998; Nokes, Fenton, and Randall 1999; Westerhoff et al. 2000). Empirical models tend to be based on water quality parameters, water chemistry, and treatment processes. When conducting their research on the formation, evolution, and modeling of THMs, Garcia-Villanova et al. (1997) based their empirical THM model on chlorine-to-precursor molar ratio, pH, temperature, and reaction time. These models are useful when examining broad trends in DBP formation. Conversely, kinetic models are based upon rate expressions. While informational, these models are very limited due to lack of information on the actual kinetics, in terms of reaction order(s) and rate constant(s).

In 1992, the USEPA Water Treatment Plant model was created to simulate DBP formation (individual and total THMs and several HAAs), natural organic matter, inorganic water quality changes, and disinfectant decay in water treatment processes (Harrington, Chowdhury, and Owen 1992). The model was developed to simulate the mean performance of water treatment processes in the United States. Although the original model had several limitations, Solarik et al. (1999) developed new algorithms to extend the model's predictive ability for conventional treatment processes, as well as for advanced treatment processes and alternative disinfectants.

## EPIDEMIOLOGY REANALYSIS

The goal of this project was to reanalyze two well-conducted epidemiology studies: one from King and Marrett (1996) and King, Marrett, and Woolcott (2000); and the other in Cantor et al. (1997). These two studies were selected because they provided the opportunity to study the effects of DBP exposure in two separate locations where DBP formation was dramatically different. These studies were both well-conducted and peer-reviewed, and were based on what was considered to be an adequate exposure assessment.

### **King and Marrett Epidemiology Study (1996)**

The King and Marrett (1996) study examined the relationship between bladder, colon, and rectal cancers and exposure to chlorination by-products in household water supplies. A population-based control study was conducted in Ontario, Canada. Cases of individuals aged 25 to 74 years and diagnosed with cancer of the bladder, colon, or rectum between September 1994 and May 1994 were identified using the Ontario Cancer Registry at the Ontario Cancer Treatment and Research Foundation. Controls were an age-gender frequency-matched sample of the population in the same area and were selected randomly from a computerized database of residential phone number listings. The main analysis considered 696 bladder, 754 colon, and 639 rectal cancer cases, and 1,545 controls with 30 or more years of estimated exposure data. A computer-assisted telephone interview was conducted and information was ascertained on the exposures of interest (residence and water source history, tap water consumption) and potential confounders (gender, date of birth, education, smoking history, normal diets, and body mass index).

Subject residence and water source data were augmented by water treatment data that were collected through a mailed survey of historical treatment practices at plants serving the study area between the operational years of 1950 to 1990. Treatment information was reported as an average day in August in 5-year intervals. Each water supply was characterized by three parameters: source (surface versus ground), chlorination status (chlorinated versus nonchlorinated), and levels of DBPs (estimated summer TTHM level). Source information and chlorination status were determined through the treatment surveys. Past levels of DBPs were represented by historical TTHM levels using a model that was developed to predict TTHM formation in treated drinking waters.

The model was developed using data contained in the DWSP between the years 1988 and 1992. Data from 114 treatment plants were incorporated into the model. Predictors, including raw water characteristics (source, depth of intake pipe, and water temperature), pretreatment procedures (prechlorination or prechloramination), treatments employed (coagulation, polyelectrolytes, or activated carbon), and posttreatment procedures (postchlorination, postchloramination, or dechlorination) were selected to develop the model. Separate models were created for different types of utilities: surface water source with prechlorination, surface water without prechlorination, and groundwater source. These three models allowed the researchers to estimate historical TTHM levels for each plant in the study.

An individual's exposure was estimated by linking a subject's residence and water source history to the relevant treatment-plant data by time and geographic area. Duration of exposure was calculated by summing the number of years in each exposure category. Although the analysis was conducted over a 40-year time period, to limit error, the analysis was restricted to subjects with a known water history of 30 years or more.

From the analysis of water source, it was concluded that chlorinated surface water was used for 35 or more years by 35% of the controls and 45% of the cases. Among the controls, exposure for 35 or more years to estimated THM levels  $\geq 25$ , 50, and 75  $\mu\text{g/L}$  is observed for 14%, 5%, and 4% of the control subjects, respectively. For the cases subjects, at the same exposure time period, the percentages are 21%, 8%, and 6%, respectively.

A pattern of increasing risk with the years exposed to chlorinated surface water was observed, where exposure to chlorinated surface water for 35+ years resulted in an odds ratio (OR) of 1.41 with a 95% confidence interval (CI) of 1.09–1.81, compared to subjects exposed to chlorinated drinking water for 10 or less years. Risk estimates also tended to increase for each of the TTHM measurements. However, only the 35+ years of exposure resulted in significantly higher ORs. A statistically higher increase in bladder cancer risk was also observed for the highest quartiles of cumulative exposure compared to the lowest quartile of exposure. Subjects exposed to chlorinated surface water for 30+ years (OR = 1.39 with a CI = 1.09–1.79) also showed a significant increase in risk when compared with subjects exposed to a groundwater source. A pattern of risk between the volume of water consumed and duration of exposure could not be defined. However, statistically significant risk estimates representing more than a doubling of risk were observed for those individuals with 35+ years of exposure who consumed 1.54–2.08 L of tap water per day (OR = 2.58 with a CI = 1.28–5.21) or >2.08 L per day (OR = 2.28 with a CI = 1.12–4.67).

Among males, colon cancer risk was associated with cumulative exposure to THMs, duration of exposure to chlorinated surface water, and duration of exposure to a THM level >50  $\mu\text{g/L}$  and 75  $\mu\text{g/L}$ . Males exposed to chlorinated surface water with an estimated THM level of 50  $\mu\text{g/L}$  for 35 to 40 years had an increased risk of colon cancer compared with those exposed for less than 10 years (OR = 1.53, with a 95% CI = 1.13–2.09). Males exposed to an estimated THM level of 75  $\mu\text{g/L}$  for 35 or more years had double the risk of those exposed for less than 10 years (OR = 2.1, CI = 1.21–3.66). In contrast, these relationships were not observed among females. No relationship was observed between rectal cancer risk and any of the measures of exposure to chlorination by-products.

The results from the King and Marrett study indicated that there was an increase in the risk of bladder cancer associated with exposure to chlorinated surface water. The duration of the exposure appeared to be a direct indicator of the risk, as the researchers only started finding slightly elevated rates of risk after 20+ years of exposure, with the highest risks occurring with exposure rates greater than 35 years. An excess risk of colon cancer among males was observed with long-term exposure to chlorination by-products. No risk of colon cancer was observed for females, nor of rectal cancer for males or females.

### **Cantor et al. Epidemiology Study (1998)**

The study performed by Cantor et al. (1998) studied the incidence of bladder cancer associated with drinking water in Iowa. Identified through the State Health Registry of Iowa, cases were Iowa residents between the ages of 40 and 85 years who were diagnosed with bladder cancer between the years 1986 and 1989. Controls were randomly selected, and frequency matched to cases by sex and 5-year age groups. Controls under the age of 65 were selected using the state's driver's license records, and controls 65 and older were selected from listings of the Health Care Financing agency. Persons who reported any previous cancer other than basal or squamous cell skin cancer were excluded as controls. The study was conducted in two separate phases. The first



phase (1986–1987) entailed six cancer sites, including bladder, and the second phase was restricted to bladder cancer cases only with a control series.

After obtaining consent for the cases, study participants were sent a questionnaire asking for demographic information, smoking history, and various lifestyle and medical issues. Questions were also asked about frequency of fluid consumption, both inside and outside the home, of beverages using tap water, such as water per se, coffee, hot and iced tea, reconstituted fruit juices, fruit drinks made from powdered mixes, and soups from concentrate or dry mix; and other beverages. Also requested were the dates, geographic locations, and water sources for each place of residence and each place of employment for jobs held at least 5 years. The study was able to obtain 1,452 bladder cancer cases and 817 controls younger than 65 years of age and 1,617 controls over the age of 65.

During the spring and summer of 1987, all Iowa utilities serving more than 1,000 people were surveyed for information on the utility's source water and treatment information since the utility was built and implemented. Overall, the survey was able to acquire information for 345 utilities that served a total population in 1980 of 1.94 million (the overall state population equaled 2.92 million). Also, each utility was sampled for the four THMs.

Exposure for each respondent was calculated using information from the participant's utility survey, THM measurements, and personal questionnaire data. Two classes of exposure index were created. The first accounted for water use from chlorinated surface sources, chlorinated groundwater sources, and any chlorinated water source. The second consisted of estimated lifetime intakes of THMs. THM exposure was estimated by summing the concentrations of the four THM species for those utilities that had a single source and one type of treatment. Approximately 222 utilities fit these criteria. THM levels were found to be generally higher in surface waters than in groundwaters, higher in shallow wells than in deep wells, and higher when chlorine was added at an earlier stage (prechlorination) than only at a later stage (postchlorination). Past levels of THMs were estimated by applying the geometric mean levels to the respective type of water source(s) and treatment(s) used historically.

Similar to the King and Marrett study, ORs were used to estimate the association between THM exposure and bladder cancer risk. Results were adjusted for smoking, high-risk occupation, education, age, study period, and sex. Little association of risk was found between THMs and bladder cancer in both men and women. [Table 2.1](#) shows ORs by increasing duration of exposure to chlorinated surface water, chlorinated groundwater, and any chlorinated water.

Risk increased among men but not women with duration of chlorinated surface water, of chlorinated groundwater, and of any chlorinated water source. Therefore, overall, ORs increased with each of the measures of exposure duration to chlorinated water sources. The association between bladder cancer risk with duration of chlorinated surface water was present only among ever-smokers. There was no evidence of elevated risk among never-smokers.

**Table 2.1**

**ORs and 95% CIs for bladder cancer (by gender and duration of residences served by chlorinated surface water, chlorinated groundwater, or all chlorinated water sources; and by estimates of total lifetime THM intake from water and lifetime average TTHM level)**

Exposure measure	Men			Women			Total
	Cases	Controls	OR (95% CI)	Cases	Controls	OR (95% CI)	OR (95% CI)
<b>Chlorinated surface water (years)</b>							
Never used	537	875	1.00	152	400	1.00	1.0
0–19	192	268	1.10 (0.80–1.30)	65	160	0.90 (0.60–1.40)	1.0 (0.8–1.2)
20–39	73	84	1.30 (0.90–1.80)	14	55	0.70 (0.30–1.30)	1.1 (0.8–1.4)
40–59	48	57	1.50 (0.95–2.30)	13	44	0.70 (0.30–1.40)	1.2 (0.8–1.7)
>60	25	24	1.90 (1.10–3.60)	4	16	0.70 (0.20–2.40)	1.5 (0.9–2.6)
P (trend)			0.009			0.18	0.13
<b>Chlorinated groundwater (years)</b>							
Never used	251	443	1.00	61	181	1.0	1.0
0–19	216	328	1.10 (0.90–1.40)	52	172	0.8 (0.5–1.3)	1.0 (0.8–1.3)
20–39	261	330	1.40 (1.10–1.80)	71	200	0.9 (0.6–1.4)	1.3 (1.0–1.6)
40–59	132	188	1.30 (0.96–1.70)	58	112	1.1 (0.7–1.8)	1.3 (1.0–1.7)
>60	15	19	1.40 (0.70–3.00)	6	10	1.6 (0.5–5.2)	1.5 (0.8–2.8)
P (trend)			0.04			0.1	0.008
<b>All chlorinated water sources (years)</b>							
Never used	174	337	1.00	43	112	1	1
0–19	168	282	1.00 (0.80–1.40)	47	149	0.7 (0.4–1.1)	1.0 (0.8–1.2)
20–39	237	319	1.20 (0.90–1.60)	56	180	0.7 (0.4–1.1)	1.1 (0.9–1.4)
40–59	222	297	1.30 (0.96–1.70)	78	181	0.8 (0.5–1.3)	1.2 (0.9–1.5)
>60	74	73	1.90 (1.30–2.80)	24	53	1.0 (0.5–2.0)	1.6 (1.2–2.3)
P (trend)			0.002			0.88	0.006

(Continued)

**Table 2.1 (Continued)**

Exposure measure	Men			Women			Total
	Cases	Controls	OR (95% CI)	Cases	Controls	OR (95% CI)	OR (95% CI)
<b>Total lifetime THM (grams)</b>							
<0.04	257	478	1.0	67	203	1.0	1.0
0.05–0.12	234	323	1.3 (1.0–1.7)	66	162	1.2 (0.8–1.8)	1.3 (1.0–1.6)
0.13–0.34	115	181	1.1 (0.8–1.5)	44	11	0.9 (0.6–1.6)	1.1 (0.9–1.4)
0.35–1.48	133	187	1.2 (0.9–1.6)	41	104	1.0 (0.6–1.7)	1.1 (0.9–1.4)
1.49–2.41	43	62	1.3 (0.8–2.0)	12	35	0.9 (0.9–2.0)	1.2 (0.8–1.7)
>2.42	60	56	1.8 (1.2–2.7)	10	41	0.6 (0.3–1.4)	1.3 (0.9–2.0)
Unknown	33	21		8	19		
P (trend)			0.05			0.54	0.08
<b>Lifetime average THM level (micrograms per liter)</b>							
<0.7	269	501	1.0	71	194	1.0	1.0
0.8–2.2	244	314	1.3 (1.0–1.6)	68	181	0.9 (0.6–1.3)	1.2 (1.0–1.5)
2.3–8.0	123	188	1.1 (0.9–1.5)	42	110	0.8 (0.5–1.3)	1.1 (0.8–1.4)
8.1–32.5	133	194	1.1 (0.8–1.5)	44	103	0.9 (0.6–1.5)	1.1 (0.8–1.4)
32.6–46.3	53	54	1.7 (1.1–2.6)	11	45	0.6 (0.3–1.3)	1.3 (0.9–1.8)
>46.4	53	57	1.5 (1.0–2.4)	12	42	0.6 (0.3–1.3)	1.2 (0.8–1.8)
P (trend)			0.02			0.33	0.04



## CHAPTER 3

### NATIONAL DBP DATABASES FOR UNITED STATES AND CANADA

#### INTRODUCTION

A number of national databases, relevant to the United States and Canada, have been assembled to evaluate present and historical trends in DBP formation and disinfection practice. These databases, largely originating from full-scale studies, provide an informative overview of DBPs, DBP precursors (natural organic matter and  $\text{Br}^-$ ), and disinfection practice (e.g.,  $\text{Cl}_2$  dose and point(s) of application). Although national databases are useful in discerning trends induced by national regulations and for developing robust models and correlations for linking DBPs and DBP species to water quality and treatment, the use of national (U.S. and/or Canadian) central tendency models may not always be appropriate for improving exposure assessment in specific regions of North America (an issue addressed in chapter 5).

Throughout U.S. drinking water history, several databases have been assembled to survey DBP occurrence ([Figure 3.1](#)). In our study, several national databases were obtained, with a particular focus on the ICR database. Data from the NORS, NOMS, 35-Utility Study, AwwaRF THM Survey, and WATER\STATS (previously known as the Water Industry Database or WIDB) study were also incorporated into the analysis.

For Canada, two national databases were obtained, both from Health Canada: A National Survey of Chlorinated Disinfection By-Products in Canadian Drinking Water (Health Canada 1995; hereafter, referred to as Health Canada's 53-Utility Survey) and One-Year Survey of Halo-genated Disinfection By-Products in the Distribution System of Treatment Plants Using Three Different Disinfection Processes (Health Canada 1996; hereafter, referred to as Health Canada's One-Year Survey). A Canadian timeline is presented in [Figure 3.2](#). Because Canada has only began to investigate DBP exposure over the last decade and only has a THM guideline of  $100 \mu\text{g/L}$  that was enacted in 1993, the historical timeline is slightly more condensed compared to its U.S. counterpart.

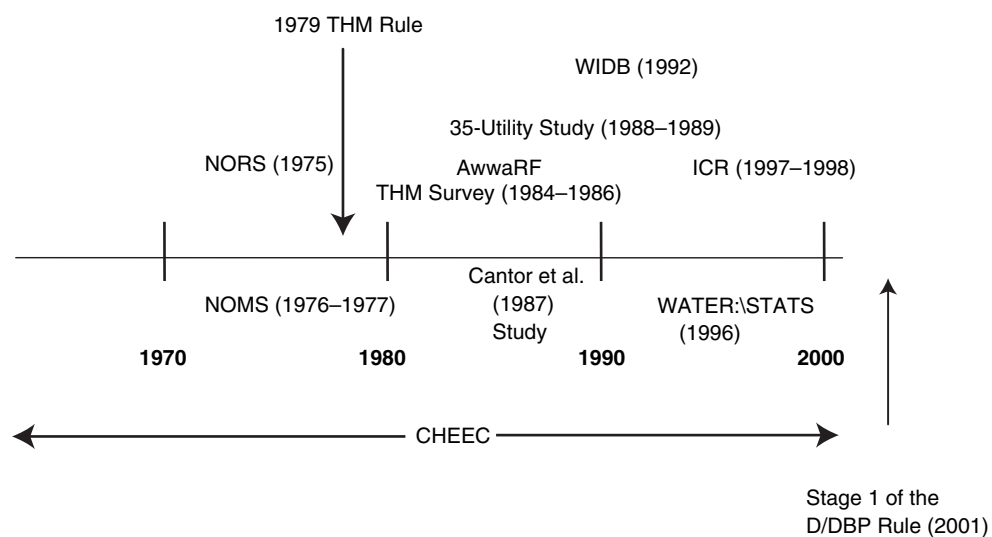
#### DESCRIPTION OF DATABASES

The NORS study, conducted by USEPA in 1975, had three main objectives:

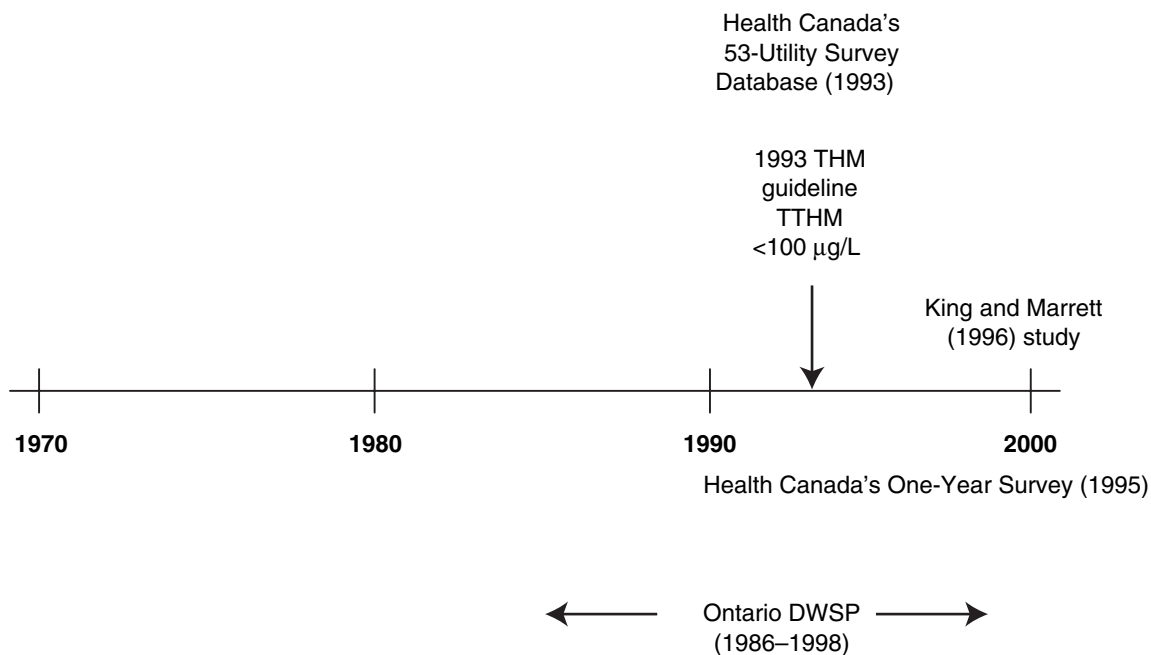
- To determine the occurrence of the four THM species
- To determine the effects of raw water source and water treatment practice on THM formation
- To characterize the organic content of finished drinking water produced from raw water sources in the United States

The NORS provides information for utility source and treatment, treatment practices, chlorine dosages employed, and water quality data pertaining to the four THM species ( $\text{CHCl}_3$ ,  $\text{BDClM}$ ,  $\text{DBClM}$ , and  $\text{CHBr}_3$ ) and TOC.

The NOMS study, conducted by USEPA from 1976 to 1977, had the main objectives of determining the frequency of THMs in drinking water supplies and providing data for the possible establishment of MCLs of DBPs found in drinking water. The study was based on 113 drinking



**Figure 3.1 Historical timeline of acquired U.S. databases**



**Figure 3.2 Historical timeline of acquired Canadian databases**

water supplies, representing various types of sources and treatment processes, and was conducted over an 18-month period. This database provides information on TTHM levels, THM species, and temporal THM variations.

In the mid-1980s, AwwaRF conducted a survey of approximately 1,300 utilities that served a population greater than 10,000 people. Samples were monitored for TTHM formation quarterly during the years 1984–1986 in the AwwaRF survey database.

In 1988–1989, a U.S. nationwide study of 35 utilities was conducted for USEPA and the California Department of Health Services. For the first year of the study, baseline data were gathered from 35 water utilities across the United States by performing four quarters of seasonal sampling, with DBPs sampled in the treatment plant effluents (i.e., finished waters).

The 35-Utility Study is part of the larger regulation negotiation (reg neg) database (RNDB), compiled in 1992 by American Water Works Association (AWWA 1992b). The RNDB contains a large quantity of DBP data from 130 utilities, representing approximately 50 million people from around the United States.

The ICR was promulgated to collect data regarding all aspects of drinking water utilities that served populations greater than 100,000 people. Monitoring began in July 1997 and continued through December 1998. Eighteen months of data were collected in four seasonal quarters for treatment parameters, water quality, DBP formation, and microbial contamination.

While the NORS, NOMS, 35-Utility Study, and ICR databases were analyzed, other informative databases and study reports were also used. WATER:\STATS, a database developed by AWWA in 1996, is composed of a large variety of utilities. An earlier version of WATER:\STATS, the WIDB, released in 1992, was also obtained. A limited portion of the WIDB is part of the RNDB; extracted information included limited data on whether a utility treated surface or groundwater; whether chlorine or chloramines were used for secondary disinfection; distribution system pH; and average distribution system concentration of TTHM.

While data from large full-scale studies have been collected, bench-scale data from two USEPA studies (Amy, Chadik, and Chowdhury 1987; Amy et al. 1998) have been assembled. The earlier study contains THM data (TTHM and THM species) from chlorination of a range of untreated source waters; the more recent study contains both THM (TTHM and THM species) and HAA data from chlorination of both untreated and conventionally treated source waters. The first study is indicative of prechlorination practice, whereas the second study includes both pre- and postchlorination.

Health Canada performed a 53-site DBP study in Canada (1995), of which 13 Ontario utilities treating surface waters were surveyed. This study was conducted over two seasons in 1993 and included sampling of the plant effluents (finished waters) and distribution systems. Data were collected on the concentrations of THMs, HAAs, and DBP precursors, as well as a summary of the plants' treatment and disinfection practices.

The 1996 report by Health Canada, One-Year Survey, summarizes DBP data from three treatment plants and their respective distribution systems and provides insight into Canadian DBP formation and disinfection practices, especially regarding the reanalysis of the Ontario epidemiology study. Health Canada's One-Year Survey represents a regional DBP study on treatment systems utilizing three disinfection scenarios (chlorine/chloramines, chlorine only, and ozone/chlorine), with both THM and HAA data reported.

## DATABASE UTILIZATION

The NORS and NOMS studies are an important component of this study. These two databases and their information were used to describe DBP formation and treatment practices during the mid-1970s in the United States. During this time period, no federal or state regulations required the monitoring of DBPs (both THMs and HAAs). Therefore, drinking water treatment plants had no incentive for monitoring or limiting the formation of DBPs. This situation continued until 1979 when the THM Rule was enacted, establishing an MCL of 100 µg/L. This MCL was recently lowered to 80 µg/L in Stage 1 of the D/DBP Rule. HAAs, specifically HAA5, currently have an MCL set at 60 µg/L, promulgated in December 1998. These MCLs are being revised in Stage 2, which was officially published in the year 2002. “Placeholder” Stage 2 MCLs remain the same as Stage 1, but apply to a “maximum” as opposed to “average” compliance location within the distribution system.

Although the NORS and NOMS databases were utilized to describe treatment practices and DBP formation prior to and through the 1970s, the 35-Utility Study was used to investigate 35 water treatment utilities and their source water qualities and disinfection practices in the 1980s. The study, which was released and published in 1989 (Krasner et al. 1989), includes data from four sampling quarters: spring 1988, summer 1988, fall 1988, and winter 1989. This database provides information on THM and HAA levels, DBP speciation, seasonal and temporal variations in DBP formation, and chlorination and treatment practices, and helps describe the initial post-1979 THM Rule regulated era.

The NORS, NOMS, and 35-Utility studies provide insight into past disinfection practices and DBP formation. The current ICR database provides current and more up-to-date information about all aspects of drinking water quality. The goal of the ICR is to provide USEPA with occurrence and treatment data for disinfectants and DBPs (as well as microbials) in developing future regulation for controlling DBP formation. The ICR is intended to address the complex risk trade-offs between DBPs and disinfection (USEPA 1996). Composed of seven data subsets, known as auxiliary databases, the ICR database provides in-depth information about present DBP occurrence and disinfection practices for all water types and treatment facilities. The ICR also captures a transitional period, as the reg neg was ongoing and utilities were anticipating more stringent DBP regulations, especially Stage 1 of the D/DBP Rule that would be enforced in 2001.

Although several informative databases have been acquired, they are clearly biased toward a national perspective in the United States. This study augmented these efforts by soliciting site-specific DBP occurrence data relevant to Iowa and Ontario.

## ANALYSIS OF NATIONAL DATABASES

### NORS (1975)

In 1974, USEPA ordered a nationwide survey to determine THM formation and exposure in public drinking water systems. Samples were sent from participating utilities and analyzed. [Table 3.1](#) describes treatment practices employed prior to the 1979 THM Rule. It is evident that many plants were relying primarily on chlorination for disinfection purposes.

[Figure 3.3](#) demonstrates chlorine dosages employed during this time period. It can be seen that a median chlorine dosage is approximately 3.75 mg/L—a value that will be compared later to the other databases, specifically the ICR database. While the previous tables describe treatment practice, [Table 3.2](#) provides TTHM formation in finished water surveyed in the NORS database. No HAA data were available in this database.

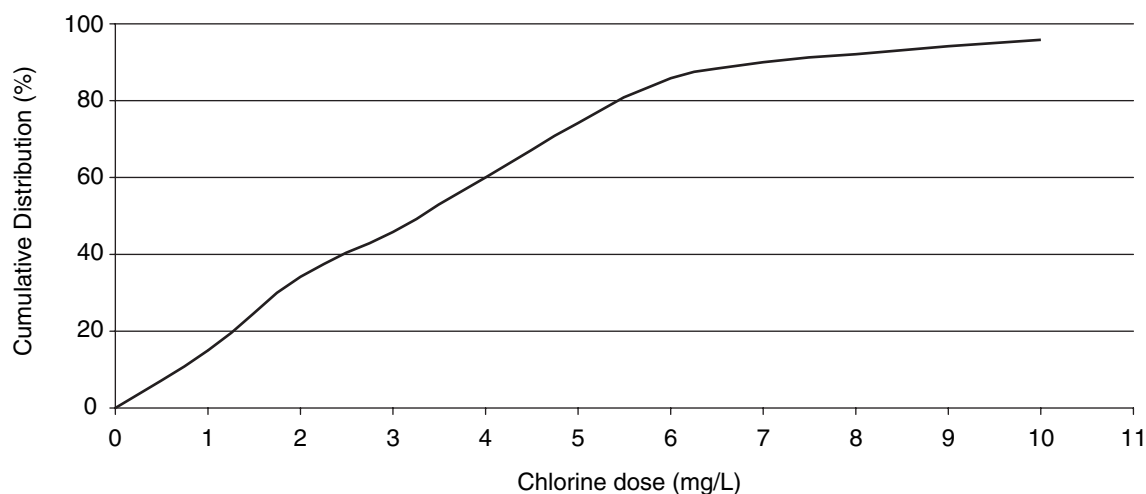


**Table 3.1**  
**Treatment practices employed in the NORS**

Treatment	Percent
Disinfection	100
Chlorination*	99
Ozonation <sup>†</sup>	1
Raw water chlorination	75
Raw water ozonation	1
Polyelectrolyte used	22
Powdered activated carbon used	25
Granular activated carbon used	10
Softening	25
Precipitative	22
Zeolite	3
Taste and odor control practiced	38

\* At some place in the treatment system.

<sup>†</sup> The only treatment practice.



**Figure 3.3 NORS cumulative distribution of total chlorine dose**

**Table 3.2**  
**Finished water THM concentrations surveyed in the NORS database**

THM	Median concentration (µg/L)	Range (µg/L)	
		Minimum	Maximum
CHCl <sub>3</sub>	21	<0.1	311
BDCM	6	BDL*	116
DBCM	1.2	BDL	100
CHBr <sub>3</sub>	Not found in 68.8% of finished waters	BDL	92

\* BDL: Below detection limit.

**Table 3.3**  
**Finished water THM concentrations surveyed by the NOMS**

Compound	Number of positives*	Mean concentration, all cities (µg/L)
CHCl <sub>3</sub>	112	27
BDCM	109	9.6
DBCM	97	1.2
CHBr <sub>3</sub>	38	2.5
TTHM		42.8

Note: Blank cell indicates information was not available.

\* Out of 113 total water samples THM concentrations.

### **NOMS (1976–1977)**

Although NORS was the first primary study to evaluate THM formation and exposure, the second survey, NOMS, conducted in 1976 and 1977, also provided valuable information such as mean and median concentrations of finished water THMs, which are shown in [Table 3.3](#). However, NOMS only provided finished water THM information; no data were provided for treatment practices or finished water HAA formation.

### **AwwaRF THM Survey (1984–1986)**

The goal of the AwwaRF THM Survey was to examine the extent and costs of compliance with the existing MCL for TTHM, which was set at 100 µg/L, as stated by the 1979 THM Rule (McGuire and Meadow 1988). A summary of the database can be found in [Table 3.4](#).

**Table 3.4**  
**Overall finished water TTHM values in the AwwaRF THM Survey**

Overall (1984–1986)	TTHM (µg/L except for minimum and count)
Average	43.12
Standard deviation	39.10
25th percentile	16.00
Median	37.00
75th percentile	61.00
Minimum	BDL *
Maximum	645.00
Count	7,237

\* BDL: Below detection limit.

**Table 3.5**  
**Median finished THM values for the 35-Utility Study database**

Quarter	Value (µg/L)
First (spring 1988)	34
Second (summer 1988)	44
Third (fall 1988)	40
Fourth (winter 1989)	30

From the analysis, McGuire and Meadow (1988) estimated that the 1979 THM Rule setting the TTHM standard at 100 µg/L reduced concentrations of TTHM in larger systems by 40%–50%.

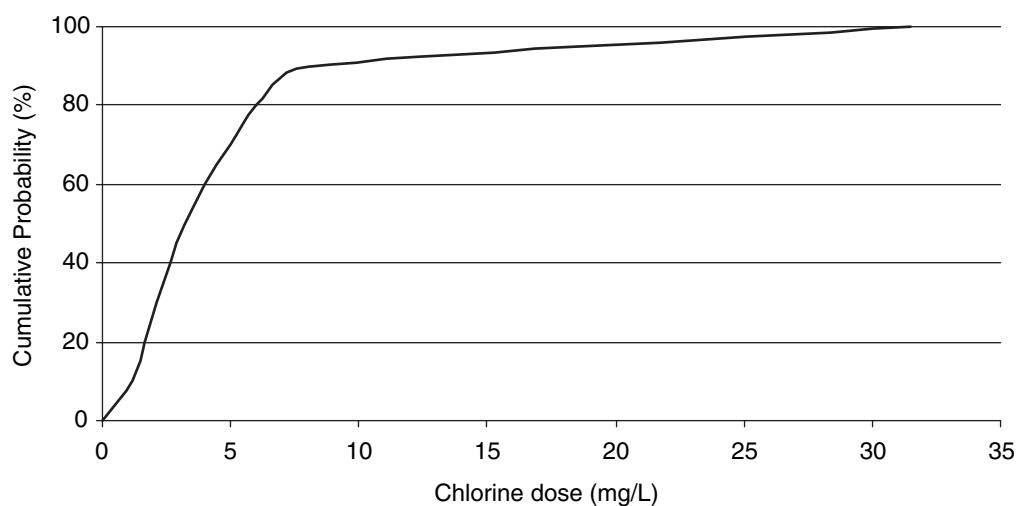
### 35-Utility Study (1988–1989)

The 35-Utility Study, which was conducted by Krasner et al. (1989), was a more comprehensive database and provided more information than either the NORS or NOMS studies. Median finished water TTHM levels for the four quarters can be found in [Table 3.5](#), while individual DBP values can be found in [Table 3.6](#). These tables illustrate seasonal variations in DBPs. The 35-Utility Study also provides information about treatments employed to meet the 1979 THM Rule. A cumulative chlorine distribution system curve can be found in [Figure 3.4](#).

**Table 3.6**  
**Median values for finished water THM and HAA species**  
**for the 35-Utility Study database**

DBPs	First quarter (spring 1988)	Second quarter (summer 1988)	Third quarter (fall 1988)	Fourth quarter (winter 1989)	Median values of four quarters
<b>THMs</b>					
CHCl <sub>3</sub>	15.00	15.00	13.00	9.60	14.00
BDCM	6.90	10.00	5.50	4.10	6.20
DBCM	2.60	4.50	3.80	2.70	3.25
CHBr <sub>3</sub>	0.33	0.57	0.88	0.51	0.54
TTHM	34.00	44.00	40.00	30.00	37.00
<b>HAAs</b>					
MCAA	<1.00	1.20	<1.00	1.20	1.20
DCAA	7.30	6.80	6.40	5.00	6.60
TCAA	5.80	5.80	6.40	5.80	
MBAA	<0.50	<0.50	<0.50	<0.50	
DBAA	0.90	1.50	1.40	1.00	1.20
HAA5	18.00	20.00	21.00	13.00	19.00

Note: Blank cells indicate information was not available.



**Figure 3.4 Total chlorine dose cumulative distribution curve for the 35-Utility Study database**

**Table 3.7**  
**Summary of TTHM and HAA5 formation in the WATER:\STATS database**

Sampling location	Median concentration (µg/L)	
	TTHM	HAA5
Finished	40	
Distribution system	32	21

Note: Blank cell indicates information was not available.

### **WATER:\STATS (1996)**

Formerly known as the WIDB, the WATER:\STATS study surveyed more than 3,200 utilities nationwide. The database provided extensive information regarding source waters, treatment, water quality, and distribution. Summary of finished water data can be found in [Table 3.7](#). Lower distribution system values, when compared to the finished water values, may be a result of biodegradation and/or pipe wall effects.

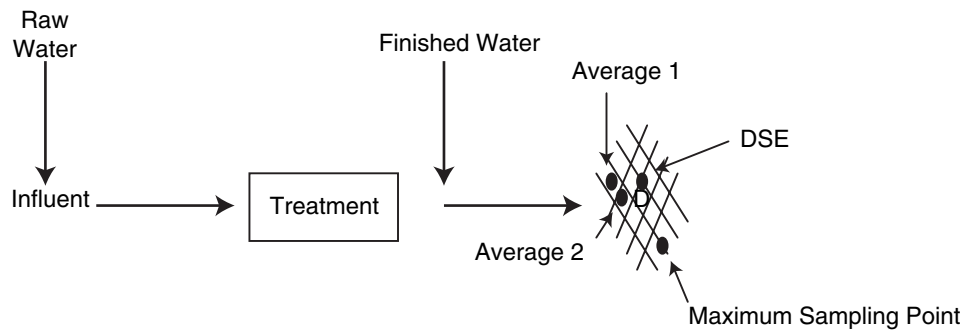
### **ICR Database (1997–1998)**

As stated earlier, the ICR database is the most complete and robust drinking water database to date and reflects various treatments that utilities employed to meet the D/DBP Rule Stage 1 regulations in the late 1990s. It will be used to compare current treatment practices with previous treatment strategies that were utilized in the NORS, NOMS, and 35-Utility Study databases.

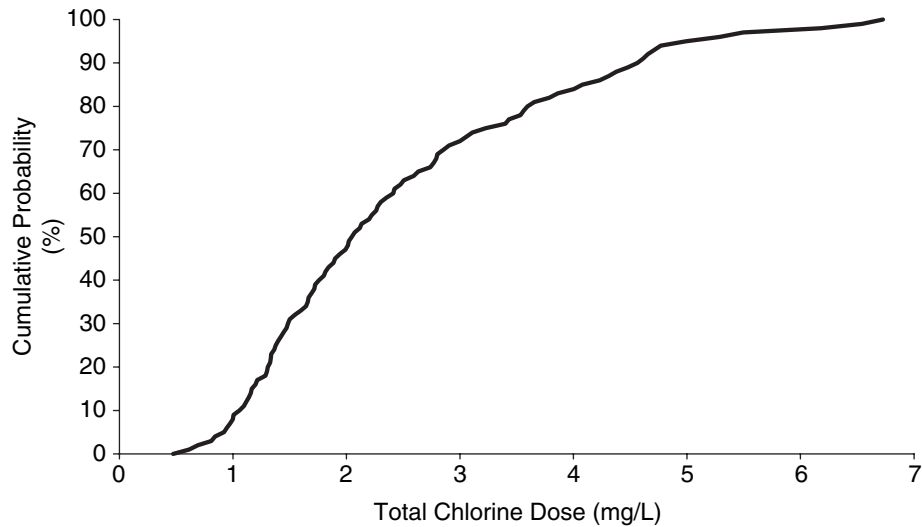
The ICR database contains monthly and quarterly water quality monitoring and operational data. Many sampling locations were monitored to provide data throughout the process train of various water treatment plants. However, for this study, six sampling locations were of importance and are identified in [Figure 3.5](#). Raw water was sampled at the head of the plant, and finished water was sampled at the end of the treatment process train. The distribution system was sampled at four different points. Average 1 and Average 2 represent two average residence times within the distribution system. The distribution system equivalent (DSE) location simulates a point within the distribution system near the water treatment plant where the residence time is well comprehended and where the impact from other water sources is limited. The maximum sampling point corresponds to the maximum residence time of the water within the distribution system.

The ICR database also classified drinking water plants into several categories, of which this study will focus on eight. The acronyms, reflecting various combinations of primary (pre-) and secondary (post-) disinfection, are as follows:

- Cl<sub>2</sub>/Cl<sub>2</sub>: Prechlorination and postchlorination
- Cl<sub>2</sub>/CLM: Prechlorination and postchloramination
- Cl<sub>2</sub>&CLM/CLM: Prechlorination followed with ammonia and postchloramination
- CLM/CLM: Prechloramination and postchloramination
- O<sub>3</sub>/Cl<sub>2</sub>: Preozonation and postchlorination
- O<sub>3</sub>/CLM: Preozonation and postchloramination



**Figure 3.5 ICR database sampling locations**

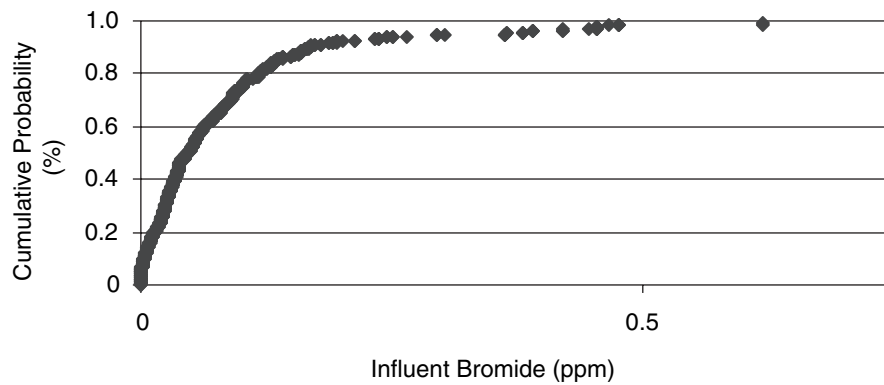


**Figure 3.6 Cumulative probability of chlorine dose in the ICR database**

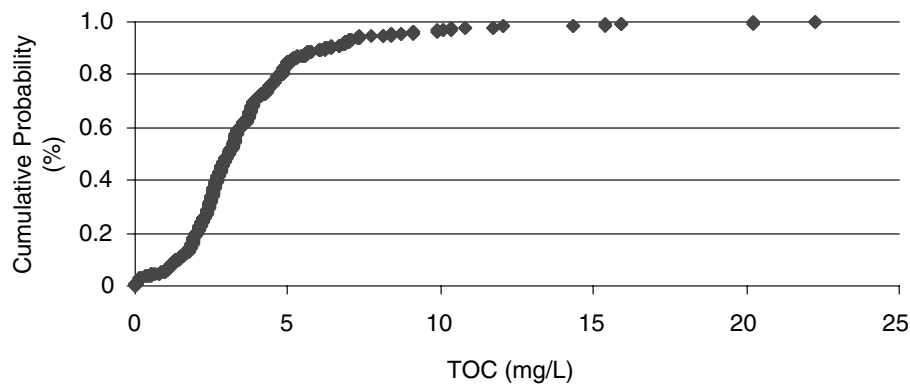
- CLX/Cl<sub>2</sub>: Prechlorine dioxide and postchlorination
- CLX/CLM: Prechlorine dioxide and postchloramination

The first parameter investigated in the ICR database was chlorine dosage as portrayed in [Figure 3.6](#), as this parameter was reported in the NORS historical database. It is known that the greater the chlorine dose employed in treatment, the more chlorinated by-products will form. Therefore, chlorine dose is important when trying to predict DBP formation. For those plants that incorporated chlorine as a disinfectant, the median total chlorine dose was approximately 2 mg/L. (This dose is representative of chlorine added anywhere within the treatment plant process train, hence pre- or postchlorination.)

Also, if a high concentration of bromide is present in the influent water, the DBP species will shift to the more brominated species. Therefore, bromide is important when modeling the formation of the brominated DBPs. [Figure 3.7](#) quantifies the cumulative distribution of influent bromide for all the plant records. The median influent bromide level was 0.047 mg/L (47 µg/L).



**Figure 3.7 Influent bromide levels in the ICR database**



**Figure 3.8 Influent TOC values in the ICR database**

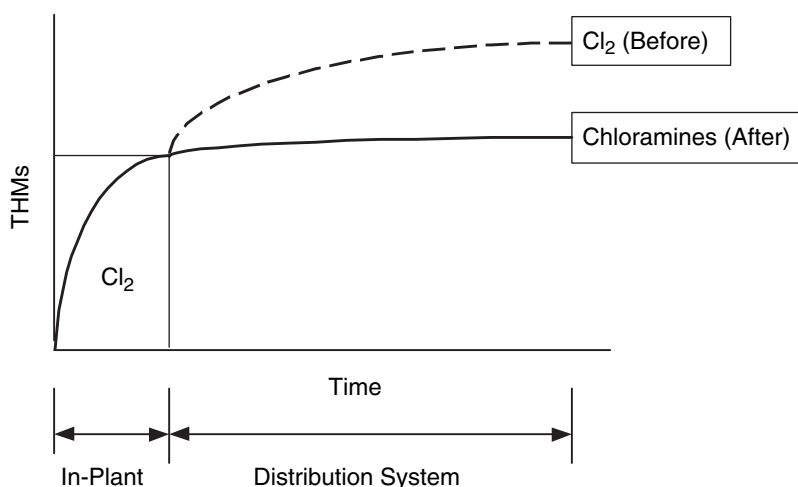
TOC as another parameter is important when considering modeling DBPs. The median influent TOC level was 3.03 mg/L, and the TOC distribution is summarized in [Figure 3.8](#).

Since an enormous amount of information is contained in the ICR database, this section will primarily focus on DBP formation (TTHM, HAA5, HAA6 [HAA5 plus bromochloroacetic acid, or BCAA], and HAA9 [HAA6 plus bromodichloroacetic acid, BDCAA; dibromochloroacetic acid, DBCAA; and tribromoacetic acid, TBAA]). DBP values for treatment categories in the ICR database are summarized in [Table 3.8](#).

Median concentrations varied with the different treatment and disinfection practices. However, the overall finished water median value for TTHM was 23.15 µg/L and 32.35 µg/L at the DSE sampling point. For HAA5 values, the median finished water concentration was 15.85 µg/L, whereas the DSE value was 16.65 µg/L. For HAA6, the finished water value was 19.10 µg/L with a DSE value of 20.15 µg/L. For HAA9, the finished water value was 23.15 µg/L with a DSE value of 26.03 µg/L. For TTHM, HAA5, HAA6, and HAA9, DBP concentrations increased from the finished water to the distribution system, as expected. This can be attributed to the postdisinfectant residual reacting with the remaining organic matter present in the finished water entering the distribution system, as illustrated in [Figure 3.9](#). It is also important to note that,

**Table 3.8**  
**Overall DBP formation in the ICR database segmented by treatment type**

Plant design	Finished median values (µg/L)				DSE median values (µg/L)			
	TTHM	HAA5	HAA6	HAA9	TTHM	HAA5	HAA6	HAA9
Cl <sub>2</sub> /Cl <sub>2</sub>	23.90	17.00	19.50	21.60	33.30	20.80	24.25	26.15
Cl <sub>2</sub> /CLM	37.10	18.35	22.90	25.20	40.50	18.60	22.50	24.00
Cl <sub>2</sub> &CLM/CLM	38.70	22.50	27.50	29.00	39.00	24.40	30.00	37.75
CLM/CLM	23.20	16.60	20.90	20.30	30.15	14.70	19.45	16.50
O <sub>3</sub> /Cl <sub>2</sub>	10.90	7.90	10.50	10.65	31.40	17.30	20.60	26.40
O <sub>3</sub> /CLM	15.50	9.15	10.95	10.90	15.50	10.70	12.50	13.80
CLX/Cl <sub>2</sub>	26.65	20.70	22.35	42.15	45.55	28.60	31.50	61.20
CLX/CLM	23.10	15.10	20.30	24.70	27.80	16.00	21.00	25.90
Overall median	23.55	16.80	20.60	23.15	32.35	17.95	21.75	26.03



**Figure 3.9 Example of a plant switching from chlorination to chloramination**

although TTHM and HAAs are being presented, THM species and HAA species will also be used in the modeling process and will be further discussed in chapter 5.

Figure 3.9 is an example of a plant that was initially employing chlorine both in the treatment plant as a predisinfectant and in the distribution system as a postdisinfectant. After DBPs formed both within the plant and in the distribution system, the plant is shown switching to postdisinfection using chloramines. In this scenario, a majority of the DBPs form only in the plant with minimal DBP formation in the distribution system.

However, from Figure 3.9, it is evident that disinfection schemes result in different rates of DBP formation. Plants that incorporate alternative disinfectants, such as ozone, chlorine dioxide,



**Table 3.9**  
**Summary of DBP formation in the Health Canada 53-Utility Survey**

Compound (µg/L)	Treatment	Site	Winter mean	Winter median	Winter range	Summer mean	Summer median	Summer range	Overall median
TTHM	Cl <sub>2</sub> /Cl <sub>2</sub>	Treated	16.8	10.9	2.0–67.9	33.5	17.2	1.6–120.8	14.1
	Cl <sub>2</sub> /CLM	Treated	12.1	10.1	0.6–40.3	31.2	19.7	2.9–80.1	
	O <sub>3</sub> /CLM or Cl <sub>2</sub>	Treated	6.8	5.7	1.7–12.3	44.0	57.4	2.5–74.9	
DCAA	Cl <sub>2</sub> /Cl <sub>2</sub>	Treated	13.2	9.0	0.3–45.4	21.1	12.5	0.6–163.3	9.8
	Cl <sub>2</sub> /CLM	Treated	9.8	7.7	1.2–23.3	12.5	10.5	5.3–27.6	
	O <sub>3</sub> /CLM or Cl <sub>2</sub>	Treated	6.9	6.4	1.6–15.0	21.2	22.6	5.3–27.6	
TCAA	Cl <sub>2</sub> /Cl <sub>2</sub>	Treated	27.8	13.0	0.1–139.8	34.0	11.9	0.04–273.2	10.6
	Cl <sub>2</sub> /CLM	Treated	13.7	6.9	0.5–66.2	25.1	9.3	2.1–85.9	
	O <sub>3</sub> /CLM or Cl <sub>2</sub>	Treated	5.8	1.5	0.7–16.9	24.6	21.6	1.3–66.1	
TTHM	Cl <sub>2</sub> /Cl <sub>2</sub>	DS	33.4	21.8	2.8–221.1	62.5	33.8	0.3–342.4	21.8
	Cl <sub>2</sub> /CLM	DS	13.7	10.9	1.5–42.1	32.8	21.7	4.3–85.2	
	O <sub>3</sub> /CLM or Cl <sub>2</sub>	DS	9.9	11.0	2.4–15.4	66.7	90.9	4.9–107.8	
DCAA	Cl <sub>2</sub> /Cl <sub>2</sub>	DS	15.6	11.8	0.2–63.6	19.0	10.4	0.3–120.1	10.6
	Cl <sub>2</sub> /CLM	DS	10.0	9.9	1.2–22.6	11.4	10.8	4.2–23.8	
	O <sub>3</sub> /CLM or Cl <sub>2</sub>	DS	4.6	4.8	0.4–9.3	14.1	10.7	0.9–42.6	
TCAA	Cl <sub>2</sub> /Cl <sub>2</sub>	DS	56.7	24.7	0.1–473.1	48.9	25.1	0.1–263.4	11.0
	Cl <sub>2</sub> /CLM	DS	13.2	7.0	0.5–57.9	21.4	8.7	1.9–71.5	
	O <sub>3</sub> /CLM or Cl <sub>2</sub>	DS	4.1	2.0	0.9–12.8	28.3	13.3	0.7–77.3	

and even chloramines, will generally have lower DBP formation rates in the distribution system than plants that employ only traditional chlorination.

Again, it is important to note that the ICR database contains substantial amounts of information about water source, water quality, treatment processes, and DBP formation. Important parameters used for the modeling efforts, such as Br<sup>−</sup>, will be discussed in further detail in a later chapter.

### Health Canada's 53-Utility Survey (1993)

As stated earlier, Health Canada surveyed 53 utilities in nine provinces during the winter and summer seasons in 1993. Raw, finished water, and distribution system water samples were analyzed. Pre- and postchlorination was practiced at 35 facilities, and prechlorination coupled with postchloramination was employed at 10 facilities. Pre-ozonation coupled with postchlorination or chloramination was utilized at the remaining utilities. Treated values correspond to finished water and DS corresponds to the distribution system. The results of the study can be found in [Table 3.9](#).

**Table 3.10**  
**Treatments employed by the three utilities in Health Canada's One-Year Survey**

Plant	Treatment type	Water source	Population served
Ottawa	Prechlorination and postchloramination	Ottawa River	>300,000
Hull	Prechlorination and postchlorination	Ottawa River	>100,000
Buckingham	Prechlorination and postchlorination	La Lievre River	>15,000

**Table 3.11**  
**DBP formation in Health Canada's One-Year Survey**

Plant	Site	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	MCAA	DCAA	TCAA	MBAA	DBAA	HAA5
		(µg/L)										
Ottawa	Treated	29.2	2.4	0.3	<0.1	32.0	2.1	15.7	7.9	<0.01	<0.01	25.7
Hull	Treated	21.3	3.1	0.3	<0.1	24.8	2.0	16.2	13.0	<0.01	<0.10	31.2
Buckingham	Treated	19.2	1.0	0.1	<0.1	20.3	1.9	18.3	5.9	<0.01	<0.01	26.1
	Median	21.3	2.4	0.3	BDL*	24.8	2.0	16.2	7.9	BDL	BDL	26.1
Ottawa	DS	35.4	2.2	0.1	<0.1	37.7	1.8	17.3	7.7	<0.01	<0.01	26.8
Hull	DS	55.9	5.1	0.3	<0.1	61.4	0.8	3.7	4.3	<0.01	<0.10	8.8
Buckingham	DS	47.3	2.1	0.1	<0.1	49.5	1.0	6.1	9.8	<0.01	<0.01	16.9
	Median	47.3	2.2	0.1	BDL	49.5	1.0	6.1	7.7	BDL	BDL	16.9

\* BDL: Below detection limit.

TTHM and HAAs were found in all the waters tested in this survey, and the HAA levels often equaled or exceeded TTHM concentrations. As expected, DBP formation levels were higher in the summer than in the winter for all of the treatment processes. DBP formation also increased in the distribution system, excluding the prechlorination and postchloramination plants.

In 1995, Health Canada performed a study (One-Year Survey; Health Canada 1996) examining DBP concentrations in drinking water as a function of treatment practice, season, and location in the distribution system for three water treatment plants that applied different treatment processes. Summaries of the treatments employed are presented in [Table 3.10](#).

Samples from the raw water influent, treated water, and distribution system were analyzed over a 13-month time period. Median DBP values can be found in [Table 3.11](#).

Similar to the 53-Utility Survey performed in 1993, HAA levels often equaled or exceeded TTHM concentrations in the One-Year Survey. Chloroform was the main THM detected and represented approximately 90% of the TTHM concentration in each of the three systems. For HAA formation, MCAA, TCAA, and DCAA were found in all of the finished water and distribution system samples. MBAA and DBAA were not detected in any of the samples. The most

noticeable trend in this study was that DCAA levels observed at the end point in the distribution system decreased compared to the treated effluent, a trend usually attributed to bacterial degradation and pipe wall effects.

## NATIONAL TRENDS IN U.S. DISINFECTION PRACTICES

From the databases accumulated, some general chlorination trends can be discerned. However, it should be noted that only the NORS and ICR databases clearly define chlorination practice. The 35-Utility Study contained some information on chlorination practices. The RNDB, which included the 35-Utility Study, provided information and data on the chlorine, ammonia, and ozone dose, and application points for each treatment plant in the 35-Utility Study. The NORS study also indicated which utilities used chloramines (combined chlorine), summarized under the postdisinfectant type category. According to the ICR database, 33% of the plants surveyed use chloramines at some point in the plant's process train. The NORS study also indicated that many treatment plants were utilizing treatment processes such as raw water chlorination, powdered and granular activated carbon (PAC and GAC) for taste and odor control (mostly PAC), and/or softening. In addition, some utilities in the NORS used ozone or chlorine dioxide. According to the ICR database, where raw water chlorination and PAC continue to be utilized, alternative disinfectants (ozone, chlorine dioxide, and chloramines) are being more frequently employed. Therefore, there has been a trend from multiple-point chlorination practices to utilizing alternative disinfectants. This difference can be explained by the fact that current DBP regulations are driving drinking water treatment utilities to explore the possibility of utilizing alternative disinfectants to meet current and projected THM and HAA regulations.

## COMPARISON OF NATIONAL DATABASES

### Comparison of U.S. Databases

The occurrence of DBPs was first reported in 1974 for chloroform and other THMs in drinking water. Since this time, DBPs have been monitored in several databases, including the NORS, NOMS, AwwaRF THM Survey, 35-Utility Study, WATER\STATS, and ICR. Such databases have provided crucial information regarding DBP occurrence trends over time. A comparison of these databases can be found in [Table 3.12](#).

In evaluating trends exhibited in [Table 3.12](#), it is important to note that some of the samples (Phase II) obtained in the NOMS survey were unquenched, and thus THM levels may be somewhat overstated. Nevertheless, it is clear that chlorinated DBPs have decreased somewhat over time. As stated before, the NOMS study was conducted in 1976 when no THM or HAA regulations existed. However, when THM regulations were first promulgated, there was an evident decrease in THM median concentrations. Although there are no clear historical trends for HAAs, it is known from the RNDB that the Stage 1 (and now Stage 2) MCL, promulgated at 60 µg/L, required a significant number of drinking water treatment utilities to make treatment changes (especially to meet the MCL with a 20% safety factor, i.e., HAA5 <48 µg/L). (For the RNDB, the 10th, 50th, and 90th percentiles for TTHM were, respectively, 1.5, 34, and 71 µg/L; corresponding values for HAA sums were, respectively, 4.7, 27, and 67 µg/L). This, in turn, will cause facilities to alter their drinking water treatment process trains, including disinfection practices, to decrease HAA formation. Stage 1 of the D/DBP Rule also affects drinking water utilities for THM formation. With

**Table 3.12**  
**Overall comparison of U.S. databases**

Study	Median concentrations of THMs and HAAs (µg/L)							
	TTHM	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	HAA5	HAA6	HAA9
NORS (1975)		21.00	6.00	1.20				
NOMS* (1976–1977)	42.80	27.00	9.60	1.20	2.50			
AwwaRF THM Survey (1984–1986)	37.00							
35–Utility (1988–1989)	37.00	13.15	6.60	3.40	0.57	18.00		
WATER\STATS finished water (1996)	40.00							
WATER\STATS DS	32.00					21.00		
ICR finished water (1997–1998)	32.32	16.33	7.13	2.52	0.00	20.40	23.40	24.78
ICR DSE	40.02	23.35	8.85	2.80	0.00	22.28	26.28	28.15

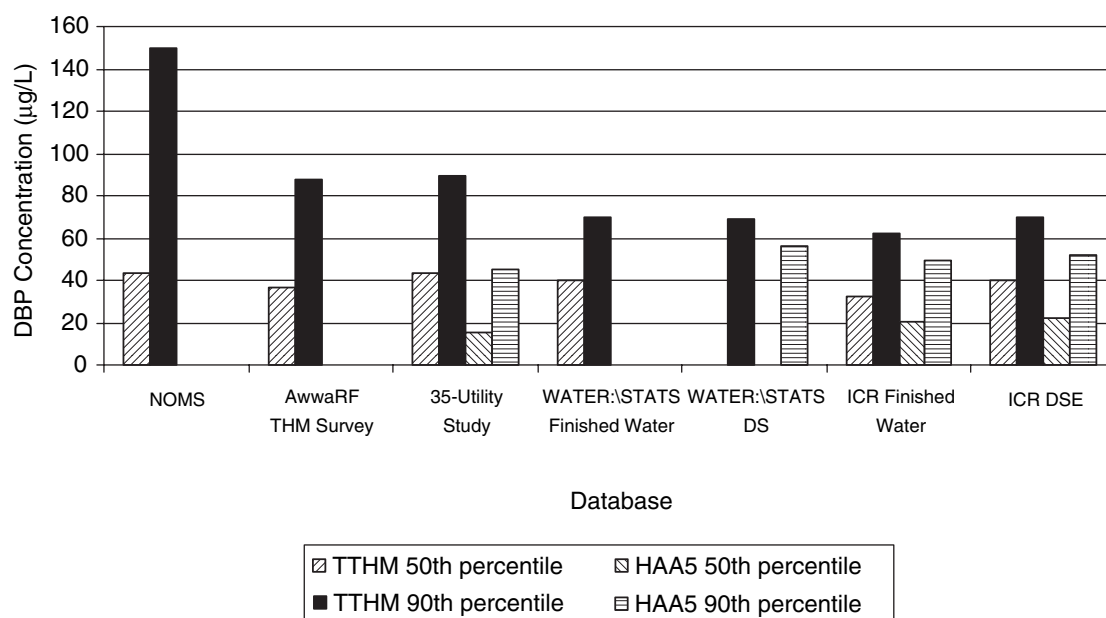
Note: Blank cells indicate information was not available.

\* Phase II samples were quenched.

promulgation of the Stage 1 (and now Stage 2) MCL of 80 µg/L, a significant number of utilities have been making treatment changes (especially to meet the MCL with a 20% safety factor, i.e., TTHM <64 µg/L). [Figure 3.10](#) further supports this point.

While median concentrations have decreased somewhat over time, 90th percentile values have decreased even more dramatically, as evident in [Figure 3.10](#). Seasonal trends are also an important consideration; higher concentrations of DBPs typically form in the summer compared to winter.

Although the present D/DBP regulations have promoted changes in treatment and disinfection practice, the 1979 THM Rule had a similar effect. Many utilities had to change their treatment practices to comply with the rule. In the AwwaRF THM Survey (McGuire and Meadow 1988), more than 700 utilities reported THM data. Of these, more than 200 utilities reported more than 500 changes in treatment, reflecting more than one change per respondent. [Table 3.13](#) shows what treatment changes were selected for those systems requiring modification to comply with the THM Rule. (The sum of the percentages is greater than 100%, as the survey reflected more than one treatment change per respondent.) The majority of utilities changed their chlorine dosage and/or moved the point of disinfection. A significant number of utilities that responded to the AwwaRF THM Survey had changed to chloramines as the primary or secondary disinfectant. Some utilities enhanced their coagulation process or switched to chlorine dioxide for primary disinfection. Only a small number of utilities switched to ozone for primary disinfection or incorporated GAC treatment. On average, promulgation of the THM Rule resulted in a 40%–50% reduction in THMs at larger utilities.



**Figure 3.10 50th and 90th percentile concentration of TTHM and HAAs over time in the U.S. databases**

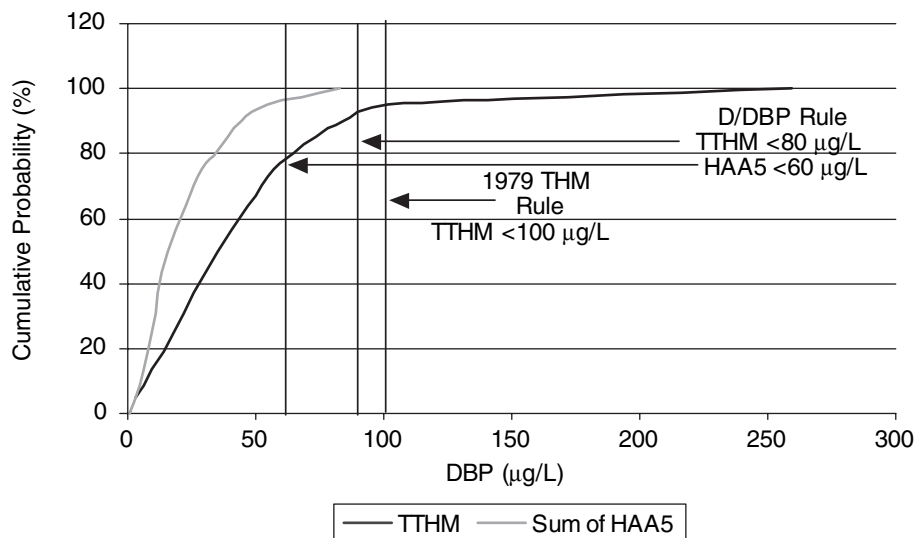
**Table 3.13**  
**Percentage of utilities that made treatment modifications to comply with the 1979 THM Rule**

Treatment technology	Percentage of utilities that made changes*
Changed chlorine dosage	54.00
Changed point of disinfection	67.00
Chloramines	440.18
Chlorine dioxide	12.00
Enhanced coagulation	20.00
GAC	4.00
Miscellaneous <sup>†</sup>	28.00
Ozone	1.00
PAC	11.00

Source: Data from AwwaRF THM survey (1984–1986); McGuire and Meadow 1988.

\* 543 treatment changes made by 225 utilities; changes reflect more than one change per utility.

<sup>†</sup> Off-line storage, alternate source, aeration, other.



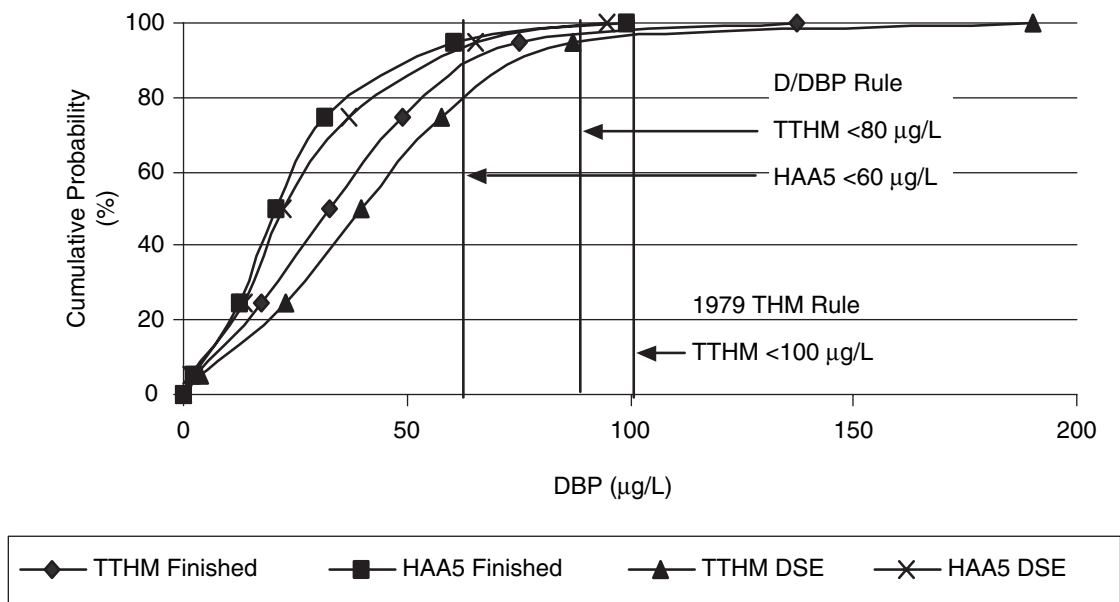
**Figure 3.11 Cumulative probability of TTHM and HAA5 formation in the 35-Utility Study**

Evidently, all the median values fall within the mandatory regulations for that specific time period. The AwwaRF THM Survey, 35-Utility Study, and WATER:\STATS data for median THM values are well below the 1979 THM Rule standard of TTHM <100 µg/L. The ICR database median values also fall below the D/DBP Rule standards of TTHM <80 µg/L and HAA5 <60 µg/L. The reason for this is because the median values represent a majority of the treatment plants across the nation, and the regulations primarily affected plants operating at the higher end of DBP formation. The 1979 THM Rule and D/DBP Rule primarily affected plants that formed higher concentrations of DBPs, as demonstrated in Figure 3.11.

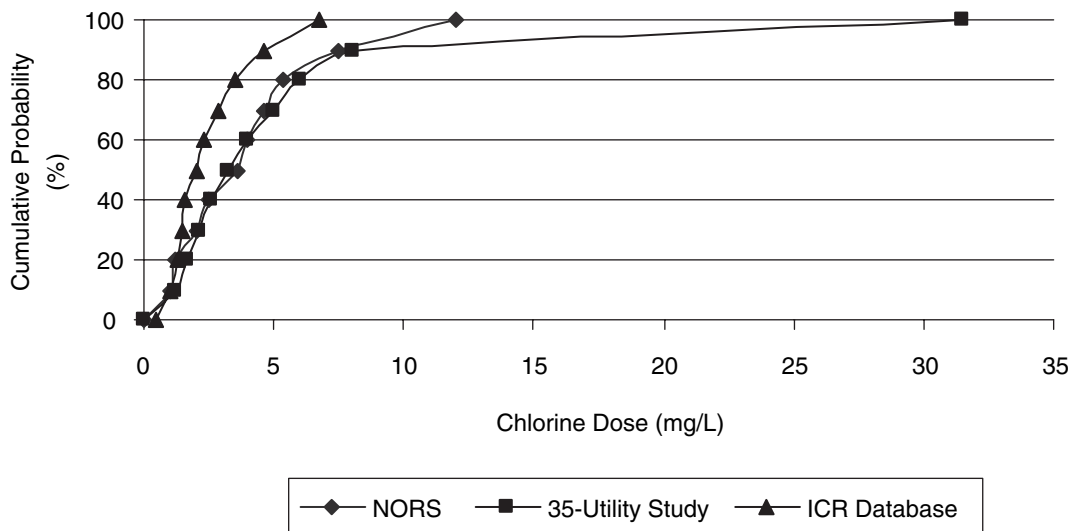
In 1997–1998, when the 35-Utility Study was performed, less than 5% of the plants surveyed were in violation of both the 1979 THM Rule and D/DBP Rule. Therefore, it was these plants that were forced to decrease their THM levels. Similarly, for HAA5 formation, less than 5% of the plants surveyed were in violation of the D/DBP Rule. This trend was also apparent in the current ICR database, as illustrated in Figure 3.12.

Similar to the 35-Utility Study, plants in the ICR database with higher concentrations of DBP formation were the most affected by the DBP regulations. Less than 10% of the utilities were in violation of the current D/DBP Rule. However, Figure 3.12 illustrates that distribution system levels, which correspond to monitoring points for the D/DBP Rule, are more affected. For example, approximately 10% of the plants surveyed violated the D/DBP Rule that requires HAA5 <60 µg/L. Therefore, the regulations are forcing a small minority of treatment plants that form higher DBP levels to meet the regulations. While DBP formation has remained somewhat constant over the last several decades, one slightly significant change has occurred regarding chlorine dose ( $\Delta \text{CL}_2 \approx -1$  mg/L based upon the median values) (Figure 3.13).

Figure 3.13 illustrates that plants typically employed the same chlorine dosage during the 1970s to the 1980s. However, in order to meet the newly enacted D/DBP Rule, plants decreased their chlorine dose to decrease their DBP formation.



**Figure 3.12 Cumulative probability curves of TTHM and HAA5 in the ICR database**



**Figure 3.13 Comparative cumulative probability curves for chlorine dose**

### Comparison of Canadian Databases

While the United States began to monitor DBP formation starting in the mid-1970s, Canada started in the mid-1980s. Therefore, this study was unable to acquire many national Canadian historical databases, and there is minimal comparison between the Health Canada 53-Utility Survey (1993) and the Health Canada One-Year Survey (1996). These data are summarized in [Table 3.14](#).

**Table 3.14**  
**Median values of DBPs for Canadian databases**

Study	Site	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	MCAA	DCAA	TCAA	MBAA	DBAA	HAA5
		(µg/L)										
Health Canada's 53-Utility Survey (1993)	Treated*					14.1		9.8	10.6			
	DS					21.8		10.6	11.0			
Health Canada's One-Year Survey (1995)	Treated	21.3	2.4	0.3	BDL <sup>†</sup>	24.8	2.0	16.2	7.9	BDL	BDL	26.1
	DS	47.3	2.2	0.1	BDL	49.5	1.0	6.1	7.7	BDL	BDL	16.9

Note: Blank cells indicate information was not available.

\* Treated: Finished water.

† BDL: Below detection limit.

Because Canada issued a national guideline of TTHM <100 µg/L in 1993, Health Canada's 53-Utility Survey can be used to describe current treatment practices and DBP formation prior to the guideline being promulgated. Health Canada's One-Year Survey can then be used to describe DBP occurrence after the guidelines were enacted. This database can also be used to examine the formation of DBPs under the use of different disinfectants.

When the two surveys are compared with each other, it appears that TTHM concentrations increased over time. However, this comparison is biased because the Health Canada's One-Year Survey only examined three utilities. The 53-Utility Survey was more comprehensive due to the larger number of utilities incorporated in the analysis. This same phenomenon occurred with the values for HAA formation, with a less significant difference. While the One-Year Survey analyzed fewer utilities, the 53-Utility Survey provided national data regarding TTHM and species and HAA5 and species formation.

## COMPARISON OF NATIONAL U.S. AND CANADIAN DATABASES

A comparison of the U.S. with the Canadian national databases can be found in [Table 3.15](#).

Compared with U.S. drinking waters, Canadian drinking waters tend to differ in two main areas. Canadian waters generally have lower levels of influent bromide, resulting in lower levels of brominated DBPs and natural organic matter properties that promote the formation of HAAs and species over TTHM and species. This is evident from [Table 3.15](#). Both of the Canadian databases showed significantly less TTHM formation than the U.S. surveys. The Canadian databases also indicate similar amounts of HAAs compared to TTHM, a result not observed in the U.S. databases.



**Table 3.15**  
**Comparison of national U.S. and Canadian databases for DBP formation**

Databases	Median concentrations of THMs and HAAs (µg/L)							
	TTHM	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	HAA5	HAA6	HAA9
<b>U.S. studies</b>								
NORS (1975)		21.00	6.00	1.20				
NOMS* (1976–1977)	42.80	27.00	9.60	1.20	2.50			
AwwaRF	37.00							
THM Survey (1984–1986)								
35-Utility Study (1988–1989)	37.00	13.15	6.60	3.40	0.57	18.00		
WATER\STATS finished water (1996)	40.03							
WATER\STATS DS	33.80					23.06		
ICR finished water (1997–1998)	32.32	16.33	7.13	2.52	0	20.40	23.40	24.78
ICR DSE	40.02	23.35	8.85	2.80	0	22.28	26.28	28.15
Median	37.00	21.00	7.13	2.52	0.285	21.34	24.84	26.465
<b>Canadian studies</b>								
Health Canada's 53-Utility treated water (1993)	14.10					20.40		
Health Canada's 53-Utility DS	21.80					21.60		
Health Canada's One-Year Survey treated water (1995)	24.80	21.30	2.40	0.30		26.10		
Health Canada's One-Year Survey DS	49.50	47.30	2.2	0.1		16.90		
Median	23.30	34.30	2.30	0.20		21.00		

Note: Blank cells indicate information was not available.

\* Phase I samples were quenched.



## **CHAPTER 4**

### **SITE-SPECIFIC DATABASES FOR IOWA AND ONTARIO**

#### **INTRODUCTION**

Although informative national databases have been acquired, they are clearly biased toward a national perspective, both in the United States and Canada. Soliciting site-specific data relevant to Iowa and Ontario augmented these national databases for the purpose of improving the exposure assessment of the two indicated epidemiology studies. Site-specific databases were acquired from CHEEC that assembled pertinent data for an extensive Iowa database. Other Iowa-specific databases included the Iowa DNR (1993–1998) and Iowa utilities contained in the WATER:\STATS (1996) and ICR (1997–1998) databases. For Ontario, the Ontario Ministry of the Environment provided an extensive database, designated as the DWSP. The 53-Utility Survey database from Health Canada was also obtained, which provided information on 13 utilities within Ontario. These site-specific databases helped elucidate present and past historical trends in DBPs and treatment and disinfection practices for Iowa and Ontario.

#### **DESCRIPTION OF SITE-SPECIFIC DATABASES**

##### **Iowa**

In Iowa, dissolved organic carbon (DOC) and  $\text{Br}^-$  levels are near and below (U.S.) national averages, respectively. TTHM levels as well as individual THM species are presently below national averages. Three Iowa utilities are included in the ICR database: Des Moines, Cedar Rapids, and Iowa American. One Iowa utility that treated surface water was included in the NORS study (Davenport); this was further studied in the NOMS study along with Des Moines. Limited TTHM data are available from the WIDB and WATER:\STATS. Participating Iowa utilities in the WATER:\STATS study included Cedar Rapids and West Des Moines. An Iowa DNR database was obtained, which covers the period 1993–1998 and consists of THM compliance data from Iowa utilities. The database was used in another study to evaluate population exposure in the three main hydrologic units (watersheds) that comprise the state of Iowa. Cantor et al. (1998) assembled two THM databases (1979 and 1987 data) that were used in Cantor et al.'s previous epidemiology study in Iowa.

However, the key source of Iowa data was CHEEC, which compiles data from the Iowa DNR. The Iowa Groundwater Protection Act established CHEEC in 1987, with the goal, as quoted in the mission statement, “to determine the levels of environmental contamination which can be specifically associated with human health effects” (CHEEC 1998). CHEEC assembled a database of historical water supply source and treatment information, with information dating back to the early 1900s.

Because the ICR database contained information only for Cedar Rapids, Des Moines, and Davenport, the ICR database was expanded, through an extensive process, to include information for an additional 10 plants in the proximity of Iowa. This is discussed in more detail later in this chapter.

## Ontario

Ontario waters have are two distinguishing factors: low bromide levels, leading to lower levels of brominated DBPs; and natural organic matter properties promoting more HAA than THM formation and higher levels of TCAA than DCAA. These trends are in contrast to U.S. waters where the THM class dominates the HAA class; BDCM is a significant THM species; and DCAA and TCAA typically have similar concentrations. The 13-utility Ontario database for the year 1993, from the 53-Utility Survey, includes TTHM (and species) and HAA5 (and species) in both finished waters and distribution systems. The King and Marrett (1996) epidemiology study, which will be reanalyzed using the improved exposure assessment in this study, contains a THM database.

A key database is the Ontario DWSP, which initially consisted of approximately 22 plants and has grown in each subsequent year. In 1997, roughly 150 of Ontario's more than 400 treatment facilities, serving a population of 7.1 million (67% of Ontario's total population), were included in the survey. The 150 plants that are sampled each serve populations greater than 10,000 and account for 88% of the Ontario population served by public water. From 1988 to 1992, only TTHM (and specific THM species) were monitored, along with other parameters such as chlorine residual, pH, color, and DOC. TTHM sampling data consists of four to eight samples per distribution system per year. In addition to these parameters, HAA5 was measured at many of the plants from 1993 to 1997. In Canada, a TTHM guideline of 100 µg/L was not instituted until 1993, and plants began to comply with this guideline, in general, by 1995. Therefore, monitoring data between 1988 and 1994 was, in all probability, generally representative of historical levels, before regulatory pressure.

## ANALYSIS OF THE SITE-SPECIFIC DATABASES

### Iowa

The earliest Iowa database acquired was the Cantor et al. (1998) database. This database contained parameters including the 1990 population, person-years (defined as the exposure duration summed over the study population or selected subset) for each utility, plant maximum formation of the four THM species (samples were allowed to continue to form THMs instead of being quenched), quenched plant samples for the four THM species (chemicals were added to cease THM formation), TOC, TDS, total organic halides (TOX), temperature, pH, sample dates ranging from April to August 1987, sample location, and treatment types. Finished water samples were analyzed for quenched THM values. Quenched samples represented the amount of THMs that consumers were exposed to at those sampling locations, whereas "maximum formation" THM samples may not represent actual exposure but are more representative of the formation potential of the water. [Table 4.1](#) summarizes DBP values compiled in the Cantor et al. (1998) study.

The time period in which the Iowa DNR surveyed utilities is representative of utilities treating water to meet the 1979 THM Rule and trying to meet the soon-to-be enacted Stage 1 of the D/DBP Rule. [Table 4.2](#) represents overall DBP occurrence for Iowa during this period.

This database provides important information regarding DBP formation for Iowa utilities. The high *n* (count) values make this database a crucial one.

In 1996, the WATER:STATS database was compiled, which surveyed 3,200 utilities nationwide, of which 13 utilities were located in Iowa. [Table 4.3](#) describes TTHM values for the Iowa plants.

**Table 4.1**  
**DBP formation in Cantor et al. (1998) study**

Water type	Statistic	Quenched CHCl <sub>3</sub>	Quenched DBCM	Quenched BDCM	Quenched CHBr <sub>3</sub>
		(µg/L)			
Surface water (SW)	Median	85.0	2.0	13.0	0.0
	Range	2.0–160.0	0.0–3.0	0.0–24.0	0.0
Shallow groundwater systems with high-brominated THM concentrations (ShGW/HiBr)	Median	6.0	3.0	5.0	1.0
	Range	2.0–17.0	2.0–6.0	2.0–10.0	0.0–1.0
Shallow groundwater systems with low- brominated THM concentrations (ShGW/LoBr)	Median				
	Range				
Nonalluvial groundwater systems with high-brominated THM concentrations (NAGW/HiBr)	Median	0.0	0.0	0.0	0.0
	Range	0.0–12.0	0–4.0	0–5.0	0–1.0
Nonalluvial groundwater systems with low-brominated THM concentrations (NAGW/LoBr)	Median	0.0	0.0	0.0	0.0
	Range	0.0–1.0	0.0	0.0	0.0
SW/groundwater (GW)	Median	25.5	1.0	5.5	0.0
	Range	2.4–593.0	0.0–2.0	0.0–16.0	0.0
Overall	Median	12.0	1.0	5.0	0.0
	Range	0.0–160.0	0–6.0	0–24.0	0.0–1.0

Note: Blank cells indicate information was not available.

**Table 4.2**  
**THM formation in the Iowa DNR database**

Iowa statistic	CHBr <sub>3</sub>	CHCl <sub>3</sub>	DBCM	BDCM	TTHM
	(µg/L)				
Count	340	2,108	1,652	1,949	1,649
Average	1.72	37.18	3.07	10.12	58.27
Standard deviation	1.81	39.52	2.64	6.82	40.43
Median	1.00	29.00	2.20	9.90	53.60
Minimum	0.07	0.10	0.10	0.10	0.00
Maximum	14.00	550.00	22.00	69.00	297.00

**Table 4.3**  
**TTHM formation in the WATER:\STATS database**

Statistic	TTHM (µg/L) average
Count	5.0
Average	62.0
Standard deviation	18.4
Median	63.0
Minimum	43.0
Maximum	91.0

**Table 4.4**  
**DBP formation for the three Iowa plants in the ICR database**

Plant design	Finished median values (µg/L)				DSE median values (µg/L)			
	TTHM	HAA5	HAA6	HAA9	TTHM	HAA5	HAA6	HAA9
Iowa American (Davenport)	65.20	17.40	18.70		69.50	18.30	19.70	
Des Moines	19.50	5.80	7.60		34.25	7.60	11.10	
Cedar Rapids	0.50	1.80	1.80		0.50	2.15	2.15	
Overall for three plants	16.20	5.00	6.80		30.05	7.10	10.15	

Note: Blank cells indicate information was not available.

This database also supports the theory that Iowa-treated waters exhibit less TTHM formation than found in the national average. However, these data are biased due to the low count (*n*) value. Only 5 utilities out of the 13 surveyed reported a TTHM formation value. Because of this low *n* value, the database will not be used in the improved exposure assessment for the eventual reanalysis. However, the data can be utilized to better grasp an understanding of Iowa DBP formation over time by comparing WATER:\STATS with other Iowa databases. Also, it is important to note that the Iowa DNR data for TTHM are similar to the WATER:\STATS data, even considering WATER:\STATS' limitations. This was expected since the databases were created around the same time period when utilities were treating water to meet the 1979 THM Rule and Stage 1 of the D/DBP Rule.

ICR DBP data for the three Iowa utilities found in the ICR database are shown in [Table 4.4](#).

Because the ICR database contains information on only three Iowa utilities, the ICR database was expanded. To do this, the three Iowa utilities and their DBP precursors and relevant water quality parameters were first summarized. [Table 4.5](#) represents the findings for influent water quality parameters.

**Table 4.5**  
**Influent values for the three ICR–Iowa plants**

Plant averages and ID number	Statistic	Influent values					
		pH	Alkalinity (mg/L)	TOC (mg/L)	UVA <sub>254</sub> (cm <sup>-1</sup> )	Br <sup>-</sup> (mg/L)	SUVA* (L/cm-mg)
Cedar Rapids, 338 (groundwater)	Average	7.37	258	2.0	0.042	0.057	0.021
	Standard deviation	0.08	33	0.3	0.012	0.011	0.003
	25th percentile	7.32	239	1.8	0.034	0.052	0.018
	Median	7.35	250	2.0	0.036	0.058	0.021
	75th percentile	7.43	284	2.2	0.050	0.063	0.023
	Range	7.3–7.5	208–300	1.6–2.5	0.032–0.062	0.04–0.07	0.016–0.025
Davenport, 341 (surface water)	Average	8.05	163	4.7	0.162	0.028	0.022
	Standard deviation	0.20	13	0.8	0.035	0.011	0.020
	25th percentile	7.93	154	4.4	0.142	0.015	0.030
	Median	8.03	158	4.8	0.144	0.030	0.030
	75th percentile	8.17	174	5.1	0.173	0.034	0.030
	Range	7.80–8.30	150–179	4.0–5.5	0.141–.203	0.01–0.040	0.000–0.037
Des Moines, 340 (surface and groundwater)	Average	8.29	225	5.3	0.124	0.042	0.024
	Standard deviation	0.17	40	2.4	0.048	0.010	0.004
	25th percentile	8.20	208	3.8	0.104	0.033	0.022
	Median	8.26	245	4.4	0.117	0.042	0.023
	75th percentile	8.33	251	5.2	0.125	0.048	0.026
	Range	8.10–8.60	156–254	3.7–10.0	0.070–0.212	0.03–0.06	0.019–0.031
Three plants overall	Average	7.87	217	3.9	0.095	0.043	0.024
	Standard deviation	0.43	50	2.2	0.059	0.016	0.006
	25th percentile	7.43	170	2.2	0.043	0.030	0.021
	Median	8.00	230	3.7	0.086	0.040	0.023
	75th percentile	8.22	257	4.8	0.129	0.053	0.027
	Range	7.3–8.6	150–300	1.6–10.0	0.032–0.212	0.010–0.073	0.016–0.037

Note: SUVA (specific UV absorbance) is an indicator of humic content. Typically, SUVA at <0.03 L/cm-mg (<3.0 L/m-mg) contains largely nonhumic material, whereas SUVA in the range of 0.04–0.05 L/cm-mg (4.0–5.0 L/m-mg) contains mainly humic material. The surface water treated by Davenport is intermediate in humic content (based on SUVA), whereas the groundwater treated at Cedar Rapids is nonhumic. The mixed surface/groundwater treated by Des Moines is typically nonhumic (based on the median SUVA).

The ICR database was then queried for all plants throughout the United States that fell within a range for selected influent water quality parameters (pH, alkalinity, TOC, UV absorbance at 254 nanometers [UVA<sub>254</sub>], and bromide concentrations) corresponding to an average value plus or minus one standard deviation. From this analysis, a total of 29 plants that had similar water quality and DBP precursors as the Iowa plants were identified in the ICR database. These plants were then evaluated for their proximity to national watersheds (see U.S. Geological Survey [USGS] map in [Figure 4.1](#)) corresponding to the regional area of Iowa. For example, utilities located in California, Florida, Pennsylvania, and Utah were immediately dismissed. Upon these eliminations, utilities that fell into the regional watersheds encompassing Iowa were included to expand the Iowa–ICR database; 10 additional utilities located in proximate states—Illinois, Indiana, Kansas, Missouri, and Nebraska—were included. Each of these utilities lay within the two major watersheds of Iowa, which are the Upper Missouri and the Lower Missouri. Other USGS maps were used to verify similar vegetation and topographic soil characteristics of the added utilities outside of Iowa and compared with Iowa. At this point in the process, no utilities were eliminated due to differences in vegetation or topography.

In terms of bromide, the Iowa utilities tend to contain a typical level of bromide. Based on the ICR database, the nationwide median bromide level is ~0.04–0.05 mg/L. On the other hand, the Iowa waters exhibit relatively high alkalinity. Nationwide, high-alkalinity waters have >120 mg/L of alkalinity.

[Tables 4.6](#) and [4.7](#) list the expanded ICR–Iowa database (the 3 Iowa plants plus the additional 10 plants from surrounding areas and within the regional Upper Missouri and Lower Missouri watersheds) and the treatment and disinfection practices of each utility.

The expanded ICR–Iowa plants had a median TOC value of 3.63 mg/L and a bromide value of 0.051 mg/L. These data support the findings that Iowa waters have TOC and bromide values similar to the national U.S. median: 2.17 mg/L and 0.037 mg/L, respectively. Overall DBP formation for the expanded ICR database can be found in [Table 4.8](#).

Although values for TTHM and species are reported here, the eventual reanalysis will also focus on individual HAA species.

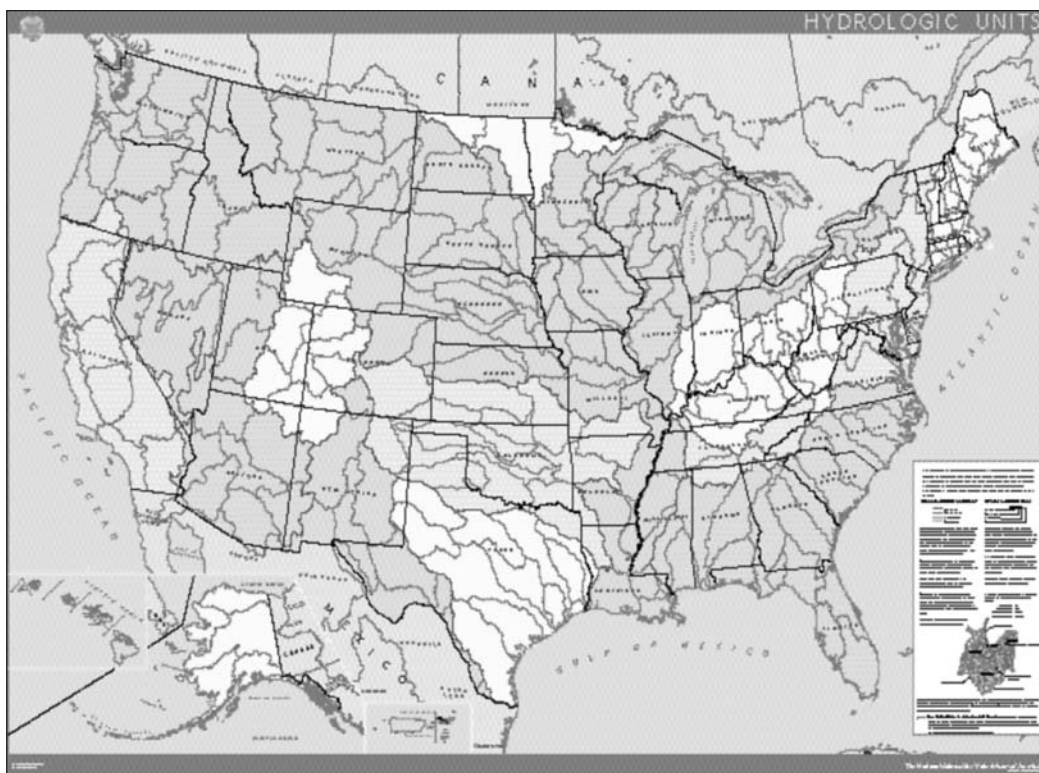
## Ontario

The 53-Utility Survey's time period, 1993, is of importance because this was the year that the Canadian government enacted a guideline of TTHM <100 µg/L. Therefore, this database was used to describe treatment prior to the enacted guidelines. A summary of this database can be found in [Table 4.9](#).

These Ontario waters had a median TOC value of 1.85 mg/L and an influent bromide level of 0.005 mg/L. These data support the finding that, when compared to the U.S. national averages, Ontario waters tend to have low influent bromide concentrations, and consequently low brominated DBP formation, and higher concentrations of HAA5 and HAA species than TTHM and THM species. The Ontario waters also tend to have a lower median TOC value compared to the United States.

While the Health Canada database surveyed DBPs over a 1-year time period, the DWSP has monitored Ontario utilities from 1986 to the present date and is still ongoing. A summary of DBP occurrence can be found in [Table 4.10](#).





**Figure 4.1 USGS map of major watersheds across the nation**

**Table 4.6  
Treatment type for the expanded Iowa plants in the ICR database**

Treatment type			
Conventional	Coagulation/sedimentation/ softening	Softening	Split/softening
360*—Peoria, Ill.	380—Johnston County, Kan.	437—St. Louis, Mo.	453—Omaha, Neb.
371—Indianapolis, Ind.	430—Kansas City, Mo.	438—St. Louis	
372—Indianapolis	340—Des Moines, Iowa	338—Cedar Rapids, Iowa	
373—Indianapolis			
384—Kansas City, Kan.			
341—Davenport, Iowa			

\* ICR numbers used to identify utilities.

**Table 4.7**  
**Disinfection practices for the expanded Iowa plants in the ICR database**

Disinfection practice				
Cl <sub>2</sub> /Cl <sub>2</sub>	Cl <sub>2</sub> /CLM	Cl <sub>2</sub> &CLM/CLM	CLM/CLM	CLX/CLM
360—Peoria, Ill.	438—St. Louis, Mo.	371—Indianapolis, Ind.	338—Cedar Rapids, Iowa	380—Johnston County, Kan.
453—Omaha, Neb.	341—Davenport, Iowa	372—Indianapolis		384—Kansas City, Kan.
340—Des Moines, Iowa		373—Indianapolis		
		430—Kansas City, Mo.		
		437—St. Louis		

Note: For the utilities in the ICR database that used prechlorination, most of the DBPs will have formed prior to the addition of chloramines. For systems that did not use free chlorine (primary disinfection with chlorine dioxide or chloramines), DBP formation will have been due to chloramine addition. Thus, the relationships between DBP precursors and DBP formation should be examined separately for these two disinfection scenarios.

**Table 4.8**  
**Overall DBP formation for the expanded ICR–Iowa plants**

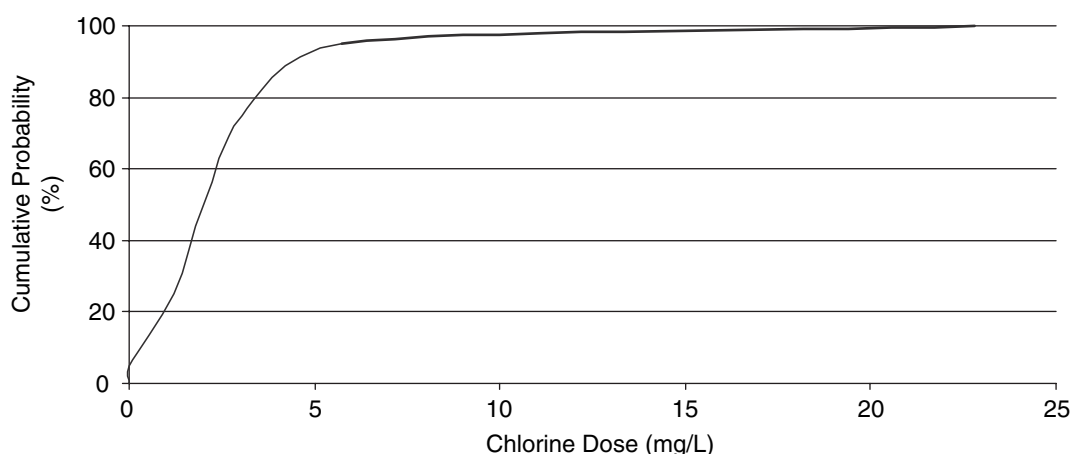
	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	HAA5	HAA6	HAA9
Expanded ICR–Iowa	(µg/L)							
ICR finished	16.50	6.50	1.50	0.50	26.70	12.00	13.00	38.00
ICR DSE	21.00	8.70	2.00	0.50	31.10	15.00	19.00	40.85

**Table 4.9**  
**TTHM and species information for the 13 Ontario utilities in Health Canada’s 53-Utility Survey**

	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	HAA5
	(µg/L)					
Finished water	5.55	3.60	1.20	0.50	12.15	16.45
Distribution system	8.70	3.85	2.50	0.70	14.55	19.72

**Table 4.10**  
**Overall DBP formation in the Ontario DWSP database**

Statistic	Treated water							Distribution system				
	CHCl <sub>3</sub>	CHBr <sub>3</sub>	DBCM	BDCM	TTHM	HAA6	CHCl <sub>3</sub>	CHBr <sub>3</sub>	DBCM	BDCM	TTHM	HAA6
	(µg/L)											
Count	923	923	923	923	923	427	833	833	833	833	833	405
Minimum	0.1	0.2	0.1	0.1	0.5	0.5	0.2	0.2	0.1	0.1	0.5	0.1
5th percentile	3.7	0.2	0.1	1.8	10.8	4.5	3.7	0.2	0.1	1.1	6.5	2.9
25th percentile	11.6	0.4	0.2	3.7	25.5	12.5	8.5	0.3	0.2	2.6	17.7	9.5
50th percentile	22.2	0.5	2.8	7.6	36.0	21.8	18.9	0.5	1.9	5.1	28.5	19.0
75th percentile	57.4	0.5	4.4	10.5	66.7	40.7	48.8	0.5	3.4	7.4	55.1	36.1
95th percentile	119.0	0.9	6.4	14.2	123.7	91.3	105.0	0.6	4.9	12.0	111.3	83.5
Maximum	283.2	17.0	30.0	38.8	289.4	316.0	229.3	12.5	28.7	36.7	234.0	299.5
Average	40.7	0.6	2.7	7.5	51.1	34.1	34.4	0.5	2.0	5.5	42.0	28.3
Standard deviation	42.9	0.8	2.4	4.3	41.4	36.9	37.8	0.5	1.9	3.6	37.0	32.9



**Figure 4.2 Cumulative probability of chlorine dose in the Ontario DWSP database**

HAA species data are available and discussed in chapter 5. The cumulative probability curve for total chlorine dose is shown in [Figure 4.2](#) for DWSP data. For TOC, the DWSP database showed an average of 2.13 mg/L.

Similar to the process of expanding the ICR–Iowa database from 3 to 13 utilities, using DWSP water quality, the ICR database was queried for all plants in the ICR database that fell within ranges for pH, temperature, turbidity, and DOC within plus or minus one standard deviation of the DWSP median values. This database was called the expanded ICR–Ontario database. A statistical summary of DWSP water quality parameters can be found in [Table 4.11](#).

**Table 4.11**  
**DWSP raw water quality parameters**

Statistic	Raw water average annual values			
	DOC (mg/L)	pH	Temperature (°C)	Turbidity (ntu)
Count	931	926	930	908
Minimum	0.2	6.0	0.2	0.0
5th percentile	0.9	6.9	4.4	0.3
25th percentile	1.8	7.3	8.0	0.9
50th percentile	2.1	7.6	10.0	2.1
75th percentile	5.4	8.0	11.5	6.4
95th percentile	10.0	8.3	15.0	31.8
Maximum	23.6	9.0	26.5	140.7

From this initial query, 39 plants were identified in the ICR database that had water quality parameters and DBP formation similar to plants within the DWSP database. These plants were then evaluated regarding their proximity to the Great Lakes region, using an atlas, and their corresponding watershed, using the USGS map ([Figure 4.1](#)). Utilities located in Indiana, central portions of New York, southern sections of Ohio, and Pennsylvania were immediately dismissed. However, 19 plants matching the DWSP water quality data and DBP formation and that fell within the Great Lakes watersheds were retained. This analysis provided a basis for estimating crucial missing data in the DWSP database, such as influent bromide analysis (discussed in chapter 5). Based on previous research and knowledge of existing models (e.g., the USEPA Water Treatment Plant Model), influent bromide, even in low-level bromide water, plays an important role in the modeling process. For example, the USEPA model, which is a five-parameter model, incorporates influent bromide in the modeling analysis. Summary statistics for the expanded ICR–Ontario database can be found in [Tables 4.12 to 4.14](#).

As stated earlier, the purpose of extracting plants from the ICR database that were similar to utilities in the DWSP database was to provide crucial model information that the DWSP database did not contain, specifically influent bromide. Thus, the expanded ICR–Ontario database could be used to further the development of multiple regression models.

## COMPARISON OF SITE-SPECIFIC WITH NATIONAL DATABASES

An overall summary of the Iowa databases is presented in [Table 4.15](#). HAA data were available and used in the improved exposure analysis in chapter 5. These values can be directly compared with the national U.S. databases. A summary of the national databases can be found in [Table 4.16](#).

From the two tables, it is evident that the Iowa waters tend to form less DBPs than are found in U.S. national levels. The TTHM and HAA5 for Iowa fall slightly below the median

**Table 4.12**  
**Influent water quality parameters for the expanded ICR–Ontario database**

Statistic	pH	Alkalinity (mg/L as CaCO <sub>3</sub> *)	Turbidity (ntu)	Temperature (°C)	TOC (mg/L)	UV (cm <sup>-1</sup> )	Br <sup>-</sup> (ppm)	NH <sub>3</sub> (mg/L as N)†	TOX (µg/L as Cl)
Count	225	225	225	225	225	200	225	220	210
Maximum	8.08	264	20.0	13.1	5.90	0.1285	0.16	0.3	88.5
95th percentile	8.008	250	15.1	13.0	5.08	0.1163	0.13	0.3	25.0
75th percentile	7.9	112	7.17	12.0	2.65	0.0695	0.057	0.1	25.0
50th percentile	7.65	88	3.7	10.5	1.90	0.045	0.029	0.1	25.0
25th percentile	7.42	46	1.81	9.1	1.70	0.031	0.01	0.1	25.0
5th percentile	7.26	10	0.46	7.5	0.95	0.01215	0.01	0.1	25.0
Minimum	7.19	10	0.01	6.5	0.90	0.0045	0.01	0.1	25.0

\* CaCO<sub>3</sub>: Calcium carbonate.

† NH<sub>3</sub>: Ammonia.

**Table 4.13**  
**Spatial distribution of THMs in the expanded ICR–Ontario database**

Statistic	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM
	(ppb)				
Count	194	194	194	194	194
95th percentile	40.8	15.5	6.5	2.1	58.1
75th percentile	23.8	9.9	4.4	0.5	35.7
50th percentile	15.3	7.2	2.9	0.5	25.3
25th percentile	6.5	4.2	1.7	0.5	16.6
5th percentile	1.8	2.4	0.5	0.5	9.0
Minimum	0.5	1.0	0.5	0.5	2.5

values for all of the national databases. This can be further illustrated by comparing the three ICR–Iowa plants against the ICR database as a whole. Although the ICR database showed a finished water TTHM concentration of 32.2 µg/L and a DSE value of 40.02 µg/L, the three Iowa plants in the ICR database exhibited a lower finished TTHM concentration of 16.2 µg/L and a DSE value of 30.1 µg/L. This same trend was observed for HAAs. The overall ICR database showed a median finished water HAA value of 20.4 µg/L and a DSE value of 22.28 µg/L, whereas

**Table 4.14**  
**Spatial distribution of HAAs in the expanded ICR–Ontario database**

	TCAA	DCAA	MCAA	BCAA	DBAA	MBAA	BDCAA	DBCAA	TBAA	HAA5	HAA6	HAA9
Statistic	(ppb)											
Count	207	212	203	212	212	212	82	80	72	198	198	72
Maximum	59.0	33.0	15.0	7.8	6.6	2.2	8.2	2.9	2.0	85.0	88.6	45.6
95th percentile	38.0	24.2	5.8	6.8	2.6	1.0	7.7	2.4	2.0	64.3	68.6	43.9
75th percentile	18.3	14.0	1.5	3.8	1.1	0.5	4.7	1.0	2.0	34.2	38.3	25.6
50th percentile	8.8	9.1	1.0	2.9	0.5	0.5	3.8	1.0	2.0	19.2	23.0	17.2
25th percentile	2.8	3.8	1.0	1.8	0.5	0.5	1.6	1.0	2.0	8.2	10.7	8.3
5th percentile	0.5	0.5	1.0	0.5	0.5	0.5	0.5	1.0	2.0	1.8	2.7	1.2
Minimum	0.5	0.5	1.0	0.5	0.5	0.5	0.5	1.0	2.0	0.5	0.5	0.5

**Table 4.15**  
**Overall summary of the formation of DBPs in the Iowa databases**

	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	HAA5	HAA6	HAA9
Study	(µg/L)							
Cantor et al. (1998)	117.0	23.0	6.0	0.0				
Iowa DNR (1993–1998)	29.0	9.9	2.2	1.0	53.6			
WATER\STATS (1996)					63.0			
ICR database (three Iowa plants) treated (1997–1998)	11.2	4.2	1.1	0.5	16.2	5.0	6.8	
ICR database (three Iowa plants) DSE (1997–1998)	19.1	6.6	1.6	0.5	30.1	7.1	10.2	
Expanded ICR database (13 plants) treated	16.5	6.5	1.5	0.5	26.7	12.0	13.0	38.0
Expanded ICR database (13 plants) DSE	21.0	8.7	2.0	0.5	31.1	15.0	19.0	40.9
Median	20.0	7.7	1.8	0.5	30.6	9.6	11.6	39.4

Note: Blank cells indicate information was not available.

the three ICR–Iowa plants had lower HAAs values: 5.0 µg/L and 7.1 µg/L, respectively. These data support the finding that TTHM levels in Iowa are lower than the national averages.

A summary for the Canadian databases is presented in [Table 4.17](#).

Similar to the other databases, HAA data were available and utilized in the improved reanalysis. A comparison is difficult to make because the only national Canadian database was the Health Canada survey conducted in 1993. However, from these data, it is apparent that Ontario DBP formation is very similar to national Canadian trends.

**Table 4.16**  
**Overall summary of national U.S. databases**

Study	Median concentrations of THMs and HAAs (µg/L)							
	TTHM	CH <sub>3</sub> Cl	BDCM	DBCM	CHBr <sub>3</sub>	HAA5	HAA6	HAA9
NORS (1975)		21.00	6.00	1.20	BDL*			
NOMS (1976–1977)	42.80	27.00	9.60	1.20	2.50			
AwwaRF THM Survey (1984–1986)	37.00							
35-Utility Study (1988–1989)	37.00	13.15	6.60	3.40	0.57	18.00		
WATER\ASTATS finished (1996)	40.03							
WATER\ASTATS DS	33.80					23.06		
ICR finished water (1997–1998)	32.32	16.33	7.13	2.52	0.00	20.40	23.40	24.78
ICR DSE	40.02	23.35	8.85	2.80	0.00	22.28	26.28	28.15
Median	37.00	21.00	7.13	2.52	0.285	21.34	24.84	26.465

Note: Blank cells indicate information was not available.

\* BDL: Below detection limit.

**Table 4.17**  
**Comparison of the national Canadian databases with the site-specific Ontario databases (median values)**

Study	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	HAA5	HAA6	HAA9
	(µg/L)							
53-Utility Survey treated (1993)	5.6	3.6	1.2	0.5	12.2	16.5		
53-Utility Survey DS (1993)	8.7	3.9	2.5	0.7	14.6	19.7		
Ontario DWSP treated (1990–1998)	22.2	5.1	2.8	0.5	36.0	21.8		
Ontario DWSP DS (1990–1998)	18.9	7.6	1.9	0.5	28.5	19.0		
Median	13.8	4.5	2.2	0.5	21.5	19.4		

Note: Blank cells indicate information was not available.

**Table 4.18**  
**Site-specific database DBP summary (median values) comparing Iowa**  
**and Ontario databases**

Study	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	HAA5	HAA6	HAA9
	(µg/L)							
Canter et al. (1998)	117.0	23.0	6.0	0.0				
Iowa DNR (1993–1998)	29.0	9.9	2.2	1.0	53.6			
WATER\STATS (1996)					63.0			
ICR database (three Iowa plants) treated (1997–1998)	11.2	4.2	1.1	0.5	16.2	5.0	6.8	
ICR database (three Iowa plants) DSE (1997–1998)	19.1	6.6	1.6	0.5	30.1	7.1	10.2	
Expanded ICR database (13 plants) treated	16.5	6.5	1.5	0.5	26.7	12.0	13.0	38.0
Expanded ICR database (13 plants) DSE	21.0	8.7	2.0	0.5	31.1	15.0	19.0	40.9
Median	20.0	7.7	1.8	0.5	30.6	9.6	11.6	39.4
53-Utility Survey treated (1993)	5.6	3.6	1.2	0.5	12.2	16.5		
53-Utility Survey DS (1993)	8.7	3.9	2.5	0.7	14.6	19.7		
Ontario DWSP treated (1990–1998)	22.2	5.1	2.8	0.5	36.0	21.8		
Ontario DWSP DS (1990–1998)	18.9	7.6	1.9	0.5	28.5	19.0		
Median	13.8	4.5	2.2	0.5	21.5	19.4		

Note: Blank cells indicate information was not available.

## COMPARISON OF IOWA WITH ONTARIO SITE-SPECIFIC DATABASES

While the site-specific databases were compared with their nation's counterparts, a comparison of the Iowa databases with the Canadian databases can be found in [Table 4.18](#).

When comparing Iowa with Ontario, it is evident that the Ontario waters tended to form less TTHM than the Iowa waters. The Ontario waters also tended to form higher levels of HAAs and HAA species, presumably because the Ontario waters contain natural organic matter that promotes HAA formation over TTHM formation. The Ontario waters also contained less bromide, resulting in lower formation of brominated DBPs and thus higher formation of



**Table 4.19**  
**Comparison of DOC, pH, and temperature for the DWSP, ICR, and expanded ICR–Iowa databases**

Database	DOC ( $\cong$ TOC) (mg/L)			pH			Temperature ( $^{\circ}$ C)		
	10th percentile	50th percentile	90th percentile	10th percentile	50th percentile	90th percentile	10th percentile	50th percentile	90th percentile
DWSP	1.42	2.13	8.63	7.03	7.64	8.17	5.75	9.96	13.33
Expanded ICR–Iowa	1.86	3.65	5.85	7.42	8.18	8.35	17.00	15.50	26.00
Overall ICR	0.00	2.35	5.30	6.70	7.60	8.27	8.00	18.00	26.70

chlorinated DBPs. This is evident in the observation that the overall Ontario databases exhibited lower BDCM, DBCM, and  $\text{CHBr}_3$  concentrations compared to the overall Iowa values.

The Ontario phenomenon of forming HAAs preferentially over THMs may also be attributed to other factors, including pH, temperature, and TOC ( $\cong$ DOC). [Table 4.19](#) illustrates a comparison of pH, TOC ( $\cong$ DOC), and temperature for the DWSP database, the ICR database overall, and the expanded ICR–Iowa database. Although not reported, SUVA may be another influential property.

Higher pH favors THM formation over HAA formation. For the Ontario utilities in the DWSP database, the median pH was 7.64. However, the percentile value was 7.03. Under this lower pH condition, HAA formation would be preferential over THM formation. A high TOC or DOC concentration will also affect DBP formation, as TOC or DOC serves as a DBP precursor. DWSP DOC levels are comparable to the U.S. levels, as described by the ICR database overall value. However, most importantly, temperature can significantly affect DBP formation. Lower temperatures tend to suppress THM formation. As evident in [Table 4.19](#), the DWSP database reported lower temperatures than those reported in the ICR database. Therefore, at the lower temperatures, the DWSP utilities promoted HAA formation over THM formation. Although natural organic matter properties may play an important role in preferential HAA formation over THM formation, other factors, such as TOC or DOC, pH, and temperature, may also contribute to this phenomenon.



## CHAPTER 5

### AN IMPROVED EXPOSURE ASSESSMENT: CENTRAL TENDENCY MODELS

#### INTRODUCTION

The goal of this study is to provide an improved exposure assessment for a reanalysis of two well-conducted, peer-reviewed epidemiology studies, conducted in the mid-1990s, investigating the risk of cancer associated with drinking water (Cantor et al. 1998; King and Marrett 1996). To perform this task, it was necessary to first construct and reconstruct exposure to chlorination DBPs over the past 50 years both in Iowa and Ontario. A 50-year time period allows adequate time for cancer to be induced. National databases were used to define regulatory transitions backward through time, which resulted in a historical and chronological overview of DBP formation and disinfection practices. Once this historical timeline was created, statistical models were utilized to fill in any missing gaps of data (e.g., projecting past HAA occurrence). These data gaps occurred because the historical data tended to be extremely fragmented or did not exist at all, especially for HAAs, due to the lack of regulations or monitoring requirements present at that specific time. With an improved exposure assessment, the Iowa and Ontario epidemiology studies can be fully reanalyzed in terms of DBP occurrence and exposure and its link to cancer.

#### PRIORITIZATION OF MODELING PARAMETERS

Before the modeling could be initiated, it was first necessary to decide which specific dependent and independent variables would be utilized in the modeling efforts. [Table 5.1](#) represents the prioritization of parameters and the exposure components they are intended to address.

Each exposure component listed in [Table 5.1](#) represents what was ideally preferred in terms of the modeling efforts. Many of the ideal parameters can be found in the newer and more robust databases such as the ICR database, whereas the older databases do not contain as much information on DBP precursor or water quality parameters. In lieu of actual parameter values, surrogate parameters can be considered. For example, although TTHM and species can readily be found in databases created in the early 1980s to the present, HAAs and species can only be found in databases from the mid- to late 1990s, especially for HAA5 and HAA9 formation. Predictive parameters such as pH, UVA, and DOC, can be used to calculate these missing HAA values through statistical relationships. DOC (or TOC,  $\approx$  DOC) and pH data are parameters that are readily available throughout the time line of the various databases. When examining brominated versus chlorinated species, the best parameter to determine such a relationship is bromide concentration. However, bromide data for Iowa can currently only be found extensively in the ICR database and slightly less in the 35-Utility Study database. Similar to HAA data, bromide concentration is a relatively newly monitored DBP precursor and water quality parameter. Surrogates such as chloride and TDS or calculated bromide incorporation associated with measured THM species (THM-Br) can be utilized to calculate the missing bromide data for other databases. The calculated bromide concentration can then be utilized to determine the relationships between brominated and chlorinated DBPs. Regarding the accumulated databases, both the Iowa and Ontario databases contain a significant amount of data in terms of the desired parameters. Only parameters such as bromide concentration and UVA values are missing.

**Table 5.1**  
**Prioritization of modeling parameters**

Exposure component	Predictive parameters	Surrogates
TTHM versus HAAs	pH SUVA = UVA/DOC	
Brominated versus chlorinated THMs	Br <sup>-</sup> Br <sup>-</sup> /TOC	Cl <sup>-</sup> , * THM-Br, TDS
	Time	Distance from treatment plant in distribution system, chlorine demand
	Temperature	Season
	pH	
DBPs	TOC	Coagulant dose, Cl <sub>2</sub> dose
	UVA	Color
	Br <sup>-</sup>	TDS, Cl <sup>-</sup> , THM-Br
	Cl <sub>2</sub> dose	Cl <sub>2</sub> usage
	NH <sub>3</sub>	

Note: DOC and TOC are considered to be approximately equal, and hence are considered synonymous.

\* Cl<sup>-</sup>: Chloride ion.

For the prioritization of parameters, the first focus should be on examining TOC and chlorine dose. TOC is the most significant indicator (and predictor) of THM and HAA levels. Although bromide affects DBP speciation, bromide concentrations generally do not vary significantly over time in the Iowa and Ontario waters. Chlorine dose is important, as it affects DBP formation and is an indicator (and predictor) of the reactivity of the TOC. Next, THMs and HAAs should be analyzed to develop general and/or site-specific relationships for DBP formation in Iowa and Ontario waters. DBP occurrence data are needed for representative surface and groundwaters, including those with relatively high levels of brominated species. At a minimum, data are needed on the five regulated HAAs, although it would be advantageous if utilities have data on the six, or even the nine, HAAs, as reported by some utilities in the ICR database. In other research (Singer et al. 2002), equations have been developed to predict other HAA species based on THM and HAA5 data. For systems that only have THM data, it is necessary to estimate their HAA occurrence based on similarities to other waters with HAA data.

The next grouping of priority parameters encompasses pH, temperature, and ammonia. DBP formation (THMs versus HAAs at a higher versus lower pH, respectively) is affected, in part, by pH, and DBP formation is temperature dependent. Ammonia can significantly affect chlorine demand; however, if current data on pH, temperature, and ammonia are obtained, these data can be used to represent historical water quality, unless there have been significant climatic variations and/or significant variation in ammonia sources over time.

The last priority relates to bromide, UVA, and (reaction) time. Although bromide and UVA are important parameters, it is apparent that such data do not readily exist, except in the ICR database. However, the current bromide and UVA data that are currently available have been assembled. Whenever possible, surrogate parameters for bromide (e.g., TDS, chloride) and UVA (i.e., color) are utilized. Bromide concentrations can also be estimated from bromide incorporation into the THM species (i.e., calculating estimates of  $\text{Br}^-$  from measured values of THM-Br). Although (reaction) time is an important parameter, contact time within a treatment plant or a distribution system is highly variable (due to diurnal and seasonal variations). Contact times can be approximated based on plant flows (water demands), size of basins in the plants, distances in the distribution system, chlorine demand, and so forth.

## HIT LIST OF IOWA UTILITIES

Because of the large numbers of Iowa utilities represented in the CHEEC and Cantor et al. databases (1998), a smaller number of Iowa utilities were identified, through an extensive selection process, as a part of an optimal and workable “hit list.” The hit list is a smaller subset of utilities representative of all the utilities listed in both the Cantor study and the CHEEC database. Due to the large numbers of utilities in these databases, modeling and reanalysis of all 356 utilities would be extremely time-consuming and tedious. Therefore, plants were selectively chosen that were representative of all the utilities, and these were used to create the smaller hit list. Since bladder cancer is highly associated with chlorinated surface water, chlorinated surface waters tend to have significantly higher THMs and other DBPs than chlorinated groundwaters and were weighted more in the selection process.

For the first Iowa database provided by Cantor et al., the database of 356 utilities was segmented into six primary water types:

- 39 surface water (SW) systems
- 50 shallow groundwater systems with high-brominated THM concentrations (ShGW/HiBr)
- 19 shallow groundwater systems with low-brominated THM concentrations (ShGW/LoBr)
- 41 nonalluvial groundwater systems with high-brominated THM concentrations (NAGW/HiBr)
- 190 nonalluvial groundwater systems with low-brominated THM concentrations (NAGW/LoBr)
- 17 mixed systems in which utilities utilize both surface water and groundwater

Bromide was not measured in this survey; rather, groundwaters were divided into those with relatively high proportions of brominated THMs and those with relatively low proportions of brominated THMs. This database was selected for inclusion in the Iowa hit list, because it included a utility’s 1990 census population and person-years of exposure information. In a case control study, the term *person-years* refers to exposure duration summed over the study population (or selected subset). For example, if 100 persons were exposed for 5 years each, there would be 500 person-years of exposure. In actuality, each person typically would have a different duration of exposure, and the total person-years of exposure would be exposure duration summed over all persons in the group.

**Table 5.2**  
**Population of Iowa utilities contained in the Cantor et al. (1998) database**

	1990 population	Plants serving:								
		>1,000	>1,500	>2,000	>2,500	>3,000	>4,000	>5,000	>7,500	>10,000
Population summary	1,863,348	1,794,853	1,702,856	1,640,110	1,582,150	1,524,534	1,471,494	1,403,828	1,265,860	1,146,459
Percentage of population	100.00	96.32	91.39	88.02	84.91	81.82	78.97	75.34	67.93	61.53
Number of plants	340	255	179	142	116	95	80	65	42	28
Percentage of total plants	100.00	75.00	52.65	41.76	34.12	27.94	23.53	19.12	12.35	8.24

Note: Although the Cantor et al. database contained information regarding 356 utilities, only 340 utilities contained 1990 census population statistics.

**Table 5.3**  
**Person-years contained in the Cantor et al. (1998) database**

	Person-years (PY)	PY >100	PY >200	PY >500	PY >1,000	PY >5,000	PY >10,000
Sum of PY	140,229	132,953	132,953	99,975	79,513	44,178	16,632
Number of plants	356	196	196	50	21	5	1
Percent of PY	100.00	94.81	94.81	71.29	56.70	31.50	11.86
Percent of plants	100.00	55.06	55.06	14.04	5.90	1.40	0.28

Note: The sum of person-years is a relatively small number because the entire population in Iowa (approximately 2 million) did not respond to Cantor et al.'s survey.

The first approach was to base the hit list on population data for each utility, regardless of water type. [Table 5.2](#) describes cutoff levels for utilities for populations served.

Next, the same approach was applied for utilities versus person-years, as detailed in [Table 5.3](#).

Both of these methods were unacceptable. If utilities were only selected on the basis of population or person-years, variability in DBP exposure (high versus low) was lost among the different water types. Since both methods were not able to reflect variances among DBP exposure within the database, it was concluded that the hit list should be based on the water type, and then a reasonable number of representative utilities could be chosen from each category. A careful balance between person-years data and variability in DBP exposure, both high and low, was considered to ensure that the utilities included were both representative of their water-type grouping category and contained an adequate number of person-years. The selection process was as follows:

- *SW systems.* Nine utilities were identified that represent 87% of the person-years in the surface water grouping (see hit list summarized in [Table 5.4](#)). Riverdale (7% of the person-years) was included because it is served by Davenport (the Iowa American plant from the ICR database and many others), and therefore Davenport should extend to Riverdale. Several smaller systems were included that reported very high THM values. These may be representative of large surface water systems prior to the 1979 THM regulations.
- *ShGW/HiBr systems.* Included were five systems that served more than 10,000 people, representing 46% of the person-years in this grouping.
- *ShGW/LoBr systems.* The only utility included was Cedar Rapids, because Cedar Rapids represents 81% of the person-years in the grouping and serves a large population greater than 100,000 people. The next largest system is significantly smaller and serves less than 3,000 people. It is believed that this group of utilities is representative of low THM exposure. From the databases, it is evident that the smaller systems contain significantly less data than the larger utilities.
- *NAGW/HiBr systems.* The six utilities selected represent 64% of the person-years in this grouping and each serves more than 10,000 people. Each utility is expected to have been collecting THM data as required by the SDWA.
- *NAGW/LoBr systems.* Five utilities were included, representing 29% of the person-years in this grouping. Because they all serve more than 10,000 people, all are expected to have been collecting THM data. These systems are expected to produce the lowest levels of TTHM and brominated THM species; thus these five systems may be useful in predicting exposure for the remaining systems in this group, which is by far the largest number of systems.
- *SW/GW systems.* Six utilities serve more than 10,000 people and should have THM data. A number of the other communities obtain their water from some of the larger ones, so they were also included. Some mixed systems that use >50% surface water and that serve more than 10,000 people were included. Again, they are considered representative of pre-1979 treatment and disinfection conditions. The eight utilities in this group represent 94% of the person-years in this classification.

The total hit list encompasses 34 Iowa utilities, representing 63% of the total person-years in the study, and is summarized in [Table 5.4](#). Anywhere from 87% to 94% of the person-years for the surface water or mixed systems are represented. Because most groundwater systems represent low DBP exposure, focusing on key utilities in these groupings may be adequate.

## HIT-LIST SELECTION OF ONTARIO UTILITIES

For the Ontario epidemiology study conducted by King and Marrett (1996), a different approach was taken when considering the modeling and correlation analysis. Since the Ontario database contained approximately 170 utilities, it was decided that all of the utilities would be incorporated into the modeling process applied to the improved exposure assessment.

**Table 5.4**  
**Iowa hit-list utilities\***

SW	ShGW/HiBr	ShGW/LoBr	NAGW/HiBr	NAGW/LoBr	SW/GW
Iowa American (Davenport)	Muscatine	Cedar Rapids	Waterloo	Mason City	Des Moines
Ottumwa	Newton		Sioux City	Clinton	Council Bluffs
Keokuk	Spencer		Dubuque	Fort Dodge	Burlington
Creston	Boone		Cedar Falls	Marshalltown	Iowa City
Iowa American (Bettendorf)	Oskaloosa		Marion	Ames	Fort Madison
Centerville			Indianola		West Des Moines
Clarinda					Fairfield
Osceola					Winterset
Chariton					Des Moines (Norwalk)
Spirit Lake					Des Moines (Urbandale)
Iowa American (Riverdale)					Des Moines (Johnston)
					Des Moines (Clive)
					Burlington (Middleton)
					Des Moines (Windsor Heights)

\* Selected utilities (communities) and corresponding water types from the Cantor et al. (1998) database.

## MODELING APPROACH

### Overview

The major objective of the modeling effort was to provide estimates of chlorination and chloramination DBPs (TTHM, individual THM species, HAA5, HAA6, HAA9, and individual HAA species) and DBP-related parameters (DXAA, TXAA, THM-Br, HAA-Br, DBP-Br [sum of THM-Br and HAA-Br], and TOX) over an approximately 50-year period of record (1940–1990). DXAA and TXAA are the total sums of the di- and trihaloacetic acids. More specifically, the objective was to estimate missing values within the Iowa and Ontario databases. The approach was to chronologically progress backward along a time line from the present (robust database with few missing values) to the past (sparse database with many more missing values), recognizing historical milestones of significance (e.g., promulgation of the 1979 THM Rule and



subsequent compliance over the promulgation period for Iowa and the 1993 THM guideline for Ontario). Where possible, site-specific (or regional) models and correlations were used in those cases where adequate data were available to define such relationships; otherwise, national models and correlations were used. A key issue was relating finished water DBPs to distribution-system DBPs, upon which an exposure assessment should be based; this was an issue for data sets and subsets containing only finished water DBPs. Ideally, the final product from the modeling efforts would be a three-dimensional matrix (spreadsheet[s]) describing DBPs versus time (ranging from 1940 until the year of the epidemiology study to be subjected to reanalysis) and location (utility); for earlier years within the time line (e.g., before 1975), DBP estimates were provided according to yearly increments and were regionally based (as opposed to individual-utility based).

### **Level 1: Site-Specific Models and Correlations**

Data from the Iowa and Ontario databases were used to develop both simple (single) and multiple regression models. Multiple regression models can be constructed to describe each dependent variable; the individual DBPs and DBP-related parameters, in terms of independent variables comprising DBP precursors (DOC, UVA<sub>254</sub>, Br<sup>-</sup>); disinfectant conditions (Cl<sub>2</sub> [or chloramines] dose[s], residual[s], and demand[s]; contact/residence time); and water quality conditions (pH and temperature). While single regressions were used to model one parameter against another (i.e., TTHM as a function of influent TOC), multiple regressions were performed stepwise, with sequential inclusion of the most statistically significant parameters up to a defined point of diminishing returns (e.g.,  $\Delta R^2$ ) (i.e., TTHM as a function of influent TOC, pH, temperature, influent bromide, and chlorine dose). Simple correlations can be established among individual DBPs/DBP-related parameters (e.g., BCAA versus BDCM) or between DBPs/DBP-related parameters and DBP precursors (e.g., DCAA versus DOC), disinfectant conditions (e.g., DCAA versus Cl<sub>2</sub> dose), and water quality (e.g., TCAA versus pH). In some cases, simplistic multiple regression models may evolve (e.g.,  $\text{TTHM-Br} = f[\text{TOC and Br}^-]$ ).

### **Level 2: Regional Models and Correlations**

The expanded ICR database, consisting of the three Iowa utilities in the ICR database and 10 other utilities in surrounding states and situated within the two main USEPA-defined watersheds comprising Iowa (Upper Missouri River and Lower Missouri River), were used to define models and correlations similar to those described for level 1 for the Iowa hit list. (The expanded ICR–Ontario database was defined similarly to Iowa except for the use of DWSP utilities.) These utilities were identified and described according to the disinfection scheme described in chapter 4. Because the ICR database contains much more HAA5 and HAA6 data than HAA9 data, the approach of Singer et al. (2002) can be used to estimate the remaining brominated HAA species from brominated THM species (e.g.,  $\text{BDCAA} = (\text{BDCM}/\text{CHCl}_3) * \text{TCAA}$ , as a molar basis; discussed in more detail later). However, because of lower levels of Br<sup>-</sup> (based on limited data), HAA5 or HAA6 may be sufficient. Given the pH dependencies of TTHM, DXAA, and TXAA, pH would also be potentially useful in defining HAA speciation. The 34 utilities within the hit list were first categorized according to watershed and next according to surface water versus groundwater, and finally according to disinfection scheme. This data stratification approach allows application of the most appropriate models and correlations.

For Ontario, one approach for a level 2 analysis consisted of extracting the data from the King and Marrett (1996) database. Unfortunately, this database only provided data for chlorine dose dating back to the early 1940s. While a multiple regression analysis could not be performed on this data, the database provided important information regarding chlorine dose over time. However, the King and Marrett database helps to support assumptions for the Ontario model that will be discussed later regarding historical treatment changes and DBP formation.

The second approach for the level 2 analysis for Ontario was completed through the creation of the expanded ICR–Ontario database. The expanded database provided additional modeling parameters such as influent bromide concentration and other data that would be utilized for the Ontario modeling development.

### **Level 3: National Models and Correlations**

National (U.S.) DBP prediction models exist but require robust input data for accurate predictions. Given that TOC and Br<sup>-</sup> data are significantly lacking for the overall Iowa database, it does not appear that these models play an important role. However, these databases provide important information regarding DBP formation trends over time.

As stated earlier, Canada has no federal regulations regarding DBPs, and a robust national Canadian DBP database does not exist.

### **Accounting for Historical Milestones**

National databases to describe DBP occurrence trends span the time frame from 1975 through 1998 for the United States (i.e., Iowa): NORS, NOMS, AwwaRF THM Survey, WIDB, 35-Utility Study, WATER\STATS, and ICR. National trends in terms of average and median DBP levels were tracked to provide a basis for estimating past DBP occurrence from present occurrence. These chronological DBP trends were interpreted in terms of important historical milestones affecting DBP levels (e.g., the THM levels before and after promulgation of the 1979 THM MCL). For example, the 1979 THM MCL was not only a time point of interest, but also sensitized utilities and marked gradual changes of treatment throughout the 1980s.

For Ontario, the only historical DBP milestone was the TTHM MCL enacted in 1993. The King and Marrett database was used to support the assumption that, prior to 1993, general treatment and disinfection practice had not changed from approximately 1940 to 1993. This can be reflected in the fact that chlorine levels remained approximately the same over a five-decade period. Because the treatment plants did not have to meet any regulated DBP standards, they had no incentive to significantly change their general treatment and disinfection practice, as illustrated in [Table 5.5](#).

### **Role of Full ICR Database**

One important role of the full ICR database is to define relationships between distribution system and finished water DBPs in terms of a ratio and/or increment, for both postchlorination and postchloramination. (Data for both the finished water and distribution system are readily available in the ICR database.) Such adjustments would permit estimates of distribution system DBPs when only finished water DBPs is available.

**Table 5.5**  
**Disinfectant dosage over time in the King and Marrett (1996) database**

Time period	Predisinfection dose*		Pre-other dose†		Postdisinfection dose‡	
	50th percentile	95th percentile	50th percentile	95th percentile	50th percentile	95th percentile
1975–1990	1.0	5.5	1.3	14.1	0.9	5.0
1950–1973	1.0	9.3	0.8	1.5	0.7	3.7
1940–1945	1.2	7.0	0.0	0.0	0.5	2.4

\* Chlorine or sodium hypochloride/ite.

† Ammonia, chlorite, or sodium hypochloride/ite.

‡ Chlorine or chlorine dioxide.

Another potential role of the ICR database is to determine relationships between levels and distributions of chloramination versus chlorination DBPs. (For example, the USEPA Water Treatment Plant Model simply assumes that, all factors equal, TTHM levels under chloramination are 20% of those formed under free chlorination.) Also, as suggested above, the full ICR database can be used to estimate ratios and incremental levels of chloramination DBPs formed in the distribution system versus the finished water.

The ICR database was the only U.S. national database to provide extensive HAA information, because utilities were required to report HAA5 and species information. Approximately 90 utilities also reported HAA6 (and species) and HAA9 (and species) data.

## MODELING EFFORTS

### Ontario Modeling Efforts

The DWSP database was the first site to undergo the improved exposure analysis. However, before the model development could begin, several issues had to be dealt with regarding the software package Statistica. When using the program to develop models, the first question that arises is which relationship to use: linear or nonlinear. A simple linear relationship is very convenient and provides straightforward interpretations as “the more of  $x$  (e.g.,  $\text{Cl}_2$  dose), the more there is of  $y$  (HAA5 formation); and, given a particular increase in  $x$ , a proportional increase in  $y$  can be expected.” Nonlinear relationships cannot usually be interpreted and verbalized in such a simple manner. The second issue that needs to be addressed is how to exactly compute the relationship; that is, how to arrive at results that determine whether or not there is a nonlinear relationship, as predicted.

A linear multiple regression model can be constructed as

$$y = a + b_1 * x_1 + b_2 * x_2 + \dots + b_n * x_n$$

where  $y$  = dependent variable (i.e., HAA6 formation)

$a$  = intercept

$x$  values = the independent variables (i.e., water quality parameters or other DBPs)

$b$  values = coefficients for the independent variables

In general, whenever the simple linear regression model does not appear to adequately represent the relationships between variables, then the nonlinear regression model approach is appropriate.

A nonlinear multiple regression can be constructed as

$$y = a * x_1^{b^1} * x_2^{b^2} * \dots * x_n^{b^n}$$

where the variables are the same as in the linear multiple regression example.

Developed models were analyzed and sorted according to their  $R^2$  (R-square), F, and p-level. The  $R^2$  value is an indicator of how well the model fits the data. For example, if an  $R^2 = 0.5$ , 50% of the variability has been accounted for, whereas 50% of the variability has not been accounted for and is considered as residual variability. An  $R^2$  value close to 1.0 indicates that almost all of the variability with the variables specified in the model has been accounted for. The F value, or F distribution, is most commonly used in tests of variance. High F values are desired. The value of the p-level represents a decreasing index of the reliability of a result. The higher the p-level, the less reliable the result. Specifically, the p-level represents the probability of error that is involved in the observation result as valid, that is, as representative of the population.

After it was decided to develop models based on both the linear and nonlinear approach, a hierarchy of the modeling approach first had to be established before the models could be developed, and dependent variables had to be defined. In this study, the dependent variables included TTHM, HAA9, HAA6, HAA5, the four THM species, and the nine HAA species. To model the independent variables, independent variables were identified, which included TOC ( $\cong$ DOC), influent bromide, pH, temperature, and chlorine dose.

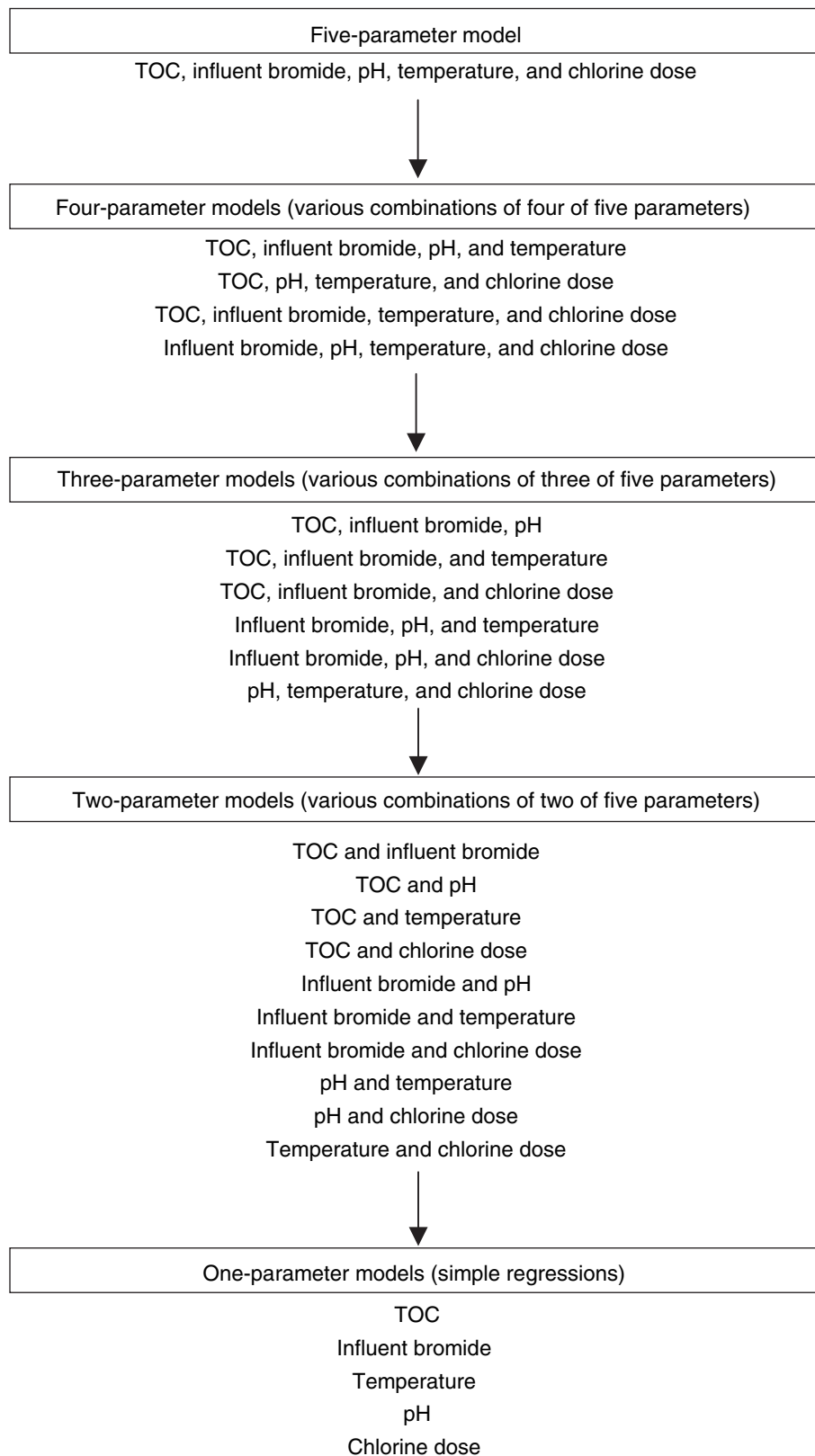
As stated earlier, USEPA's Water Treatment Plant Model was based upon a five-parameter model that was the basis of the initial modeling hierarchy. Subsequently, four-parameter, three-parameter, two-parameter, and one-parameter models were developed. Statistically, a five-parameter model would provide less error than models with lesser numbers of parameters.

A schematic of the hierarchy of models can be found in [Figure 5.1](#).

Multiple-parameter and single-parameter models for the dependent variable HAA5 for Iowa (HAA6 for Ontario) and HAA species were also developed using the independent variable TTHM and the four THM species.

After the modeling hierarchy was established, it was decided that DWSP utilities should be segmented into those that treated Great Lakes water and those that did not. For each data subset, both linear and nonlinear multiple regression models were developed using Statistica. After examining the initial Statistica run, it was decided to develop models based on the entire DWSP database. For both the linear and nonlinear models, models were run using the entire DWSP database instead of separating the database into those systems that treated Great Lakes water and those that did not. This approach resulted in higher  $R^2$  values (it was predetermined that for models to be considered, an  $R^2$  value must be greater than 0.5). Therefore, subsequent models were based on using the DWSP database as a whole.

Once it was decided to use the entire DWSP database for modeling, it then had to be decided which modeling approach would be used, either the linear or the nonlinear approach.



**Figure 5.1 Hierarchy of water quality models used for the improved exposure analysis**

**Table 5.6**  
**Linear DWSP models using water quality variables: intercept (*a*) and coefficients (*b<sub>i</sub>*)**

Dependent variable	Intercept ( <i>a</i> )	Independent variables ( <i>b<sub>i</sub></i> )					Statistics	
		pH	Temperature (°C)	DOC (mg/L)	Total Cl <sub>2</sub> (mg/L)	Color (cu <sup>*</sup> )	R <sup>2</sup>	Count
TOC (mg/L)	1.60					0.17	0.85	931
CHCl <sub>3</sub> (µg/L)	129.95	−18.24	0.74	7.00	3.25		0.55	779
CHCl <sub>3</sub> (µg/L)	128.73	−17.16		7.09	3.21		0.55	779
CHCl <sub>3</sub> (µg/L)	129.50	−17.45	0.70	7.91			0.52	823
CHCl <sub>3</sub> (µg/L)	128.02	−16.40		7.99			0.52	823
CHCl <sub>3</sub> (µg/L)	−5.96			8.06	3.11		0.51	784
TTHM (µg/L)	104.64	−14.18	1.01	6.64	3.48		0.50	779

Note: Blank cells indicate a specific independent variable was not included in the given model.

\* cu: Color units.

After both approaches were run and analyzed, it was evident that modeling should be performed using the linear approach, because the nonlinear approach only provided a limited number of statistically acceptable (i.e.,  $R^2 > 0.50$ ) models for a few dependent variables, such as TTHM, TCAA, and HAA6. The linear approach provided more models regarding TTHM, HAA5, and four of the six HAA species (DBAA data was not provided in the DWSP database and models and correlations could not be developed using the MBAA data) with an  $R^2$  value  $> 0.5$ .

The linear models derived from the DWSP database can be found in [Tables 5.6](#) and [5.7](#), summarizing the intercepts (*a*) and coefficients (*b<sub>i</sub>*) for various models.

Although the DWSP provided a variety of models for DBP predictions, specifically HAA6 and selected HAA species (DCAA and TCAA), as stated in chapter 4, the DWSP database was missing crucial influent bromide data. This helps explain the absence of brominated species models. Models were developed for the brominated DBPs, but the resulting  $R^2$  values were  $< 0.50$ . The ICR database was subsequently queried for plants within the ICR database that had water quality data similar to those plants in the DWSP database. The ICR database was also used to develop a model for predicting influent bromide concentration that could be applied to the DWSP database. The expanded ICR–Ontario database was also used to develop a bromide incorporation factor (i.e., a percent conversion of THM-Br) to estimate influent bromide. Using the percent conversion of THM-Br, it was determined in the expanded ICR–Ontario database that influent bromide equaled approximated 12.6% of the TTHM value. Statistica was also used to develop a multiple regression model for influent bromide. The equation is as follows:

$$\text{Influent Br}^- (\text{ppm}) = 0.01377 + 0.000421 * \text{DBCM} + 0.004438 * \text{BDCM} - 0.001021 \\ * \text{CHCl}_3 + 0.005896 * \text{CHBr}_3$$

**Table 5.7**  
**Linear DWSP models using DBP values: intercept ( $a$ ) and coefficients ( $b_i$ )**

Dependent variable ( $\mu\text{g/L}$ )	Intercept ( $a$ )	Independent variables ( $b_i$ )						Statistics	
		$\text{CHCl}_3$	BDCM	DBCM	$\text{CHBr}_3$	TTHM	HAA6	$R^2$	Count
$\text{CHCl}_3$	-8.02					1.01		0.98	828
DCAA	-0.44					0.28		0.63	402
	1.47	0.28						0.64	402
	2.30	0.28	-0.16					0.65	402
	2.14	0.29	-0.34	0.40				0.65	402
	0.52						0.35	0.90	403
HAA6	-3.75					0.83		0.75	402
	2.21	0.82						0.75	402
	-0.05	0.84		0.89				0.75	402
	0.98	0.86	-0.66	1.79				0.75	402
TCAA	-6.23					0.54		0.76	402
	-2.38	0.54						0.77	402
	-3.54	0.55		0.46				0.77	402
	-2.88	0.56	-0.43	1.04				0.77	402
	-3.09						0.63	0.95	403
TTHM	8.55	0.97						0.98	828

Note: Blank cells indicate a specific independent variable was not included in the given model.

Actual versus predicted bromide values and the corresponding errors are illustrated in [Table 5.8](#).

Two bromide models (influent bromide based on THM-Br and bromide based on a linear multiple regression model) were developed for the DWSP database using the influent bromide data contained in the expanded ICR–Ontario database. The DWSP linear models were not run using the estimated bromide data, because this would increase the modeling error. Instead, the models that incorporated influent bromide as an independent parameter were run using the expanded ICR–Ontario database, thus reducing any modeling error.

After the missing bromide data were estimated, the expanded ICR–Ontario database was then input into Statistica and run using the linear modeling approach to develop models for HAAs and HAA species. [Tables 5.9](#) and [5.10](#) contain the expanded ICR–Ontario database models, portraying the intercept ( $a$ ) and coefficients ( $b_i$ ) for various models.

The models from the DWSP database were then merged with the models from the expanded ICR database and sorted according to  $R^2$ . This resulted in a series of multiple-parameter prediction models that were composed of THM species or HAA species as a function of precursor, chlorination, and water quality conditions and HAA species as a function of THM



**Table 5.8**  
**Summary statistics for influent bromide modeling in the expanded ICR–Ontario database**

Statistic	Based on constant percent of 12.6% bromide (ppm)	Percent error*	Based on regression model bromide (ppm)	Percent error*	Actual bromide (ppm)
Count	194	54	194	54	225
Maximum	0.106	278.0	0.136	298.2	0.160
95th percentile	0.073	268.0	0.065	263.6	0.130
75th percentile	0.045	146.0	0.044	74.3	0.057
50th percentile	0.032	71.2	0.037	33.1	0.029
25th percentile	0.021	32.9	0.027	16.8	0.010
5th percentile	0.011	6.1	0.009	4.0	0.010
Minimum	0.003	3.7	–0.016	2.9	0.010

\* Percent error defined as absolute value of (actual Br – predicted Br) / actual Br.

species. The models were used to fill in missing data for each year of record and utility in the DWSP database, progressing backward through time until 1990. Because there was minimal missing TTHM and species data, TTHM and THM species were not modeled. And because the database contained 927 sets of TTHM and THM species data, the missing data did not affect the analysis. This also minimized modeling error by not using predicted THM values in the HAA multiple regression models. It is important to note that DWSP monitored a different HAA5 group than the U.S. HAA5 group and included BCAA instead of DBAA. National databases have shown DBAA, along with MBAA and MCAA, to be minor species.

The models were then applied to input from the DWSP database. The most important goal was to be able to model HAA5 and HAA species, because HAA monitoring under DWSP only began in 1995. For HAA5 and each HAA species, the model with the highest  $R^2$  value corresponding to the dependent variable was used. These models were then compared to the actual HAA5 and HAA species data for model validation purposes. [Table 5.11](#) contains the actual TTHM and THM species and HAA5 and HAA species formation in the DWSP database, dating from 1990 to 1998 (HAA5 and HAAs date back to only 1995).

Predicted HAA5 and HAA species data can be found in [Table 5.12](#).

Per [Table 5.12](#), HAA species such as MCAA and DCAA were modeled by using actual HAA5 data that existed only after 1995 and predicted HAA5 data that was modeled based on the four THM species. The goal of this exercise was to determine if modeling HAA species based on modeled HAA5 would significantly increase the modeling error. However, in examining [Table 5.12](#), it is apparent that no significant statistical difference exists between the MCAA data that were modeled using actual HAA6 values and the MCAA modeled using predicted HAA5 values, especially regarding the 50th percentile value. If the error between the two calculated values is not significantly different, the modeled HAA5 data can be used to predict the remaining HAA species due to the higher count values.



**Table 5.9**  
**Linear expanded ICR database models using water quality variables:**  
**intercept (*a*) and coefficients (*b<sub>i</sub>*)**

Dependent variable (µg/L)	Intercept ( <i>a</i> )	Independent variables ( <i>b<sub>i</sub></i> )						Statistics	
		pH	Temperature (°C)	TOC (mg/L)	Br <sup>-</sup> (mg/L)	Cl <sub>2</sub> dose (mg/L)	UVA (cm <sup>-1</sup> )	R <sup>2</sup>	Count
BDCM	-27.62	2.87	0.66	1.50	53.27	0.37		0.50	194
CHCl <sub>3</sub>	3.19			8.02	-122.04			0.54	194
	-2.85		0.58	8.22	-131.85			0.55	194
	37.42	-4.57		8.40	-124.69			0.55	194
	36.21	-5.40	0.71	8.72	-137.25			0.56	194
	1.79			7.19	-157.08	1.37		0.56	194
	-7.94		0.90	-7.31	-179.91	1.67		0.56	194
	22.41	-4.14	0.98	7.76	-180.45	1.54		0.58	194
DBCM	-9.48	1.32	0.18		50.43	-0.35		0.51	194
	-15.15	2.06	0.19	-0.48	43.03			0.52	194
	-13.06	1.87	0.15	-0.34	49.56	-0.23		0.54	194
HAA6	91.50	-12.46	0.38	15.45	-140.79			0.52	198
	-5.48		0.41	12.09	-195.14	3.08		0.53	198
	-0.92			11.89	-188.09	3.03		0.53	198
	62.47	-9.16	0.53	12.93	-185.45	2.69		0.55	198
TOC	0.69						30.47	0.81	200
TTHM	-14.71	0.53	1.50	8.64		1.09		0.50	194
	-13.94		1.87	8.58	-68.54	1.75		0.51	194
	-17.21	0.45	1.86	8.53	-68.48	1.76		0.51	194

Note: Blank cells indicate that an independent parameter was not considered in the model.

After the model predictions, the predicted HAA values were compared with the actual HAA values. The error analysis can be found in [Table 5.13](#).

After performing the error analysis, it was concluded that using modeled HAA5 data instead of actual HAA5 data would not significantly affect the analysis. For example, DCAA was modeled using actual and predicted HAA5 data. At the 50th percentile value, DCAA that was based on the predicted HAA5 data had a 50th percentile error value of 46.5%, whereas DCAA that was based on actual HAA5 data had a 50th percentile error value of 48.6%. Thus, there is little difference between the two models. Due the higher count values, the modeled HAA5 data

**Table 5.10**  
**Linear expanded ICR database models using DBP values: intercept (*a*) and coefficients (*b<sub>i</sub>*)**

Dependent variable (µg/L)	Intercept ( <i>a</i> )	Independent variables ( <i>b<sub>i</sub></i> )						Statistics	
		CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	HAA6	R <sup>2</sup>	Count
BCAA	0.76	0.04	0.07	0.45	−0.63			0.54	181
BDCM	1.01					0.24		0.61	194
CHCl <sub>3</sub>	−4.65					0.77		0.85	194
	−4.65					0.77		0.85	194
DCAA	1.42	0.51						0.75	181
	4.68	0.47		−0.79				0.80	181
	0.78						0.34	0.85	198
HAA6	−1.21					1.00		0.54	167
	2.95	1.44						0.73	167
	10.25	1.01	1.38	−4.56	3.96			0.77	167
TCAA	−1.21	0.83						0.66	198
	4.68	0.52	1.00	−3.52	3.27			0.73	176
	−3.20						0.68	0.92	198
TTHM	9.46	1.11						0.85	194

Note: Blank cells indicate that an independent parameter was not considered in the model.

**Table 5.11**  
**Actual DBP formation in the DWSP database**

Statistic	Actual THMs (µg/L)					Actual HAAs (µg/L)					
	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	MCAA	DCAA	TCAA	BCAA	MBAA	HAA5
Count	827	827	827	827	827	402	402	402	402	402	402
Maximum	229.25	36.68	28.72	12.50	233.95	8.33	82.38	230.15	11.71	3.05	299.50
95th percentile	105.34	12.02	4.87	0.63	111.35	3.25	32.87	46.95	4.17	0.49	83.77
75th percentile	49.26	7.39	3.40	0.50	55.50	1.50	12.50	18.98	2.50	0.23	36.15
50th percentile	19.06	5.10	1.85	0.50	28.68	0.75	6.50	6.73	1.32	0.10	19.00
25th percentile	8.50	2.57	0.20	0.33	17.72	0.50	3.00	2.99	0.61	0.05	9.46
5th percentile	3.76	1.09	0.10	0.20	6.58	0.50	0.80	0.85	0.20	0.05	2.90
Minimum	0.17	0.05	0.10	0.20	0.53	0.50	0.10	0.05	0.10	0.05	0.05

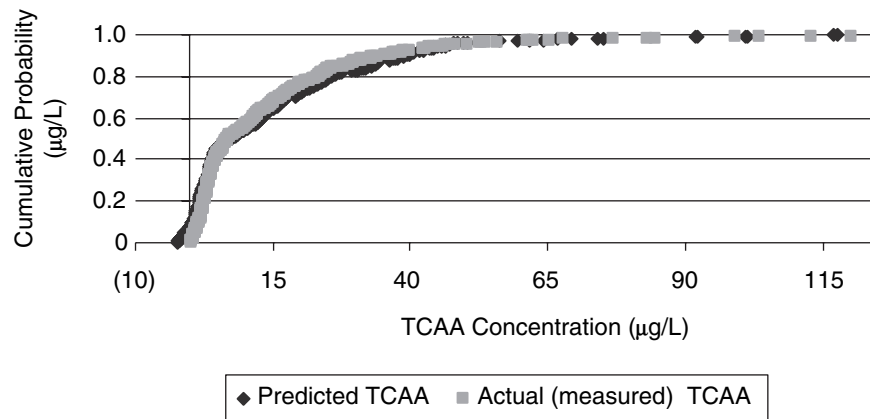
**Table 5.12**  
**Predicted HAA5 and HAA species formation in the DWSP database**

Statistic	MCAA		DCAA		TCAA	BCAA	HAA5
			Predicted values (µg/L) based on:				
	Actual HAA5 data	Predicted HAA5 data	Actual HAA5 data	Predicted HAA5 data	Actual THM data	Actual THM data	Actual THM data
Count	402	827	402	827	827	827	827
Maximum	7.19	5.93	156.33	84.35	123.51	14.31	242.34
95th percentile	2.43	3.23	43.73	42.12	54.50	5.37	120.25
75th percentile	1.37	1.95	18.87	22.12	23.39	3.74	62.43
50th percentile	0.99	1.13	9.92	9.29	7.73	2.90	25.36
25th percentile	0.78	0.83	4.94	4.57	2.83	2.23	11.72
5th percentile	0.64	0.72	1.52	2.76	-0.26	1.13	6.46
Minimum	0.58	0.10	0.03	-6.93	-2.50	-5.93	-21.55

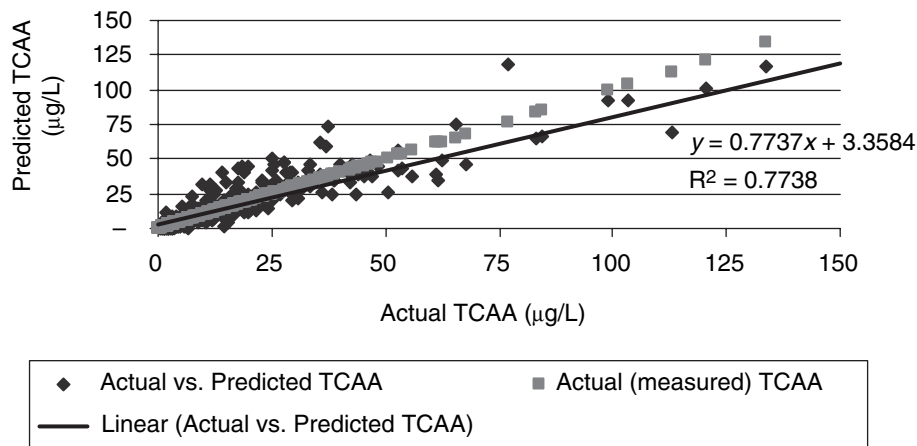
**Table 5.13**  
**Error analysis of the modeled DWSP data**

Statistic	Unit	TCAA	DCAA		MCAA		BCAA	HAA5
		Actual THMs	Predicted HAA5	Actual HAA5	Percent error* based on:		Actual THMs	Actual THMs
					Actual HAA5	Predicted HAA5		
Count		402	402	402	402	402	402	402
Maximum	%	3,789.0	1,8677.3	2,405.5	450.2	525.7	4,935.3	2,447.2
95th percentile	%	233.7	896.8	226.4	125.5	261.6	1,238.4	366.3
75th percentile	%	71.4	149.5	81.1	60.7	86.1	336.5	115.5
50th percentile	%	32.2	46.5	48.6	34.4	51.0	87.7	52.2
25th percentile	%	13.5	20.6	25.6	18.0	26.4	26.2	24.6
5th percentile	%	2.5	4.2	8.0	3.2	6.3	4.2	4.9
Minimum	%	0.1	0.0	0.7	0.1	0.4	0.2	0.1

\* Percent error defined as (actual value – predicted value) / actual value.



**Figure 5.2 Cumulative probability curve of actual and predicted TCAA in DWSP database**



Note: When dealing with scatterplots, an ideal model will have an  $R^2 = 1.0$ , intercept = 0, and a slope = 1.

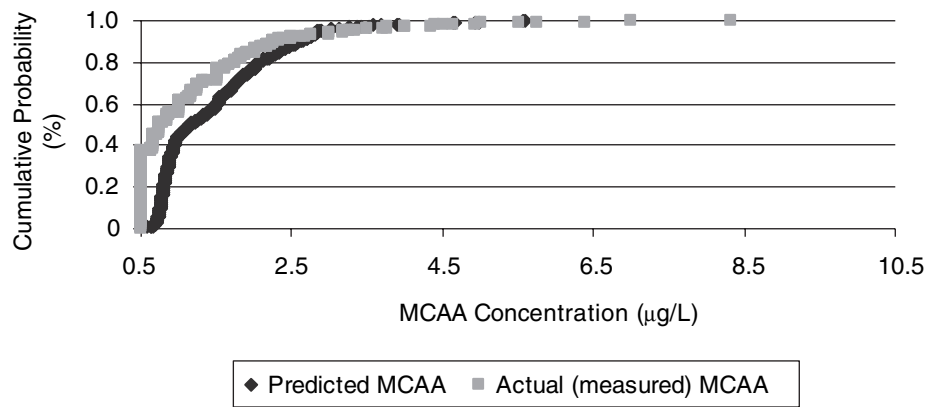
**Figure 5.3 Scatterplot of actual versus predicted TCAA in DWSP database**

were used for the modeling of HAA species where appropriate. For HAA5 and HAA species, the median error ranged from approximately 30% to 50%. Although, at first, these values may appear to be high, these models have been developed primarily for use as central tendency trends. Under this scenario, these levels are considered acceptable on a site-specific basis.

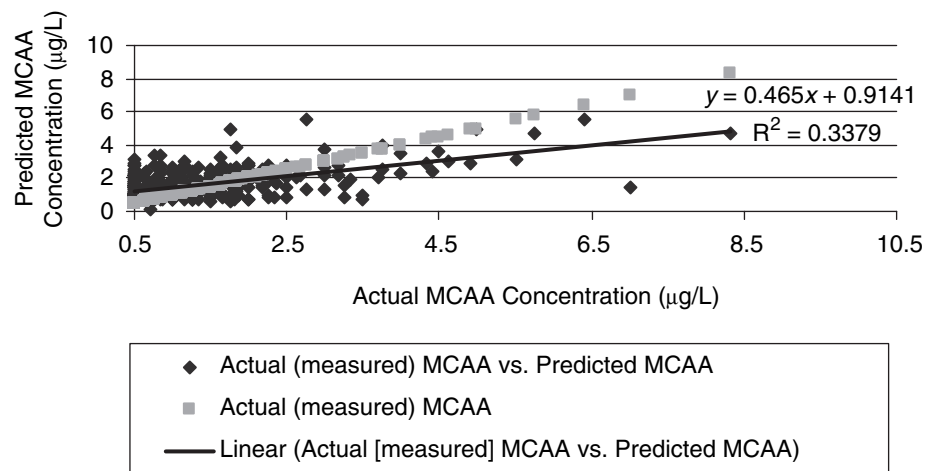
Once values were predicted using the appropriate model, the models were verified, similar to the error analysis, by plotting predicted values against actual (measured) values. Cumulative probability curves and scatterplots of actual versus predicted values were developed for HAA5 and each HAA species. [Figures 5.2 to 5.11](#) represent this modeling verification effort.

From these figures, it can be concluded that the models derived from the DWSP database and the expanded ICR–Ontario database can predict HAA5 and species (excluding MBAA and DBAA) formation on a central tendency basis.

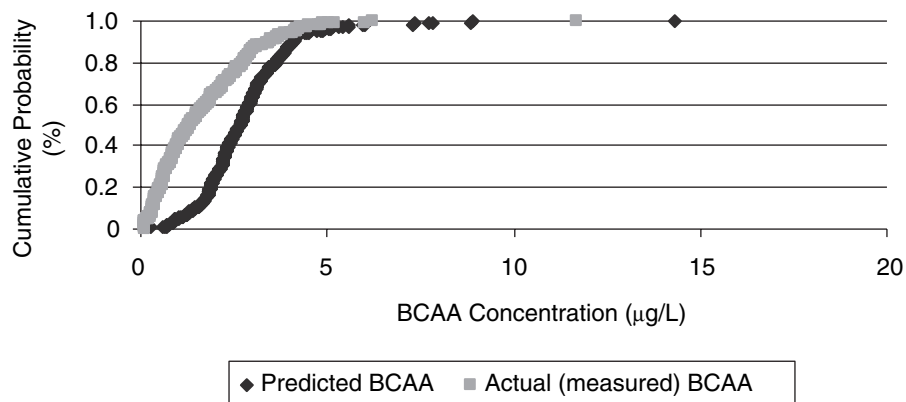
Although the aforementioned figures adequately represent the data based on the error analysis, they may not be accurate, because the error analysis was based on the models derived



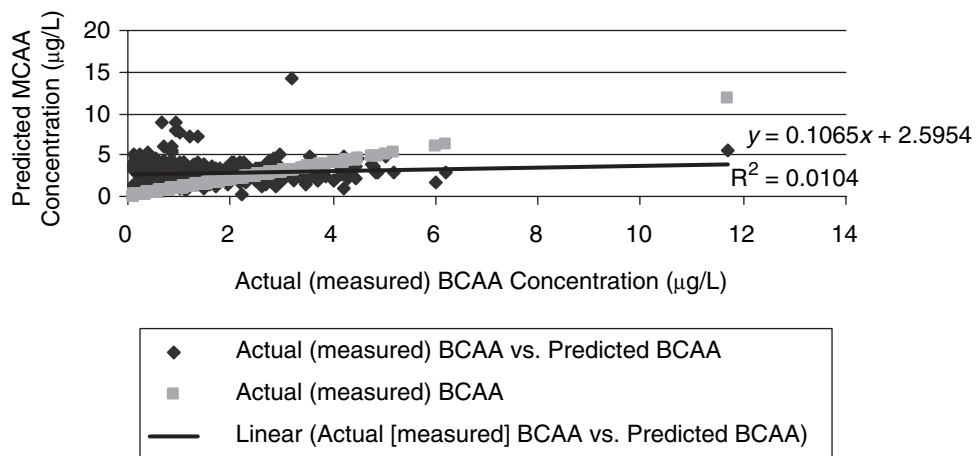
**Figure 5.4 Cumulative probability curve of actual and predicted MCAA in DWSP database**



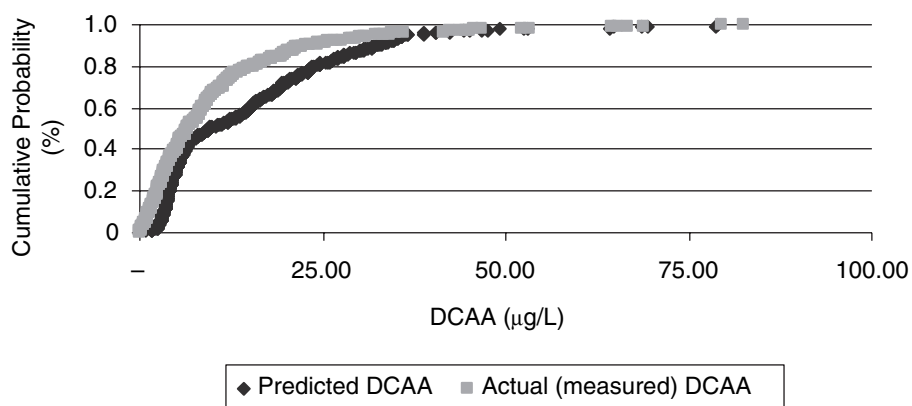
**Figure 5.5 Scatterplot of actual versus predicted MCAA in DWSP database**



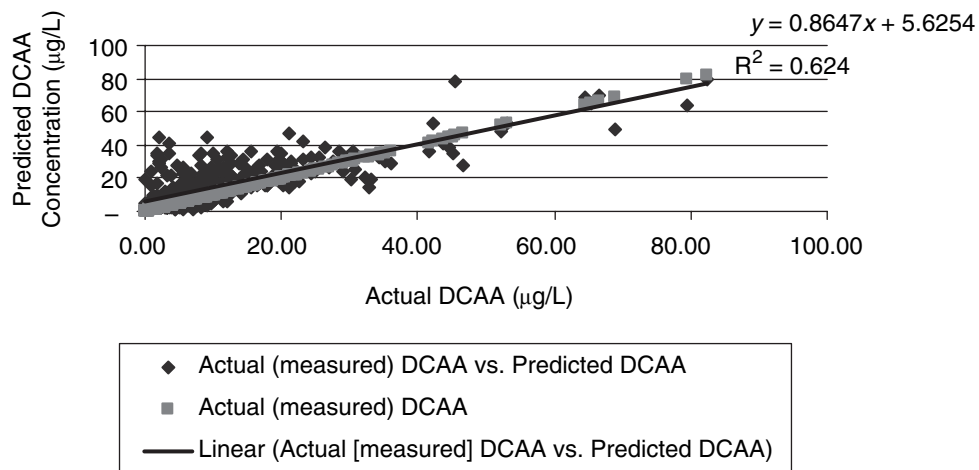
**Figure 5.6 Cumulative probability curve of actual and predicted BCAA in DWSP database**



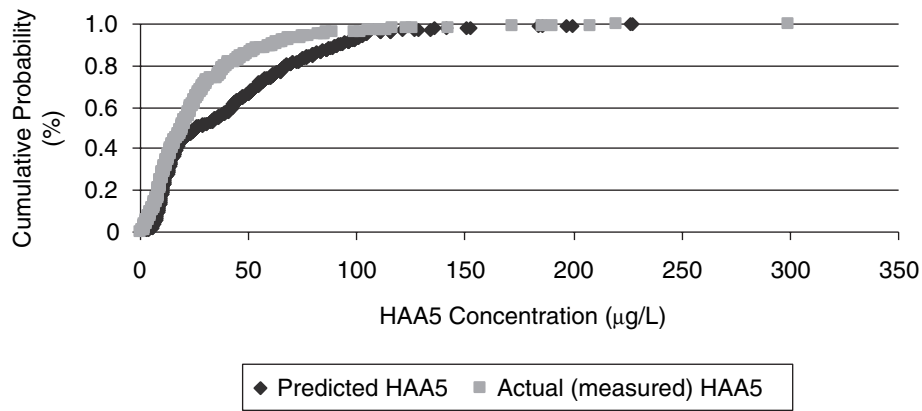
**Figure 5.7 Scatterplot of actual versus predicted BCAA in DWSP database**



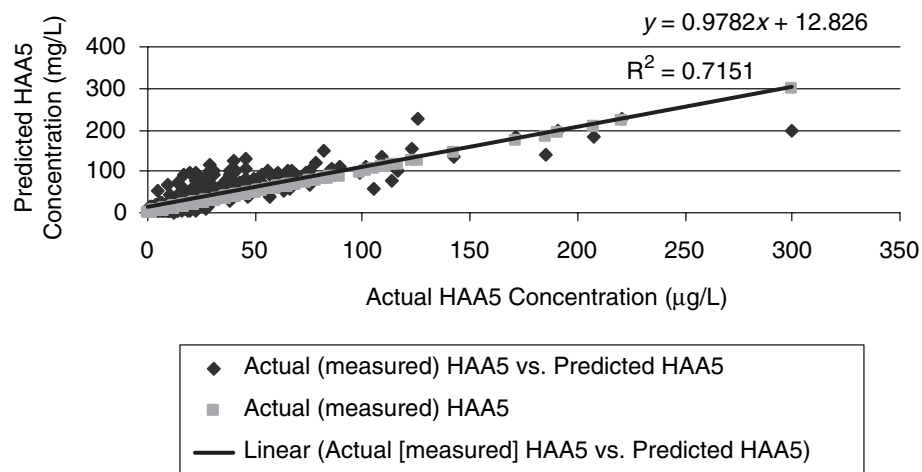
**Figure 5.8 Cumulative probability curve for actual and predicted DCAA**



**Figure 5.9 Scatterplot of actual versus predicted DCAA in DWSP database**



**Figure 5.10 Cumulative probability curve of actual and predicted HAA5**



**Figure 5.11 Scatterplot of actual versus predicted HAA5 in DWSP database**

with the specific dataset. Typically, when models are derived from a dataset, a percentage of the data is reserved or set aside (e.g., 10%). The models are then derived using the remaining 90% of the data, after which the models are applied to the reserved data and the error analysis is then performed. However, in the case of Ontario, this could not be done because the DWSP database was not large enough to “bank” 10% of the data. Instead, in this case, it was decided that, if a model was derived from the DWSP database, it would be applied to the expanded ICR–Ontario database, and a subsequent error analysis would be performed. In turn, if the model was derived using the expanded ICR–Ontario database, the model would be applied to the DWSP database and the error analysis would then be performed.

When modeling HAA5 and HAA species in the DWSP database, the models for MCAA, DCAA, and TCAA were developed using the DWSP database. Therefore, to perform a more accurate error analysis, the models were applied to the expanded ICR–Ontario database. This error analysis is summarized in [Table 5.14](#).

**Table 5.14**  
**Error analysis for MCAA, DCAA, and TCAA models applied to expanded ICR–Ontario database**

Statistic	Unit	Predicted MCAA error*	Predicted DCAA error*	Predicted TCAA error*
Count		198	198	181
Maximum	%	153.2	92.6	569.5
95th percentile	%	85.8	90.3	178.9
75th percentile	%	50.1	87.1	68.5
50th percentile	%	32.6	84.8	48.6
25th percentile	%	15.6	77.2	32.1
5th percentile	%	4.9	23.9	8.4
Minimum	%	0.4	7.9	3.4

\*Percent error defined as (actual value – predicted value) / actual value.

**Table 5.15**  
**Error analysis for BCAA and HAA5 models applied to DWSP database**

Statistic	Unit	Predicted BCAA error*	Predicted HAA6 error*
Count		827	827
Maximum	%	14.3	242.3
95th percentile	%	5.4	120.3
75th percentile	%	3.7	62.4
50th percentile	%	2.9	25.4
25th percentile	%	2.2	11.7
5th percentile	%	1.1	6.5
Minimum	%	–5.9	–21.5

\* Percent error defined as (actual value – predicted value) / actual value.

The models for BCAA and HAA5 were derived using the expanded ICR–Ontario database. These models were then applied to the DWSP database, and an error analysis was again performed, as shown in [Table 5.15](#).

Similar to the initial error analysis, it was concluded that the models derived from the DWSP database and the expanded ICR–Ontario database can predict HAA5 and HAA species (excluding MBAA and DBAA) formation on a central tendency basis.

After HAA5 and the individual species were modeled (excluding HAA5), the analysis continued in order to predict the remaining HAA species: BDCAA, DBCAA, and TBAA. These



**Table 5.16**  
**Predicted HAA3 species for the DWSP database**

	BDCAA	DBCAA	TBAA	BDCAA	DBCAA	TBAA
	Based on:					
	Actual TCAA	Actual TCAA	Actual TCAA	Predicted TCAA	Predicted TCAA	Predicted TCAA
Statistic	(µg/L)					
Count	401	401	401	827	827	827
Maximum	17.71	11.67	21.38	28.85	21.55	3.93
95th percentile	4.56	1.82	0.35	4.97	1.90	0.23
75th percentile	2.52	1.08	0.23	2.78	1.06	0.19
50th percentile	1.67	0.49	0.18	1.68	0.43	0.14
25th percentile	0.86	0.09	0.14	0.93	0.08	0.09
5th percentile	0.30	0.05	0.08	0.00	0.00	0.00
Minimum	0.05	0.01	0.03	-5.22	-9.96	-5.17

species are also referred to as HAA3. Instead of using Statistica and creating multiple regression models, an estimation approach developed by Singer et al. (2002) was utilized. In this approach, each of the HAA3 species can be estimated on a molar basis, using the following models:

- $BDCAA = TCAA * (BDCM/CHCl_3)$
- $DBCAA = TCAA * (DBCM/CHCl_3)$
- $TBAA = TCAA * (CHBr_3/CHCl_3)$

These equations were applied to the DWSP database. Similar to the HAA5 and species modeling, HAA3 was estimated from actual and predicted TCAA concentrations. The analysis is provided in [Table 5.16](#).

Because the predicted species concentrations that were based on the actual versus modeled TCAA values were not significantly different, with respect to the 50th percentile range, HAA3 species were estimated based on TCAA values. As illustrated in [Table 5.16](#), the use of modeled TCAA values provided 827 cases, compared to the actual TCAA values, which only provided 401 cases.

The DWSP database did not contain any data regarding the HAA3 species; thus no associated error analysis could be performed. In order to verify the HAA3 equations developed by Singer et al. (2002), the approach was applied to the expanded ICR–Ontario database that contained limited HAA3 data. Predicted concentrations, actual concentrations, and percent error can be found in [Tables 5.17](#) and [5.18](#).

The equation for BDCAA provides adequate estimations with a median error of 16%. In contrast, the DBCAA and TBAA equations have median errors ranging from 70% and 85%, respectively. This error is not necessarily attributed to the model but may also be attributed to

**Table 5.17**  
**Actual and predicted HAA3 concentration in the expanded ICR–Ontario database**

Statistic	Predicted HAA3 concentrations (µg/L)			Actual (measured) HAA3 concentrations (µg/L)		
	BDCAA	DBCAA	TBAA	BDCAA	DBCAA	TBAA
Count	81	81	81	81	79	71
Maximum	8.44	3.89	1.28	8.20	2.90	2.00
95th percentile	7.63	2.42	0.47	7.70	2.40	2.00
75th percentile	4.54	2.07	0.37	4.70	1.00	2.00
50th percentile	3.76	1.73	0.29	3.80	1.00	2.00
25th percentile	1.25	1.32	0.23	1.60	1.00	2.00
5th percentile	0.58	0.60	0.19	0.50	1.00	2.00
Minimum	0.16	0.04	0.03	0.50	1.00	2.00

**Table 5.18**  
**Error analysis of actual versus predicted HAA3 values**

Statistic	Unit	BDCAA error*	DBCAA error*	TBAA error*
Count		81	79	71
Maximum	%	270.01	182.39	98.48
95th percentile	%	84.67	132.72	90.33
75th percentile	%	41.95	98.36	87.93
50th percentile	%	16.49	71.69	85.42
25th percentile	%	6.47	45.15	81.54
5th percentile	%	1.05	11.38	72.06
Minimum	%	0.02	1.89	35.87

\* Percent error defined as (actual value – predicted value) / actual value.

values being substituted for nondetect values in the ICR database for these two species. CDBAA has a detection limit of 2 ppb, and TBAA has a detection limit of 4 ppb. In the expanded ICR–Ontario database, nondetect values were substituted with a concentration equal to one half of the detection limit. Therefore, the error can be partly attributed to these substituted values.

After the DWSP database was “completed” with the estimation of HAA5 and HAA species and HAA3 species, the next issue investigated was projecting DBP formation trends in Ontario before 1990. The only national Canadian database that could be obtained was the 53-Utility Survey upon which the THM goal of 100 µg/L was based. Therefore, the use of historic databases or a milestone approach could not be used in constructing a chronological

**Table 5.19**  
**Actual THM data in the DWSP database for 1990–1993 and 1994–1998**

Statistic	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM
<b>1990–1993</b>	(µg/L)				
Count	325	325	325	325	325
Maximum	229.3	22.2	7.7	1.2	234.0
95th percentile	113.2	13.6	5.2	0.6	118.6
75th percentile	55.1	8.6	3.7	0.4	59.6
50th percentile	19.9	6.0	2.2	0.3	31.3
25th percentile	9.8	3.2	0.2	0.2	20.1
5th percentile	5.0	1.3	0.1	0.2	12.3
Minimum	1.0	0.1	0.1	0.2	1.4
<b>1994–1998</b>					
Count	502	502	502	502	502
Maximum	216.5	36.7	28.7	12.5	218.6
95th percentile	91.9	10.5	4.3	0.6	99.7
75th percentile	46.5	6.7	3.1	0.5	51.9
50th percentile	17.8	4.3	1.6	0.5	26.8
25th percentile	7.5	2.4	0.2	0.5	15.7
5th percentile	3.2	1.0	0.2	0.5	5.7
Minimum	0.2	0.2	0.2	0.5	0.5

timeline for Ontario. Because the TTHM guideline was promulgated in 1993, the DWSP database was segmented into two parts: 1990–1993 and 1994–1998. [Tables 5.19](#) and [5.20](#) provide information regarding actual TTHM and THM species data and HAA5 and HAA species data in the DWSP database.

The modeled HAA5, HAA species, and HAA3 species data were then incorporated into the DWSP database. Overall summary statistics are provided in [Table 5.21](#).

These tables support the premise that Ontario DBP formation was not significantly different prior to the 1993 TTHM guideline. To further support this notion, 50th percentile and 95th percentile graphs for CHCl<sub>3</sub>, TCAA, and DCAA were plotted for the expanded ICR–Ontario database and DWSP segmented into the years 1990–1993 and 1994–1998 in [Figures 5.12](#) through [5.17](#); these compounds were selected because they are the most prominent in Ontario waters.

Similar to trends in the United States, it is apparent from these graphs that the 50th percentile values of the treatment plants in Ontario were not significantly affected by the 1993 TTHM guideline. Even the 95th percentile values of utilities appear to be unaffected. The three 95th percentile graphs only show a decrease in DBP primarily occurring after the guideline was enacted in 1994. This supports the assumption that, prior to 1993, Ontario utilities had no incentive to alter their drinking water process train to limit DBP formation. Therefore, the years 1990–1993, when averaged together, should adequately represent DBP formation progressing

**Table 5.20**  
**Actual HAA data in the DWSP database for 1995–1998**

	MCAA	DCAA	TCAA	BCAA	MBAA	HAAs*
	(µg/L, except for count)					
Count	402	402	402	402	402	402
Maximum	8.3	82.4	230.2	11.7	3.1	299.5
95th percentile	3.2	32.9	47.0	4.2	0.5	83.8
75th percentile	1.5	12.5	19.0	2.5	0.2	36.2
50th percentile	0.8	6.5	6.7	1.3	0.1	19.0
25th percentile	0.5	3.0	3.0	0.6	0.1	9.5
5th percentile	0.5	0.8	0.9	0.2	0.1	2.9
Minimum†	0.5	0.1	0.1	0.1	0.1	0.1

\* HAAs: sum of MCAA, MBAA, DCAA, BCAA, and TCAA. DBAA was not measured.

† Analytes that were not detected are reported at one half of their minimum reporting level rather than zero.

backward through time until 1940. This assumption can be further supported by [Figures 5.18 to 5.20](#), which plot the cumulative probability of the three DBPs—chloroform, TCAA, and DCAA—respectively.

Again, little difference is observed between the 50th and 95th percentile values for  $\text{CHCl}_3$ , TCAA, and DCAA. Therefore, assuming that DBP formation remained unchanged prior to 1993 is justified.

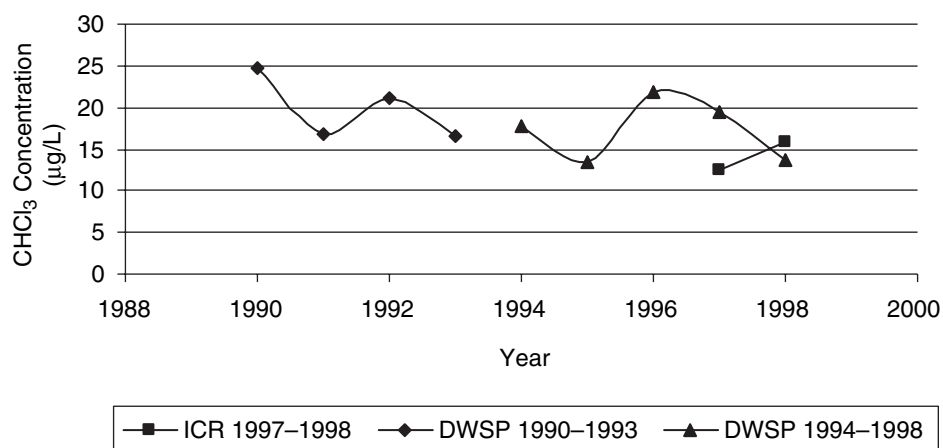
Based on a central tendency modeling approach, the Ontario improved exposure analysis can be considered complete at this stage and potentially applicable to reanalysis of the relevant epidemiology study for DBP formation and its link to bladder cancer. Such a reanalysis is based on a table that provides the median values for all the utilities for all the DBPs (both measured and predicted). [Table 5.22](#) represents the 50th percentile values for all the Ontario utilities dating from before 1990 to 1998.

This improved exposure assessment analysis can provide a more accurate assessment of DBP formation in the Ontario utilities by providing additional information regarding HAA5 and species. HAA5 and species data were filled in, progressing backward through time to 1990. The three remaining HAA3 species (BDCAA, DBCAA, and TBAA) were also modeled back to 1990 using the Singer et al. (2002) approach. The DWSP database had no prior data for these DBPs. Before 1990, averages of each DBP, both THMs and HAAs, from 1990 to 1993 can be used for values progressing backward until 1940.

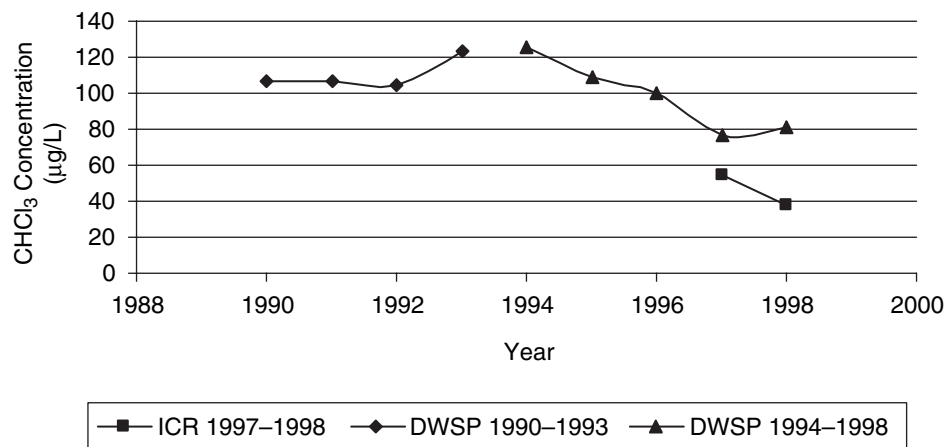
In applying the central tendency models, this approach was found to be inadequate in describing more extreme (e.g., 10th and 90th percentile) as opposed to more typical (i.e., central tendency) conditions. Thus, a decision was made midway through the project to explore the development of another complementary modeling approach, the case study approach, as will be discussed in chapter 6.

**Table 5.21**  
**HAA5 and species and HAA3 species for 1990–1993 and 1994–1998**

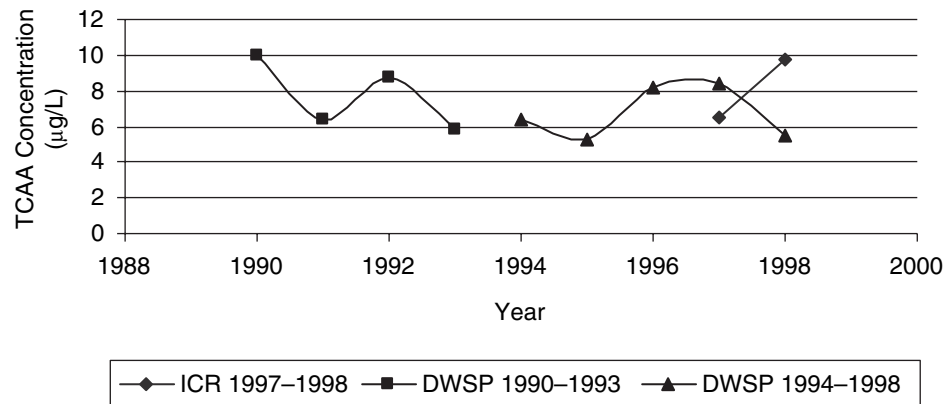
Statistic	MCAA	DCAA	TCAA	BCAA	MBAA	HAA5	BDCAA	DBCAA	TBAA
<b>1990–1993</b>	(µg/L, except for count)								
Count	325	325	325	325		325	325	325	325
Maximum	5.9	84.3	123.5	9.5	0.0	242.3	9.5	3.8	0.5
95th percentile	3.4	44.7	59.3	5.7	0.0	127.6	5.6	2.1	0.2
75th percentile	2.0	23.6	26.3	4.1	0.0	66.7	3.2	1.2	0.1
50th percentile	1.1	9.3	8.3	3.2	0.0	25.4	2.1	0.6	0.1
25th percentile	0.9	4.9	3.5	2.6	0.0	12.5	1.2	0.1	0.1
5th percentile	0.7	2.7	0.6	1.5	0.0	6.3	0.0	0.0	0.0
Minimum	0.6	0.8	–2.2	0.7	0.0	0.7	–1.7	–1.3	–0.4
<b>1994–1998</b>									
Count	502	502	502	502	402	502	502	502	502
Maximum	5.6	78.9	117.7	14.3	3.1	226.7	28.9	21.6	3.9
95th percentile	3.0	38.4	47.7	4.9	0.5	109.4	4.3	1.6	0.2
75th percentile	1.9	21.4	21.8	3.4	0.2	60.5	2.4	0.9	0.2
50th percentile	1.1	9.3	7.1	2.7	0.1	25.3	1.4	0.3	0.2
25th percentile	0.8	4.4	2.2	2.0	0.1	11.2	0.8	0.1	0.1
5th percentile	0.7	2.8	–0.5	1.0	0.1	6.6	0.0	0.0	0.0
Minimum	0.1	–6.9	–2.5	–5.9	0.1	–21.5	–5.2	–10.0	–5.2



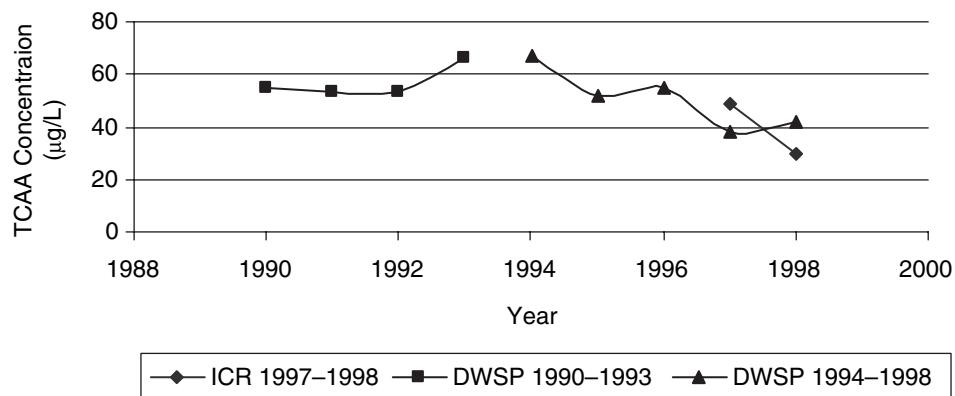
**Figure 5.12 50th percentile of CHCl<sub>3</sub> over time in Ontario databases**



**Figure 5.13 95th percentile of  $\text{CHCl}_3$  over time in Ontario databases**



**Figure 5.14 50th percentile of TCAA over time in Ontario databases**



**Figure 5.15 95th percentile of TCAA over time in Ontario databases**

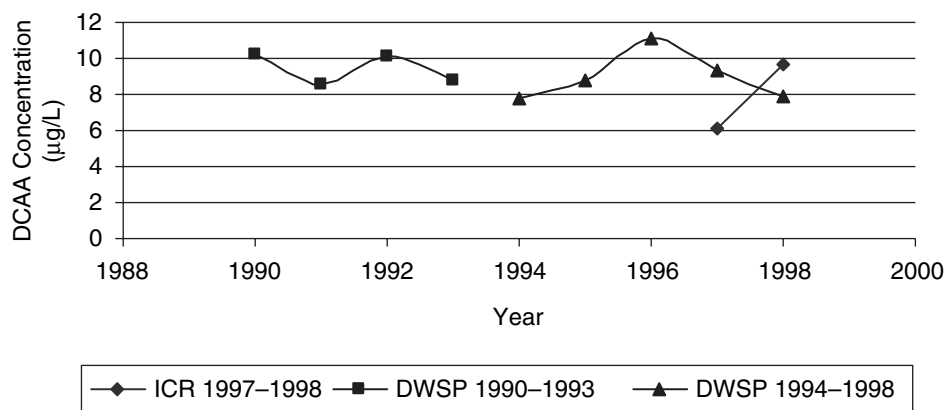


Figure 5.16 50th percentile of DCAA over time in Ontario databases

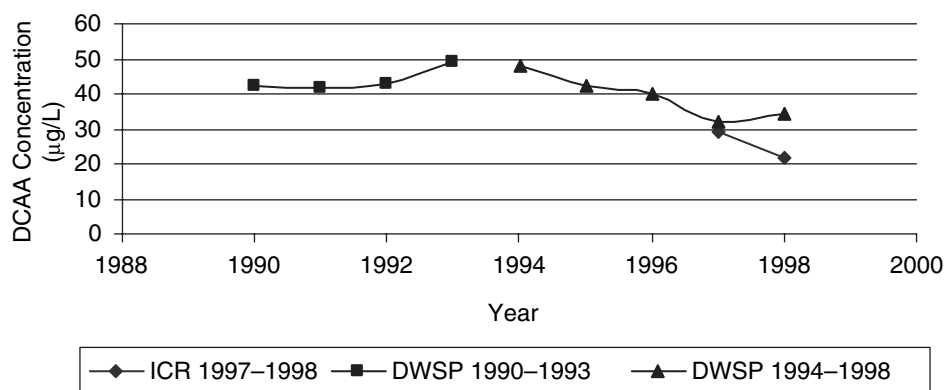


Figure 5.17 95th percentile of DCAA over time in Ontario databases

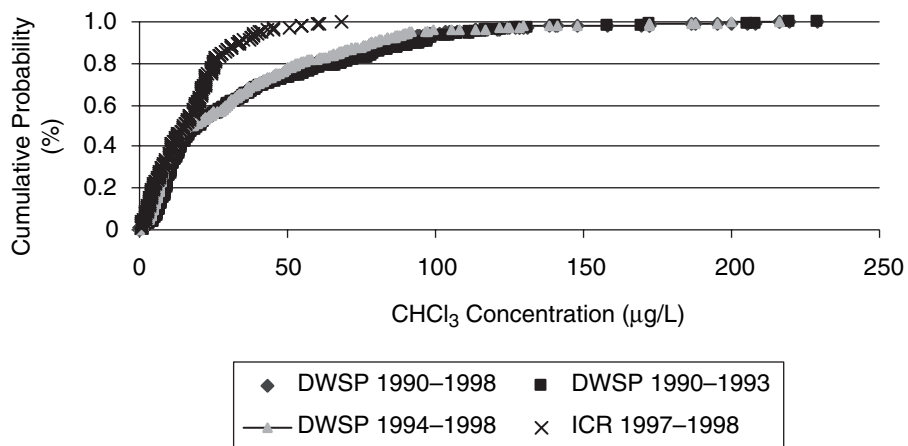
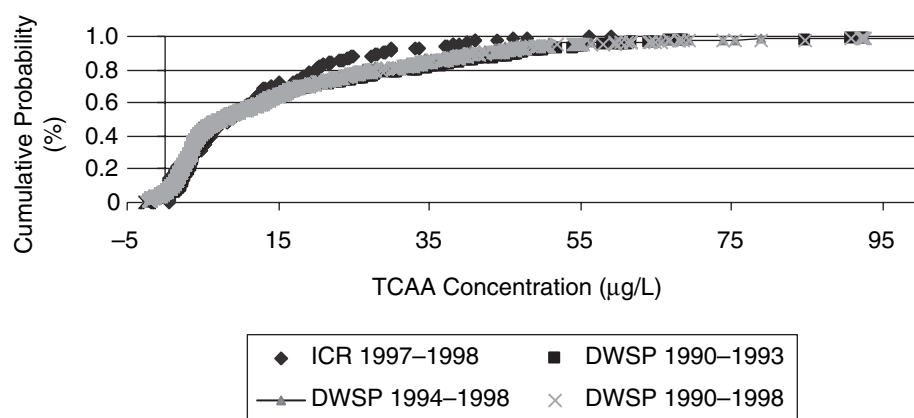
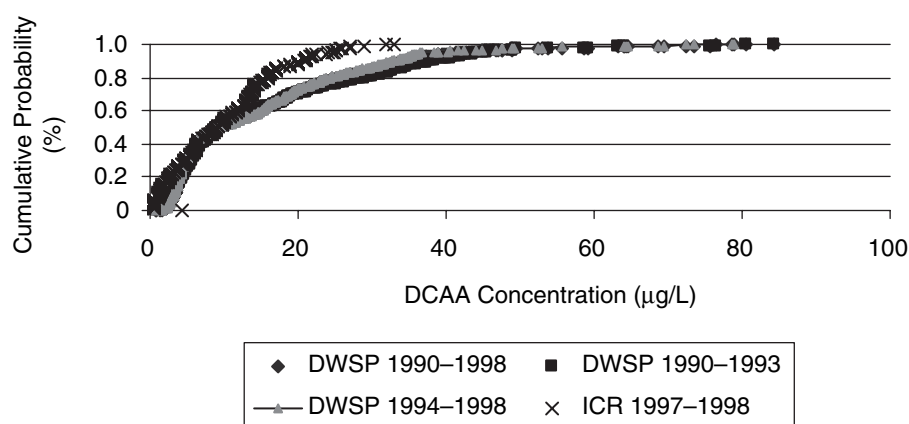


Figure 5.18 Cumulative probability curves of CHCl<sub>3</sub> in Ontario databases



**Figure 5.19 Cumulative probability curves of TCAA in Ontario databases**



**Figure 5.20 Cumulative probability curves of DCAA in Ontario databases**

## Iowa Modeling Efforts

After Ontario underwent the improved exposure analysis, Iowa data such as chlorine dose, chlorine demand, temperature, alkalinity, dissolved solids, and some limited color data were acquired by obtaining the CHEEC database. These data were then input into a spreadsheet, which is summarized in appendix 5.1 describing the number of annual average values out of a possible 34 (except for the time intervals covering a 5-year time span, which may include up to five values). Appendices 5.2 and 5.3 illustrate spreadsheets for the Cantor et al. (1998) study and the expanded ICR–Iowa database, respectively. Appendix 5.4 contains the overall spreadsheet describing a compilation of the three databases. The indicated DBPs include finished water and distribution system levels for the expanded ICR–Iowa database, the CHEEC database, and the Cantor et al. data. The initial prognosis for the overall Iowa database (appendix 5.4), comprising the hit list of 34 utilities as described by CHEEC database, (appendix 5.1), Cantor et al. (appendix 5.2), and ICR–Iowa (appendix 5.3) databases, indicated that site-specific multiple regression modeling was not a viable approach, given the scarcity of data for many independent variables. It



**Table 5.22**  
**50th percentile values for all DWSP utilities prior to 1990 until 1998**

	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	MCAA	DCAA	TCAA	BCAA	HAA5	BDCAA	DBCAA	TBAA
	(µg/L)												
Before 1990	19.9	6.0	2.2	0.3	31.3	1.1	9.3	8.3	3.2	25.4	2.1	0.6	0.1
1990	24.8	8.2	3.3	0.3	38.0	1.2	10.2	10.4	3.8	28.0	3.0	1.0	0.1
1991	16.8	5.8	2.4	0.2	27.2	1.1	8.5	6.4	3.1	23.1	2.0	0.7	0.1
1992	21.0	5.9	2.0	0.2	31.6	1.2	10.1	8.6	3.3	27.7	2.0	0.6	0.1
1993	16.5	5.3	1.7	0.3	26.8	1.1	8.8	6.5	2.9	24.0	1.8	0.5	0.1
1994	17.8	5.1	2.0	0.5	26.4	1.0	7.8	6.9	2.9	21.1	1.6	0.5	0.2
1995	13.4	4.7	1.5	0.5	24.3	1.1	8.8	5.0	2.8	24.0	1.3	0.2	0.2
1996	21.8	4.1	1.5	0.5	30.5	1.3	11.1	8.5	2.7	30.6	1.4	0.2	0.2
1997	19.5	4.2	1.7	0.5	27.9	1.1	9.3	7.9	2.7	25.5	1.3	0.3	0.2
1998	13.7	4.0	1.4	0.5	24.0	1.0	7.7	5.7	2.4	20.8	1.2	0.2	0.2

appeared that it was more viable to develop simple correlations between individual DBPs/DBP-related parameters; however, only limited HAA data were available (ICR–Iowa and 1999 CHEEC databases). There were several iterations in soliciting data and assembling the Iowa database. Earlier, when it was apparent that TOC data were very limited, a request was made for coagulant dose and chlorine dose data from the CHEEC database, with little additional data realized. However, TOC data were available for one year (1987) for 33 of the 34 utilities (presumably part of a special sampling campaign).

Consolidation of the CHEEC database revealed serious data deficiencies, such as DOC and other water quality parameters. It was then concluded that a questionnaire should be created, as illustrated in appendix 5.5. In order to test the questionnaire’s terminology, ease of interpretation, and the time it would take to complete the survey, the questionnaire was sent to three utilities: Des Moines, Cedar Rapids, and Iowa City. All three utilities reported that the survey was easy to follow and could be completed in a reasonable amount of time. However, the amount of time was estimated to be dependent on a variety of factors, including

- *The amount of data electronically stored.* Utilities were expected to have only electronic files dating back over the last 5 years.
- *The accessibility of the data.* The utility would have to exert some effort when going back through paper records. In addition, certain events might have resulted in lost historical files. For example, Des Moines lost all of its paper files when the facility was flooded in 1993.
- *Personnel effort.* Some of the utilities would not have enough personnel necessary to complete the survey. These utilities might need additional assistance.

Once the survey was completed and reviewed by the three utilities, they were notified by phone by Peter Weyer, one of the co-investigators, and his colleagues at the University of Iowa, who explained the purpose of the study and asked for permission to send the survey for review.

After the utilities were sent the survey, the mailing was followed up with phone calls to discuss the survey in depth. From this approach, 33 of the 34 utilities agreed to participate in the survey (Osceola refused). Twelve utilities were able to complete the survey without any assistance, and 21 utilities required a site visit.

Once the site visits were conducted, all of the data were compiled electronically. After the first compilation, several deficiencies were discovered in the files, including missing sample names and unit discrepancies. To remedy this problem, the surveys were subjected to quality assurance/quality control (QA/QC). However, the Iowa/CHEEC database modeling and exposure assessment could not be performed.

However, using the ICR–Iowa database, linear multiple regression models were developed using Statistica. [Table 5.23](#) describes models based on water quality values, and [Table 5.24](#) describes models based on DBPs. Similar to Ontario, the models were developed using the hierarchical parameter approach described in [Table 5.23](#) and [Table 5.24](#) and ranked according to  $R^2$ .

### Modeling Based on Iowa Databases

While the Iowa survey was undergoing compilation, the two Iowa databases—Cantor et al. (1998) and the expanded CHEEC—underwent initial modeling analysis. Because neither the Cantor et al. database nor the original CHEEC database contained HAA5 and HAA species data, the Iowa models were derived using the expanded ICR–Iowa database. MBAA could not be modeled due to low  $R^2$  values. The Singer et al. (2002) model that was used previously in this chapter was also used to predict the remaining HAA3 species (BDCAA, DBCAA, and TBAA). These models were then applied to the Cantor et al. database. A summary of the predicted values is shown in [Table 5.25](#).

It is important to note that these values only reflect the period in which the THM data were collected by Cantor et al. (1998), specifically 1987. Neither the THM data in this database nor the modeled HAA values represent DBP exposure over a historic time line, unless both the quality of the water supply and treatment and disinfection processes used at a particular plant did not significantly change over time. Also, because HAA5 and species were not measured in 1987, a direct error analysis could not be performed on the dataset.

After the Cantor et al. (1998) database was modeled, the original CHEEC data underwent a similar analysis. After the Iowa survey was completed, the survey data were integrated into the original CHEEC database. Again, the expanded ICR–Iowa database models were applied to the data. HAA5 and HAA species were then predicted based on THM data. Again, MBAA could not be modeled due to low  $R^2$  values. A summary of the measured THM data and the predicted HAA values is presented in [Table 5.26](#).

The main advantage of the expanded CHEEC database is that THM measurements date back to 1979. Such data allow the prediction of DBP formation over a wider DBP time line, dating back to the 1979 THM Rule. However, the earlier data (i.e., 1970s) is somewhat sparse. Most of the integrated CHEEC data were measured in the late 1980s and 1990s.

Subsequent to the development of the original CHEEC database, HAA data were collected in Iowa (in 1999 and 2000). After being compiled into the CHEEC database, selected HAA5 and HAA species data were extracted from selected plants for selected sample dates in 1999. A summary of these selected data can be found in [Table 5.27](#). It should be noted that these are averaged values of samples collected on the same day at several different locations for the same plant.

**Table 5.23**  
**Expanded ICR–Iowa database models based on influent water quality parameters**

Dependent variable (µg/L)	Independent variables					Statistics	
	pH	Temperature (°C)	TOC (mg/L)	Br <sup>-</sup> (mg/L)	Cl <sub>2</sub> dose (mg/L)	R <sup>2</sup>	Count
BCAA	X		X	X		0.50	166
	X	X	X	X		0.58	166
			X	X	X	0.62	76
	X	X			X	0.63	100
		X	X	X	X	0.67	76
	X			X	X	0.74	88
	X	X		X	X	0.76	88
	X	X	X	X	X	0.79	76
DBAA		X	X	X	X	0.54	72
	X		X	X		0.54	162
	X	X	X	X		0.55	162
	X	X	X	X	X	0.57	72
DCAA	X	X			X	0.53	100
	X	X		X	X	0.55	88
			X	X	X	0.68	76
		X	X	X	X	0.69	76
HAA5	X	X	X	X	X	0.72	76
	X	X		X	X	0.71	84
	X			X	X	0.71	84
			X	X	X	0.72	72
	X	X			X	0.73	96
	X	X	X	X	X	0.84	72
TCAA		X	X	X	X	0.84	72
			X	X	X	0.70	76
	X			X	X	0.77	88
	X	X		X	X	0.78	88
		X	X	X	X	0.79	76
	X	X	X	X	X	0.80	76
	X	X			X	0.80	100
	X	X	X	X		0.56	153
TTHM	X	X		X		0.59	165
		X	X	X	X	0.73	73
			X	X	X	0.73	73
	X			X	X	0.74	85
	X	X			X	0.78	97
	X	X	X	X	X	0.84	72
	X	X		X	X	0.87	85

Note: X indicates independent variable was considered in model (coefficients not shown).

**Table 5.24**  
**Expanded ICR–Iowa database models based on DBP values**

Dependent variable (µg/L)	Independent variables (µg/L)						R <sup>2</sup>	Count
	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	HAA5		
BCAA	X	X	X	X			0.61	173
DBAA	X	X	X	X			0.63	169
DCAA						X	0.76	186
HAA5	X	X	X	X			0.59	169
MCAA	X	X	X	X			0.51	173
						X	0.68	186
TCAA	X	X	X	X			0.63	173
						X	0.92	186

Note: X indicates independent variable was considered in model (coefficients not shown).

**Table 5.25**  
**Predicted DBP values except count and standard deviation: Cantor et al. database**

Statistic	Modeled BCAA	Modeled DBAA	Modeled DCAA	Modeled MCAA	Modeled TCAA	Modeled HAA5	Modeled BDCAA	Modeled DBCAA	Modeled TBAA
	(µg/L)								
Count	266	266	266	266	266	266	142	142	142
Maximum	7.79	4.88	24.58	5.62	46.42	78.40	11.62	3.28	0.27
95th percentile	5.01	1.65	10.10	2.40	15.44	31.49	6.33	1.27	0.00
75th percentile	1.48	0.60	1.96	1.07	−0.57	5.11	0.44	0.00	0.00
50th percentile	1.00	0.38	1.96	1.07	−0.57	5.11	0.00	0.00	0.00
25th percentile	1.00	0.38	1.38	0.77	−2.58	3.25	−2.46	−2.34	−0.06
5th percentile	0.65	0.25	−2.26	−0.46	−12.85	−8.55	−26.46	−34.16	−9.88
Minimum	−7.29	−0.87	−37.42	−16.84	−99.30	−122.44	−166.94	−258.80	−201.78
Standard deviation	1.44	0.56	4.74	1.40	10.67	15.34	17.29	29.82	23.92

Also, when a particular HAA was not detected, a value equal to half of the minimum reporting level was used (e.g., <1.0 entered as 0.50).

The full set of measured HAA data were to be used to validate which portions of the CHEEC database were best modeled by a central tendency model. For example, the central tendency model based on the expanded ICR–Iowa database had a significant number of negative predictions for certain HAAs (e.g., at least 50% of the TCAA predictions in the CHEEC database).

After the survey underwent a final QA/QC, it was determined that data was insufficient to perform a regression analysis on the dataset. Based on this conclusion, all of the Iowa data were merged into one comprehensive database to try to develop a dataset that could undergo regression

**Table 5.26**  
**Predicted DBP values: Original CHEEC database**

Statistic	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	BCAA	DBAA	DCAA	MCAA	TCAA	HAA5
(µg/L)											
Count	1,654	1,645	1,631	1,611	1,649	1,663	1,663	1,663	1,663	1,663	1,663
Maximum	201.00	69.00	21.00	14.00	219.00	26.38	6.61	132.61	36.18	291.99	428.31
95th percentile	76.00	19.00	8.00	2.00	95.87	6.34	2.18	15.81	3.99	27.83	49.99
75th percentile	30.00	10.00	3.33	0.50	45.67	3.45	1.01	6.53	1.75	8.04	19.93
50th percentile	8.00	4.00	1.00	0.50	16.00	1.38	0.57	1.57	0.99	-0.78	3.85
25th percentile	1.00	0.50	0.50	0.50	2.50	1.03	0.57	1.43	0.91	-0.78	3.39
5th percentile	0.63	0.50	0.50	0.50	2.00	0.47	0.35	-3.34	-0.85	-14.72	-12.05
Minimum	0.05	0.10	0.10	0.25	0.25	-4.91	-1.14	-36.73	-11.05	-100.10	-120.22
Average	19.92	6.18	2.35	0.72	28.72	2.39	0.88	4.13	1.30	3.45	12.15

**Table 5.27**  
**Selected HAA data compiled in 1999 and 2000: CHEEC database**

Iowa town	Sample date	Sample code	DBAA	DCAA	MBAA	MCAA	TCAA	HAA5
			(µg/L)					
Fairfield	5/17/99	Routine sample	1.00	0.50	0.50	1.00	0.50	1.00
Burlington	5/17/99	Routine sample	1.25	10.75	0.50	1.00	2.75	14.75
Council Bluffs	5/23/99	Routine sample	0.50	11.50	0.50	1.00	1.25	12.75
Sioux City	5/25/99	Routine sample	2.50	6.50	0.50	1.00	4.50	13.50
Ottumwa	6/1/99	Routine sample	1.00	17.00	0.50	1.00	2.67	20.67
Ottumwa	6/1/99	Maximum potential	0.50	16.00	0.50	1.00	2.00	18.00
Oskaloosa	6/1/99	Routine sample	1.00	3.75	0.50	1.00	3.25	8.00

analysis. This new dataset was deemed the Iowa composite database. To do this, the CHEEC database was augmented with data from

- NORS
- NOMS
- ICR database (Iowa utilities only)
- AWWA WATER:STATS
- AWWA WIDB

The Iowa data were also divided into two groups: alluvial and nonalluvial systems. This is because the nonalluvial systems, due to their hydrogeological formation, formed little or no DBPs. The alluvial systems, on the other hand, had high DBP formation potential.

**Table 5.28**  
**Average finished water and distribution system THMs for**  
**the Iowa composite alluvial systems**

Source	Finished water (µg/L)					Distribution system (µg/L)				
	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM
Boone	3.50	2.17	1.17	0.00	7.67	12.08	7.82	3.64	0.00	24.08
Burlington	30.75	7.60	1.55	0.00	39.75	51.43	12.86	3.86	0.00	62.50
Cedar Rapids	0.45	0.00	0.00	0.00	0.73	0.13	0.00	0.00	0.00	2.67
Centerville	160.00	24.00	2.00	0.00	186.00	38.78	8.33	1.33	0.06	48.53
Chariton	55.62	13.73	2.58	0.04	71.73	54.60	14.00	2.00	0.00	69.60
Clarinda	37.93	12.71	1.93	0.00	52.57	27.50	4.00	0.75	0.00	32.50
Council Bluffs	33.55	10.40	4.75	1.00	48.14	21.00	6.00	2.25	0.38	32.90
Creston						23.00	2.00	0.00	0.00	25.00
Des Moines	13.69	5.12	2.94	0.13	20.88	24.71	10.00	5.29	0.71	39.38
Fairfield	6.86	2.14	0.86	0.14	10.57	1.00	1.00	1.60	2.60	5.20
Fort Madison	20.80	5.60	1.50	0.00	28.10	9.62	2.92	0.62	0.00	13.86
Iowa American (Davenport)	65.75	7.74	0.88	0.00	71.76	57.04	6.32	1.83	0.00	61.83
Iowa City	35.87	13.63	3.71	0.11	54.81	31.00	12.83	4.83	0.33	74.75
Keokuk	53.50	9.50	0.50	0.00	63.50	27.64	7.82	2.09	0.09	37.82
Muscatine	2.00	2.00	2.00	0.00	6.00					
Newton	3.81	4.00	3.75	0.94	12.38	4.00	3.60	3.20	0.60	12.00
Osceola	70.87	15.77	2.48	0.00	89.29	28.67	10.33	2.80	0.00	41.50
Oskaloosa	9.00	4.80	3.00	0.40	17.00	10.75	9.00	1.75	26.60	32.75
Ottumwa	1.33	0.00	0.00	0.00	1.33					
Spencer	3.00	3.80	4.00	0.80	11.60	4.00	4.92	4.77	1.31	14.86
Spirit Lake	27.00	9.00	3.00	0.00	39.00	12.63	5.75	2.88	0.33	21.38
West Des Moines	4.00	0.00	0.00	0.00	4.50					
Winterset	55.50	19.25	8.75	0.75	84.00	32.67	7.00	2.00		40.50

Note: Blank cells indicate information was not available.

Once the data were aggregated into a centralized database, the entire dataset was imported into Statistica to perform the regression analysis. Similar to the first attempt, it was discovered that too many data values were missing to perform a linear regression analysis. Only the expanded Iowa–ICR database had sufficient paired data for such an analysis. This attempt further justifies a case study approach for the Iowa utilities, as discussed in chapter 6.

Even though the Iowa composite database could not be used to develop DBP formation models, similar to all of the other databases acquired, it could still be used to provide insight on DBP formation for each utility. [Tables 5.28](#) through [5.30](#) present average DBP values for each utility.

**Table 5.29**  
**Average finished water and distribution system HAAs for the Iowa composite alluvial systems**

Source	Finished water (µg/L)								Distribution system (µg/L)							
	MCAA	DCAA	TCAA	BCAA	MBAA	DBAA	HAA5	HAA6	MCAA	DCAA	TCAA	BCAA	MBAA	DBAA	HAA5	HAA6
Boone																
Burlington								1.00	9.80	4.80		0.00	1.00	15.00		
Cedar Rapids	1.00	1.86	0.00	0.00	0.00	0.00	1.86	1.86	1.00	2.67	0.00	0.00	0.00	0.00	2.67	2.67
Centerville																
Chariton																
Clarinda																
Council Bluffs								1.00	10.57	1.57		0.00	0.50	12.29		
Creston								38.75	16.00	4.75	0.00	59.75	59.75			
Des Moines	1.00	5.33	0.50	2.50	0.00	0.60	6.20	8.40	1.00	6.13	1.13	2.60	0.00	1.13	8.00	10.20
Fairfield								1.00	0.00	0.00		0.00	1.00	1.00		
Fort Madison								1.00	12.00	1.50		0.00	0.00	14.00		
Iowa American (Davenport)	1.83	7.00	13.00	1.33	0.00	0.00	21.29	19.67	1.60	8.50	14.58	2.00	0.09	0.00	24.58	21.13
Iowa City								1.00	10.88	12.88		0.00	0.86	24.38		
Keokuk								1.00	14.14	5.00		0.00	0.33	19.14		
Muscatine																
Newton																
Osceola																
Oskaloosa							11.00	4.50	2.00		0.00	1.50	8.00		10.67	
Ottumwa																
Spencer																
Spirit Lake		8.00	3.00	0.00		1.00	12.00	12.00		9.00	3.00	0.00		1.00	13.00	13.00
West Des Moines																
Winterset	1.50	16.00	11.50		0.00	1.00	29.50									

Note: Blank cells indicate information was not available.

**Table 5.30**  
**Average finished water THMs for the Iowa composite nonalluvial systems**

Utility (database)	Finished water				
	CHCl <sub>3</sub>	BDCM	DBCM	CHBr <sub>3</sub>	TTHM
	(µg/L)				
Clinton	0.00	0.00	0.06	0.00	0.51
Dubuque	7.82	1.36	0.27	0.00	10.00
Fort Dodge	1.25	0.13	0.13	0.00	2.29
Indianola	0.00	0.00	0.00	0.00	0.50
Marion	0.00	0.00	0.00	0.00	0.50
Marshalltown	0.21	0.00	0.00	0.00	0.84
Sioux City	9.67	10.67	10.00	2.33	32.83

## SUMMARY

Based on the demonstrated inadequacy of the central tendency modeling approach, a decision was made midway through the project to explore the development of another complementary modeling approach, the case study approach, as discussed in the next two chapters for Ontario and Iowa, respectively.



## **CHAPTER 6**

### **CASE STUDY APPROACH TO MODELING DBP EXPOSURE IN ONTARIO**

#### **INTRODUCTION**

It was decided that a complementary modeling effort would be made for the Ontario DWSP database that used both the central tendency model, discussed in chapter 5, and a new case study approach, developed by Stuart Krasner (co-investigator), representing an expert system approach. The motivation for this new approach was the poor prediction capability of the central tendency model(s) for lower and higher levels of independent parameters.

#### **PREDICTIONS OF HISTORICAL DBP OCCURRENCE IN ONTARIO**

For the prediction of historical DBP occurrence in Ontario (Canada), a central tendency model and a case study approach (described in previous chapters) were used where appropriate to make primary predictions.

The central tendency model was used for plants that treat surface waters with low concentrations of DOC, which use conventional treatment or direct filtration with  $\text{Cl}_2$  at some point in the disinfection process. The central tendency model requires measured THM data and can only be used for plants that use the same treatment/disinfection scheme in the DWSP database that was used historically (during the period of exposure).

The case study approach was used for plants that treat moderate- or high-DOC surface waters and for plants that treat groundwaters. In addition, the case study approach was used for low-DOC surface waters for which the central tendency model could not be used. Because HAA monitoring did not begin until 1995, changes in treatment/disinfection prior to the collection of HAA data required a “secondary” modeling effort in the case study approach.

If the treatment/disinfection process changed prior to the 1990s, a secondary modeling effort was needed for both the THMs and HAAs. For plants with no current DBP data, a secondary modeling effort was also required.

Table 6.1 represents a summary of the DWSP database, which includes monitoring data from 132 water treatment facilities supplying 73% of the population (7.2 million) of the total of Ontario (10 million). As originally planned, the approximate population served by the central tendency plants is 5.3 million; however, it became necessary to subject some of these plants to case study predictions because of a change in disinfection practice over the relevant prediction period.

#### **Central Tendency Model**

A decision was made to use the central tendency model for plants that treat surface waters with low concentrations of DOC and employ conventional treatment or direct filtration with  $\text{Cl}_2$  at some point in the disinfection process. These plants represent a significant portion of the plants in the Ontario DWSP and, thus, correspond to the central tendency plants and waters for which the central tendency model should be most accurate.

**Table 6.1**  
**Segmentation of Ontario treatment plants**

Group population*	Statistic	Raw DOC (mg/L)	Raw color (cu)	Treated color (cu)	DCAA (µg/L)	TCAA (µg/L)	BCAA (µg/L)	HAA5 (µg/L)
DWSP 7,183,990	Minimum	0.2	0.2	0.2	0.1	0.1	0.1	0.1
	10th (percentile)	1.4	1.0	0.5	1.3	1.5	0.3	4.9
	25th	1.8	1.8	0.8	3.0	3.0	0.6	9.5
	50th	2.1	4.3	1.7	6.5	6.7	1.3	19.0
	75th	5.4	18.4	3.6	12.5	19.0	2.5	36.2
	90th	8.6	34.4	5.6	22.6	35.4	3.5	60.6
	Maximum	23.6	103.0	41.8	82.4	230.2	11.7	299.5
Central tendency 5,270,721	Minimum	1.2	0.2	0.2	0.3	0.6	0.1	2.0
	10th	1.6	0.8	0.4	1.5	1.9	0.8	6.0
	25th	1.8	1.3	0.5	2.6	2.5	1.3	8.5
	50th	1.9	2.1	0.8	4.2	3.6	2.2	12.0
	75th	2.1	3.4	1.3	6.9	6.0	3.0	18.0
	90th	2.6	6.9	2.0	10.0	11.3	3.8	24.7
	Maximum	5.1	17.4	6.2	32.9	30.9	6.0	66.1
High-DOC surface waters 64,440	Minimum	5.8	15.7	1.5	0.1	0.1	0.1	0.1
	10th	7.9	21.5	2.5	4.5	15.0	0.2	23.5
	25th	8.6	26.1	3.3	9.8	20.2	0.3	34.2
	50th	9.5	36.8	4.0	14.2	25.9	0.6	44.6
	75th	10.3	48.3	6.2	24.3	36.9	1.2	63.1
	90th	11.3	61.4	11.9	50.8	74.6	2.6	125.0
	Maximum	23.6	103.0	30.3	82.4	230.2	3.4	299.5
Moderate-DOC surface waters 859,491	Minimum	3.3	2.2	0.6	1.7	6.0	0.1	13.8
	10th	4.2	11.8	1.7	6.3	9.7	0.4	20.6
	25th	4.8	14.7	2.4	8.6	12.7	0.5	24.8
	50th	5.4	18.7	3.5	12.7	18.5	0.9	31.3
	75th	6.0	29.2	4.4	21.3	31.3	1.5	52.6
	90th	6.4	34.3	6.8	31.3	47.2	2.8	85.3
	Maximum	9.0	38.5	9.5	69.0	113.0	5.0	185.5
Groundwaters and springs 339,758	Minimum	0.2	0.2	0.2	0.1	0.1	0.1	0.5
	10th	0.3	0.6	0.6	0.2	0.2	0.1	1.6
	25th	0.6	1.3	1.1	0.5	0.5	0.5	2.3
	50th	0.9	2.4	2.2	1.7	1.1	0.8	4.8
	75th	1.2	4.5	3.9	5.9	4.6	2.1	14.0
	90th	2.4	8.5	6.1	9.1	9.2	2.9	24.0
	Maximum	11.9	85.2	41.8	33.2	50.5	11.7	105.5
Miscellaneous 494,200	Minimum	1.2	0.8	0.2	0.3	0.7	0.1	1.0
	10th	1.4	1.5	0.6	0.7	1.0	0.3	2.3
	25th	1.9	2.0	0.8	1.2	1.3	0.5	3.9
	50th	2.1	3.5	1.3	4.0	3.8	0.9	12.9
	75th	2.9	7.8	4.7	7.6	10.1	1.7	22.0
	90th	3.1	8.5	5.6	14.3	12.6	2.4	27.9
	Maximum	3.5	22.3	9.1	22.7	16.5	6.2	38.0

\* Some municipalities in Ontario are served by more than one plant. In those cases, the entire population was assigned to the first plant listed in DWSP for that municipality. Thus, the populations in each group are approximate.

In 1995, DWSP monitored 132 water treatment facilities, which supplied 73% of Ontario's total population of 10 million. The approximate population served by the plants in DWSP was 7.2 million and by the central tendency plants was 5.3 million. In DWSP, the median raw water DOC was 2.1 mg/L (25th and 75th percentiles of 1.8 and 5.4 mg/L, respectively) (Table 6.1). For the central tendency plants, the median raw water DOC was 1.9 mg/L (25th and 75th percentiles of 1.8 and 2.1 mg/L, respectively). The central tendency plants also treated waters low in color (25th percentile, median, and 75th percentile values of 1.3, 2.1, and 3.4 cu, respectively).

The central tendency model predicts HAA occurrence based on measured THM values. Table 6.1 summarizes measured HAA data for the Ontario treatment plants. The central tendency plants tended to produce similar levels of DCAA and TCAA. In addition, they tended to produce a fair amount of BCAA because the bromide-to-DOC ratios in these waters were relatively high because the denominator (DOC) was relatively low. For example, the median concentrations of DCAA, TCAA, and BCAA for this group were 4.2, 3.6, and 2.2 µg/L, respectively.

Because the central tendency model requires measured THM data, it cannot be used for plants that treat low-DOC surface waters where there are no THM data in DWSP. In addition, it cannot be used for plants that used a different treatment and disinfection scheme in the DWSP database than was used historically.

If the treatment and disinfection process changed at some time in the 1990s, THM data and HAA (central tendency) predictions for the time period prior to the changes are used for modeling historical DBP exposure.

## Case Study Approach

The case study approach was used for plants that treat high-DOC surface waters. The median raw water DOC for this group was 9.5 mg/L (25th and 75th percentiles of 8.6 and 10 mg/L, respectively) (Table 6.1). Although these plants represent a small portion of the Ontario population (approximately 65,000), they treat the waters with the highest DBP exposure. For example, HAA5 for plants that treated high-DOC surface waters was 45 µg/L on a median basis (90th percentile = 125 µg/L). In comparison, the central tendency plants had median and 90th percentile HAA5 values of 12 and 25 µg/L, respectively.

The central tendency model did not accurately predict HAA formation, especially the speciation, for the plants that treat high-DOC waters. Because these plants treat high-color waters (25th percentile, median, and 75th percentile values of 26, 37, and 48 cu respectively), they typically produced much more TCAA than DCAA. In addition, they tended to produce relatively low amounts of BCAA because the bromide-to-DOC ratios were relatively low. For example, the median concentrations of DCAA, TCAA, and BCAA for this group were 14, 26, and 0.6 µg/L, respectively.

The case study approach was used for plants that treat moderate-DOC surface waters. The median raw water DOC for this group was 5.4 mg/L (25th and 75th percentiles of 4.8 and 6.0 mg/L, respectively). These plants represent a larger portion of the Ontario population (approximately 1 million) than the plants that treat high-DOC waters, and they treat waters with moderately high DBP exposure. For example, HAA5 for plants that treated moderate-DOC surface waters was 31 µg/L on a median basis (90th percentile = 85 µg/L).

The central tendency model did not accurately predict HAA formation, especially the speciation, for the plants that treat moderate-DOC waters. Because these plants treat moderately colored waters (25th percentile, median, and 75th percentile values of 14, 19, and 29 cu,

respectively), they typically produced much more TCAA than DCAA. In addition, they tended to produce relatively low amounts of BCAA because the bromide-to-DOC ratios were relatively low. For example, the median concentrations of DCAA, TCAA, and BCAA for this group were 13, 18, and 0.9 µg/L, respectively.

The case study approach was used for plants that treat groundwaters or springs. Most of these plants used little or no treatment other than disinfection. The central tendency model did not accurately predict HAA formation for the plants that treated groundwaters. The population served by plants that treated groundwaters or springs was less than half a million.

Most of the groundwaters (and springs) in DWSP were low in DOC (25th percentile, median, and 75th percentile values of 0.6, 0.9, and 1.2 mg/L, respectively). However, some of the groundwaters were high in DOC and/or color. In addition, some of the groundwaters appeared to be relatively high in bromide, as the DBP speciation was shifted to the more brominated THMs and HAAs. Thus, many of the groundwaters were significantly different than the central tendency surface waters (e.g., much lower or higher in DBP precursors).

As noted above, some plants that treated low-DOC surface waters that could not be modeled with the central tendency model were handled by the case study approach. The population served by these miscellaneous plants was approximately half a million.

If the treatment and disinfection process changed at some time in the 1990s, THM and HAA data for the time period prior to the changes were used for modeling historical DBP exposure. Because HAA monitoring did not begin until 1995, changes in treatment and disinfection prior to the collection of HAA data required a secondary modeling effort. If the treatment and disinfection process changed prior to the 1990s, a secondary modeling effort was needed for both the THMs and HAAs. For plants with no current DBP data, a secondary modeling effort was also required.

### **Secondary Modeling Effort**

The secondary modeling effort is an extrapolation of the case study approach. Plants that treated water from the same or similar watersheds and whose current treatment and disinfection scheme was the same as another plant's were used to predict historical DBP exposure for the latter plant. Likewise, other secondary modeling efforts were made for plants that treat surface or groundwater, as appropriate. The approach was to take the median DBP occurrence for the plants upon which the predictions were being made. In some cases, median DBP relationships (e.g., THM/HAA ratio, TCAA/HAA ratio) were used in predicting historical DBP occurrence. Each of the assumptions in this effort is documented.

Another outstanding issue is predicting DBP exposure for municipalities that treat more than one source of water (e.g., surface and groundwater). In most cases, the secondary source represents a small portion of the overall demand. In those cases, most of the exposure is due to the primary source. In some municipalities, the two treated waters are blended, whereas in other cases one source may serve one portion of the system. A decision was made in each case as to which approach would be most appropriate to minimize misclassification in each system. Each of the assumptions in this decision-making process is documented.

### **Tertiary Modeling Effort**

The tertiary modeling effort represents an extrapolation of the secondary approach, focusing on utilities not in the DWSP database, but part of the King and Marrett (1996) study.

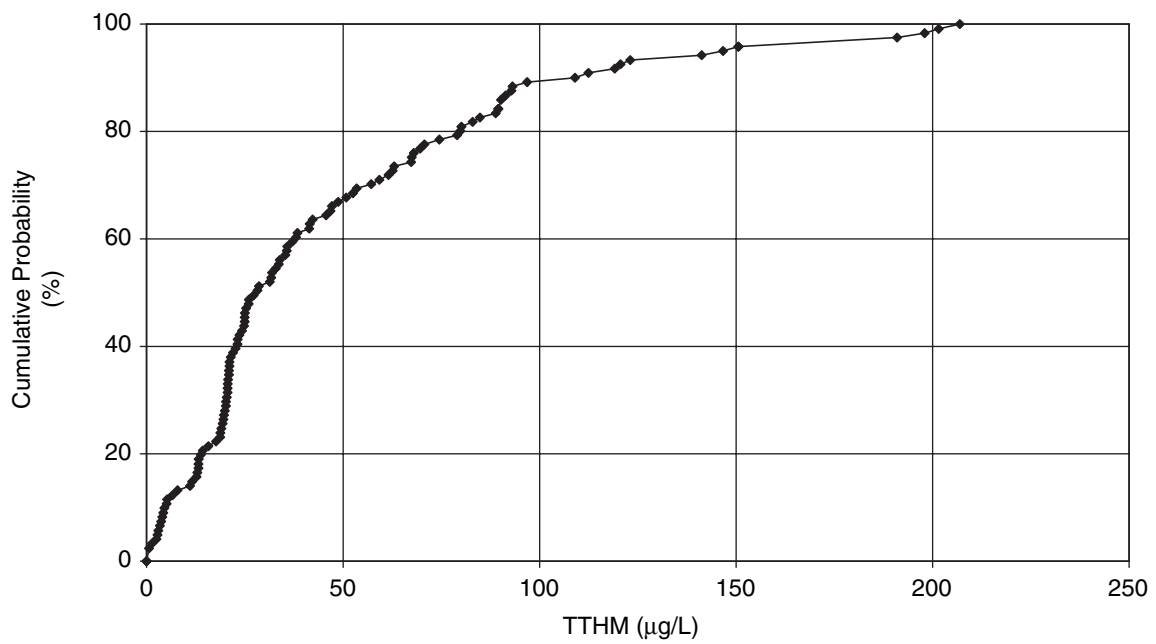
## CASE STUDY PREDICTION OF HISTORICAL DBP OCCURRENCE IN ONTARIO

### Primary Predictions

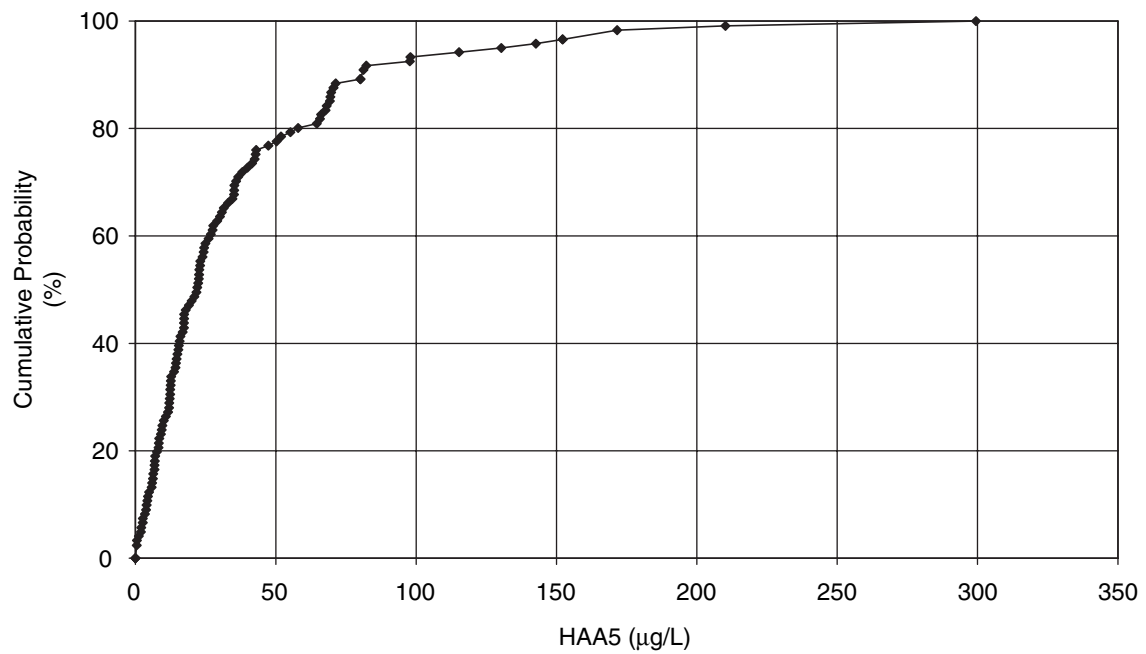
A tabular summary of primary predictions of historical DBP occurrence in Ontario appears in appendix 6.1 as a Microsoft Excel spreadsheet. From the relevant Excel file in appendix 6.1, one set of DBP predictions for each distribution system was copied, excluding individual predictions for separate water supplies feeding into common distribution systems. If predictions were for different time periods, predictions for the oldest period (even if this period did not result in the highest DBP exposure for that system) were taken. From these, cumulative probability distributions (CPDs) were calculated for certain DBPs (Figures 6.1 to 6.6); these CPDs are not population weighted. Table 6.2 summarizes some of the key percentiles in each CPD.

The following items address the compilation and interpretation of this tabular summary:

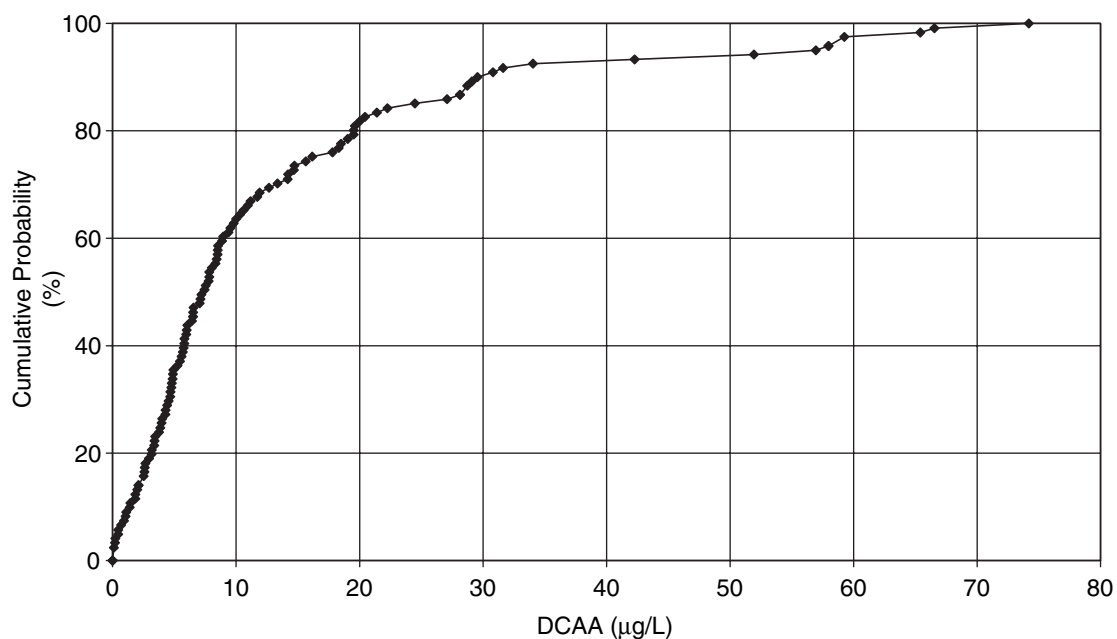
- For each of the water treatment plants (WTPs) or water supply systems (WSSs) in the DWSP, historical DBP occurrence was predicted.
- For each WTP or WSS, the source water and treatment and disinfection process associated with the DBP predictions is provided. If a particular municipality used a different source water and/or treatment and disinfection process during some portion of the King and Marrett (1996) historical database, then the predictions are not valid for that period of time. Additional secondary DBP predictions have been developed for those municipalities with alternative source waters or treatment and disinfection processes in some portion of their historical records (discussed later in this chapter).
- Most surface water plants used conventional treatment or direct filtration. Both these treatment systems use coagulation to remove turbidity and some portion of the DOC and color. Some surface water plants used disinfection only and had no physical process (coagulation or filtration) to remove turbidity, DOC, or color. However, at such plants, there was still some reduction in the level of color, as  $\text{Cl}_2$  can oxidize a certain portion of the color.
- Most groundwater plants used disinfection only. Some groundwater plants employed a process to remove iron and/or manganese; however, those processes would not impact DBP formation or control.
- In addition to the use of  $\text{Cl}_2$ , which was in the form of gaseous  $\text{Cl}_2$  or liquid  $\text{Cl}_2$  (i.e., hypochlorite), some plants used chloramines in the form of monochloramine ( $\text{NH}_2\text{Cl}$ ), and other plants used chlorine dioxide ( $\text{ClO}_2$ ).
- Predictions were made for TTHM and the two major THM species ( $\text{CHCl}_3$  and BDCM). For most municipalities, predictions were also made for the other two THMs (DBCM and  $\text{CHBr}_3$ ). Because the occurrence of the latter two brominated species is quite low or below the reporting limits, associations between the occurrence of these THMs (on an individual basis) with cancer will not be examined in the reanalysis of the King and Marrett (1996) study.
- Predictions were made for HAA5 and the three major HAA species (DCAA, TCAA, and BCAA). It should be noted that, in the Ontario DWSP, HAA5 corresponds to the sum of DCAA, TCAA, BCAA, MCAA, and MBAA, whereas in the United States,



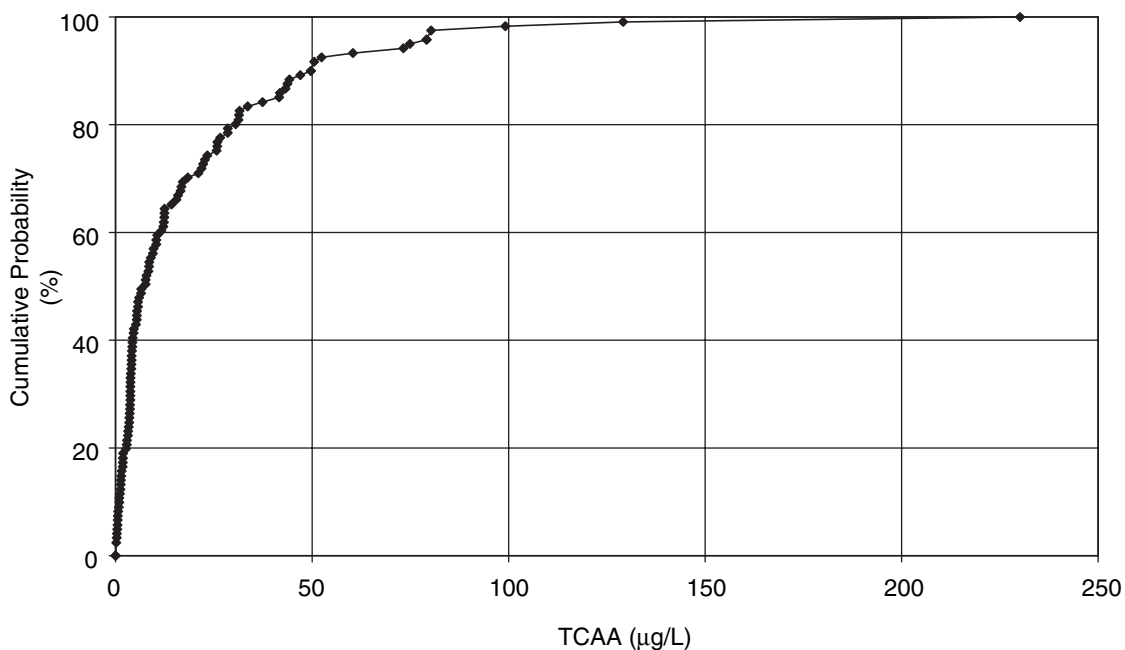
**Figure 6.1 Cumulative probability distribution of historical TTHM in Ontario DWSP**



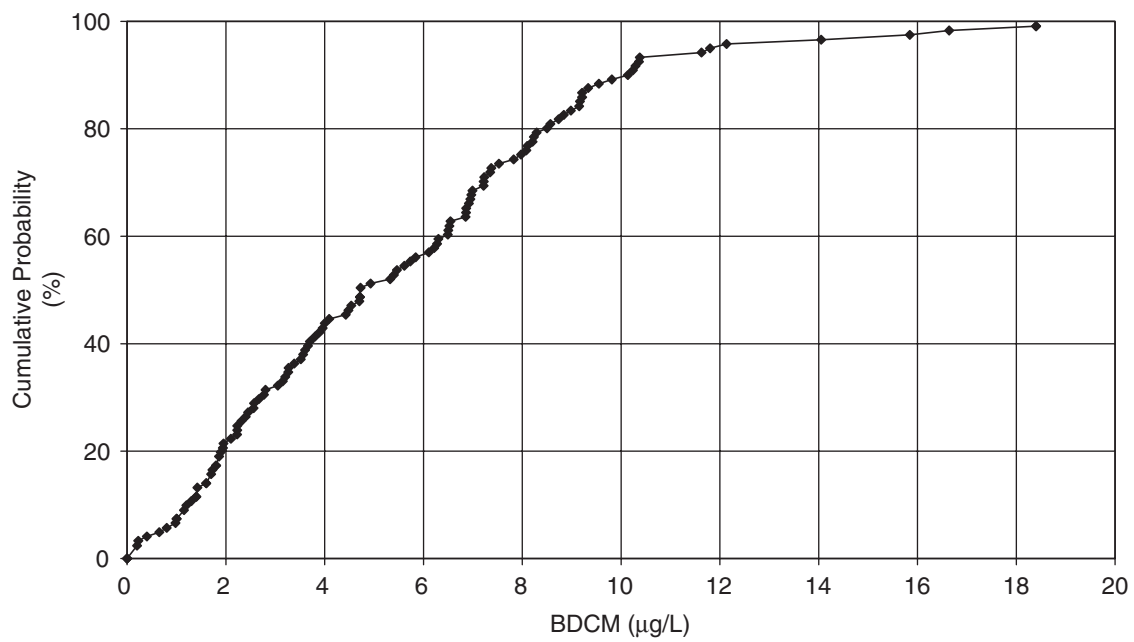
**Figure 6.2 Cumulative probability distribution of historical HAA5 in Ontario DWSP**



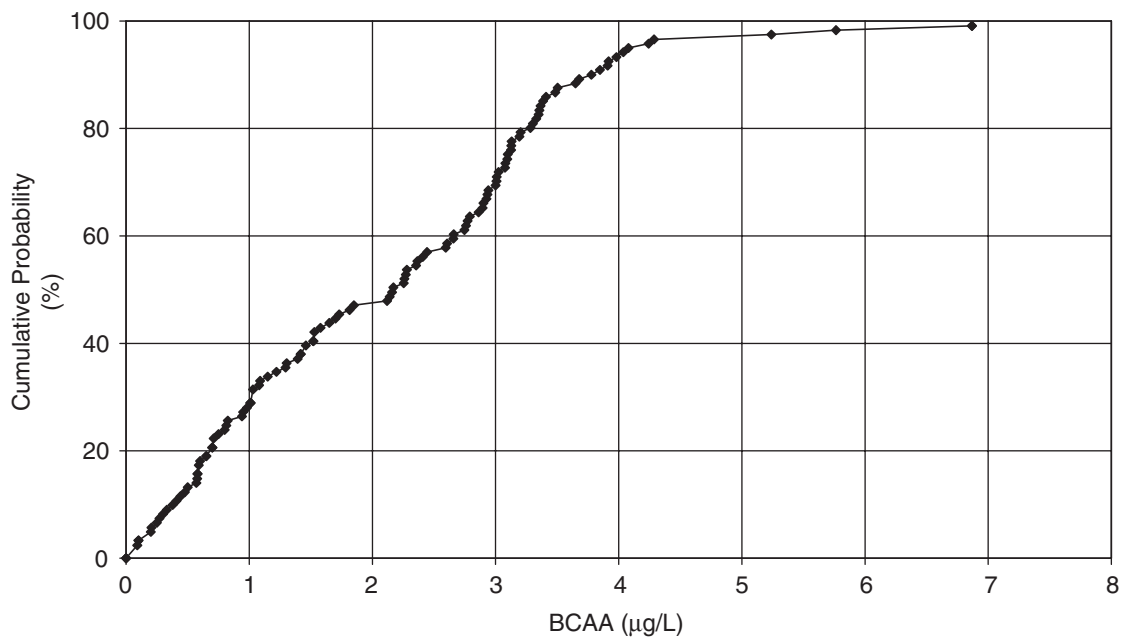
**Figure 6.3 Cumulative probability distribution of historical DCAA in Ontario DWSP**



**Figure 6.4 Cumulative probability distribution of historical TCAA in Ontario DWSP**



**Figure 6.5 Cumulative probability distribution of historical BDCM in Ontario DWSP**



**Figure 6.6 Cumulative probability distribution of historical BCAA in Ontario DWSP**



**Table 6.2**  
**Percentiles in CPDs of historical occurrence of DBPs in Ontario DWSP**

	TTHM	HAA5	DCAA	TCAA	BDCM	BCAA
Percentile	(µg/L)					
Maximum	207	299	74	230	18	6.9
90th	109	81	30	50	10	3.8
75th	67	42	16	26	8.0	3.1
Median	28	22	7.5	7.0	4.7	2.2
25th	19	10	3.9	3.5	2.2	0.8

HAA5 is the sum of DCAA, TCAA, MCAA, MBAA, and DBAA. In the United States, BCAA is included in HAA6, which equals HAA5 plus BCAA. In low-bromide waters, the relative occurrence of the DXAAs is  $DCAA > BCAA \gg DBAA$ . Because most of the waters in Ontario are low in bromide,  $HAA5 \sim HAA6$ . This is an important point to remember when comparisons are made to HAA data in the Iowa database.

- DBP predictions are for the distribution system, not for the plant effluents. Most consumers, except those close to the WTPs, have DBP exposure reflective of the distribution system.
- DBP predictions are annual average values. These values are typically lower than the peak DBP occurrence (in August) studied by King and Marrett (1996).
- Predictions are typically based on the time period of 1940 to 1990, which is the period studied by King and Marrett (1996). Changes in source waters and/or treatment and disinfection processes after 1990 are not included in these predictions.
- If a municipality changed its source water and/or treatment and disinfection process during the historical period of interest, two (or more) sets of DBP predictions are provided, representing the two (or more) sets of conditions.
- If a municipality has two sources of water feeding its distribution system, a distribution system prediction of DBP occurrence was made based on the relative proportion of each water feeding the distribution system.
- If the relative proportion of two different source waters feeding a common distribution system was not provided, an estimate of the blend ratio was made.
- No information was provided on when a switch was made to alternative disinfectants for certain systems.
- The King and Marrett (1996) database does not go back to 1940 for some systems in which historical treatment and disinfection practices were probed. Therefore, DBP predictions were only provided back to the earliest date for which there is information.

Although the maximum historical occurrence of HAA5 was greater than that of the TTHM, the 25th through 90th percentiles were greater for the TTHM. The relative proportion of THMs to HAAs was different for some of the low-DBP waters compared to some of the high-DBP waters.

Although the median historical occurrences of DCAA and TCAA were similar, the 75th percentile to maximum values were greater for TCAA. In many of the high-DOC, high-color waters, TCAA occurrence was much greater than DCAA occurrence.

The major THM formed in most of the Ontario waters was  $\text{CHCl}_3$ . However, various toxicology studies suggest that BDCM has a much greater cancer risk than  $\text{CHCl}_3$ . The range of historical BDCM occurrence (up to 18  $\mu\text{g/L}$ ) was greater than the range of BCAA occurrence (up to 6.9  $\mu\text{g/L}$ ), the major brominated HAA in this study.

## Secondary Predictions

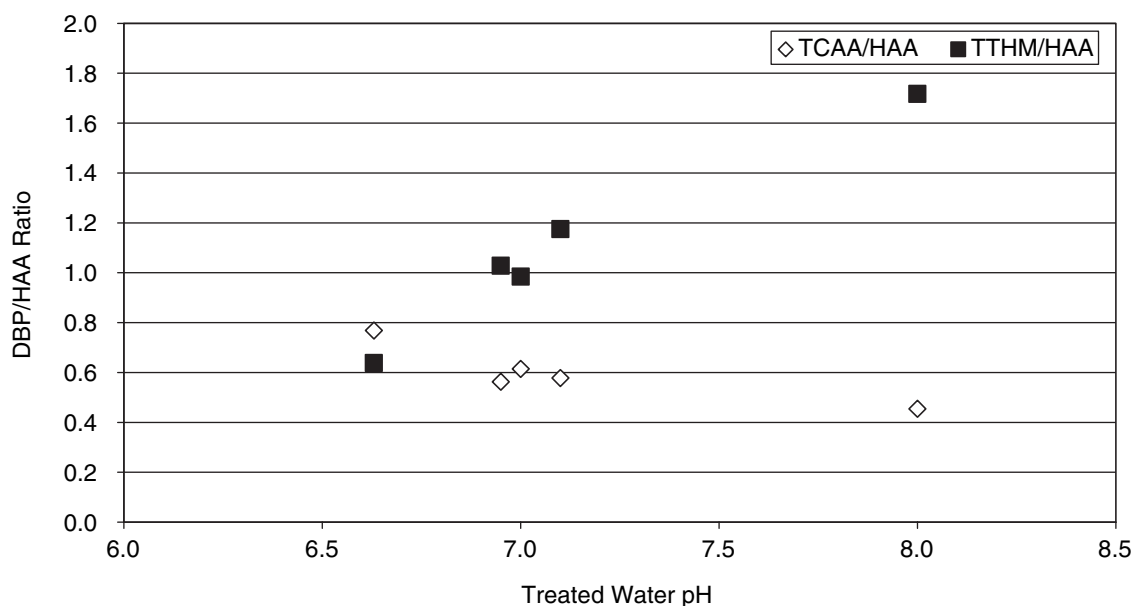
A secondary modeling effort represents an extrapolation of the case study approach. Plants that treated water from the same or similar watersheds whose current treatment and disinfection scheme was the same as another plant's historical treatment and disinfection scheme were used to predict historical DBP exposure for the latter plant. The predictions are summarized in appendix 6.2. This section of the chapter gives examples of how the secondary modeling effort was performed.

### *Atikokan*

This WTP treats a high-DOC (8.6 mg/L), high-color (40 cu) surface water. Settling (sedimentation basins) was added in 1995. Improved physical removal resulted in a lower treated-water color (4.8 versus 10 cu) and lowered the level of THMs from 198 to 71  $\mu\text{g/L}$ . Currently, HAA5 is 46  $\mu\text{g/L}$ , with 15 and 25  $\mu\text{g/L}$  of DCAA and TCAA, respectively. Five WTPs in DWSP (Balmertown [Sandy Bay], Cochenour, Hudson, Madsen, and Red Lake) treat high-DOC (6.8–11 mg/L), high-color (16–46 cu) surface waters with chlorine and no physical treatment and produce treated water with 10–25 cu and 141–207 and 82–299  $\mu\text{g/L}$  of THMs and HAA5, respectively. The latter five plants treat water with a similar amount of DOC, color (raw and treated), and THMs, as Atikokan did before the addition of settling. However, these five plants had a wide range of HAA data, with THM/HAA ratios of 0.6–1.7  $\mu\text{g}/\mu\text{g}$ . Figure 6.7 shows the impact of treated-water pH on the THM/HAA and TCAA/HAA ratio for these five plants.

Stevens, Moore, and Miltner (1989) evaluated the effect of pH on DBP formation. They evaluated chlorination pH levels of 5, 7, and 9.4. THM formation was higher with increasing pH. For the HAAs, pH (in the range of 5–9.4) had no significant effect on DCAA formation, whereas TCAA formation was lower at pH 9.4 than at the lower pH levels. The results in Figure 6.7 are consistent with the findings of Stevens, Moore, and Miltner, with an increase in pH resulting in an increase in the THM/HAA ratio and a decrease in the TCAA/HAA ratio. Atikokan's historical pH for the treated water was 8.0, which is similar to that of Hudson WTP. Hudson had a THM/HAA ratio of 1.7  $\mu\text{g}/\mu\text{g}$ , where 45% and 52% of the HAA5 was TCAA and the dihalogenated HAAs (DXAAs), respectively. At Hudson, 51% of the HAA5 was DCAA and the remainder was BCAA. It was assumed that Atikokan's historical DBP ratios were similar to those at Hudson. Thus, using a historical THM value of 198  $\mu\text{g/L}$  at Atikokan and assuming that the THM/HAA ratio was 1.7  $\mu\text{g}/\mu\text{g}$ , historical HAA5 at Atikokan was determined to be 115  $\mu\text{g/L}$ . Assuming that 52%, 51%, and 45% of HAA5 was DXAA, DCAA, and TCAA, respectively, historical HAA5 speciation was determined to be 59, 52, and 1.0  $\mu\text{g/L}$  of DCAA, TCAA, and BCAA, respectively.

Note that after Atikokan installed settling, it was operated at a lower pH (7.3). A conventional WTP (with sedimentation basins) can handle a higher coagulant dose, where the additional



**Figure 6.7 Effect of pH on TTHM/HAA and TCAA/HAA ratios for high-DOC surface waters with chlorination only**

coagulant dose will suppress the pH. With a lower pH, Atikokan has a higher TCAA/HAA ratio (60%). Also note that historical BCAAs were predicted to be low based on HAA speciation at Hudson. Alternatively, this prediction was checked by assuming that the shift in speciation to brominated DXAAs was similar to the shift in speciation to brominated THMs (from  $\text{CHCl}_3$  to BDCM) (i.e.,  $\text{BCAA} = \text{DCAA} * [\text{BDCM}/\text{CHCl}_3]$ , on a molar basis). Based on the Atikokan speciation for THMs, BCAA was predicted to be 1.1  $\mu\text{g}/\text{L}$ , which is similar to the prediction based on the Hudson HAA speciation.

### **DWSP Springs**

In DWSP, there are four low-DOC (0.3–1.1 mg/L), low-color (0.6–4.0 cu) springs (Bracebridge [leader spring], Delhi [spring supply], Owen Sound [spring supply], and Port Dover [spring supply]) that are chlorinated and typically receive no physical treatment. Three of the spring supplies have THM data, whereas only one has HAA data. All have DOC and color data; however, older THM data were obtained from Will King (co-investigator) for Port Dover for which there were no DOC/color data. Therefore, the Port Dover data were segmented into two groups: one with DOC and color data and the other without.

First, the ratio of color to DOC in the raw water was examined, which would provide an indication of the reactivity of the DOC to form DBPs. Three of the springs had color/DOC ratios of 1.2–2.6 cu/mg, whereas Port Dover had a ratio of 4.4 cu/mg. Second, the amount of THMs produced per unit DOC was examined. For two of the springs, the THM/DOC was 7.5–17  $\mu\text{g}/\text{mg}$  (median = 12  $\mu\text{g}/\text{mg}$ ), whereas Port Dover had a ratio of 44  $\mu\text{g}/\text{mg}$ . The higher reactivity of the Port Dover DOC is consistent with its higher color/DOC ratio. Thus, the amount of THMs produced per unit of color was examined. For two of the springs (which have lower raw water color), the THM/color was 6.1–6.7  $\mu\text{g}/\text{cu}$  (median = 6.4  $\mu\text{g}/\text{cu}$ ), whereas Port Dover had a ratio of

9.9 µg/cu. For these springs, color appears to be a better indicator than DOC of the reactivity of the water. Because Bracebridge (leader spring) has a similar color/DOC ratio (2.0 cu/mg) as the other two low-color springs, it was assumed that it would have a similar THM reactivity (i.e., 6.4 µg/cu). Thus, it was predicted that Bracebridge had 3.8 µg/L of THMs, which corresponds to 13 µg THMs/mg DOC, similar to the median ratio for the other two low-color springs.

Next, THM speciation data were examined. As there were no TDS or chloride data available for Bracebridge (leader spring), there was no surrogate parameter for the bromide of the water. For the two low-color springs,  $\text{CHCl}_3$  was 34%–54% of the THMs (median = 44%) and BDCM was 29%–30% of the THMs (median = 29%). It was assumed that the THM speciation of Bracebridge was similar to that of the other two low-color springs (i.e., 44% and 29%  $\text{CHCl}_3$  and BDCM, respectively).

Finally, HAA predictions for all three of the springs with no HAA data were developed. Owen Sound (spring supply) had a THM/HAA ratio of 3.19 µg/µg, and the HAA speciation was 54% DXAAs (40% DCAA) and 37% TCAA. As all four of the springs treat relatively low-DOC, low-color water with  $\text{Cl}_2$ , and little or no physical treatment at ambient pH (7.4–7.7), it was assumed that the relative formation of HAAs would be similar for all of the springs. (Note: In low-color waters, THMs tend to be higher than HAAs and DCAA higher than TCAA, which was what was observed for Owen Sound.)

### ***Brantford***

This WTP treats water from the Grand River (DOC ~5 mg/L, color ~20 cu) with conventional treatment,  $\text{Cl}_2$ , and  $\text{NH}_2\text{Cl}$ . In 1993, THM formation was reduced (on a median basis) from 131 to 83 µg/L (to comply with the 100-µg/L guideline). Brantford implemented chloramines in 1952, so a historical prediction of its DBPs levels with  $\text{Cl}_2$  is needed. Cayuga WTP and Ohsweken WTP also treat water from the Grand River (DOC = 4.6–4.9 mg/L, color = 14–15 cu) with conventional treatment and  $\text{Cl}_2$ . In addition, Ohsweken WTP uses UV disinfection. Cayuga and Ohsweken have THM levels of 151 and 74 µg/L, respectively. Ohsweken may have relatively low THM formation due to the use of a combination of UV and  $\text{Cl}_2$  disinfection, whereas Cayuga uses  $\text{Cl}_2$  only. As Brantford and Cayuga have similar raw water DOC and color and similar treated water color (4.6–5.9 cu) and pH (7.5–7.8), it was assumed Brantford's historical THMs with  $\text{Cl}_2$  only were similar to Cayuga's current THMs with  $\text{Cl}_2$  (i.e., 151 µg/L). Thus, it was predicted that a switch from  $\text{Cl}_2$  only to  $\text{Cl}_2/\text{NH}_2\text{Cl}$  at Brantford historically reduced THMs from 151 to 131 µg/L. Because Brantford used a relatively high pre- $\text{Cl}_2$  dose (7 mg/L on a median basis), there was still significant THM formation with pre- $\text{Cl}_2$  before post- $\text{NH}_2\text{Cl}$ .

Next, historical HAAs were predicted for Brantford. With  $\text{Cl}_2/\text{NH}_2\text{Cl}$ , the THM/HAA ratio was 0.99 µg/µg, with 43% DXAAs (38% DCAA) and 52% TCAA. Because most of the THM formation occurred with pre- $\text{Cl}_2$ , it was assumed that the relative formation of THMs to HAAs was similar when  $\text{Cl}_2$  only was used. Thus, it was predicted that historical HAAs were 152 µg/L (with 58 µg/L DCAA, 79 µg/L TCAA, and 6.9 µg/L BCAA). Alternatively, BCAA was predicted (based on the relative speciation of BDCM) to be 8.3 µg/L, which is consistent with the former prediction. Finally, it was assumed that the current HAA occurrence at Cayuga with  $\text{Cl}_2$  only was similar to the historical prediction at Brantford with  $\text{Cl}_2$  only (i.e., 152 µg/L). Note: For Brantford—which is a high-color water—THM and HAA formation were similar, and TCAA was greater than DCAA, which is consistent with other high-color waters.

## *Cambridge Well Supply*

A low-DOC (1.2 mg/L), low-color (2.4 cu), high-chloride (132 mg/L), high-TDS (666 mg/L) groundwater is treated with  $\text{Cl}_2$ . This water is probably also high in bromide, as the THM with the highest concentration is DBCM. For this water, 18% and 31% of the THMs were  $\text{CHCl}_3$  and BDCM, respectively. Cambridge was compared to three other low-DOC (0.3–0.7 mg/L), potentially high-bromide groundwaters (Midland Well Supply, Penetanguishene Well Supply, and Kitchener Well Supply [1990–1998]). For the latter three waters, 9%–10% and 20%–23% of the THMs were  $\text{CHCl}_3$  and BDCM, respectively. For Midland and Penetanguishene, the THM/HAA ratios were 0.86–1.7  $\mu\text{g}/\mu\text{g}$  (median = 1.3  $\mu\text{g}/\mu\text{g}$ ), with 52%–57% DXAAs (15%–20% DCAA) and 5%–9% TCAA. Because BDCM formation is greater than that of  $\text{CHCl}_3$  in these waters, it is consistent that DCAA was only a small portion of the DXAAs. Likewise, the formation of TCAA was relatively small, as there should be a shift in the speciation of the TXAAs to brominated species not measured by DWSP.

HAAs for Cambridge and Kitchener (1990–1998) were predicted, assuming a similar THM/HAA ratio and HAA speciation as that of Midland and Penetanguishene. Secondary predictions of BCAA (based on the relative speciation of BDCM) in Cambridge and Kitchener (1990–1998) were consistent with the predictions based on the relative HAA speciation of Midland and Penetanguishene.

## *Chapleau*

This WTP treats a high-DOC (10 mg/L), high-color (59 cu) surface water with conventional treatment (pH = 7.4, treated water color = 4.0 cu) with  $\text{NH}_2\text{Cl}$  only. As a result, THMs are typically quite low (median = 6.0  $\mu\text{g}/\text{L}$ ), except for 1997 (i.e., 52  $\mu\text{g}/\text{L}$ ) when it is likely that a combination of  $\text{Cl}_2$  and  $\text{NH}_2\text{Cl}$  were used. Chloramines were implemented between 1985 and 1990, so predictions are needed for historical DBPs with  $\text{Cl}_2$  only.

Twelve other WTPs in DWSP treat high-DOC, high-color surface waters with conventional treatment and  $\text{Cl}_2$  (Alexandria, Atikokan [1995–1998], Beardmore, Casselman, Ear Falls, Emo, Fort Francis, Kenora, Odessa, Plantagenet, Rainy River, and Smooth Rock Falls). Atikokan's current treatment (conventional with  $\text{Cl}_2$ ) was used rather than its historical treatment (direct filtration with  $\text{Cl}_2$ ), as its current treatment is comparable to Chapleau's historical treatment (conventional with  $\text{Cl}_2$ ). In addition, three WTPs (Ear Falls, Kenora, and Odessa) changed their treatment in the 1990s to lower THM levels <100  $\mu\text{g}/\text{L}$ , so their historical treatment and DBP data were used rather than their current treatment and DBP levels. Smooth Rock Falls WTP was excluded, as its raw water DOC (24 mg/L) was much higher than that of Chapleau (10 mg/L). Finally, DBP results for three WTPs (Casselman, Odessa, and Plantagenet) were excluded, as they formed much more BDCM (12%–20% of the THMs) than that of Chapleau (3% of the THMs when  $\text{Cl}_2/\text{NH}_2\text{Cl}$  was used in 1997). The remaining WTPs had comparable water quality to that of Chapleau (median values for relevant WTPs: DOC = 9.7 mg/L DOC, raw and treated water color = 41 and 3.2 cu, respectively, pH = 7.2) and THM speciation when using  $\text{Cl}_2$  (median values for relevant WTPs: 97.6%  $\text{CHCl}_3$ , 2.7% BDCM).

For the relevant WTPs, the median THM formation per unit DOC was 8.2  $\mu\text{g}/\text{mg}$ . It was assumed that Chapleau had a similar historical amount of THMs formed per unit DOC when  $\text{Cl}_2$  only was used. Note, for Chapleau, THM formation per unit DOC was 5.6 and 0.6  $\mu\text{g}/\text{mg}$  for  $\text{Cl}_2/\text{NH}_2\text{Cl}$  and  $\text{NH}_2\text{Cl}$  only, respectively. Thus, it was predicted that historical THM formation at



Chapleau was 83 µg/L, which is consistent with the median THM formation for the relevant WTPs (i.e., 78 µg/L). It was then assumed that 97.6% of the THMs was CHCl<sub>3</sub> and 2.7% was BDCM, similar to the speciation of the relevant WTPs.

Next, predictions for HAAs were developed. For the relevant WTPs, the median THM/HAA ratio was 1.5 µg/µg, with 32% DXAAs (31% DCAA) and 64% TCAA. For high-color waters, TCAA is greater than DCAA. Also, for waters in which almost all of the THMs are CHCl<sub>3</sub>, almost all of the DXAAs should be DCAA. When Chapleau used Cl<sub>2</sub>/NH<sub>2</sub>Cl in 1997, the HAA/THM ratio was 2.0 µg/µg, with 35% DXAAs (34% DCAA) and 62% TCAA. This HAA speciation is comparable to that of the other relevant WTPs; however, the THM/HAA ratio was somewhat higher for Chapleau. Thus, it was decided to use the HAA ratios for Chapleau for 1997 to predict the historical Chapleau HAA data, as this small difference may reflect site-specific differences at Chapleau. It was predicted that historical HAAs at Chapleau were 42 µg/L (with 14 µg/L DCAA, 26 µg/L TCAA, and 0.5 µg/L BCAA). This is similar to the median HAA levels for the other relevant WTPs (i.e., 49 µg/L, with 15 µg/L DCAA, 29 µg/L TCAA, and 0.5 µg/L BCAA). A secondary prediction of BCAA (based on the relative speciation of BDCM) was 0.4 µg/L, which is consistent with the prediction based on the relative HAA speciation.

### ***Clarence Creek Well Supply***

A moderate DOC (3.1 mg/L), colored (17 cu) groundwater is treated with Cl<sub>2</sub>/NH<sub>2</sub>Cl. In 1998, THMs in DWSP were 28 µg/L, whereas Will King obtained data for 1998–1999 with an average THM of 52 µg/L. Thamesville Well Supply also treats a moderate DOC (3.0 mg/L), colored (17 cu) groundwater with Cl<sub>2</sub> only and produced 32 µg/L of THMs. These limited data suggest that pre-Cl<sub>2</sub> at Clarence Creek is able to form as much or more THMs than Cl<sub>2</sub> only at Thamesville. Thus, the 1998–1999 average THM level of 52 µg/L was used for the historical THMs with Cl<sub>2</sub> only. Note: Clarence Creek has used Cl<sub>2</sub> since 1981; no records are available prior to this date. The epidemiology study data includes residences from this community from 1953 to 1958. However, there is no information to know if Cl<sub>2</sub> was used or not during that time period.

Likewise, the 1998–1999 average HAA level of 34 µg/L was used for the historical HAAs with Cl<sub>2</sub> only. Finally, it was assumed that historical THM and HAA speciation were similar to current speciation.

Note: The Thamesville Well Supply treats wells adjacent to the Thames River. It is likely that this well supply is under the influence of surface water. Groundwaters under the influence of surface waters often have moderate levels of DOC (and color) for groundwaters and, thus, produce relatively high levels of DBPs for groundwaters.

### ***Elmira Well Supply***

This treats a low-DOC (0.9 mg/L), low-color (1.2 cu) groundwater with Cl<sub>2</sub>. In 1992–1994—while treating the North Aquifer—14 µg/L of THMs were produced, with 52% and 30% as CHCl<sub>3</sub> and BDCM, respectively. The South Aquifer was taken out of service in 1989. In 1988, THMs were similar to that measured in the 1990s (13 µg/L, with 30% each of CHCl<sub>3</sub> and BDCM). The average THM speciation in this well supply was 41% and 30% as CHCl<sub>3</sub> and BDCM, respectively. Three other well supplies (Port Perry, Kitchener [1987–1989], and Stouffville) also treat low-DOC (0.7–0.9 mg/L), low-color (1.0–2.8 cu) groundwaters with Cl<sub>2</sub>. The latter well supplies produced 7.4–19 µg/L THMs, with 39%–43% and 26%–29% as CHCl<sub>3</sub>

and BDCM, respectively. Although  $\text{CHCl}_3$  is the THM produced in the highest concentration in each of these well supplies, the sum of the brominated THMs is equal to or greater than the concentration of  $\text{CHCl}_3$ .

Port Perry was the only one with HAA data: THM/HAA ratio of 1.95  $\mu\text{g}/\mu\text{g}$ , with 61% DXAAs (27% DCAA) and 27% TCAA. The relative speciation of BCAA and DCAA is consistent with that of BDCM and  $\text{CHCl}_3$ . The THM/HAA ratio and HAA speciation for Port Perry was used for the other well supplies in this subgroup.

Note: Kitchener Well Supply in 1987–1989 had 39% and 26% of the THMs as  $\text{CHCl}_3$  and BDCM, respectively, whereas in 1990–1991 it only had 9% and 20% of the THMs as  $\text{CHCl}_3$  and BDCM, respectively. Without additional information on why the speciation varied between these two time periods, historical predictions for this well supply (when  $\text{Cl}_2$  was used) were based on an average set of values between these two distributions.

### ***Metro Toronto***

Of four WTPs (F.J. Horgan, Island, R.C. Harris, and R.L. Clark), two (F.J. Horgan and R.L. Clark) originally used  $\text{ClO}_2$  only (generated on-site from the addition of chlorine and sodium chlorite) for disinfection. Although pure  $\text{ClO}_2$  produces little to no THMs, old  $\text{ClO}_2$  generators were notoriously inefficient and produced  $\text{ClO}_2$  with significant amounts of free chlorine present. It was assumed that DBP formation with  $\text{ClO}_2$  (due to the presence of excess free  $\text{Cl}_2$ ) was 25% of that formed with  $\text{Cl}_2$  only.

### ***Midland Well Supply***

Historically, this supply treated surface water from the Georgian Bay—supplemented by wells—with  $\text{Cl}_2$ . Two WTPs (Collingwood and Owen Sound [R.H. Neath]) also treat water from Georgian Bay with  $\text{Cl}_2$ . The latter two plants produced 14–24 and 8–17  $\mu\text{g}/\text{L}$  of THMs and HAAs, respectively. It was assumed that Midland produced a similar level of DBPs historically (the average values of the two other WTPs are used here). The latter prediction was then adjusted with a blend prediction based on the supplemental use of well water.

### ***Oakville***

This WTP treats water from Lake Ontario with  $\text{Cl}_2/\text{NH}_2\text{Cl}$  and produces 15  $\mu\text{g}/\text{L}$  of THMs. It used  $\text{Cl}_2$  (and no other treatment) from 1958 to 1962 and  $\text{Cl}_2/\text{ClO}_2$  (plus coagulation) from 1962 to 1988; chloramination began in 1988. As part of the analysis, 11 WTPs were examined that treat water from Lake Ontario with  $\text{Cl}_2$  (and conventional treatment or direction filtration).

The median, 75th, and 90th percentile THM formation for the latter WTPs were 20, 24, and 26  $\mu\text{g}/\text{L}$ , respectively. It was assumed that Oakville's historical DBP formation with  $\text{Cl}_2$  only (and no other treatment) was the same as the 90th percentile DBP formation for the 11 Lake Ontario WTPs. Because Oakville used no physical treatment from 1958 to 1962, its DBP formation should correspond to a higher percentile for the 11 WTPs. With the use of  $\text{ClO}_2$  and coagulation, it was assumed its DBP formation was the same as the median DBP formation for the 11 WTPs.

## ***Ottawa***

Two WSSs (Britannia and Lemieux Island) treat moderate-DOC (~6 mg/L), high-color (~30 cu) water from the Ottawa River with conventional treatment and  $\text{Cl}_2/\text{NH}_2\text{Cl}$ . Three WTPs (Hawkesbury, Pembroke, and Rockland) also treat water from the Ottawa River. The Ottawa WSSs and two of the other WTPs (Pembroke and Rockland) have treated water pH ~8, whereas Hawkesbury has a treated water pH of 7.6. Thus, Pembroke and Rockland were used for developing HAA predictions for the Ottawa WSSs. These two WTPs have THM/HAA ratios of 1.3–1.4  $\mu\text{g}/\mu\text{g}$ , with 47%–54% DXAAs (42%–51% DCAA) and 40%–49% TCAA. These HAA relationships were used to predict historical HAAs at the Ottawa WSSs.

## ***Perth and Smith Falls***

These two WTPs treat moderate-DOC (6.1–6.5 mg/L), high-color (22–23 cu) surface waters with conventional treatment (pH = 7.0–7.1) with  $\text{ClO}_2/\text{Cl}_2$ . Their current levels of THMs are 60 and 41  $\mu\text{g}/\text{L}$ , respectively. Historical predictions are needed for the use of  $\text{Cl}_2$  only. Six WTPs (Belleville, Deseronto, Huntsville, Peterborough, Picton, and Trenton) treat moderate-DOC (median = 5.4 mg/L), high-color (median = 16 cu) water with conventional treatment (median pH = 7.0) with  $\text{Cl}_2$  only and produce moderate amounts of THMs (median = 90  $\mu\text{g}/\text{L}$ ). It was assumed that the historical THM formation for Perth and Smith Falls with  $\text{Cl}_2$  only was the same as the median THMs for the latter subgroup of WTPs.

For the six WTPs, the median THM/HAA ratio was 1.1  $\mu\text{g}/\mu\text{g}$ , with 37% DXAAs (35% DCAA) and 62% TCAA. It was assumed that the historical HAA relationships for Perth and Smith Falls with  $\text{Cl}_2$  only were the same as for the latter subgroup of WTPs.

## ***Prescott***

This WTP treats water from the St. Lawrence River, which is low in DOC (2.0 mg/L) and color (3.0 cu). Since 1987, direct filtration with  $\text{ClO}_2/\text{Cl}_2$  has been used. Historically, it used  $\text{Cl}_2$  with no physical treatment. Currently, it produces 20 and 13  $\mu\text{g}/\text{L}$  of THMs and HAAs, respectively. Four WTPs (Brockville, Charlottenburg, Cornwall, and Morrisburg) treat water from the St. Lawrence River (median DOC = 2.1 mg/L, median color = 2.6 cu) with  $\text{Cl}_2$ . Two of these WTPs use conventional treatment, one uses direct filtration, and one has partial treatment. These WTPs produce 19–21  $\mu\text{g}/\text{L}$  THMs (median = 21  $\mu\text{g}/\text{L}$ ). It was assumed that Prescott had a historical THM level of 21  $\mu\text{g}/\text{L}$ , where an upgrade in treatment had very little impact on THM formation based on the other WTPs. The other four WTPs produce 8–17  $\mu\text{g}/\text{L}$  HAAs (median = 12  $\mu\text{g}/\text{L}$ ). The pH of the WTPs were similar (7.4–7.8, median = 7.6). The highest HAA formation was at the WTP with partial treatment, whereas the lowest HAA formation was at a WTP with conventional treatment. It was assumed that the historical HAAs were the same as the WTP with partial treatment (17  $\mu\text{g}/\text{L}$ ), as Prescott had no physical treatment in the past.

## ***Sault Ste. Marie***

This WTP disinfects a low-DOC (1.0 mg/L), low-color (3.9 cu) well water at pH 7.8 and produce 5.3  $\mu\text{g}/\text{L}$  THMs (72% and 21%  $\text{CHCl}_3$  and BDCM, respectively). Two well supplies (Blezzard Valley and Simcoe) add  $\text{Cl}_2$  to low-DOC (0.6–1.0 mg/L), low-color (1.5–4.2 cu) wells



at pH 7.4–7.5 and produce 4.5–6.0 µg/L THMs (53%–91% and 9%–30% CHCl<sub>3</sub> and BDCM, respectively). All of these wells produce primarily CHCl<sub>3</sub>. The median percentages (72% and 19% CHCl<sub>3</sub> and BDCM, respectively) for the latter two wells is similar to that of Sault Ste. Marie. The latter two wells had THM/HAA ratios of 0.7–1.0 µg/µg (median = 0.9 µg/µg), with 56%–81% DXAAs (median = 68%), 49%–75% DCAA (median = 62%), and 10%–31% TCAA (median = 20%). It was assumed that Sault Ste. Marie had HAA relationships similar to that of the median values for the other two wells.

Sault Ste. Marie WTP treats water from Lake Superior (DOC = 1.4 mg/L, color = 1.8 cu) with direct filtration and NH<sub>2</sub>Cl only and produces 5.8 and 3.1 µg/L THMs and HAAs, respectively. Two WTPs (Red Rock and Thunder Bay [Bare Point]) treat water from Lake Superior (DOC = 4.5 and 1.6 mg/L, respectively; color = 12 and 3.0 cu, respectively) with conventional treatment and direct filtration, respectively, with Cl<sub>2</sub>. The DOC and color at Red Rock is much higher than that of the other two WTPs. Thunder Bay (Bare Point) produces 22 and 23 µg/L of THMs and HAAs, respectively. It was assumed that historical DBP formation at Sault Ste. Marie was similar to that of Thunder Bay (Bare Point), as the DOC and color of the raw water were similar.

### ***Sudbury (Wanapitei)***

This WSS treats a moderate-DOC (4.6 mg/L), high-color (22 cu) surface water with conventional treatment (pH = 8.2) with ClO<sub>2</sub>/Cl<sub>2</sub> and produces 57 and 33 µg/L of THMs and HAAs, respectively. Perth WTP treats a moderate-DOC (6.1 mg/L), high-color (23 cu) surface water with conventional treatment (pH = 7.0) with ClO<sub>2</sub>/Cl<sub>2</sub> and produces 60 and 45 µg/L of THMs and HAAs, respectively. A major difference between these two plants is the treated water pH (8.2 versus 7.0).

It was predicted that historical THMs for Perth with Cl<sub>2</sub> only was 90 µg/L, which represents a 33% reduction in THMs with the switch to predisinfection with ClO<sub>2</sub>. It was assumed that a similar switch at Sudbury (Wanapitei) had a similar reduction in THMs and that historical THMs were 84 µg/L.

With current treatment, Sudbury (Wanapitei) has a THM/HAA ratio of 1.7 µg/µg, with 66% and 29% DXAAs and TCAA, respectively, whereas Perth has a THM/HAA ratio of 1.4 µg/µg, with 39% and 56% DXAAs and TCAA, respectively. These differences in HAA relationships are consistent with their differences in pH: at the lower pH, Perth has a lower THM/HAA ratio, and TCAA formation is greater than that of the DXAAs. Therefore, the current HAA relationships for Sudbury (Wanapitei) were used to predict historical HAAs at this WSS.

### ***Thunder Bay (Loch Lomond)***

This WTP treats a moderate-DOC (5.0 mg/L), moderate-color (13 cu) surface water with Cl<sub>2</sub> and no physical treatment. THMs of 119 and 85 µg/L were produced in 1990–1992 and 1993–1998, respectively. Thunder Bay was able to lower its THM formation somewhat in order to comply with the 100-µg/L guideline. Currently, it produces 80 µg/L of HAAs, with a THM/HAA ratio of 1.0 µg/µg, and 45% and 52% DXAAs and TCAA, respectively. As there was no major change in treatment at this WTP, it was assumed that the historical HAA relationships were similar to the current ones.

## ***Wallaceburg***

This WTP treats water from the St. Clair River (DOC = 1.5 mg/L, color = 1.4 cu) with conventional treatment (pH = 7.1) and  $\text{ClO}_2/\text{Cl}_2$  and produces 19 and 11  $\mu\text{g/L}$  of THMs and HAAs, respectively. Two WTPs (Sarnia [Lambton Area] and Walpole Island) treat water from the St. Clair River (DOC = 1.5 mg/L, color = 0.7–0.8 cu) with direct filtration or conventional treatment (pH = 7.5–7.7) with  $\text{Cl}_2$  and produce 20–21 and 5–10  $\mu\text{g/L}$  of THMs and HAAs, respectively. It was assumed that historical THM formation at Wallaceburg was the same as the average value for the other two WTPs (i.e., 20  $\mu\text{g/L}$ ), which implied that the addition of  $\text{ClO}_2$  had little impact on THM formation for this low-DOC, low-color water. HAA formation at the other two WTPs were relatively low, especially at Walpole Island. The other two WTPs had somewhat higher pH levels than Wallaceburg. As there appeared to be no major change in THM formation at this WTP, it was assumed that the historic HAA relationships at Wallaceburg were similar to the current ones.

## **Tertiary Predictions**

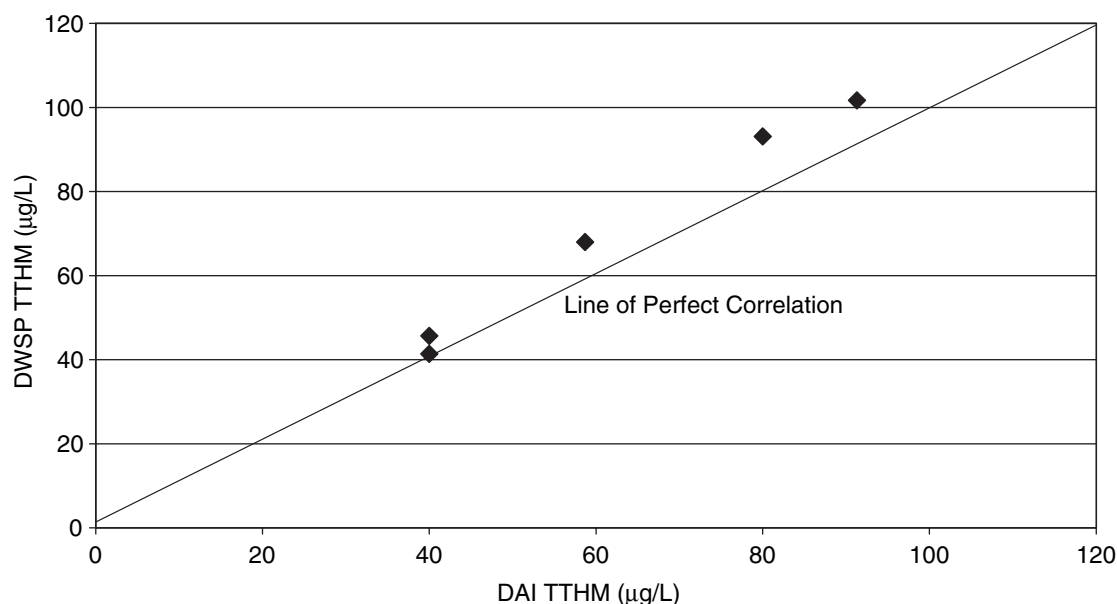
Tertiary predictions were made for selected surface water systems in Ontario that are not in the DWSP. The predictions are summarized in an Excel spreadsheet in appendix 6.3 and are discussed below according to groupings.

### ***Deep River, Petawawa, and Arnprior***

Deep River and Petawawa WTPs treat water from the Ottawa River, whereas Arnprior WTP treats water from the Madawaska River very close to its break from the Ottawa River. All three WTPs use coagulation. Arnprior and Petawawa use chlorine only, whereas Deep River uses prechlorination/postchlorine dioxide (where chlorine and sodium chlorite are added to form the chlorine dioxide). Deep River and Petawawa are close to the Pembroke WTP, which is in DWSP. Four other WTPs in DWSP treat water from the Ottawa River (Hawkesbury WTP, Ottawa [Britannia] WSS, Ottawa (Lemieux Island) WSS, and Rockland WTP).

Historically (1977–1982), THMs were measured in Ontario with a direct aqueous injection (DAI) of unquenched samples. For the five WTPs in DWSP that treat water from the Ottawa River, the DAI data were compared to DWSP distribution system THMs from 1990 to 1998 (Figure 6.8). The results were comparable. The Ottawa River is relatively high in DOC and color. Although the chlorine residuals were not quenched in the DAI method, it is possible that the chlorine residuals in these samples were relatively low, where the DOC and color of the Ottawa River consumed most of the chlorine sedimentation.

Arnprior had an average DAI THMs of 99.5  $\mu\text{g/L}$ , which was quite similar to the DAI THMs of Ottawa (Britannia) WSS (i.e., 91.3  $\mu\text{g/L}$ ). Thus, it was assumed that the THMs and HAAs of Arnprior were the same as that of Ottawa (Britannia) WSS. There were no DAI THM data for Deep River and Petawawa. Thus, these two WTPs were assigned THM and HAA values that equaled the median values for the five WTPs in DWSP that treat Ottawa River water with coagulation and chlorination. Although Deep River uses postchlorine dioxide disinfection, it was assumed that most of the THMs and DBPs formed during prechlorination of this high-DOC, high-color water. Note: The median THM and HAA values were essentially the same values as that of Pembroke, which is the closest WTP.



**Figure 6.8 Relationship between DAI/unquenched TTHM and DWSP distribution system TTHM for five WTPs on the Ottawa River**

### ***Beamsville***

This WTP treats water from Lake Ontario with coagulation and chlorination. It is geographically between the Grimsby WTP, which treats water from Lake Ontario, and St. Catharines (de Cew) WSS, which treats water from the Welland Canal. Eleven WTPs in DWSP treat water from Lake Ontario with coagulation and chlorination (Ajax WTP, Bowmanville WTP, Burlington WSS, Cobourg WTP, Grimsby WTP, Kingston WTP, Napanee WTP, Oshawa WSS, Port Hope WTP, South Peel [Lorne Park] WSS, and Whitby WTP). It was assumed that the THMs and HAAs at Beamsville were equal to the median values for these 11 WTPs. Note: The median values were quite similar to the levels at Grimsby. Although the levels of THMs at St. Catharines (de Cew) WSS were similar, the concentrations of the HAAs were different than that of the Lake Ontario WTPs.

### ***Gananoque***

This WTP treats water from the St. Lawrence River with chlorine and no physical treatment. It is near the Kingston WTP, which treats water from Lake Ontario, and Prescott WTP and Brockville WTP, which also treat water from the St. Lawrence River. In addition, three other WTPs in DWSP treat water from the St. Lawrence River (Charlottenburg, Cornwall, and Morrisburg). All use chlorine, with varying amounts of physical treatment. Morrisburg only does partial treatment, whereas Prescott historically did not use physical treatment. In this low-DOC, low-color water, the use of physical treatment appeared to have no significant impact on THM formation, but did result in somewhat lower HAA formation. Thus, predictions for Gananoque were based on Morrisburg and the historical data for Prescott. Note: The DBP levels at Kingston, which

has conventional treatment, were similar to that of the St. Lawrence River WTPs with conventional treatment or direct filtration.

### ***Kincardine WTP and Petrolia WSS***

Although both treat water from Lake Huron with chlorine, Petrolia uses coagulation, whereas Kincardine does not. Goderich WTP and Port Elgin WTP are along the same shore of Lake Huron as Kincardine. Sarnia (Lambton Area) WTP, which treats water from the St. Clair River, is close to Petrolia. In addition, London (Lake Huron) WSS and Southampton WTP also treat water from Lake Huron. All of the WTPs in DWSP that treat water from Lake Huron use conventional treatment. In this low-DOC, low-color water, there is probably a relatively low amount of DBP precursors that are removed by the conventional treatment. It was predicted that Kincardine and Petrolia have the same concentration of THMs and HAAs as the median concentrations of the four WTPs in DWSP that treat water from Lake Huron. The amount of DBPs at Sarnia was somewhat similar to the levels at the Lake Huron WTPs.

### ***Meaford, Parry Sound, and Wiarton***

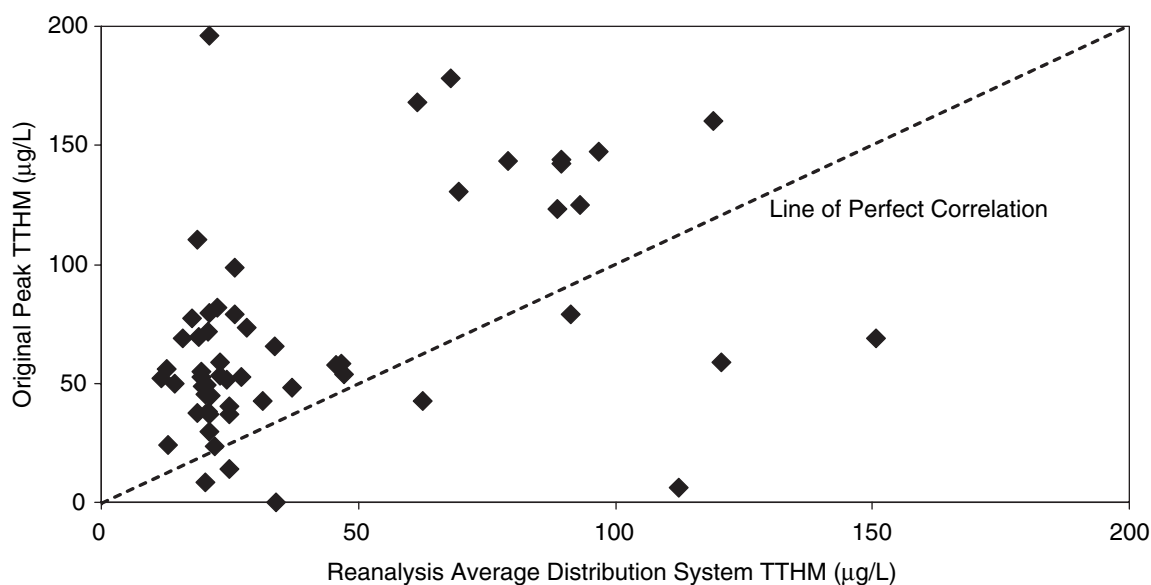
These WTPs all treat water from the Georgian Bay or bays off of the Georgian Bay. They all use chlorination but no physical treatment. Two WTPs in DWSP treat water from the Georgian Bay with chlorine (Collingwood WTP, Owen Sound [R.H. Neath] WTP). Collingwood does not use physical treatment, whereas Owen Sound has direct filtration. Even though Owen Sound uses physical treatment, it produces more DBPs than Collingwood (24 versus 14 µg/L of THMs and 17 versus 8 µg/L of HAAs). Historically, DAI THMs were also higher for Owen Sound than Collingwood (46 versus 23 µg/L). In this case, unquenched DAI THMs were higher than the DWSP THMs. In this low-DOC, low-color water, chlorine demand is probably low, such that THM formation probably continued in the unquenched samples collected historically. Meaford and Parry Sound had DAI THMs of 16–24 µg/L, which is similar to that of Collingwood. Thus, it was assumed that Meaford and Parry Sound had similar levels of DBPs to that of Collingwood. Because Wiarton had no DAI THM data, it was assumed that its DBP levels were equal to the median concentrations of Owen Sound and Collingwood.

### ***Sutton WSS***

Likewise, this was assumed to have the same concentration of DBPs as Beaverton WTP, both of which treat water from Lake Simcoe. Note: Although Orillia WTP treats water from Lake Couchiching, which is part of Lake Simcoe, that WTP treats a blend of surface and groundwater and has a different amount of DOC and color than that of Lake Simcoe.

### ***Thorold Main Water System***

This was assumed to have the same concentration of DBPs as the median levels of St. Catharines and Welland WSSs, which all treat water from the Welland Canal.



**Figure 6.9 Comparison of predicted TTHM for DWSP surface waters: Case study average distribution system versus King and Marrett peak values**

Most of the WTPs in this tertiary analysis treat water from low-DOC, low-color surface waters, and low levels of DBPs should be formed, as evidenced by other WTPs in DWSP treating the same water. Alternatively, WTPs on the Ottawa River were predicted to produce moderate levels of DBPs, consistent with that of other plants in DWSP treating the same water.

### COMPARISON OF CASE STUDY AND KING AND MARRETT PREDICTIONS FOR ONTARIO

A comparison was made of the original King and Marrett (1996) predictions and the new set of case study predictions. The compilation of the two datasets permitted a comparison of 184 sets of results, which were segmented into four groups for comparisons:

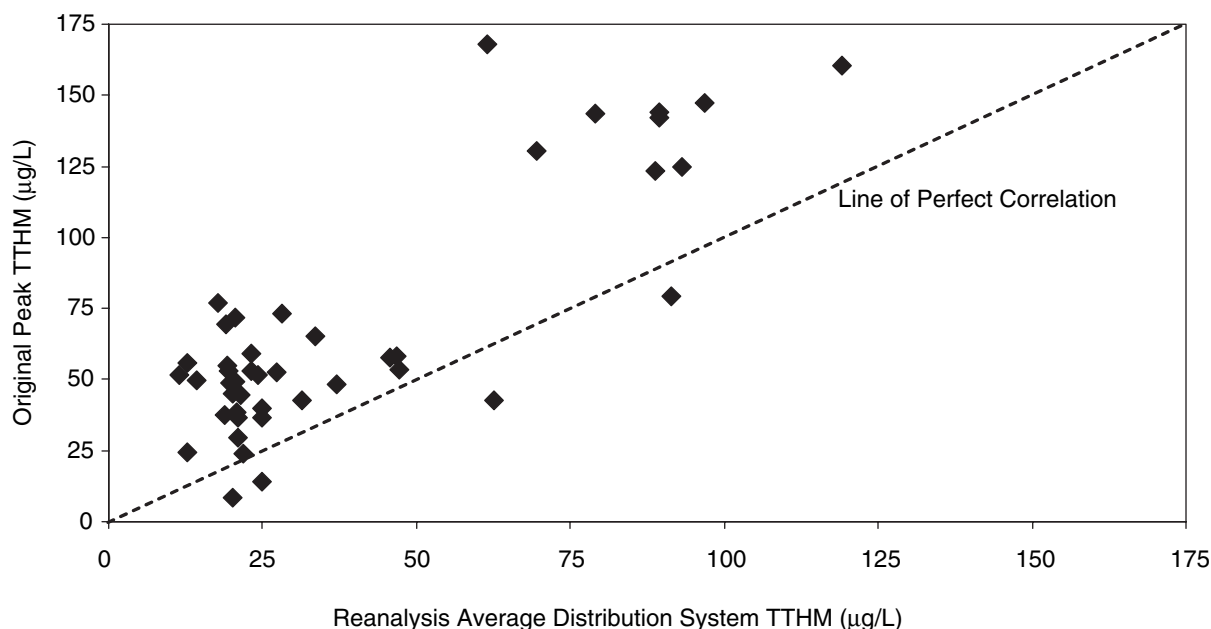
- Surface waters in DWSP
- Groundwaters in DWSP
- Other surface waters
- Other groundwaters

Figure 6.9 shows a comparison of predicted TTHM for DWSP surface waters. One would expect that the King and Marrett (1996) peak values would tend to be somewhat higher than case study average distribution system values. However, some sets of values differed significantly.

The DWSP surface waters in which the difference between the two predicted values was much greater than 2:1 or much lower than 1:2 were next examined. Also included were historical (1977–1982) TTHM data from a DAI of unquenched samples. The following observations were made:

- The case study prediction for Bracebridge (112 µg/L) was based on a combination of surface water (Lake Muskoka) supplemented by spring water. King and Marrett's (1996) prediction for this system (6.1 µg/L) appears to be based on the spring water only.
- The Brantford WTP treats water from the Grand River (average DOC = 5.2 mg/L, color = 20 cu). In 1987–1992, the plant's average TTHM with chlorine/chloramines was 135 µg/L. The case study approach predicted that its historical TTHM with chlorine only was 151 µg/L. King and Marrett's (1996) prediction for this system was 69 µg/L. That value is low when compared to current treatment with postchloramination. In addition, its historical DAI TTHM was 197 µg/L (an unquenched sample), which is consistent with the case study predicted value. In 1987–1992, Brantford's average prechlorine dose was 7.5 mg/L.
- The Brockville, Cornwall, Morrisburg, and Prescott WTPs treat water from the St. Lawrence River (average DOC for each WTP ranged from 1.9 to 2.1 mg/L, color = 2.0–2.9 cu). Currently, the average THMs for the three WTPs with chlorine only were 19–23 µg/L. At Prescott, the current average TTHM with chlorine dioxide and chlorine was 23 µg/L, and it had a higher result for the year 1990 (35 µg/L). These results are consistent with a low-DOC, low-color surface water. Alternatively, King and Marrett's (1996) predictions for these systems were 80–196 µg/L, results that are too high for such a low-DOC surface water. In addition, their historical DAI TTHM (where available) were 15–35 µg/L (unquenched samples), which are consistent with the case study predicted values.
- The Mitchells Bay WTP treats water from Lake St. Clair (average DOC = 3.0 mg/L, color = 9.2 cu, postchlorine dose = 2.8 mg/L). There were no distribution system TTHM data for this system in DWSP in the 1990s. Based on a secondary prediction, the case study approach predicted that the plant's historical TTHM would be 34 µg/L. King and Marrett's (1996) prediction was 0 µg/L. The plant's historical DAI TTHM was 82 µg/L (an unquenched sample).
- The Burlington WSS, Grimsby WTP, Oakville WTP, Port Hope WTP, South Peel (Lakeview and Lorne Park) WSS, and Whitby WTP treat water from Lake Ontario (average DOC for each WTP ranged from 1.7 to 2.1 mg/L, color = 1.6–2.4 cu). Currently, the average TTHM for these WTPs with chlorine only were 13–27 µg/L. South Peel (Lorne Park) had a higher result for the year 1990 (42 µg/L). Alternatively, King and Marrett's (1996) predictions for these systems were 50–79 µg/L. Their historical DAI TTHM (where available) were 30–33 µg/L (unquenched samples). For some of these systems, King and Marrett's predictions appeared to be too high (especially when compared to the DAI unquenched results), and in other cases the predictions are possible (when compared to the Lorne Park 1990 results).
- The Odessa WTP treats water from the Millhaven Creek (average DOC = 7.2 mg/L, color = 22 cu). In 1990–1993, its average TTHM with chlorine only was 126 µg/L. King and Marrett's prediction for this system was 59 µg/L. That value is low when compared to treatment in the early 1990s. In addition, Odessa's historical DAI TTHM was 187 µg/L (an unquenched sample), which is consistent with the case study predicted value (121 µg/L). In 1990–1993, its average pre- and postchlorine doses were 1.9 and 3.5 mg/L, respectively.
- The Pembroke WTP treats water from the Ottawa River (average DOC = 6.5 mg/L, color = 37 cu). In 1992–1995, its average TTHM with chlorine only was 68 µg/L. King

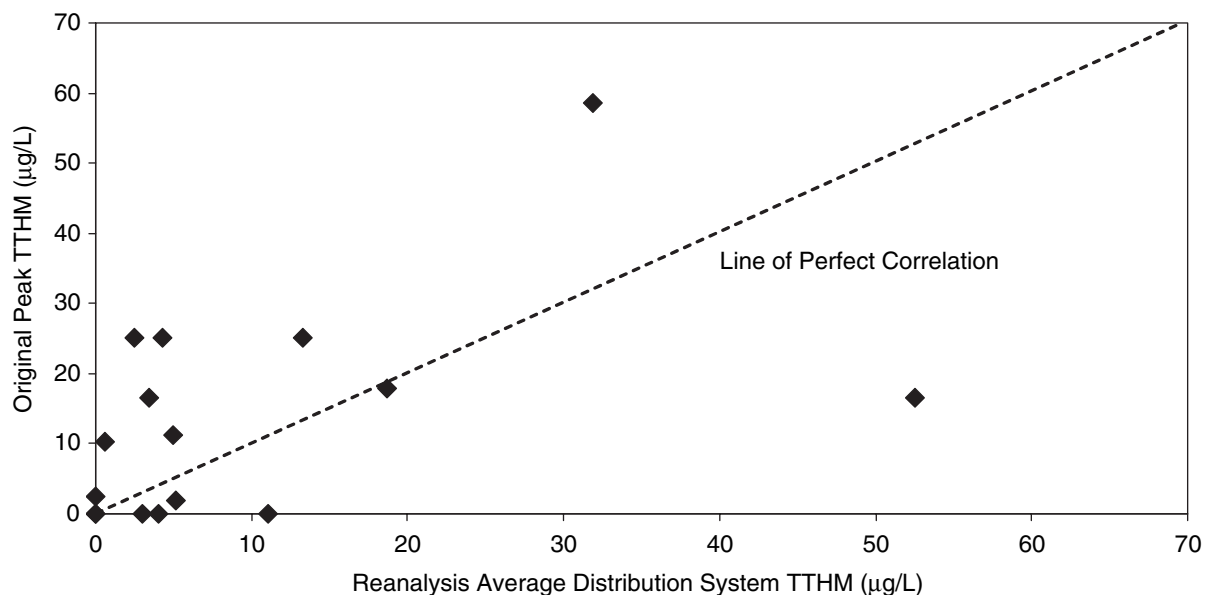




**Figure 6.10 Comparison of predicted TTHM for DWSP surface waters: Case study average distribution system versus King and Marrett peak values**

and Marrett's prediction for this system was 178  $\mu\text{g/L}$ . That value is high when compared to treatment in the early 1990s. In addition, Pembroke's historical DAI TTHM was 59  $\mu\text{g/L}$  (an unquenched sample), which is consistent with the case study predicted value (68  $\mu\text{g/L}$ ). In 1992–1995, its average pre- and postchlorine doses were 2.3 and 0.6  $\text{mg/L}$ , respectively.

- The Amherstburg and Windsor WSSs treat water from the Detroit River (average DOC for each WSS ranged from 1.8 to 1.9  $\text{mg/L}$ , color = 1.8–3.1 cu). Currently, the average THMs for the two WSSs with chlorine only were 19–26  $\mu\text{g/L}$ . Windsor had a higher result for 1990 (39  $\mu\text{g/L}$ ), which is consistent with a low-DOC, low-color surface water. Alternatively, King and Marrett's predictions for these systems were 70–100  $\mu\text{g/L}$ . Those results appear to be too high for such a low-DOC surface water. In addition, their historical DAI TTHM were 43–58  $\mu\text{g/L}$  (unquenched samples), which are more consistent with the case study values than with the King and Marrett peak values.
- The other WTPs, when similarly examined, could possibly have such differences between annual average results and peak values. For those systems for which it was believed likely that the King and Marrett peak values are too high or too low, they were deleted, and replotted for a comparison of King and Marrett predicted values versus the case study predicted results (Figure 6.10). In this figure, all of the King and Marrett predicted peak values are either approximately equal to or greater than the case study distribution system annual average results. In general, there are two clusters of results, those with very high levels of THMs and those with significantly less THMs.
- It should be noted that quite a number of plants in DWSP were not in the list of systems in the King and Marrett list of 184.



**Figure 6.11 Comparison of predicted TTHM for DWSP groundwaters: Case study average distribution system versus King and Marrett peak values**

Figure 6.11 shows a comparison of predicted TTHM for DWSP groundwaters. One would expect that the King and Marrett peak values would tend to be somewhat higher than the case study average distribution system values. However, some sets of values differ significantly.

Next, the DWSP groundwaters in which the difference between the two predicted values was much greater than 2:1 or much lower than 1:2 were examined. There were no DAI TTHM data for these samples. Some of the observations were

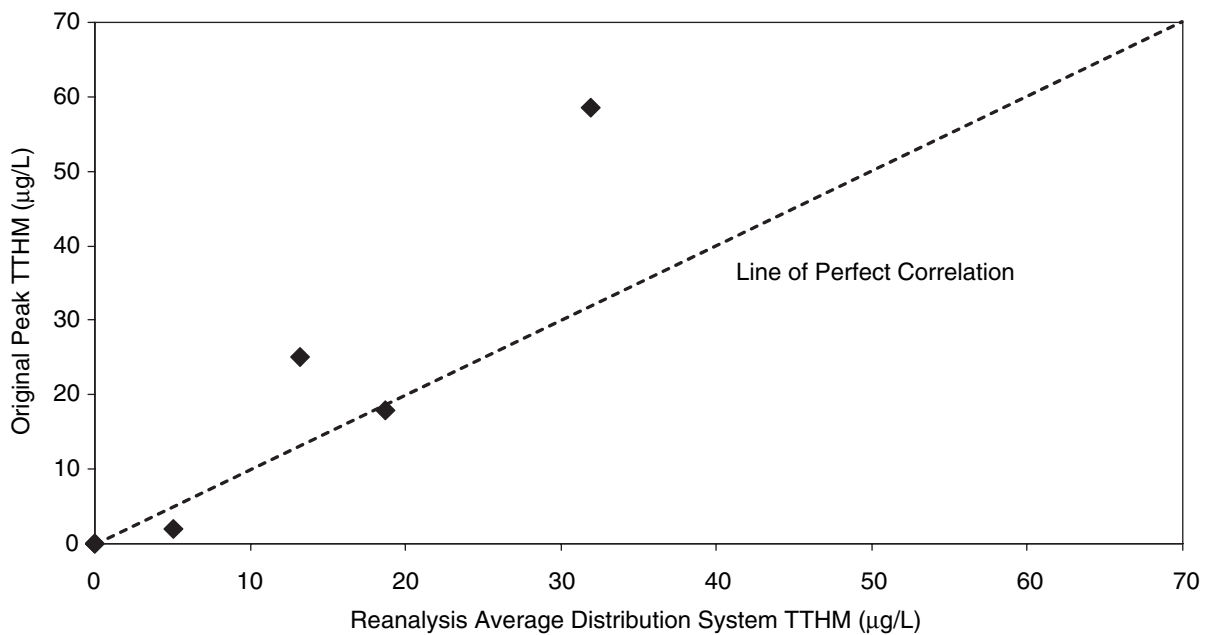
- The Clarence Creek Well Supply currently treats a high-color (17 cu) groundwater (DOC = 3.1 mg/L) with chlorine and chloramines. In 1998, it produced (on average) 28 µg/L of TTHM. In 1997, chlorine only was used, but there was no TTHM data. Clarence Creek has no chemical treatment to remove the color, other than to add chlorine, which should form THMs. The case study approach predicted that its TTHM with chlorine only would be 52 µg/L. King and Marrett's prediction was 17 µg/L, which is what is produced by many low-DOC, low-color groundwaters in DWSP.
- The Dorchester Well Supply, Orangeville Well Supply, and Waterloo Well Supply currently produce levels of TTHM that range from 3 to 12 µg/L. King and Marrett's prediction for these three systems was 0 µg/L.
- The Ingersoll Well Supply and Norwich Well Supply treat low-DOC (0.9 µg/L), low-color (2–7 cu) groundwaters with high chlorine doses (11–21 mg/L). Currently, their average distribution system TTHM are low (3 µg/L). Ingersoll aerates the water and Norwich does iron sequestration. One reason that some groundwater systems aerate their water is that they contain a high amount of hydrogen sulfide. It takes a significant amount of chlorine to oxidize hydrogen sulfide. Also, reduced iron has a chlorine



- demand. Norwich has a problem with iron, which may include reduced iron (many groundwaters are high in reduced species (e.g., ammonia, hydrogen sulfide, reduced iron) that can have a high demand for chlorine. King and Marrett's predictions for these two systems was 17–25 µg/L, which is much higher than their current average distribution system values. It is suspected that they were predicted to have high values of THMs because of their high chlorine doses. However, for these waters, most of the chlorine is probably consumed to meet the chlorine demand from inorganic chemicals in the water and is not due to reactions with the low amount of DOC and color in the water.
- The Fergus Well Supply treats a low-DOC (0.6 mg/L), low-color (3.1 cu) groundwater that is probably high in bromide. Of the 16 µg/L of TTHM currently produced, 12 µg/L were bromoform. Even though the DOC and color is low, if there is high bromide, there can be a significant formation of brominated THMs. (Also note that bromoform weighs approximately twice that of chloroform on a molar basis.) King and Marrett's prediction for this system was 2.5 µg/L; their model is probably not able to accurately predict THM formation for a high-bromide groundwater.
  - There were other DWSP groundwater systems with low current THM formation for which King and Marrett had relatively high THM predictions. Again, it is suspected that these waters had higher chlorine demand from inorganic chemicals than from the DOC and color, which were low. For those systems for which it was believed that the King and Marrett peak values are too high or too low, they were deleted, and a comparison of the King and Marrett predicted values versus the case study predicted results were replotted ([Figure 6.12](#)). In this figure, all of King and Marrett predicted peak values are either approximately equal to or greater than the case study distribution system annual average results.
  - It was noted that quite a number of groundwaters in DWSP were not in the list of systems in the King and Marrett list of 184.

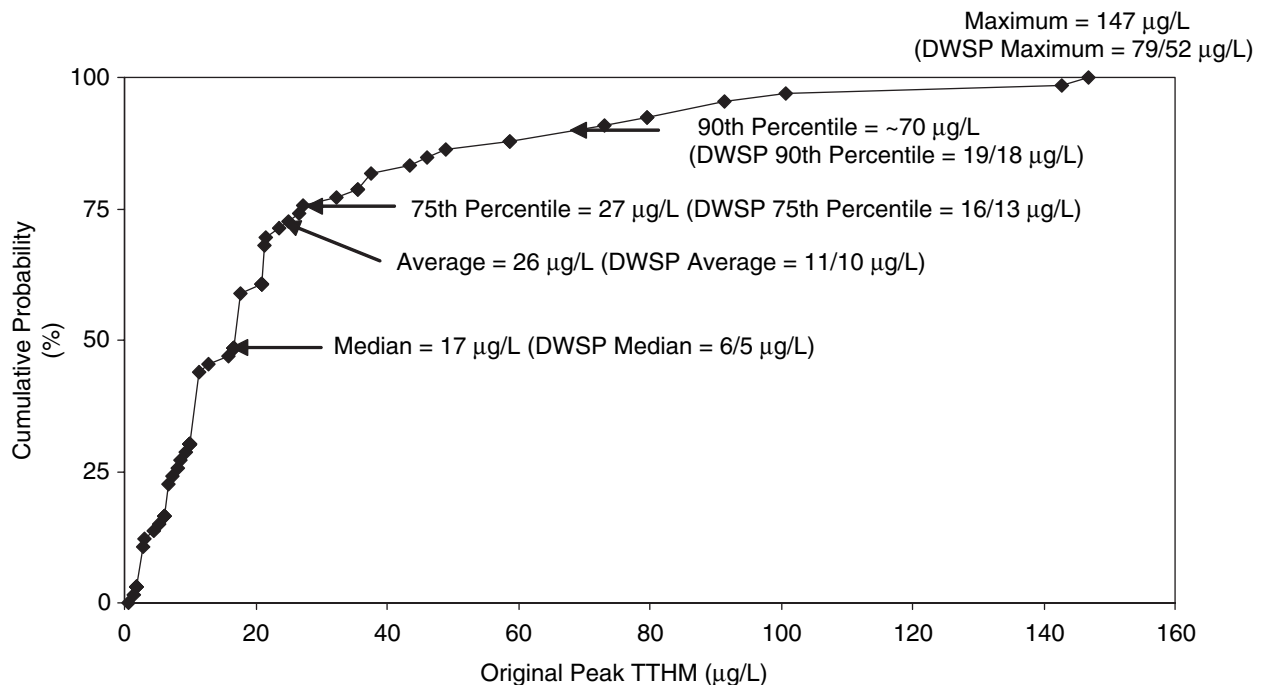
Next, other groundwaters in the list of 184 were examined. [Figure 6.13](#) shows the CPD of the THMs for the other groundwaters that chlorinate (i.e., THMs >0 µg/L). Data are provided in this figure for the CPD for the DWSP groundwaters, both the statistics from the measured values in the DWSP database and the statistics for the portion of DWSP groundwaters for which case study predictions were developed. The CPD of THMs for the other groundwaters is significantly different than that of DWSP. In DWSP, the median value for the measured results was 6 µg/L, whereas the median for the other groundwaters was 17 µg/L. The 75th and 90th percentile values in DWSP were 16 and 19 µg/L, respectively, whereas the 75th and 90th percentile values for the other groundwaters were 27 and ~70 µg/L, respectively. In DWSP, the maximum value (for a colored groundwater) was 79 µg/L, whereas the maximum value for the other groundwaters was 147 µg/L. Based on the DWSP groundwaters, it appears that many of the King and Marrett predictions for other groundwaters were too high, as chlorine dose is not a good surrogate parameter for THM formation in groundwaters.

Note that DAI TTHM data exist for three of these other groundwaters: L'Original Well Supply (DAI = not detected, King and Marrett prediction = 36 µg/L), Paris Well Supply (DAI = 17 µg/L, King and Marrett prediction = 13 µg/L), and Vankleek Hill Well Supply (DAI = 5 µg/L,



Note: Results deleted that appeared too high or too low.

**Figure 6.12 Comparison of predicted TTHM for DWSP groundwaters: Case study average distribution system versus King and Marrett peak values**



Note: DWSP groundwater statistics (statistics for measured value/statistic for predicted value) provided for comparison.

**Figure 6.13 Cumulative probability distribution of predicted TTHM for non-DWSP groundwaters: King and Marrett peak values**

King and Marrett prediction = 36 µg/L). These limited DAI results are consistent with the DWSP data: most groundwaters produce low amounts of THMs.

Finally, the other surface waters in the list of 184 were examined. There were DAI data for three of these systems:

- Arnprior (Madawaska River): DAI = 100 µg/L, case study prediction = 102 µg/L, King and Marrett prediction = 100 µg/L
- Meaford WTP (south end of Georgian Bay): DAI = 16 µg/L, case study prediction = 14 µg/L, King and Marrett prediction = 90 µg/L
- Parry Sound WTP (Georgian Bay): DAI = 24 µg/L, case study prediction = 14 µg/L, King and Marrett prediction = 60 µg/L

For the Madawaska River, all of the results were similar. For the two WTPs using water from the Georgian Bay, which is a low-DOC surface water, King and Marrett predictions appeared to be too high, unless the plants' August peak values are much greater than the annual average or DAI results. Likewise, King and Marrett's prediction (117 µg/L) for Wiarton WTP (which treats water off of the Georgian Bay) was much higher than the case study prediction (19 µg/L). Other surface water plants had results that were similar or quite different, as was observed with the DWSP systems. For example, King and Marrett's prediction (92 µg/L) for the Gananoque WTP (which treats water from the St. Lawrence River) was much higher than the case study prediction (20 µg/L), which was similar to what was observed with DWSP WTPs treating water from the St. Lawrence River. Alternatively, the case study prediction for Beamsville (20 µg/L) (which treats water from Lake Ontario) is consistent with King and Marrett's peak prediction of 31 µg/L.



## CHAPTER 7

### CASE STUDY APPROACH TO MODELING DBP EXPOSURE IN IOWA

#### DBP PREDICTIONS

##### Surface Waters and Alluvial Groundwaters on the Iowa Hit List

This initial effort focuses on surface water and alluvial groundwater systems; deep groundwater systems will be examined later in this chapter.

##### *TTHM Formation and Speciation*

Many of the utilities in Iowa have changed their treatment and disinfection processes over time. Moreover, many of these systems first used physical treatment only (e.g., coagulation, sedimentation, and/or filtration) and did not add chlorination ( $\text{Cl}_2$  or hypochlorite [ $\text{OCl}^-$ ]) until sometime in the 1900s (Table 7.1). For each utility, prior to the implementation of chlorination, DBP occurrence is set at zero.

Table 7.2 shows a summary of the impact of changes in disinfection practices on TTHM control in Iowa. Moving the point of chlorination at Davenport resulted in a 13% reduction in TTHM formation. The implementation of  $\text{ClO}_2$  resulted in a median reduction in TTHM formation of 37%. Implementation of postchloramination resulted in a median reduction in TTHM formation of 32%, whereas elimination of free chlorine and conversion to chloramines at Centerville resulted in a 94% reduction in TTHM formation.

Table 7.3 shows the relationship of finished water TOC to TTHM formation at chlorination plants in Iowa. Finished water TOC was examined because raw water TOC data are not available for many of the Iowa source waters. However, the 1987 database in Cantor et al. (1998) does have finished water TOC data for most of the Iowa plants in the May, June, or July time period in 1987.

The plants in Table 7.3 were segmented into several groups. Initially, the following three groups were created:

- Coagulation plants with pre- and postchlorination (Figure 7.1)
- Coagulation or softening plants with intermediate and/or postchlorination, with finished water pH levels between 7.5 and 8.7 (Figure 7.2)
- Softening plants with intermediate and/or postchlorination, with finished water pH levels between 9.4 and 10.0 (Figure 7.3)

It should be noted that Spirit Lake, Oscaloosa, Des Moines at pH 9.1, and Spencer were not used in the model development. Spirit Lake produced a relatively low level of THMs (53  $\mu\text{g/L}$ ) for a water with a moderate finished water TOC concentration (4.8 mg/L). Oscaloosa produced a relatively high level of THMs (21  $\mu\text{g/L}$ ) for a water with a low finished water TOC concentration (0.9 mg/L). Oscaloosa produced a relatively high amount of brominated THMs (13 of the 21  $\mu\text{g/L}$ ), so this water is probably relatively high in bromide, which would explain the relatively high TTHM formation. Des Moines at pH 9.1 and Spencer had finished water pH levels that were intermediate compared to that of the other softening plants, so they did not quite fit in with the other groups.

**Table 7.1**  
**Implementation of chlorination**

Utility	Source water	%	Date	Treatment	Disinfection begun		
					SW	GW/A*	GW/C†
Iowa American (Davenport)	Mississippi River		1901	Coagulation, sedimentation, filtration			
			1910	Coagulation, sedimentation, filtration, OCl <sup>-</sup>	1910		
Ottumwa-3	Des Moines River	100	1900–1983				
			1904	Disinfection, sedimentation, filtration	1904		
Keokuk	Mississippi River		1900	Sedimentation, filtration			
			1917	Coagulation or softening, sedimentation, filtration, disinfection	1917		
Creston-2	Green Valley Lake to Summit Lake		1894	Sedimentation, filtration			
			1936	Coagulation, sedimentation, disinfection, filtration	1936		
Centerville-3	Well	100	1893–1912				
Centerville-2	Two-stage impounding reservoir	100	1912				
			1924	Alkali/coagulation/lime, sedimentation, sedimentation, filtration, Cl <sub>2</sub>	1924		
Clarinda	Nodaway River		1936	Coagulation, sedimentation, filtration, stabilization			
Clarinda	Nodaway River		1955	Coagulation, Cl <sub>2</sub> , sedimentation, sedimentation+alkali, sedimentation, filtration, Cl <sub>2</sub>	1955		
Osceola	West Lake		1933	Coagulation, sedimentation, disinfection, filtration, stabilization	1933‡		
Chariton-4	Well	100	1906–1916				
Chariton-1	Lake Ellis	100	1916–1942	Coagulation, sedimentation, Cl <sub>2</sub> , alkali, filtration, Cl <sub>2</sub>	1916		
Spirit Lake	Spirit Lake		1949	Coagulation/Na aluminate, sedimentation, lime, acid, filtration, Cl <sub>2</sub>	1949§		
Des Moines-1	Well	100	1871–1950				
		100	1911	OCl <sup>-</sup>			1911
Council Bluffs-2	Missouri River	100	1906–1957				
			1911	Coagulation/softening, sedimentation, OCl <sup>-</sup>	1911		
Burlington-1	Mississippi River	100	1878–1985				
			1900	Filtration			
			1912	Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	1912		
Iowa City-2	Iowa River	100	1880–1962				
			1922	Coagulation/lime, sedimentation, filtration, Cl <sub>2</sub>	1922		

(Continued)

Table 7.1 (Continued)

Utility	Source water	%	Date	Treatment	Disinfection begun		
					SW	GW/A <sup>*</sup>	GW/C <sup>†</sup>
Fort-Madison-2	Mississippi River	100	1918	Coagulation, sedimentation, filtration			
			1925	Coagulation, sedimentation, filtration, disinfection	1925		
Cedar Rapids-2	Cedar River	100	1900	Coagulation or softening, filtration			
			1910	Coagulation or softening, filtration, disinfection	1910		
Muscatine-2	Mississippi River		1887–1903	None			
Muscatine-1	Wells/alluvium		1903–1996				
			1952	Cl <sub>2</sub>		1952	
Newton	Wells/alluvium		1883–1996				
			1948	Cl <sub>2</sub>		1948	
Spencer-2	Wells/alluvium ( <i>d</i> = 24–45)**	100	1900–1959				
		100	1935	Cl <sub>2</sub>		1935	
Spencer-3	Wells/Camord ( <i>d</i> = 970)	19	1959–71				
			1959	Aeration/oxidation, filtration			>1971
Boone-2	Well/Camord ( <i>d</i> = 2,914–3,000)	100	1890–1911				
Boone-1	Well/alluvium ( <i>d</i> = 32–67)	100	1911	Cl <sub>2</sub>		1911	
Oscalosa-3	Wells/alluvium ( <i>d</i> = 40)	100	1906–1927				
Oscalosa-1	Wells/alluvium ( <i>d</i> = 40–59)	100	1927	Aeration/oxidation/filtration			
			1933	Lime/coagulation/lime, sedimentation, sedimentation, filtration, Cl <sub>2</sub> /CO <sub>2</sub>		1933	
Summary				Minimum	1904	1911	
				Median	1917	1935	
				Maximum	1955	1952	>1971

Note: Blank cells indicate information was not available.

\* GW/A: alluvial groundwater systems.

† GW/C: deep groundwater systems.

‡ Operations begun.

§ Treatment/disinfection practices for Spirit Lake are not known before 1949.

\*\* *d* = depth, in feet.

**Table 7.2**  
**Impact of changes in disinfection practices on TTHM control**

Utility	Source water	%	Date	Treatment	Finished pH	CHCl <sub>3</sub> (µg/L)	BDCM (µg/L)	DBCM (µg/L)	CHBr <sub>3</sub> (µg/L)	TTHM (µg/L)	Reduction in TTHM				
											Move point	→	Cl <sub>2</sub> →	Cl <sub>2</sub> →	Post-1979 (%)
											of Cl <sub>2</sub> (%)	ClO <sub>2</sub> (%)	Cl <sub>2</sub> /NH <sub>3</sub> * (%)	NH <sub>2</sub> Cl (%)	
Iowa American (Davenport)	Mississippi River		1974	Cl <sub>2</sub> , coagulation, alkali, sedimentation, filtration, Cl <sub>2</sub> , NH <sub>3</sub>		75.7	6.0	0.7	<3	81.9					
			1981	Coagulation, sedimentations/clarification, Cl <sub>2</sub> , filtration, Cl <sub>2</sub> /NH <sub>3</sub> , alkali	7.3	64.4	6.4	0.7	0.5	71.1	13				
			1995	ClO <sub>2</sub> , coagulation, sedimentation/clarification, Cl <sub>2</sub> , filtration, Cl <sub>2</sub> /NH <sub>3</sub> , alkali	7.4	54.6	11.1	1.7	0.5	61.4		14			
Creston-1	12-Mile Reservoir		1986	Coagulation, up-flow clarifier, Cl <sub>2</sub> , filtration, Cl <sub>2</sub> /alkali		111.0	33.0	5.0	0	149.0					
			5/20/87	Coagulation, up-flow clarifier, Cl <sub>2</sub> , filtration, NH <sub>3</sub> , Cl <sub>2</sub> /alkali	7.7	85.0	23	2.0	0	110.0			26		
			1988	Coagulation, ClO <sub>2</sub> , up-flow clarifier, Cl <sub>2</sub> , filtration, NH <sub>3</sub> , Cl <sub>2</sub> /alkali		37.2	16.1	4.8	0.2	57.9		47			
Centerville-2	Two-stage impounding reservoir	100	1962	Up-flow clarifier/coagulation/lime, Cl <sub>2</sub> , sedimentation, Cl <sub>2</sub> , filtration	7.7	98.5	27.0	9.5	1.0	136.0					
			1990	ClO <sub>2</sub> , up-flow clarifier/coagulation/lime, Cl <sub>2</sub> , sedimentation, Cl <sub>2</sub> , filtration		67.1	15.8	2.6	0.5	85.5		37			
			1995	ClO <sub>2</sub> , NH <sub>2</sub> Cl, filtration	8.3	4.7	0.8	0.4	0.4	5.4				94	
Clarinda	Nodaway River		1955	Coagulation, Cl <sub>2</sub> , sedimentation, sedimentation+alkali, sedimentation, filtration, Cl <sub>2</sub>	7.4	48	19	4	NF	71.0					
			1981	Coagulation, Cl <sub>2</sub> , lime, sedimentation, filtration, Cl <sub>2</sub>	7.6	35.6	11.0	1.8	0.4	48.4					32
Osceola	West Lake		1985	Coagulation, alkali, Cl <sub>2</sub> , lime, up-flow clarifier, filtration, Cl <sub>2</sub>	7.3	78.0	25.3	6.0	0.3	126.4					
			1990	ClO <sub>2</sub> , coagulation, lime, up-flow clarifier, filtration, Cl <sub>2</sub>	8.3	60.4	14.7	2.6	0.5	77.8		38			

(Continued)



Table 7.2 (Continued)

Utility	Source water	%	Date	Treatment	Finished pH	CHCl <sub>3</sub> (µg/L)	BDCM (µg/L)	DBCM (µg/L)	CHBr <sub>3</sub> (µg/L)	TTHM (µg/L)	Reduction in TTHM				
											Move point of Cl <sub>2</sub> (%)	→ ClO <sub>2</sub> (%)	Cl <sub>2</sub> → Cl <sub>2</sub> /NH <sub>3</sub> * (%)	Cl <sub>2</sub> → NH <sub>2</sub> Cl (%)	Post-1979 (%)
Chariton-1	Lake Ellis	35	1988–1989												
Chariton-2	Lake Morris	35	1988–1989												
Chariton-3	Red Haw Lake	30	1988–1989												
Chariton			1988	Sedimentation/alkali/Cl <sub>2</sub> /coagulation, filtration, Cl <sub>2</sub>						122.0					
Chariton-1,-2,-3			1989	ClO <sub>2</sub> , ClO <sub>2</sub> , coagulation, up-flow clarifier, Cl <sub>2</sub> , filtration, Cl <sub>2</sub> , alkali	8.2					87.3		28			
Council Bluffs-2	Missouri River	94	1957–1996												
Council Bluffs-1	Well/alluvium	6	1957–1996												
Council Bluffs			1974	Softening, sedimentation, coagulation/lime/Na aluminate, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	9.5	29.4	9.5	4.6	1.0	45.6					
Council Bluffs			1993	Softening, sedimentation, coagulation/lime/Na aluminate, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, Cl <sub>2</sub> /NH <sub>3</sub>	9.1	20.8	5.8	2.2	0.7	28.8			37		
All plants	Minimum											14	26		
	Median										13	37	32	94	32
	Maximum											47	37		

Note: Blank cells indicate information was not available.

\* Cl<sub>2</sub>/NH<sub>3</sub>: Postchloramination.

**Table 7.3**  
**Relationship of finished water TOC to THM formation at chlorination plants**

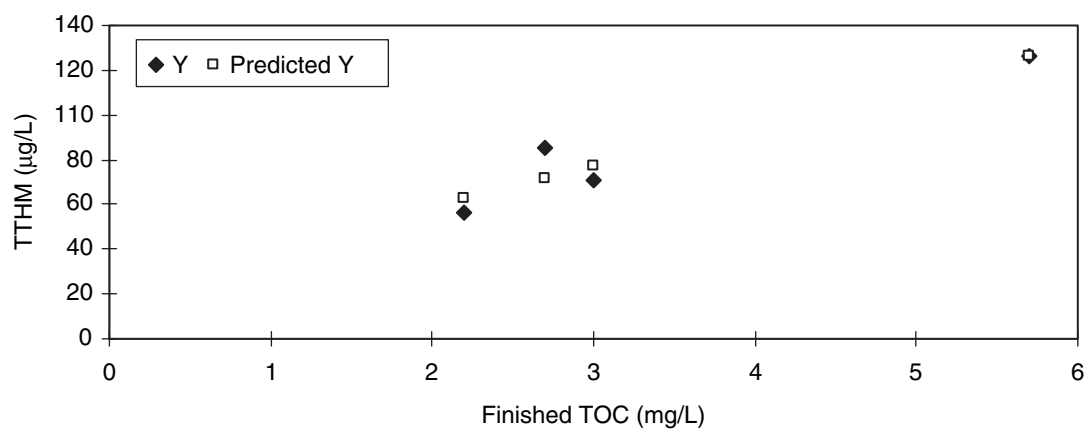
Utility	Treatment	Finished pH	Predicted TTHM (µg/L)							
			TOC (mg/L)		Measured TTHM (µg/L)	Coagulation with pre-Cl <sub>2</sub>	Coagulation/ softening	Softening	Coagulation2/ softening	Coagulation
			Raw	Finished			(pH 7–8)	(pH 9–10)	(pH 7–8)	
	Cl <sub>2</sub> only	7–8								
Muscatine-1	Cl <sub>2</sub>	7.6		1	6					
Boone	Greensand, Cl <sub>2</sub>	7.9		2.4	30					
	Coagulation with pre-Cl <sub>2</sub> and post-Cl <sub>2</sub> ; or coagulation with intermediate and/or post-Cl <sub>2</sub>	7–8								
Clarinda	Coagulation, Cl <sub>2</sub> , sedimentation, sedimentation+alkali, sedimentation, filtration, Cl <sub>2</sub>	7.4	3.5	3.0	71	77.2			74.4	80.5
Clarinda	Coagulation, Cl <sub>2</sub> , lime, sedimentation, filtration, Cl <sub>2</sub>	7.5		2.2	56	62.6			47.6	56.0
Osceola	Coagulation, alkali, Cl <sub>2</sub> , lime, up-flow clarifier, filtration, Cl <sub>2</sub>	7.4		5.7	126	126.5			165.1	163.2
Winterset	Cl <sub>2</sub> , coagulation/lime/up-flow clarifier, clarification, filtration, Cl <sub>2</sub>	7.3		2.7	85	71.7			64.4	71.3
Creston-1	Coagulation, up-flow clarifier, Cl <sub>2</sub> , filtration, Cl <sub>2</sub> /alkali			4.1	149.0		118.8		111.4	114.2
Centerville	Up-flow clarifier/coagulation/lime, Cl <sub>2</sub> , sedimentation, Cl <sub>2</sub> , filtration	7.8		5.1	186		162.8		144.9	144.8
Chariton-1,-2	Sedimentation/alkali/coagulation, filtration, Cl <sub>2</sub>	7.9		5.3	135		171.6		151.6	150.9
Iowa City	Coagulation/lime/sedimentation/solids contactor, Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	7.5		2.4	35		44.0		54.3	62.1
Burlington-1,-2	Coagulation, sedimentation, lime, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	8.7		1.8	21		17.6		34.2	
Fort Madison	Coagulation, lime/solids contactor, up-flow clarifier, Cl <sub>2</sub> , CO <sub>2</sub> , filtration, Cl <sub>2</sub>	7.8		2.9	55		66.0		71.1	

(Continued)

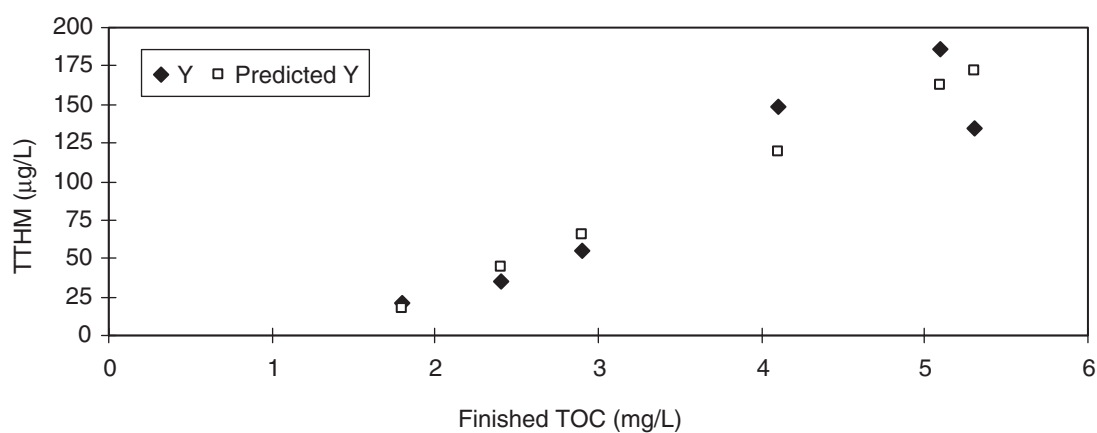
**Table 7.3 (Continued)**

Utility	Treatment	Finished pH	Predicted TTHM (µg/L)							
			TOC (mg/L)		Measured TTHM (µg/L)	Coagulation with pre-Cl <sub>2</sub>	Coagulation/softening (pH 7–8)	Softening (pH 9–10)	Coagulation2/softening (pH 7–8)	Coagulation
			Raw	Finished						
Spirit Lake	Coagulation/clarification, clarification/coagulation, up-flow clarifier/lime/Na aluminate, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	8.3		4.8	53					
Oscalooosa	Coagulation/lime, sedimentation, sedimentation, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	8.3		0.86	21					
	Softening with intermediate and/or post-Cl <sub>2</sub>	7.8–8.9								
	Softening with intermediate and/or post-Cl <sub>2</sub> :	9–10								
Des Moines	Coagulation, sedimentation, lime/coagulation/Na aluminate, sedimentation, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	9.1		1.3	7.9					
Spencer	Lime/solids contact/Na aluminate, CO <sub>2</sub> , sedimentation, filtration, Cl <sub>2</sub>	9.0		1.7	10					
Des Moines	Coagulation/Na aluminate, lime, sedimentation, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	9.6		1.6	36			28.3		
Des Moines	Coagulation/softening, sedimentation, Na aluminate/lime/coagulation/softening, CO <sub>2</sub> , filtration, ion exchange, Cl <sub>2</sub>	9.6	3.7	2	30.4			33.4		
Newton	Up-flow clarifier/Na aluminate/lime, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	9.4		0.95	17			20.1		
Council Bluffs	Softening, sedimentation, coagulation/lime/Na aluminate, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	10.0		2.1	33			34.6		

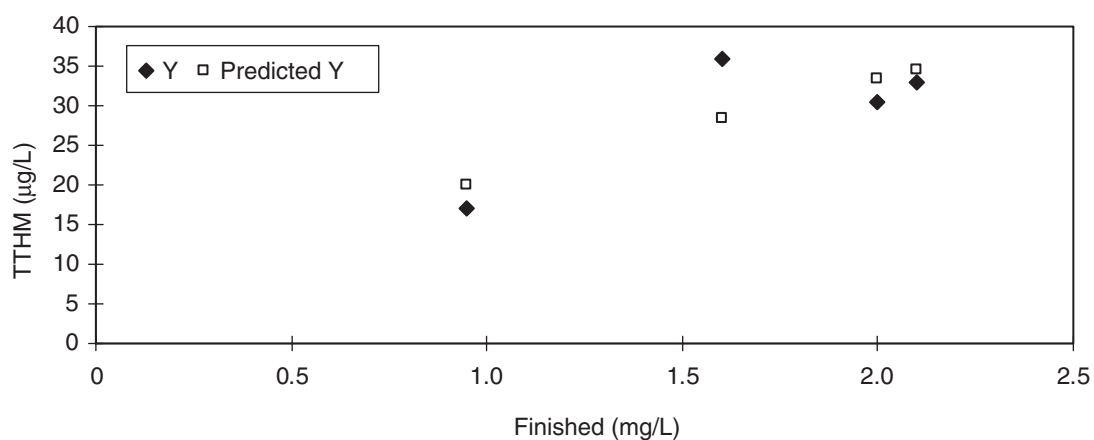
Note: Blank cells indicate information was not applicable.



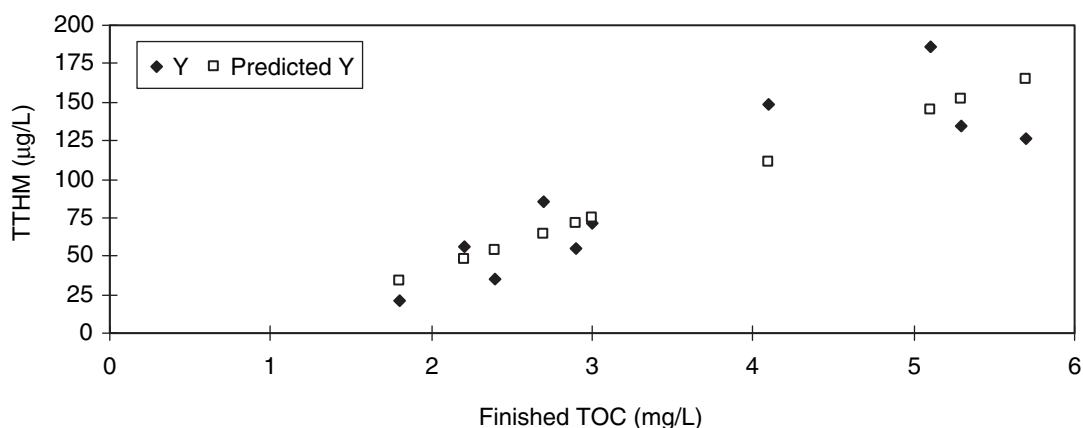
**Figure 7.1 TTHM as function of finished TOC: Coagulation plants with pre- and post-Cl<sub>2</sub>**



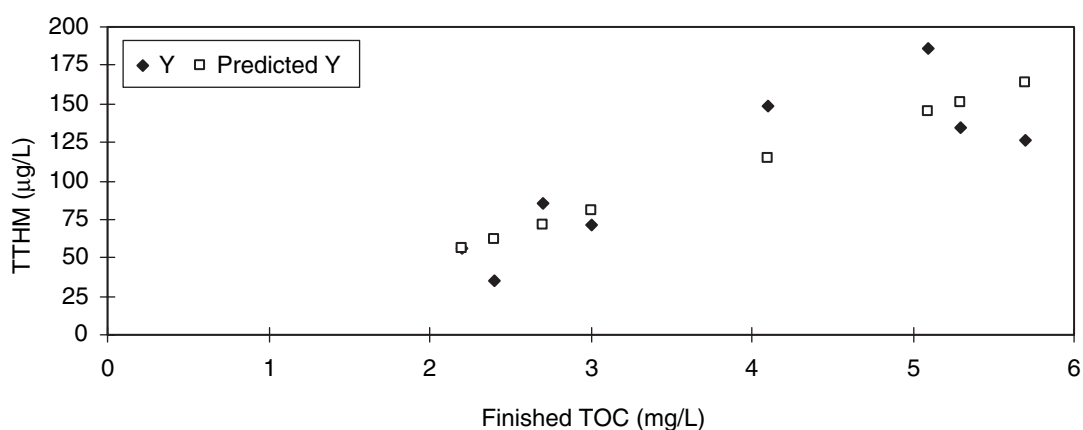
**Figure 7.2 TTHM as function of finished TOC: Coagulation or softening plants with intermediate and/or post-Cl<sub>2</sub>, pH 7.5–8.7**



**Figure 7.3 TTHM as function of finished TOC: Softening plants with intermediate and/or post-Cl<sub>2</sub>, pH 9.4–10.0**



**Figure 7.4 TTHM as function of finished TOC: Coagulation or softening plants with pre- or intermediate  $\text{Cl}_2$  and/or post-  $\text{Cl}_2$ , pH = 7.3–8.7**



**Figure 7.5 TTHM as function of finished TOC: Coagulation plant with pre- or intermediate  $\text{Cl}_2$  and/or post- $\text{Cl}_2$**

In theory, a plant with prechlorination should produce more DBPs than a plant with intermediate and/or postchlorination, as the former plant is adding chlorine before the DBP precursors can be removed. However, some of the coagulation plants with intermediate and/or postchlorination produced more THMs than the coagulation plants with pre- and postchlorination. It is possible that the latter plants were not adding a sufficient prechlorine dose to meet the chlorine demand of the water. Nevertheless, the creation of a central tendency model was evaluated for coagulation (and softening plants with pH levels <9) (Figures 7.4 and 7.5).

Table 7.3 shows a summary of the different models used to predict THM formation for the different Iowa plants. The second coagulation and softening mode (coagulation2), which was based on plants with pre- or intermediate chlorination and/or postchlorination, with pH levels of 7.3–8.7 (Figure 7.4), and the softening model, which was based on plants with pH levels of 9.4–10.0 (Figure 7.3) were selected. The predictive equation for the coagulation and softening plants with

pH <9 is  $TTHM = 33.56 * TOC - 26.24$  (Figure 7.4). The predictive equation for the softening plants with pH >9 is  $TTHM = 12.65 * TOC - 8.07$  (Figure 7.3).

Initially, the focus was on predicting TTHM for surface water plants. Predictions were based on the following:

- TTHM as a function of finished water TOC (Figures 7.3 and 7.4)
- Reductions in THM formation with changes in disinfection practices (Table 7.2)
- The same THM formation as other utilities treating water from the same watershed with similar treatment and disinfection practices

The secondary predictions are summarized in appendix 7.1. As an example, Davenport had a finished water TOC of 3.8 mg/L and a plant effluent TTHM of 101 µg/L in July 1987 (Cantor et al. 1998). The plant used coagulation with intermediate chlorination and postchloramination. The coagulation and softening model predicted 101 µg/L if Davenport was using chlorine only. The measured result with postchloramination was the same, suggesting that postammonia addition did not minimize THM formation in the plant, only in the distribution system.

From the larger database of monitoring data, Davenport produced an average of 71 µg/L of THMs in the 1980s and early 1990s with chlorine and chloramines. This represented an average of samples collected in the winter, spring, summer, and fall from the plant effluent and distribution system (at average and maximum detention times). For Davenport, the annual average THM value equaled 70% of the peak value measured in July 1987.

When Davenport used chlorine only, it was estimated that peak THM formation (e.g., in July)—using the coagulation and softening model—was 145 µg/L, based on an average finished water TOC value of 5.1 mg/L. (The average raw water TOC of Davenport was 6.6 mg/L.) Assuming that the annual average THM value equaled 70% of the peak value estimated for July, it was estimated that Davenport produced 102 µg/L of THMs (on an annual average basis) when using chlorine only.

As another example, Keokuk had a finished water TOC of 3.3 mg/L and a plant effluent TTHM of 18 µg/L in June 1987 (Cantor et al. 1998). The plant used softening with intermediate chlorination and postchloramination. The softening model predicted 50 µg/L if Keokuk was using chlorine only. The measured result with postchloramination was much lower, suggesting that postammonia addition minimized THM formation in the plant as well as in the distribution system. From the larger database of monitoring data, Keokuk produced an average of 43 µg/L of THMs in the 1980s and 1990s with chlorine and chloramines.

When Keokuk used chlorine only, initially (starting in 1917) it was likely a coagulation plant. (The database indicated coagulation or softening, but did not specify which.) It was assumed that Keokuk with coagulation and chlorine produced 102 µg/L of THMs based on the predictions cited for Davenport, as both utilities treat water from the Mississippi River.

In 1938, Keokuk definitely became a softening plant (finished water pH = 8.5), as well as adding postchloramination. Burlington, which also softens Mississippi River water with a finished water pH of 8.7, produced (on average) 90 µg/L of THMs with chlorine only. It was assumed that Keokuk would have produced a similar amount of THMs with softening and chlorine only. Then—based on a median THM reduction of 32% for going to postchloramination—it was estimated that Keokuk produced 61 µg/L of THMs with softening and chlorine and chloramines.

**Table 7.4**  
**TTHM predictions for some plant(s) treating Mississippi River water**

TOC/Treatment	Mississippi River	Plants using Mississippi River water			
Average raw TOC	6.6 mg/L (Davenport)				
Average finished TOC	5.1 mg/L (Davenport)				
Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	TTHM = 138 µg/L			Burlington (1912)	
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>	TTHM = 117 µg/L				Fort Madison (1946, 1966)
Coagulation/sedimentation, Cl <sub>2</sub>	TTHM = 102 µg/L	Davenport (1910, 1955)	Keokuk (1917)	Burlington (1961, 1963)	Fort Madison (1925)
Softening/sedimentation, Cl <sub>2</sub>	TTHM = 90 µg/L			Burlington (1967)	
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 82 µg/L	Davenport (1974)			
Coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 71 µg/L	Davenport (1967, 1981)		Burlington (1946)	
Cl <sub>2</sub> , softening/clarification, NH <sub>3</sub> , Cl <sub>2</sub>	TTHM = 61 µg/L		Keokuk (1938)		
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 61 µg/L	Davenport (1995)			
Softening/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 43 µg/L		Keokuk (1981)		

Note: Blank cells indicate information was not applicable.

Table 7.4 shows a summary of TTHM predictions for plants treating Mississippi River water. The highest TTHM formation (138 µg/L) was for Burlington in 1912, when it used chlorination and filtration (no coagulation). The second highest TTHM formation (117 µg/L) was for Fort Madison in 1946 and 1966, when coagulation was used with pre- and postchlorination. As prechlorination was eliminated and as alternative disinfectants (chlorine dioxide and/or chloramines) were introduced, it was predicted that THM formation was reduced as appropriate (Table 7.2). Note that, for a particular utility, changes in treatment and disinfection practices sometimes meant increasing THM formation rather than lowering it. For example, Davenport did not use prechlorination in 1967 and 1981 but did in 1974. Burlington used chloramines in 1946 but went back to chlorine only in 1961.

Table 7.5 shows a summary of TTHM predictions for some plants treating various river waters. In general, as the TOC (raw and/or finished) of the river decreased, it was predicted that plants on that river would produce less THMs.

**Table 7.5**  
**TTHM predictions for some plants treating various river waters**

TOC/Treatment	Source water					
	Mississippi River	Iowa River	Des Moines River	Missouri River	Nodaway River	Cedar River
Average raw TOC	6.6 mg/L (Davenport)	5.6 mg/L (Iowa City)	4.4 mg/L (Ottumwa)	4 mg/L (Council Bluffs)	3.5 mg/L (Clarinda)	DOC = 3.0 mg/L
Average finished TOC	5.1 mg/L (Davenport)	2.4 mg/L (Iowa City)	2.2 mg/L (Ottumwa)	3.5 mg/L (Council Bluffs)	2.6 mg/L (Clarinda)	
Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	TTHM = 138 µg/L					
Cl <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub>	TTHM = 117 µg/L	TTHM = 85 µg/L	TTHM = 61 µg/L		Avg. TTHM = 60 µg/L	
Coagulation/sedimentation, Cl <sub>2</sub>	TTHM = 102 µg/L	TTHM = 74 µg/L		TTHM = 64 µg/L		TTHM = 45 µg/L
Cl <sub>2</sub> , softening/sedimentation, Cl <sub>2</sub>				TTHM = 52 µg/L		
ClO <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub>		TTHM = 64 µg/L				
Softening/sedimentation, Cl <sub>2</sub>	TTHM = 90 µg/L			TTHM = 46 µg/L		
Cl <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 82 µg/L					
Coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 71 µg/L					
Cl <sub>2</sub> , softening/clarification, NH <sub>3</sub> , Cl <sub>2</sub>	TTHM = 61 µg/L		TTHM = 39 µg/L			TTHM = 35 µg/L
ClO <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 61 µg/L					
Softening/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 43 µg/L			TTHM = 29 µg/L		TTHM = 31 µg/L
NH <sub>3</sub> , softening/sedimentation, Cl <sub>2</sub>						TTHM = 3 µg/L

Note: Blank cells indicate information is not available.

Table 7.6 shows a summary of TTHM predictions for some plants treating various lakes. First note that most of these lakes had finished water TOC levels that were higher than many of the river water plants (Table 7.5). As a result, it was predicted (as well as measured) that many of these plants would produce relatively high levels (e.g., >100 µg/L) of THMs with chlorine only. As discussed above, Spirit Lake is anomalous. However, as part of this case study, Spirit Lake will include lower THM predictions, consistent with what was observed for this plant.



**Table 7.6**  
**TTHM predictions for some plants treating various lakes**

TOC/Treatment	Utility					
	Osceola	Chariton	Centerville	Spirit Lake	Creston	Fairfield
1987 Finished TOC	5.7 mg/L	5.3 mg/L	5.1 mg/L	4.8 mg/L	4.1 mg/L	3.5 mg/L
Cl <sub>2</sub> , softening/sedimentation, Cl <sub>2</sub>					TTHM = 82 µg/L	
Cl <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub>	TTHM = 126 µg/L	TTHM = 122 µg/L			TTHM = 76 µg/L	
Softening/sedimentation, Cl <sub>2</sub>			TTHM = 46 µg/L			
Coagulation/sedimentation, Cl <sub>2</sub>	TTHM = 126 µg/L	TTHM = 122 µg/L	TTHM = 136 µg/L	TTHM = 25 µg/L	TTHM = 149 µg/L	
ClO <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub>	TTHM = 78 µg/L	TTHM = 87 µg/L	TTHM = 86 µg/L			
Coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>				TTHM = 110 µg/L		
ClO <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>				TTHM = 58 µg/L		
ClO <sub>2</sub> , NH <sub>2</sub> Cl, filtration		TTHM = 5 µg/L				

Note: Blank cells indicate information was not applicable.

## Peak Versus Annual Average

In the previous section, models (equations) were developed to predict TTHM based on finished water TOC data based on samples collected by Cantor et al. (1998) in May, June, or July 1987. These months have warmer finished-water temperatures (median = 18.5°C for all utilities in the database; 21°C for surface or mixed surface and groundwater utilities). Previously, based on data from Davenport, it was assumed that annual average TTHM would be 70% of the peak TTHM values.

Table 7.7 shows the relationship of peak THM formation (based on the 1987 data from Cantor et al. 1998) to annual average THM formation for a number of utilities on the hit list. The median was 88%, with 25th to 75th percentile values of 67%–119%. One reason that the annual average value was greater than the peak value for some utilities is partly due to the fact that samples collected by Cantor et al. were plant effluents, whereas annual average values were based on a combination of plant effluent and distribution system samples. An examination of this relationship for just the utilities used in the development of the equations for THMs as a function of finished water TOC resulted in a median of 80% (and 25th to 75th percentile values of 60%–126%).

It is now assumed that annual average THM formation (in plant effluents and the distribution system) is 80% of the peak THM formation (in plant effluents sampled in May, June, or July), based on the relationship of THM formation to finished water TOC for samples collected by Cantor et al. Tables 7.8 through 7.10 show the revised predictions for various surface water systems.

**Table 7.7**  
**Relationship of peak THM formation (Cantor et al. 1998 data) to annual average THM formation**

Utility	Source water	%	Date	Treatment	Finished pH	Cantor et al. (1998) TTHM (µg/L)	Annual average TTHM (µg/L)	Annual average/ peak TTHM (%)	Coagulation/ softening models* (%)
Iowa American (Davenport)	Mississippi River		1981	Coagulation, sedimentation/clarification, Cl <sub>2</sub> , filtration, Cl <sub>2</sub> /NH <sub>3</sub> , alkali	7.3	101	71.1	70	
Keokuk	Mississippi River		1981	Coagulation, sedimentation, lime, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, NH <sub>3</sub>	9.0	18	43.0	239	
Centerville-2	Two-stage impounding reservoir	100	1962	Up-flow clarifier/coagulation/lime, Cl <sub>2</sub> , sedimentation, Cl <sub>2</sub> , filtration	7.7	186	136.0	73	73
Clarinda	Nodaway River		1981	Coagulation, Cl <sub>2</sub> , lime, sedimentation, filtration, Cl <sub>2</sub>	7.6	56	48.4	86	86
Osceola	West Lake		1985	Coagulation, alkali, Cl <sub>2</sub> , lime, up-flow clarifier, filtration, Cl <sub>2</sub>	7.3	126	126.4	100	
Chariton-1	Lake Ellis	50	1942–1988						
Chariton-2	Lake Morris	50	1942–1988						
Chariton-1,-2			1982	Sedimentation/alkali/coagulation, filtration, Cl <sub>2</sub>	7.0–8.0	135	122.0	90	90
Spirit Lake	Spirit Lake		1984	Coagulation/clarification, clarification/coagulation, up-flow clarifier/lime/Na aluminate, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	8.4	53	24.9	47	
Des Moines			1983	Coagulation/Na aluminate, lime, sedimentation, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	9.6	36	20.4	57	57
Des Moines-1	Well(s)	8–67	1983–1991						
Des Moines-2	Raccoon River, west low lift	20–52	1983–1991						
Des Moines-3	Des Moines River	12–40	1983–1991						
Council Bluffs-2	Missouri River	94	1957–1996						
Council Bluffs-1	Well(s)/alluvium	6	1957–1996						
Council Bluffs			1974	Softening, sedimentation, coagulation/lime/Na aluminate, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	9.5	33	45.6	138	138

(Continued)

Table 7.7 (Continued)

Utility	Source water	%	Date	Treatment	Finished pH	Cantor et al. (1998) TTHM (µg/L)	Annual average TTHM (µg/L)	Annual average/peak TTHM (%)	Coagulation/softening models* (%)
Burlington-1	Mississippi River	73–90	1985–1996						
Burlington-2	Well(s)/alluvium	10–27	1985–1996						
Burlington-1,-2			1985	Coagulation, sedimentation, lime, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	9.0	21	36.9	176	176
Iowa City-2	Iowa River		1973	Coagulation/lime/sedimentation/solids contactor, Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	7.8	35	73.8	211	211
Fort Madison-2	Mississippi River	40–80	1967–1996						
Fort Madison-1	Well(s)/alluvium	20–60	1967–1996						
Fort Madison-2			1979	Coagulation, lime/solids contactor, up-flow clarifier, Cl <sub>2</sub> , CO <sub>2</sub> , filtration, Cl <sub>2</sub>	8.2	55	19.3	35	35
Fairfield-5	Well/drift (depth = 140–248 ft)	0	1934–1988						
Fairfield-2	Reservoir #1	4–27	1969–1993						
Fairfield-3	Reservoir #2	13–29	1969–1993						
Fairfield-4	Walton Reservoir	21–42	1969–1993						
Fairfield-1	Well(s) (depth = 2,155 ft)	17–39	1969–1993						
Fairfield-1,-2,-3,-4,-5			1983 (5:1988)	Up-flow clarifier/solids contactor/Cl <sub>2</sub> /coagulation, up-flow clarifier/solids contactor/lime, acid, up-flow clarifier/solids contactor/coagulation, filtration, Cl <sub>2</sub> /ClO <sub>2</sub> /NH <sub>3</sub>	8.3	25	27.3	109	

(Continued)

Table 7.7 (Continued)

Utility	Source water	%	Date	Treatment	Finished pH	Cantor et al. (1998) TTHM (µg/L)	Annual average TTHM (µg/L)	Annual average/peak TTHM (%)	Coagulation/softening models*
Winterset-1	Well(s)/alluvium (depth = 30 ft)	25–33	1938–1995						
Winterset-2	Cedar Lake	67–75	1938–1995						
Winterset-2			1975	Cl <sub>2</sub> , coagulation/lime/up-flow clarifier, clarification, filtration, Cl <sub>2</sub>	7.3	85	46.8	55	55
Newton	Well(s)/alluvium		1981	Up-flow clarifier/Na aluminate/lime, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	9.5	17	12.2	72	72
Spencer-4	Well(s)/alluvium (depth = 71–80 ft)	75–85	1987–1996						
Spencer-2	Well(s)/alluvium (depth = 24–45 ft)	15–25	1987–1996						
			1987	Lime/solids contactor/Na aluminate, CO <sub>2</sub> , sedimentation, filtration, Cl <sub>2</sub>	8.5	10	11.8	118	
Boone-1	Well(s)/alluvium (depth = 32–67 ft)		1982	Greensand, Cl <sub>2</sub>	7.5	30	19.9	66	
Oscaloosa-1	Well(s)/alluvium (depth = 40–59 ft)		1979	Coagulation/lime, sedimentation, sedimentation, CO <sub>2</sub> , filtration, Cl <sub>2</sub>	8.3	21	24.9	119	
All plants						25th percentile		67	60
						Median		88	80
						75th percentile		119	126

Note: Blank cells indicate information was not available.

\* Utilities used in development of equations for THMs as function of finished water TOC.

**Table 7.8**  
**TTHM predictions for some plants treating Mississippi River water**

TOC/Treatment	Mississippi River	Plant(s) using Mississippi River water			
Average raw TOC	6.6 mg/L (Davenport)				
Average finished TOC	5.1 mg/L (Davenport)	3.8 mg/L (Cantor et al. 1998)	3.3 m/L (softening)		
Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	TTHM = 156 µg/L			Burlington (1912)	
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>	TTHM = 133 µg/L				Fort Madison (1946, 1966)
Coagulation/ sedimentation, Cl <sub>2</sub>	TTHM = 116 µg/L	Davenport (1910, 1955)	Keokuk (1917)	Burlington (1961, 1963)	Fort Madison (1925)
Softening/ sedimentation, Cl <sub>2</sub>	TTHM = 90 µg/L			Burlington (1967)	
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 82 µg/L	Davenport (1974)			
Coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 71 µg/L	Davenport (1967, 1981)		Burlington (1946)	
Cl <sub>2</sub> , softening/clarification, NH <sub>3</sub> , Cl <sub>2</sub>	TTHM = 61 µg/L		Keokuk (1938)		
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 61 µg/L	Davenport (1995)			
Softening/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 43 µg/L		Keokuk (1981)		

Note: Blank cells indicate information was not applicable.

## Bromine Speciation

For each utility, the average values for the ratio of CHCl<sub>3</sub> to TTHM and of BDCM to TTHM were determined. For example, Davenport treats water from the Mississippi River (bromide ranged from <0.02 to 0.05 mg/L) with conventional treatment and has had CHCl<sub>3</sub>/TTHM values of 89%–92% (average = 90%) and BDCM/TTHM values of 7%–18% (average = 11%) (Table 7.11). It was assumed that chloroform and BDCM represented 90% and 11%, respectively, of the TTHM for historical predictions for Davenport. Alternatively, Newton treats water from a shallow groundwater and has had CHCl<sub>3</sub>/TTHM values of 34%–35% (average = 35%) and BDCM/TTHM values of 29%–32% (average = 31%) (Table 7.11). It was assumed that chloroform and BDCM represented 35% and 31%, respectively, of the TTHM for historical predictions for Newton.

Table 7.12 shows the average THM bromine speciation for Iowa surface waters and shallow groundwaters. For the surface water systems, CHCl<sub>3</sub>/TTHM values ranged from 68% to 90% and BDCM/TTHM values ranged from 11% to 24%. These values are typical for most surface water systems in the United States. For the mixed surface water and groundwater systems, CHCl<sub>3</sub>/TTHM values ranged from 63% to 82%, and BDCM/TTHM values ranged from 17% to

**Table 7.9**  
**TTHM predictions for some plants treating various river waters**

TOC/Treatment	Source water					
	Mississippi River	Iowa River	Des Moines River	Missouri River	Nodaway River	Cedar River
Average raw TOC	6.6 mg/L (Davenport)	5.6 mg/L (Iowa City)	4.4 mg/L (Ottumwa)	4 mg/L (Council Bluffs)	3.5 mg/L (Clarinda)	DOC = 3.0 mg/L
Average finished TOC	5.1 mg/L (Davenport)	2.4 mg/L (Iowa City)	2.2 mg/L (Ottumwa)	3.5 mg/L (Council Bluffs)	2.6 mg/L (Clarinda)	
Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	TTHM = 156 µg/L					
Cl <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub>	TTHM = 133 µg/L	TTHM = 85 µg/L	TTHM = 70 µg/L		Average TTHM = 60 µg/L	
Coagulation/sedimentation, Cl <sub>2</sub>	TTHM = 116 µg/L	TTHM = 74 µg/L		TTHM = 73 µg/L		TTHM = 52 µg/L
Cl <sub>2</sub> , softening/sedimentation, Cl <sub>2</sub>				TTHM = 52 µg/L		
ClO <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub>		TTHM = 64 µg/L				
Softening/sedimentation, Cl <sub>2</sub>	TTHM = 90 µg/L			TTHM = 46 µg/L		
Cl <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 82 µg/L					
Coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 71 µg/L					
Cl <sub>2</sub> , softening/clarification, NH <sub>3</sub> , Cl <sub>2</sub>	TTHM = 61 µg/L		TTHM = 44 µg/L			TTHM = 40 µg/L
ClO <sub>2</sub> , coagulation/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 61 µg/L					
Softening/sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	TTHM = 43 µg/L			TTHM = 29 µg/L		TTHM = 35 µg/L
NH <sub>3</sub> , softening/sedimentation, Cl <sub>2</sub>						TTHM = 3 µg/L

Note: Blank cells indicate information was not applicable.

26%. For the shallow groundwater systems, CHCl<sub>3</sub>/TTHM values ranged from 26% to 52%, and BDCM/TTHM values ranged from 31% to 33%. The shallow groundwaters in [Table 7.12](#) were all part of the subgroup referred to as “shallow groundwater, high bromide” in the study by Cantor et al. (1998). Groundwaters often have higher levels of THM bromine speciation for two reasons. On average, groundwaters tend to have more bromide than do surface waters in the United States. Groundwaters also tend to have less TOC than do surface waters, so they have higher bromide/TOC ratios, which result in more bromine incorporation.

**Table 7.10**  
**TTHM predictions for some plants treating various lakes**

TOC/Treatment	Utility					
	Osceola	Chariton	Centerville	Spirit Lake	Creston	Fairfield
1987 finished TOC	5.7 mg/L	5.3 mg/L	5.1 mg/L	4.8 mg/L	4.1 mg/L	3.5 mg/L
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>	TTHM = 126 µg/L	TTHM = 122 µg/L				TTHM = 86 µg/L
Cl <sub>2</sub> , softening/ sedimentation, Cl <sub>2</sub>						TTHM = 82 µg/L
Softening/ sedimentation, Cl <sub>2</sub>				TTHM = 46 µg/L		
Coagulation/ sedimentation, Cl <sub>2</sub>	TTHM = 126 µg/L	TTHM = 122 µg/L	TTHM = 136 µg/L	TTHM = 25 µg/L	TTHM = 149 µg/L	
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>	TTHM = 78 µg/L	TTHM = 87 µg/L	TTHM = 86 µg/L			
Coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>					TTHM = 110 µg/L	
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>					TTHM = 58 µg/L	
ClO <sub>2</sub> , NH <sub>2</sub> Cl, filtration			TTHM = 5 µg/L			

Note: Blank cells indicate information was not applicable.

**Table 7.11**  
**Examples of predictions of THM bromine speciation**

Utility	Date	CHCl <sub>3</sub> (µg/L)	BDCM (µg/L)	DBCM (µg/L)	CHBr <sub>3</sub> (µg/L)	TTHM (µg/L)	CHCl <sub>3</sub> /TTHM (%)	BDCM/TTHM (%)
Iowa American (Davenport)	1910	104.6	13.1			115.9	90	11
	1974	75.7	6.0	0.7	<3.0	81.9	92	7
	1981	64.4	6.4	0.7	0.5	71.1	91	9
	7/27/87	90.0	11.0	0.0	0.0	101.0	89	11
	1995	54.6	11.1	1.7	0.5	61.4	89	18
Newton	1948	3.1	2.8			9.0	35	31
	1981	4.2	3.9	3.4	0.8	12.2	34	32
	5/19/87	6.0	5.0	5.0	1.0	17.0	35	29

Note: Blank cells indicate information was not available.

**Table 7.12**  
**Average THM bromine speciation for Iowa surface waters and shallow groundwaters**

Utility	Source water/comments	CHCl <sub>3</sub> /TTHM (%)	BDCM/TTHM (%)
Iowa American (Davenport)	SW	90	11
Ottumwa	SW	86	11
Keokuk	SW—softening	82	15
Creston	SW	72	24
Centerville	SW—Cl <sub>2</sub>	79	17
Clarinda	SW	71	24
Osceola	SW	75	17
Chariton	SW	78	19
Spirit Lake	SW	68	23
Des Moines	GW/SW	63	23
Council Bluffs	SW/GW	73	19
Burlington	SW/GW—softening	81	17
Iowa City	SW/GW	64	24
Fort Madison	SW/GW	82	17
Fairfield	SW (SW/GW)	76 (65)	20 (26)
Winterset	SW/GW—historical	80	18
Muscatine	GW	33	33
Newton	GW	35	31
Spencer	GW	26	32
Boone	GW	52	33
Oscalooosa	GW	40	32

Keokuk, Burlington, and Fort Madison historically treated Mississippi River water with conventional treatment. Currently, these three utilities use lime softening, and Burlington and Fort Madison now treat a blend of surface and groundwaters. The THM bromine speciation for Davenport was used for historical predictions of Keokuk, Burlington, and Fort Madison for the periods of time when these three utilities treated Mississippi River water only with conventional treatment.

Historically, Cedar Rapids treated water from the Cedar River (bromide has ranged from <0.01 to 0.06 mg/L). The amount of bromide in this river is similar to that in the Mississippi River. When Cedar Rapids used conventional treatment and Mississippi River water, THM bromine speciation was based on Davenport. When Cedar Rapids used lime softening and Mississippi River water, THM bromine speciation was based on Burlington, Keokuk, and Fort Madison with lime softening.



**Table 7.13**  
**THM and HAA formation and speciation for Iowa surface waters and shallow**  
**groundwaters in CHEEC database where HAA data available**

Group	TTHM (µg/L)	HAA5 (µg/L)	TTHM/HAA5 (µg/µg)	TCAA (µg/L)	DCAA (µg/L)
Median for all	32.0	13.8	2.4	1.9	9.6
Chloramines only	0.9	2.2	0.4	0.5	2.2
High-bromide GW	24.9	8.2	3.1	1.8	4.6

Historically, Spencer treated a blend of water from Stolley's Pond and groundwater. When Spencer only treated groundwater, the  $\text{CHCl}_3/\text{TTHM}$  value (on average) was 26%, and the BDCM/TTHM value (on average) was 32%. Spencer is in the same portion of Iowa as Spirit Lake. At Spirit Lake, the  $\text{CHCl}_3/\text{TTHM}$  value (on average) was 68%, and the BDCM/TTHM value (on average) was 23%. It was assumed that, when Spencer treated a blend (50/50) of water from Stolley's Pond and groundwater, the THM bromine speciation was between that of Spirit Lake and that of Spencer's groundwater (i.e.,  $\text{CHCl}_3/\text{TTHM} = 47\%$  and  $\text{BDCM}/\text{TTHM} = 27\%$ ).

### HAA Formation and Speciation

Data on THMs and HAAs were evaluated for Iowa surface waters and shallow groundwaters in the CHEEC database where HAA data were available (Table 7.13). The median ratio of THMs to HAA5 was 2.4 µg/µg. This involved data from 11 utilities, with a median finished water pH of 8.9. The pH was high because many of these Iowa utilities use lime softening. One utility used chloramines only and produced hardly any THMs (0.9 µg/L). More HAAs were produced than THMs at this utility (HAA5 = 2.2 µg/L), where all of the HAAs were DCAA. In other research, chloramines have been shown to be better at controlling the formation of THMs and TCAA than that of DCAA. Another utility was a high-bromide groundwater. As the presence of bromide shifts the speciation of the THMs from chloroform to BDCM and the other brominated THMs, the speciation of the HAAs is shifted from DCAA and TCAA to brominated analogues of these chlorinated species. However, most of the brominated HAAs are not part of HAA5. Thus, it is not surprising that the TTHM/HAA5 ratio for this groundwater was relatively high (i.e., 3.1 µg/µg).

Data on THMs and HAAs were obtained from the ICR database for three Iowa utilities and 10 other utilities with similar water quality in a similar part of the United States (referred to as the expanded ICR–Iowa database) (Table 7.14). The median ratio of THMs to HAA5 was 2.3 µg/µg, similar to the median ratio for the Iowa utilities in the CHEEC database. It should be noted that a significant number of utilities in both of these databases used alternative disinfectants (i.e., chlorine dioxide and/or chloramines). As a result, the median concentrations of TTHM for these two databases were relatively low (i.e., 32–38 µg/L).

Data on THMs and HAAs were also obtained from a database used during the negotiation of the U.S. DBP regulation (reg neg). Specifically, data were obtained for four small surface water systems—small systems were not required to comply with the THM Rule—in USEPA Regions 5 and 7 (Iowa is in Region 7; some of the expanded ICR–Iowa database utilities are in Region 5) (Table 7.15). These utilities used chlorine only and conventional treatment (median pH = 7.4).

**Table 7.14**  
**THM and HAA formation and speciation in the expanded ICR–Iowa database**

Statistic	TTHM (µg/L)	HAA5 (µg/L)	TTHM/HAA5 (µg/µg)	TCAA (µg/L)	DCAA (µg/L)
Minimum	9.3	3.8	0.4	0.5	0.5
25th percentile	28.9	11.7	1.1	1.8	7.0
Median	38.1	21.7	2.3	6.6	11.0
75th percentile	72.7	34.3	3.9	16.2	17.0
Maximum	128.8	112.2	10.8	63.7	41.9

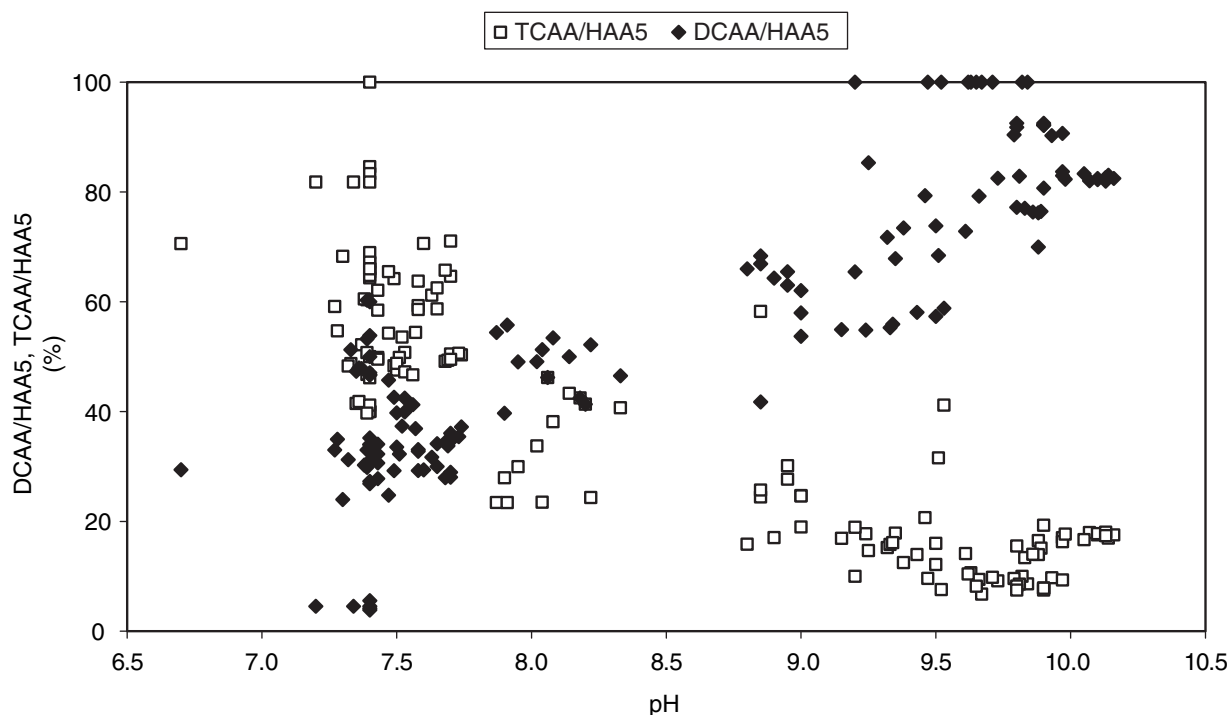
**Table 7.15**  
**THM and HAA formation and speciation in the reg neg database for  
small surface water systems in USEPA Regions 5 and 7**

Statistic	TTHM (µg/L)	HAA5 (µg/L)	TTHM/HAA5 (µg/µg)	TCAA (µg/L)	DCAA (µg/L)
25th percentile	57.4	24.7	1.4	14.2	11.8
Median	69.3	36.2	1.7	20.5	19.0
75th percentile	85.1	66.2	2.2	25.0	37.3

The median ratio of TTHM to HAA5 was 1.7 µg/µg. It should be noted that the median concentration of TTHM for this database was higher (i.e., 69 µg/L).

A decision was made to use a TTHM/HAA5 ratio of 1.7 µg/µg for historical predictions of Iowa surface water systems with conventional treatment and chlorine only, based on the reg neg database of small systems with chlorine only. A decision was made to use a TTHM/HAA5 ratio of 2.3 µg/µg for historical predictions of Iowa systems with alternative disinfectants, based on the expanded ICR–Iowa database. However, for systems that used chloramines only or for surface water systems that used chloramines and produced very low levels of THMs (e.g., <10 µg/L), a decision was made to use a TTHM/HAA5 ratio of 0.4 µg/µg, based on the Iowa utility in [Table 7.13](#) that used chloramines only. For shallow groundwaters that produced relatively high amounts of brominated THMs, a TTHM/HAA5 ratio of 3.1 µg/µg was used, based on the Iowa utility in [Table 7.7](#) that was a high-bromide groundwater. If a particular utility had site-specific TTHM/HAA5 ratios for its current treatment, this was used for some or all of the historical predictions for that utility.

[Figure 7.6](#) shows the impact of pH on HAA speciation in the expanded ICR–Iowa database (the water with chloramines only was not included). In other research, it has been shown that TCAA formation is low at high pH (e.g., 9), whereas DCAA is relatively unaffected by pH (in the range of pH 5–9). At neutral pH levels, TCAA tends to predominate. Some of the variations in HAA speciation probably reflect differences in the natural organic matter (colored waters tend to form more TCAA) and the use of alternative disinfectants.



**Figure 7.6 Impact of pH on HAA speciation in expanded ICR–Iowa database**

A regression analysis was conducted to show the impact of pH on TCAA speciation in the expanded ICR–Iowa database. The central tendency relationship is  $\text{TCAA/HAA5} = 1.94 - 0.185 * \text{pH}$ . A similar relationship was previously observed for the CHEEC database ( $\text{TCAA/HAA5} = 2.04 - 0.204 * \text{pH}$ ). In both cases, the  $R^2$  is relatively high (0.78 for CHEEC database, 0.76 for the expanded ICR–Iowa database). Because most conventional treatment plants have finished water pH levels  $<8.5$  and most lime-softening plants have finished water pH levels  $>8.5$ , the data were segmented into these two pH regions; the central tendency relationships for the two pH regions are  $\text{TCAA/HAA5} = 2.77 - 0.295 * \text{pH}$  (when  $\text{pH} < 8.5$ ) and  $1.09 - 0.097 * \text{pH}$  (when  $\text{pH} > 8.5$ ). Although the  $R^2$  for the latter two relationships are lower, the central tendency relationships mirror the typical impact of pH on TCAA formation (TCAA formation decreases as pH increases).

A regression analysis was performed to show the impact of pH on DCAA speciation in the expanded ICR–Iowa database. The central tendency relationship is  $\text{DCAA/HAA5} = 0.207 * \text{pH} - 1.21$ . A similar relationship was previously observed for the CHEEC database ( $\text{DCAA/HAA5} = 0.216 * \text{pH} - 1.20$ ). In both cases, the  $R^2$  is relatively high (0.81 for CHEEC database, 0.78 for the expanded Iowa–ICR database). Again, the data were segmented into two pH regions (Figure 7.6). The central tendency relationships for the two pH regions are  $\text{DCAA/HAA5} = 0.176 * \text{pH} - 0.967$  (when  $\text{pH} < 8.5$ ) and  $0.225 * \text{pH} - 1.37$  (when  $\text{pH} > 8.5$ ). Although the  $R^2$  for the latter two relationships is lower, the central tendency relationships mirror the typical impact of pH on TCAA formation (DCAA becomes the dominant HAA as pH increases).

A decision was made to use the TCAA/HAA5 and DCAA/HAA5 relationships from the expanded ICR–Iowa database, based on a segmentation of the finished water pH levels. However, if a particular utility had site-specific TCAA/HAA5 and DCAA/HAA5 relationships for its current treatment, that was used for some or all of the historical predictions for that utility if the

pH level of the finished water was similar. Typically, the site-specific values were quite similar to those that would have been predicted from the relationships in the expanded ICR–Iowa database. If a utility used lime softening and chloramines—or the THMs were so low as to suggest that postchloramination was the dominant factor in controlling THM formation—then it was assumed that all of the chlorinated HAAs formed were DCAA (TCAA was reported at its minimum reporting level of 0.5 µg/L).

In 1912, Burlington treated Mississippi River water (average raw water TOC = 6.6 mg/L at Davenport, which also treats Mississippi River water) with filtration and chlorine (Table 7.16). Based on the models (equations) developed to predict TTHM based on finished water TOC data, its annual average TTHM in 1912 was predicted to be 156 µg/L. Based on the median TTHM/HAA5 relationship (1.7 µg/µg) in the reg neg database for small surface water systems in USEPA Regions 5 and 7, its HAA5 was predicted to be 92 µg/L. Based on the TCAA/HAA5 and DCAA/HAA5 relationships in the expanded Iowa–ICR database, it was predicted that 42% and 44% of the HAA5 were DCAA and TCAA, respectively (for a finished water pH of 7.9). In 1961, Burlington used coagulation, sedimentation, filtration, and chlorine. Based on an average finished water TOC of 5.1 mg/L (based on Davenport with conventional treatment), its annual average TTHM in 1961 was predicted to be 116 µg/L. Based on a TTHM/HAA5 ratio of 1.7 µg/µg, its HAA5 was predicted to be 68 µg/L in 1961. In 1946, Burlington used conventional treatment with chlorine and chloramines, similar to the treatment that Davenport used in 1981. During that time period, Davenport produced 71 µg/L of TTHM. Burlington was assumed to produce a similar level in 1946. Based on the median TTHM/HAA5 relationship (2.3 µg/µg) in the expanded ICR–Iowa database—which included many systems that used chloramines—its HAA5 was predicted to be 31 µg/L in 1946. (Note: Burlington discontinued postchloramination in 1961, as discussed previously.) In 1967, Burlington used lime softening with chlorine. From the CHEEC database, its average TTHM in the late 1970s/early 1980s was 90 µg/L. Based on the median TTHM/HAA5 relationship (2.3 µg/µg) in the expanded ICR–Iowa database—which included many systems that used softening—its HAA5 was predicted to be 39 µg/L in 1967. Based on the TCAA/HAA5 and DCAA/HAA5 relationships in the expanded ICR–Iowa database, it was predicted that 58% and 25% of the HAA5 were DCAA and TCAA, respectively (for a finished water pH of 8.7). In 1985, Burlington changed to treating a mixture of surface and groundwater. As a result, it reduced its TTHM and HAA5 to 37 and 15 µg/L, respectively. This represents a TTHM/HAA5 ratio of 2.4 µg/µg, which is similar to the median value for the expanded ICR–Iowa database. In addition, DCAA and TCAA represented 63% and 31% of the HAA5 (for a finished water pH of 9.0), which is similar to what was predicted based on the HAA/pH relationships in the expanded ICR–Iowa database. The treatment, disinfection, and source water changes at Burlington resulted in lowering TTHM from 156 µg/L (1912) to 116 µg/L (1961) to 90 µg/L (1967) to 71 µg/L (1946, when it temporarily used chloramines) to 37 µg/L (1985). Likewise, it was predicted that these changes resulted in lowering HAA5 from 92 µg/L (1912) to 68 µg/L (1961) to 39 µg/L (1967) to 31 µg/L (1946) to 15 µg/L (1985). In addition, it was predicted that these changes resulted in lowering DCAA from 39 µg/L (1912) to 29 µg/L (1961) to 23 µg/L (1967) to 13 µg/L (1946) to 9.6 µg/L (1985). It was also predicted that these changes resulted in lowering TCAA from 40 µg/L (1912) to 30 µg/L (1961) and ultimately to 4.7 µg/L (1985). In 1946, it was predicted that TCAA was lowered to 14 µg/L due to postchloramination, while maintaining the finished water pH at 7.9. Alternatively, in 1967, it was predicted that TCAA was lowered to 9.8 µg/L due to raising the finished water pH to 8.7, even though Burlington had eliminated postchloramination. Thus, it was predicted that more TCAA was formed in 1946 with prechlorination/postchloramination at pH 7.9 than in 1967 with chlorine only at pH 8.7.

**Table 7.16**  
**Examples of predictions of HAA formation and speciation**

Utility	Date	Treatment	Finished pH	TTHM (µg/L)	DCAA (µg/L)	TCAA (µg/L)	HAA5 (µg/L)	TTHM/ HAA5 (µg/µg)	DCAA/ HAA5 (%)	TCAA/ HAA5 (%)
Burlington	1900	Filtration		0.0	0.0	0.0	0.0			
	1912	Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	7.9	156.2	38.9	40.4	91.9	1.7	42	44
	1946	Coagulation, sedimentation, Cl <sub>2</sub> , filtration, NH <sub>3</sub> , Cl <sub>2</sub> , acid	7.9	71.1	13.1	13.6	30.9	2.3	42	44
	1961	Coagulation, sedimentation, Cl <sub>2</sub> , filtration, Cl <sub>2</sub> , acid	7.9	115.9	28.9	30.0	68.2	1.7	42	44
	1963	Coagulation, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, Cl <sub>2</sub> , Cl <sub>2</sub> , acid	7.9	115.9	28.9	30.0	68.2	1.7	42	44
	1967	Coagulation, sedimentation, lime, sedimentation, CO <sub>2</sub> , Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	8.7	90.1	22.7	9.8	39.2	2.3	58	25
	1985	Mixed SW/GW	9.0	36.9	9.6	4.7	15.1	2.4	63	31
Cedar Rapids	1900	Coagulation or softening, filtration		0.0	0.0	0.0	0.0			
	1910	Coagulation or softening, filtration, disinfection	7.8	51.5	12.3	14.2	30.3	1.7	41	47
	1930	Coagulation or softening/sedimentation/ disinfection/filtration/ stabilization/lime/NH <sub>3</sub>	8.6	35.0	8.6	3.9	15.2	2.3	57	26
	1940	Disinfection/coagulation or softening/sedimentation/ disinfection/filtration/ stabilization/lime/NH <sub>3</sub> / disinfection	9.0	40.3	11.5	3.8	17.5	2.3	66	22
	1952	NH <sub>3</sub> , lime, sedimentation/Cl <sub>2</sub> /acid, filtration	9.2	3.1	7.8	0.5	7.8	0.4	100	

Cedar Rapids previously treated Cedar River water (average raw water DOC = 3.0 mg/L) with coagulation and chlorine in 1910 (Table 7.16). Based on the models (equations) developed to predict TTHM based on finished water TOC data, its annual average TTHM in 1910 was predicted to be 52 µg/L. Based on the median TTHM/HAA5 relationship (1.7 µg/µg) in the reg neg database for small surface water systems in USEPA Regions 5 and 7, its HAA5 was predicted to be 30 µg/L. Based on the TCAA/HAA5 and DCAA/HAA5 relationships in the expanded ICR–Iowa database, it was predicted that 41% and 47% of the HAA5 were DCAA and TCAA, respectively (for a finished water pH of 7.8). In 1930, Cedar Rapids went to lime softening and added postchloramination. Based on a median reduction in THMs of 32% from switching to postchloramination (from selected utilities in the CHEEC database), its annual average TTHM in 1930



were predicted to be 35 µg/L. Based on the median THM/HAA5 relationship (2.3 µg/µg) in the expanded ICR–Iowa database—which included many systems that used chloramines—its HAA5 was predicted to be 15 µg/L in 1930. Based on the TCAA/HAA5 and DCAA/HAA5 relationships in the expanded ICR–Iowa database, it was predicted that 57% and 26% of the HAA5 were DCAA and TCAA, respectively (for a finished water pH of 8.6). In 1940, Cedar Rapids went to prechlorination. Based on a reduction in THMs of 13% in moving the point of chlorination (from Davenport), its annual average TTHM in 1940 was predicted to be  $35/(1.0-0.13) = 40$  µg/L. Based on the median TTHM/HAA5 relationship (2.3 µg/µg) in the expanded ICR–Iowa database, its HAA5 was predicted to be 18 µg/L in 1940. Based on the TCAA/HAA5 and DCAA/HAA5 relationships in the expanded ICR–Iowa database, it was predicted that 66% and 22% of the HAA5 were DCAA and TCAA, respectively (for a finished water pH of 9). In 1952, Cedar Rapids went to pre-ammoniation. Based on a reduction in THMs of 94% in converting from chlorination to chloramination (from Centerville), its annual average TTHM in 1952 was predicted to be 3 µg/L. Based on the TTHM/HAA5 relationship (0.4 µg/µg) for Cedar Rapids in the late 1990s with pre-ammoniation, its HAA5 was predicted to be 8 µg/L in 1952. Because of the use of chloramines only at a high pH (9.2), it was predicted that all of the chlorinated HAAs were in the form of DCAA. The treatment and disinfection changes at Cedar Rapids resulted in lowering TTHM from 52 µg/L (1910) to 40 µg/L (1940, when it used prechlorination with postchloramination) to 35 µg/L (1930, when it used intermediate chlorination with postchloramination) to 3 µg/L (1952, when it used pre-ammoniation). Likewise, it was predicted that these changes resulted in lowering HAA5 from 30 µg/L (1910) to 18 µg/L (1940) to 15 µg/L (1930) to 8 µg/L (1952). In addition, it was predicted that these changes resulted in lowering DCAA from 12 µg/L (1910) to 9 µg/L (1930) and ultimately to 8 µg/L (1952). However, in 1940, it was predicted that it formed as much DCAA (12 µg/L)—by using prechlorination and postchloramination at pH 9—as in 1910—by using postchlorination at pH 7.8. It was predicted that these changes resulted in lowering TCAA from 14 µg/L (1910) to 4 µg/L (1930) and ultimately to 0.5 µg/L (1952). However, in 1940, it was predicted that it formed as little TCAA (4 µg/L)—by using prechlorination/postchloramination at pH 9—as in 1930—by using intermediate chlorination and postchloramination at pH 8.6.

Tables 7.17 through 7.19 show the HAA5 predictions for various surface water systems. Table 7.20 shows examples of predictions of BCAA and HAA6. It was assumed that a shift in bromine speciation in THMs from chloroform to BDCM was the same for a shift in bromine speciation in HAAs from DCAA to BCAA:

$$\begin{aligned} \text{BCAA} &= (\text{BDCM}/\text{chloroform}) * \text{DCAA (on a molar basis)} \text{ and HAA6} \\ &= \text{HAA5} + \text{BCAA (on a weight basis)} \end{aligned}$$

For example, in 1995, it was predicted that BCAA and HAA6 were 1.3 and 20.7 µg/L, respectively, at Davenport. The average values for BCAA and HAA6 for Davenport in the late 1990s were 1.8 and 20.7 µg/L, respectively. Alternatively, Creston, which had much more BDCM than Davenport, was predicted to have more BCAA than Davenport. Likewise, Spencer, which treats a high-bromide groundwater and has more BDCM than chloroform (except in 1971 when it treated a blend of surface and groundwater), was predicted to have more BCAA than DCAA.

**Table 7.17**  
**HAA5 predictions for plants treating Mississippi River water**

TOC/Treatment	Mississippi River	Plant(s) using Mississippi River water			
Average raw TOC	6.6 mg/L (Davenport)				
Average finished TOC	5.1 mg/L (Davenport)	3.8 mg/L (Cantor et al. 1998)	3.3 mg/L (softening)		
Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	HAA5 = 92 µg/L			Burlington (1912)	
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>	HAA5 = 78 µg/L				Fort Madison (1946, 1966)
Coagulation/ sedimentation, Cl <sub>2</sub>	HAA5 = 68 µg/L	Davenport (1910, 1955)	Keokuk (1917)	Burlington (1961, 1963)	Fort Madison (1925)
Softening/ sedimentation, Cl <sub>2</sub>	HAA5 = 39 µg/L			Burlington (1967)	
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	HAA5 = 36 µg/L	Davenport (1974)			
Cl <sub>2</sub> , softening/clarification, NH <sub>3</sub> , Cl <sub>2</sub>	HAA5 = 36 µg/L		Keokuk (1938)		
Coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	HAA5 = 31 µg/L	Davenport (1967, 1981)		Burlington (1946)	
Softening/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	HAA5 = 21 µg/L		Keokuk (1981)		
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	HAA5 = 19 µg/L	Davenport (1995)			

Note: Blank cells indicate information was not applicable.

## Nonalluvial Groundwaters on Iowa Hit List

In previous sections, predictions were developed for

- CHCl<sub>3</sub>, BDCM, TTHM, DCAA, TCAA, the sum of the five regulated HAAs (HAA5), BCAA, and the sum of six HAAs (HAA6 = HAA5 + BCAA)
- The surface waters, mixed surface and groundwaters, and alluvial groundwaters in the Iowa hit list

Appendix 7.2 summarizes DBP predictions for the nonalluvial groundwaters on the Iowa hit list. The DBP predictions were initially zeros until chlorine was first introduced (1921–1963). Whenever there was a significant change in treatment and disinfection and/or source water, a new set of DBP predictions were made. In the previous set of predictions for mixed surface and groundwaters, historical predictions had not been made for time periods when Des Moines and West Des Moines were using groundwater only. Predictions for those two utilities for those periods are now included in this analysis.

**Table 7.18**  
**HAA5 predictions for plants treating various river waters**

TOC/Treatment	Source water					
	Mississippi River	Iowa River	Des Moines River	Missouri River	Nodaway River	Cedar River
Average raw TOC	6.6 mg/L (Davenport)	5.6 mg/L (Iowa City)	4.4 mg/L (Ottumwa)	4 mg/L (Council Bluffs)	3.5 mg/L (Clarinda)	DOC = 3.0 mg/L
Average finished TOC	5.1 mg/L (Davenport)	2.4 mg/L (Iowa City)	2.2 mg/L (Ottumwa)	3.5 mg/L (Council Bluffs)	2.6 mg/L (Clarinda)	
Cl <sub>2</sub> , filtration, Cl <sub>2</sub>	HAA5 = 92 µg/L					
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>	HAA5 = 78 µg/L	HAA5 = 50 µg/L	HAA5 = 46 µg/L		Average HAA5 = 35 µg/L	
Coagulation/ sedimentation, Cl <sub>2</sub>	HAA5 = 68 µg/L	HAA5 = 43 µg/L		HAA5 = 31 µg/L		HAA5 = 30 µg/L
Cl <sub>2</sub> , softening/ sedimentation, Cl <sub>2</sub>				HAA5 = 22 µg/L		
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>		HAA5 = 26 µg/L				
Softening/ sedimentation, Cl <sub>2</sub>	HAA5 = 39 µg/L			HAA5 = 20 µg/L		
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	HAA5 = 36 µg/L					
Cl <sub>2</sub> , softening/ clarification, NH <sub>3</sub> , Cl <sub>2</sub>	HAA5 = 36 µg/L		HAA5 = 29 µg/L			HAA5 = 18 µg/L
Coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	HAA5 = 31 µg/L					
Softening/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	HAA5 = 21 µg/L			HAA5 = 12 µg/L		HAA5 = 15 µg/L
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>	HAA5 = 19 µg/L					
NH <sub>3</sub> , softening/ sedimentation, Cl <sub>2</sub>						HAA5 = 8 µg/L

Note: Blank cells indicate information is not applicable.

In the revised CHEEC database, THM data coded with the letter “M” are either maximum formation potential tests or samples collected at maximum residence time. Because of this uncertainty, those data were not used. However, in examining THM data with other sample codes for a number of nonalluvial groundwater systems, it was observed that some utilities had relatively high levels of THMs (up to 67 µg/L). In some cases, similarly high levels of THMs were reported in the AWWA WIDB (1992b). In another database from the Iowa DNR (1993–1998), many of these high THM levels were reported as maximum potential tests.



**Table 7.19**  
**HAA5 predictions for plants treating various lakes**

TOC/Treatment	Utility					
	Osceola	Chariton	Centerville	Spirit Lake	Creston	Fairfield
1987 finished TOC	5.7 mg/L	5.3 mg/L	5.1 mg/L	4.8 mg/L	4.1 mg/L	3.5 mg/L
Cl <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>	HAA5 = 74 µg/L	HAA5 = 72 µg/L				HAA5 = 51 µg/L
Cl <sub>2</sub> , softening/ sedimentation, Cl <sub>2</sub>						HAA5 = 36 µg/L
Softening/ sedimentation, Cl <sub>2</sub>				HAA5 = 23 µg/L		
Coagulation/ sedimentation, Cl <sub>2</sub>	HAA5 = 74 µg/L	HAA5 = 72 µg/L	HAA5 = 80 µg/L	HAA5 = 12 µg/L	HAA5 = 88 µg/L	
Coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>					HAA5 = 48 µg/L	
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub>	HAA5 = 34 µg/L	HAA5 = 38 µg/L	HAA5 = 37 µg/L			
ClO <sub>2</sub> , coagulation/ sedimentation, Cl <sub>2</sub> , NH <sub>3</sub>					HAA5 = 25 µg/L	
ClO <sub>2</sub> , NH <sub>2</sub> Cl, filtration			HAA5 = 14 µg/L			

Note: Blank cells indicate information was not applicable.

**Table 7.20**  
**Examples of predictions of BCAA and HAA6**

Utility	Date	CHCl <sub>3</sub>	BDCM	DCAA	HAA5	BCAA	HAA6
		(µg/L)					
Davenport	1910	104.6	13.1	23.8	68.2	2.9	71.1
	1974	75.7	6.0	12.4	35.6	1.0	36.6
	1981	64.4	6.4	10.8	30.9	1.1	32.0
	1995	54.6	11.1	6.8	19.4	1.3	20.7
Creston	1986	111.0	33.0	34.8	87.6	10.2	97.8
	1987	85.0	23.0	19.0	47.8	5.0	52.9
	1988	37.2	16.1	10.0	25.2	4.2	29.4
Spencer	1954	8.4	10.3	3.4	10.5	4.1	14.6
	1971	17.0	10.0	6.4	15.8	3.7	19.5
	1984	4.7	6.7	3.2	6.5	4.5	11.0
	1987	2.8	3.7	2.0	3.8	2.6	6.5

**Table 7.21**  
**Central tendency predictions for Iowa groundwaters**

Water	pH	CHCl <sub>3</sub>	BDCM	TTHM	DCAA	TCAA	HAA5	BCAA	HAA6
		(µg/L)							
Softening alluvial	8.7	6.8	5.9	17.7	3.4	1.4	5.7	2.9	8.6
Nonsoftening alluvial	7.6	5.7	4.4	13.0	1.7	2.5	4.6	1.3	5.9
Nonalluvial	7.7	0.6	<0.5	0.9	2.2	<0.5	2.2	0.0	2.2

Although the THM Rule requires that utilities collect four samples per plant per quarter (i.e., 16 samples per year per plant), small groundwater systems can collect as few as one sample per year (in the summer) at maximum residence time or they can run a maximum potential test. If this one sample—which represents a “worst-case scenario”—is less than the maximum contaminant level, they do not need to collect the normal full set of samples. Thus, it appears as if a number of the nonalluvial groundwater systems in Iowa took advantage of this reduced monitoring, and some of these maximum potential test data were accidentally reported or listed as plant effluent or distribution system data in the CHEEC database and/or the AWWA WIDB. The Iowa DNR database was used to cull obvious or likely maximum potential test data out of the revised CHEEC database.

[Table 7.21](#) summarizes water quality and THM data for groundwater (alluvial and nonalluvial) in the Iowa hit list. Here are some general observations for the eight nonalluvial groundwaters.

- They treat water with depths ranging from 25–44 m (83–147 ft) to 730–777 m (2,435–2,590 ft).
- Seven of the eight systems use aeration and oxidation as part of their treatment processes. These utilities probably treat groundwaters with a relatively high amount of reduced inorganic species (e.g., ammonia-nitrogen [NH<sub>3</sub>-N], hydrogen sulfide, iron or manganese) that can exert a high oxidant demand.
- Where NH<sub>3</sub>-N data were available, the median raw water concentration was 0.6 mg/L. It takes 7.6 mg/L of Cl<sub>2</sub> to “breakpoint” 1.0 mg/L of NH<sub>3</sub>-N. If the NH<sub>3</sub>-N is not removed during the aeration step, it would take 4.6 mg/L of Cl<sub>2</sub> (on average) to breakpoint oxidize the raw water NH<sub>3</sub>-N in these waters.
- The median finished water concentration of TOC was 1.0 mg/L.
- Three of these utilities serve water with a NH<sub>2</sub>Cl residual or their Cl<sub>2</sub> residual is much greater than their free Cl<sub>2</sub> residual. (Marshalltown, on average, adds 2 mg/L of Cl<sub>2</sub> to a groundwater with 1.1 mg/L of NH<sub>3</sub>-N. Because this utility does not add enough Cl<sub>2</sub> to breakpoint the NH<sub>3</sub>, it produces chloraminated water.)
- The median concentration of TTHM was 0.9 µg/L. (Under normal chlorination conditions, these utilities probably do not add enough Cl<sub>2</sub> to meet the demand of the TOC and inorganic species. Thus, they produce little to no THMs, as is observed for systems that chloramine. However, under formation potential conditions, they

probably add enough  $\text{Cl}_2$  to meet the demand of the water and, thus, can form significant levels of THM.)

Here are some general observations for the seven alluvial groundwaters with data:

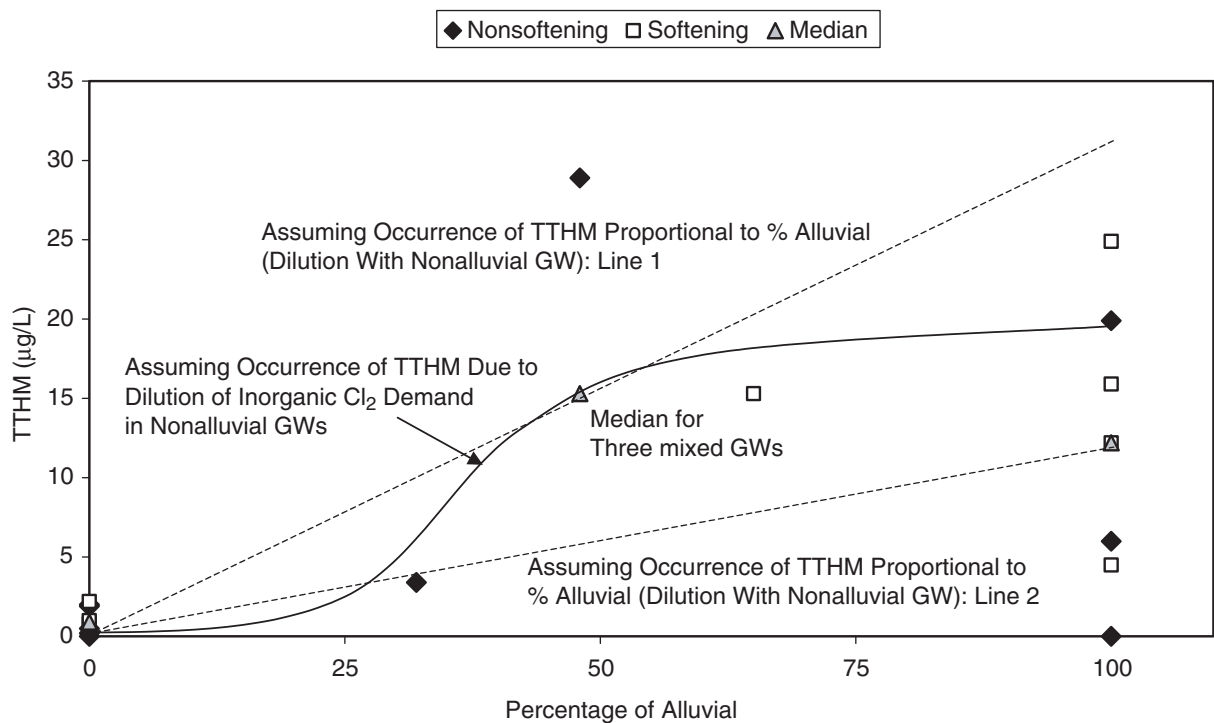
- They treat water with depths ranging from 6.6 to 24.0 m (22 to 80 ft).
- None of them use aeration and oxidation as part of their treatment processes.
- The median finished water concentration of TOC was 1.1 mg/L. Thus, on a median basis, the alluvial and nonalluvial groundwaters in the hit list had comparable levels of TOC.
- The median concentration of TTHM was 12.2  $\mu\text{g/L}$ . The average concentration of TTHM for the softening plants that use  $\text{Cl}_2$  (excluding Cedar Rapids, which uses  $\text{NH}_2\text{Cl}$  and produced 0.9–4.5  $\mu\text{g/L}$ ) was 17.7  $\mu\text{g/L}$ . (The nonalluvial groundwaters produced levels of THMs comparable to that of Cedar Rapids.) The average concentration of TTHM for the nonsoftening plants (except for Winterset, which produced 0  $\mu\text{g/L}$ ) was 13.0  $\mu\text{g/L}$ .

Here are some general observations for the three mixed groundwaters:

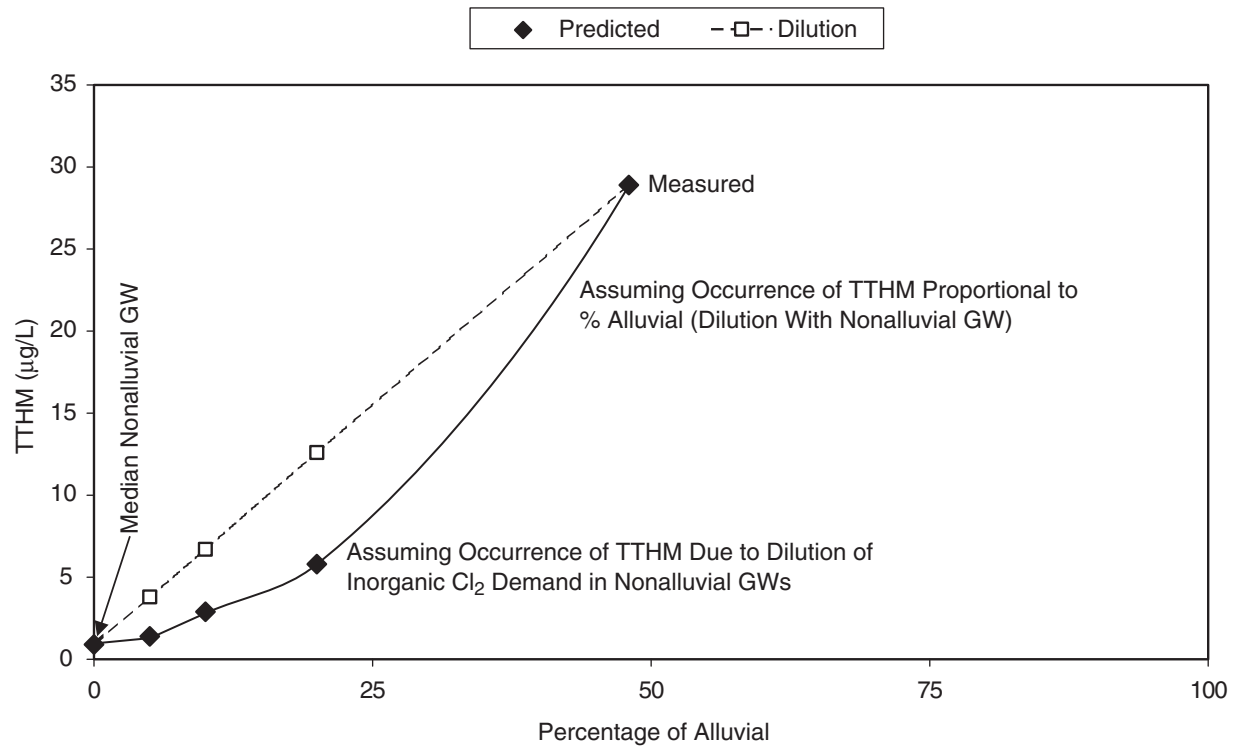
- They treat groundwater that is 32% to 65% alluvial.
- Two of the three use aeration and oxidation as part of their treatment processes.
- The median raw water  $\text{NH}_3\text{-N}$  concentration was 0.3 mg/L. On a median basis, this is less than that of the nonalluvial groundwaters.
- The raw and finished water TOC concentrations ranged from 0.6 to 2.2 mg/L.
- The concentration of TTHM ranged from 3.4 to 28.9  $\mu\text{g/L}$ .

Figure 7.7 shows the occurrence of TTHM for these three groups of groundwaters. If the occurrence of TTHM was proportional to the percent of alluvial water (i.e., “dilution” with nonalluvial groundwater), lines 1 or 2 should apply. Line 1, which was based on the median nonalluvial and the median mixed groundwater, results in too high of a TTHM level for a 100% alluvial water. Line 2, which was based on the median nonalluvial and the median alluvial groundwater, results in too low of a TTHM level for two of the three mixed groundwaters. An alternative explanation is an S-shaped curve, based on the assumption that the occurrence of TTHM in mixed groundwaters is due (in part) to the dilution of the inorganic  $\text{Cl}_2$  demand in nonalluvial groundwaters with the alluvial groundwater. Clearly, the two groundwaters with high percentages of alluvial water produced relatively high levels of TTHM, whereas the one groundwater with a low percentage of alluvial water produced a relatively low level of TTHM.

Sioux City currently treats a mixture of 48% alluvium Dakota groundwater (depth = 24–37.5 m [80–125 ft]) and 52% Dakota groundwater (47–123 m [156–410 ft]) and produces 28.9  $\mu\text{g/L}$  TTHM. Based on the results of the other alluvial and nonalluvial groundwaters in Iowa, the THM formation is most likely due to the high percent of the shallower Alluvium Dakota groundwater. Historically (1971, 1976, 1980, and 1983), Sioux City treated a mixture of groundwaters with a lower percent of Alluvium Dakota groundwater (0%, 5%, 10%, and 20%, respectively). Figure 7.8 shows the predicted occurrence of historical TTHM in the mixed groundwaters. Rather than assuming that the occurrence of TTHM was proportional to the percent of alluvial groundwater



**Figure 7.7 Occurrence of TTHM in Iowa groundwaters: alluvial versus nonalluvial, softening versus nonsoftening**



**Figure 7.8 Predicted occurrence of TTHM in Sioux City groundwater**

(based on dilution with nonalluvial groundwater), it was assumed that the occurrence of TTHM was due to a dilution of the inorganic  $\text{Cl}_2$  demand in the nonalluvial groundwater (following an S-shaped curve). For example, for 20% alluvial groundwater, it was estimated that the TTHM would be  $5.8 \mu\text{g/L}$  (instead of  $12.6 \mu\text{g/L}$ ). The lower TTHM number is consistent with another Iowa groundwater, Waterloo, with 32% alluvial groundwater ( $3.4 \mu\text{g/L}$  TTHM) (Figure 7.7).

Dubuque currently treats (aeration/oxidation, lime softening,  $\text{Cl}_2$ ) a mixture of 65% alluvial groundwater (35.5–60 m [125–200 ft]) and 35% Camord groundwater (depth = 390–543 m [1,300–1,810 ft]) and produces  $15.3 \mu\text{g/L}$  TTHM. Historically, it used  $\text{NH}_2\text{Cl}$  (concurrent addition of  $\text{Cl}_2$  and  $\text{NH}_3$  in 1924 and 1951, sequential addition of  $\text{Cl}_2$  and  $\text{NH}_3$  in 1957). Based on the median control of THMs with  $\text{NH}_2\text{Cl}$  for surface and mixed surface and groundwaters in Iowa, it was assumed that the following were true:

- The addition of post- $\text{NH}_3$  reduced the THMs by 32% (from  $15.3$  to  $10.4 \mu\text{g/L}$  for 1957).
- The concurrent addition of  $\text{Cl}_2$  and  $\text{NH}_3$  reduced the THMs by 94% (from  $15.3$  to  $0.9 \mu\text{g/L}$  for 1955—when Dubuque treated the mixture of alluvial and nonalluvial groundwaters as it currently does).

From 1947 to 1955, Dubuque treated 100% nonalluvial groundwater (with  $\text{NH}_2\text{Cl}$ ). It was assumed that the level of TTHM was  $0.9 \mu\text{g/L}$  in that time period, which is the median occurrence for the nonalluvial groundwaters, some of which use  $\text{NH}_2\text{Cl}$  (Table 7.1). It was predicted that the use of  $\text{NH}_2\text{Cl}$  would reduce the TTHM for the 65% alluvial groundwater mixture to  $0.9 \mu\text{g/L}$  (in 1955), which is the same level (on a median basis) for the nonalluvial groundwaters, some of which indirectly use  $\text{NH}_2\text{Cl}$ .

From 1900 to 1941, Marion used 100% spring water. In 1940, it started to use  $\text{OCl}^-$ . Although no data exists for spring water quality in the Iowa databases, there are several springs in the Ontario database. Because Marion appeared to use no treatment for most of the time it used the spring as its source of water, this suggests that the spring was of high quality. Thus, data for two Ontario springs with low levels of DOC ( $0.98 \text{ mg/L}$ ) and color ( $1.9 \text{ cu}$ ) were used to estimate the DBP formation ( $12.6 \mu\text{g/L}$  TTHM,  $4.9 \mu\text{g/L}$  HAA6) of the Marion spring with  $\text{OCl}^-$ .

From 1916 to 1955, Indianola used 100% drift groundwater (14–16.5 m [46–55 ft]). In 1933, it introduced aeration and oxidation, lime softening, and  $\text{OCl}^-$ . Because this shallow groundwater was similar in depth to that of the alluvial groundwaters (Table 7.1), it was assumed that Indianola produced  $12.2 \mu\text{g/L}$  TTHM in 1933, which is the median TTHM occurrence for the alluvial groundwaters (Table 7.21).

In 1955, Indianola switched to 30% drift groundwater and 70% Camord groundwater (depth = 730.5–777 m [2,435–2,590 ft]). In 1973, it switched to 100% Camord groundwater and currently produces  $0.8 \mu\text{g/L}$  TTHM. In 1955, it was assumed that it produced  $3.4 \mu\text{g/L}$  TTHM, as is produced at Waterloo with 32% alluvial groundwater (Figure 7.7).

From 1871 to 1950, Des Moines treated 100% well water. Its groundwater comes from an infiltration gallery adjacent to the Racoon River. In 1911, it added  $\text{OCl}^-$ , and, in 1917, it added alkali. In 1939, it went to lime softening. In 1911 and 1917, it was assumed that the level of TTHM was  $13.0 \mu\text{g/L}$ , which is the average level for the nonsoftening alluvial groundwaters in Table 7.21. In 1939, it was assumed that the level of TTHM was  $17.7 \mu\text{g/L}$ , which is the average level for the softening alluvial groundwaters in Table 7.21. Because THM formation increases with pH and softened water is at a higher pH, the higher average level of TTHM for the softening plants is consistent.

Previously, it was predicted that Des Moines had 7.9–30.4 µg/L TTHM (median = 22.4 µg/L) when treating a mixture of surface (e.g., Racoon River) and groundwater. Because the Des Moines well water is derived from the Racoon River, some of the DBP precursors in that surface water would also be present (in part) in the well water. Bank filtration can remove some, but not all, DBP precursors.

Once TTHM were predicted, data on speciation (specifically  $\text{CHCl}_3$  and BDCM) were developed.

Under normal  $\text{Cl}_2$  conditions, the nonalluvial groundwaters formed primarily  $\text{CHCl}_3$  (median: 0.6 µg/L  $\text{CHCl}_3$ , <0.5 µg/L BDCM), whereas the alluvial groundwaters tended to form a mixture of  $\text{CHCl}_3$  and BDCM (median: 4.2 µg/L  $\text{CHCl}_3$ , 3.9 µg/L BDCM).

Waterloo, which was 32% alluvial groundwater, had more BDCM (1.4 µg/L; 41% of the TTHM) than  $\text{CHCl}_3$  (0.6 µg/L; 16% of the TTHM). It was assumed that the 50% and 100% alluvial groundwater, which were treated in 1930–1932 and 1932–1954, respectively, had similar THM speciation.

Dubuque, which was 65% alluvial groundwater, had more  $\text{CHCl}_3$  (12.0 µg/L, 79% of the TTHM) than BDCM (2.8 µg/L, 18% of the TTHM). Historically, Dubuque used  $\text{NH}_2\text{Cl}$ . It was assumed, as TTHM were reduced by post- $\text{NH}_3$  or concurrent addition of  $\text{Cl}_2$  and  $\text{NH}_3$ , that the THM speciation was still predominantly  $\text{CHCl}_3$ .

If a utility had used water historically that it does not use currently, it was assumed that the THM speciation was the same as the median speciation of other similar waters. (The spring used by Marion was assumed to have similar speciation to that of the two Ontario springs [49%  $\text{CHCl}_3$ , 29% BDCM]). The Indianola shallow drift groundwater was assumed to have similar speciation to that of the alluvial groundwaters (34%  $\text{CHCl}_3$ , 32% BDCM, on a median basis).

Currently, the average THM speciation for Des Moines—when treating a mixture of well and surface water—is 63%  $\text{CHCl}_3$  and 23% BDCM. Because the well water is derived from one of the surface waters through an infiltration gallery, it was assumed that the well water had similar THM speciation.

Next, predictions were made for HAAs.

Currently, Sioux City, which chlorinates the water at a neutral pH (7.4), has a TTHM/HAA5 ratio of 2.8 µg/µg. (This ratio was used to predict HAA5 for other nonsoftening alluvial groundwaters. For example, the average TTHM occurrence for nonsoftening alluvial groundwaters was 13.0 µg/L. Thus, it was predicted that such a water would have  $13.0/2.8 = 4.6$  µg/L HAA5.)

Oscalosa, which has  $\text{Cl}_2$  softened water (pH 8.3), has a TTHM/HAA5 ratio of 3.1 µg/µg. (This ratio was used to predict HAA5 for other softening alluvial groundwaters. For example, the average TTHM occurrence for softening alluvial groundwaters was 17.7 µg/L. Thus, it was predicted that such a water would have  $17.7/3.1 = 5.7$  µg/L HAA5.)

Cedar Rapids, which uses  $\text{NH}_2\text{Cl}$  to disinfect the water, has a TTHM/HAA5 ratio of 0.4 µg/µg. ( $\text{NH}_2\text{Cl}$  was much better at controlling the formation of the THMs and TCAA than that of DCAA in this groundwater, which is consistent with research elsewhere for surface waters.) This ratio was used to predict HAA5 for other groundwaters that use  $\text{NH}_2\text{Cl}$ . It was also used for nonalluvial groundwaters, as some of those systems had  $\text{NH}_2\text{Cl}$  residuals. For example, the average TTHM occurrence for nonalluvial groundwaters was 0.9 µg/L. Thus, it was predicted that such a water would have  $0.9/0.4 = 2.2$  µg/L HAA5. However, when an alluvial and nonalluvial



groundwater were mixed, sometimes BDCM would predominate over  $\text{CHCl}_3$  (e.g., Waterloo). Thus, BCAA should predominate over DCAA. Although BCAA is a component of HAA6, it is not part of HAA5; in such cases—in which low levels of TTHM were produced and BDCM predominated—it was assumed that the TTHM/HAA6 ratio was  $0.4 \mu\text{g}/\mu\text{g}$ ; thus, for Waterloo ( $3.4 \mu\text{g}/\text{L}$  TTHM),  $\text{HAA6} = 3.4/0.4 = 8.2 \mu\text{g}/\text{L}$ .

Finally, data on HAA speciation (specifically DCAA, TCAA, and BCAA) were developed.

For alluvial groundwaters that use  $\text{Cl}_2$ , the HAA speciation is pH-dependent (a set of predictive equations were developed based on data in the Iowa database). The average pH for the nonsoftening alluvial groundwaters is 7.6 (Table 7.21). At this pH, 37% of the HAA5 should be DCAA, and 53% of the HAA5 should be TCAA (on a central tendency basis); for example, for an average nonsoftening alluvial groundwater with  $4.6 \mu\text{g}/\text{L}$  HAA5, the average speciation would be 1.7 and  $2.5 \mu\text{g}/\text{L}$  of DCAA and TCAA, respectively. The average pH for the softening alluvial groundwaters is 8.7 (Table 7.21). At this pH, 59% of the HAA5 should be DCAA and 25% of the HAA5 should be TCAA (on a central tendency basis); for example, for an average softening alluvial groundwater with  $5.7 \mu\text{g}/\text{L}$  HAA5, the average speciation would be 3.4 and  $1.4 \mu\text{g}/\text{L}$  of DCAA and TCAA, respectively.

For groundwaters that use  $\text{NH}_2\text{Cl}$  or for alluvial groundwaters similar to Cedar Rapids, it was assumed that 100% of the HAA5 was DCAA (or DCAA + BCAA). However, TCAA was listed at its minimum reporting level of  $0.5 \mu\text{g}/\text{L}$ . For example, for an average nonalluvial groundwater with  $2.2 \mu\text{g}/\text{L}$  HAA5, the average speciation would be 2.2 and  $0.5 \mu\text{g}/\text{L}$  of DCAA and TCAA, respectively.

After DCAA was predicted, BCAA was predicted based on the formula  $\text{BCAA} = \text{DCAA} \times (\text{BDCM}/\text{CHCl}_3)$  on a molar basis. For example, for an average nonsoftening alluvial groundwater with 5.7, 4.4, and  $1.7 \mu\text{g}/\text{L}$  of  $\text{CHCl}_3$ , BDCM, and DCAA, respectively, BCAA is predicted to be  $1.3 \mu\text{g}/\text{L}$  (and HAA6 would then be  $5.9 \mu\text{g}/\text{L}$ ). For example, for an average softening alluvial groundwater with 6.8, 5.9, and  $3.4 \mu\text{g}/\text{L}$  of  $\text{CHCl}_3$ , BDCM, and DCAA, respectively, BCAA is predicted to be  $2.9 \mu\text{g}/\text{L}$  (and HAA6 would then be  $8.6 \mu\text{g}/\text{L}$ ). For example, for an average nonalluvial groundwater with 0.6,  $<0.5$ , and  $2.2 \mu\text{g}/\text{L}$  of  $\text{CHCl}_3$ , BDCM, and DCAA, respectively, BCAA is predicted to be  $0.0 \mu\text{g}/\text{L}$  (and HAA6 would then be  $2.2 \mu\text{g}/\text{L}$ ).

However, when alluvial and nonalluvial groundwaters were mixed, and BDCM predominated over  $\text{CHCl}_3$ , HAA6 was first predicted (rather than HAA5). In such cases—in which low levels of TTHM were produced—it was assumed that  $\text{HAA6} = \text{DCAA} + \text{BCAA}$  and that TCAA was negligible. Then it was assumed that  $\text{DCAA} = \text{HAA6} \times \text{CHCl}_3/(\text{CHCl}_3 + \text{BDCM})$  and  $\text{BCAA} = \text{HAA6} \times \text{BDCM}/(\text{CHCl}_3 + \text{BDCM})$ . For example, for Waterloo, with 0.6, 1.4, and  $8.2 \mu\text{g}/\text{L}$  of  $\text{CHCl}_3$ , BDCM, and HAA6, respectively, DCAA and BCAA were predicted to be 2.3 and  $5.9 \mu\text{g}/\text{L}$ , respectively.

If nothing is known about a groundwater in Iowa, the following predictions (Table 7.21) could be made based on the central tendency observations for each group of waters. These predictions are supported by the water quality and THM data summarized in Table 7.22.

**Table 7.22**  
**Water quality and THM data for groundwaters in the Iowa hit list**

Utility	Depth (ft)	Current treatment	Raw TOC (mg/L)	Raw NH <sub>3</sub> -N (mg/L)	Finished TOC (mg/L)	Finished pH	Finished CHCl <sub>3</sub> (µg/L)	Finished BDCM (µg/L)	Finished TTHM (µg/L)	Comments
<b>Nonalluvial groundwaters</b>										
Cedar Falls	116–227	Cl <sub>2</sub>				7.0	0	0	0	Limited data
Marion	1,565–1,663	Aeration/ oxidation, Cl <sub>2</sub>		0.05	0.75	7.8	0.2	0.2	0.2	
Indianola	2,435–2,590	Aeration/oxidation, lime softening, Cl <sub>2</sub>			1.0	9.1	0.4	0.3	0.8	
Mason City	865–1,538	Aeration/ oxidation, Cl <sub>2</sub>			1.2	7.6	1.0	0.5	1.9	
Clinton	1,800–2,375	Cl <sub>2</sub> , aeration/ oxidation, Cl <sub>2</sub>			0.29	7.2	0.5	0.5	0.5	Total Cl <sub>2</sub> residual >> free Cl <sub>2</sub>
Fort Dodge	422–2,307	Aeration/oxidation, Cl <sub>2</sub> , filtration		0.82	0.93	7.6	1.0	0.5	2.0	
Marshalltown	156–270	Aeration/oxidation, lime softening, Cl <sub>2</sub> (2 mg/L)		1.1	1.2	8.8	0.6	0.5	1.0	NH <sub>2</sub> Cl residual = 2.1 mg/L as Cl
Ames	83–147	Aeration/oxidation, lime softening, Cl <sub>2</sub>		0.4	1.8	9.4	1.8	0.5	2.2	NH <sub>2</sub> Cl residual = 1.7 mg/L as Cl
Count		8		4.0	7	8	8	8	8	3
25th percentile				0.3	0.8	7.5	0.4	0.3	0.4	
Median		Aeration/oxidation, Cl <sub>2</sub>		0.6	1.0	7.7	0.6	0.5	0.9	NH <sub>2</sub> Cl residual
75th percentile		Aeration/oxidation, lime softening, Cl <sub>2</sub>		0.9	1.2	8.9	1.0	0.5	1.9	
<b>Alluvial groundwaters</b>										
Des Moines		Softening, Cl <sub>2</sub>								
West Des Moines	22–45	Cl <sub>2</sub> , lime softening								
Winterset	30	Cl <sub>2</sub>					0	0	0	

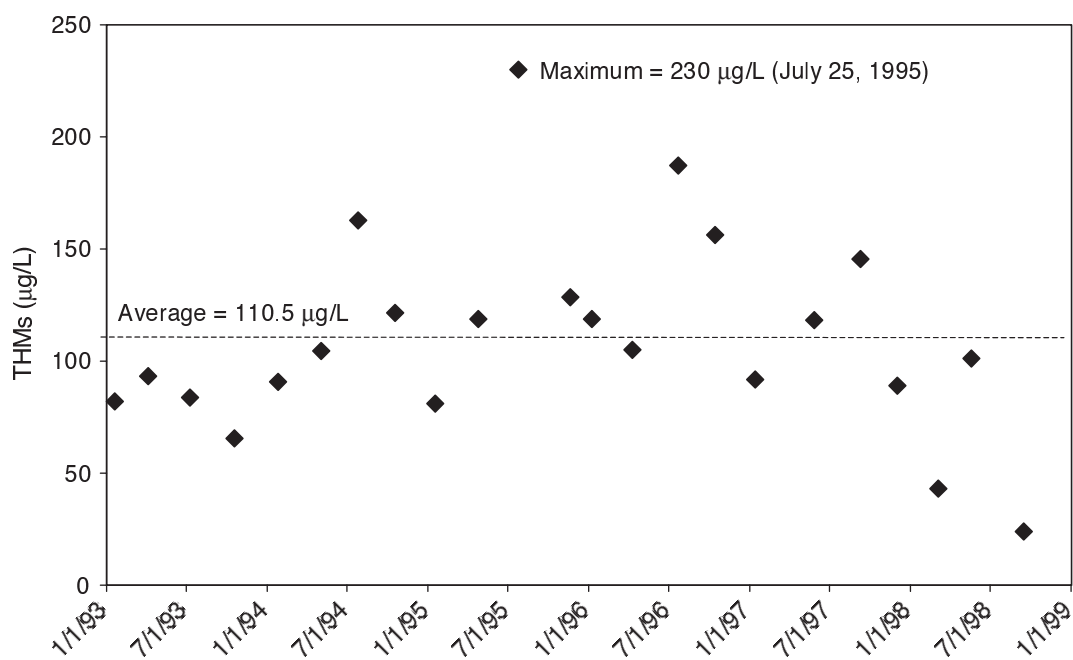
(Continued)



Table 7.22 (Continued)

Nonalluvial groundwaters			Raw TOC (mg/L)	Raw NH <sub>3</sub> -N (mg/L)	Finished TOC (mg/L)	Finished pH	Finished CHCl <sub>3</sub> (µg/L)	Finished BDCM (µg/L)	Finished TTHM (µg/L)	Comments
Utility	Depth (ft)	Current treatment								
Cedar Rapids		NH <sub>2</sub> Cl, lime softening			1.2	9.4	1.0	0.4	4.5	1995: pH = 8.2, TTHM = 0.9 µg/L
Muscatine		Cl <sub>2</sub>			1.0	7.6	2.0	2.0	6.0	
Newton		Lime softening, Cl <sub>2</sub>			0.95	9.5	4.2	3.9	12.2	
Spencer	24–80	Lime softening, Cl <sub>2</sub>			1.7	8.4	6.1	5.2	15.9	1984/87: TTHM = 20.0/11.8 µg/L
Boone	32–67	Greensand, Cl <sub>2</sub>			2.4	7.5	9.3	6.7	19.9	
Oscalooosa	40–59	Lime softening, Cl <sub>2</sub>			0.86	8.3	10.2	8.5	24.9	
Count		9			6.0	6	7.0	7.0	7.0	
25th percentile		Cl <sub>2</sub>			1.0	7.8	1.5	1.2	5.3	
Median		Lime softening, Cl <sub>2</sub>			1.1	8.4	4.2	3.9	12.2	
75th percentile					1.6	9.2	7.7	6.0	17.9	
Average/softening + Cl <sub>2</sub>					1.2	8.7	6.8	5.9	17.7	
Average/nonsoftening (except Winterset)					1.7	7.6	5.7	4.4	13.0	
<b>Mixed groundwaters</b>										
	% Alluvial									
Waterloo	32	Cl <sub>2</sub>	0.6–1.1	0.17		7.5	0.6	1.4	3.4	
Sioux City	48	Aeration/oxidation, Cl <sub>2</sub> , filtration, Cl <sub>2</sub>		0.31	2.2	7.4	8.5	10.0	28.9	
Dubuque	65	Aeration/oxidation, lime softening, Cl <sub>2</sub>	2.0–4.6	0.53	1.5	8.7	12.0	2.8	15.3	

Note: Blank cells indicate information was not available.



**Figure 7.9 Occurrence of TTHM at Mount Ayr, 1993–1998**

### Small Surface and Mixed Surface/Groundwater Systems not on Iowa Hit List

In previous sections, DBP predictions were developed:

- For  $\text{CHCl}_3$ , BDCM, TTHM, DCAA, TCAA, HAA5, BCAA, and HAA6 (HAA5 + BCAA).
- For the surface waters, mixed surface/groundwaters, and alluvial and nonalluvial groundwaters in the Iowa hit list

Appendix 7.3 summarizes DBP predictions for the non-hit list surface and mixed surface and groundwater systems in Iowa. The DBP predictions were initially set to zero until chlorine was first introduced (1920–1975). Whenever there was a significant change in treatment and disinfection and/or source water, a new set of DBP predictions were made.

Figure 7.9 shows the occurrence of TTHM at a small utility (Mount Ayr, population = 1,796) that treats a surface water (Loch Ayr) with a moderate amount of TOC (4.8 mg/L in the finished water on May 26, 1987) with free chlorine. Note, utilities that serve more than 10,000 people were not required to comply with the 1979 THM Rule.

In this example, TTHM in 1993–1998 ranged from 24 to 230 µg/L (25th to 75th percentiles = 86–125 µg/L), with an average of 110 µg/L. The maximum occurrence was in July 1995. In the 1987 database in Cantor et al. (1998), TTHM occurrence (on 5/26/87) was 188 µg/L. The latter value is more representative of peak occurrence than average occurrence. For this utility, it was assumed that historical TTHM occurrence on an annual average basis was 110 µg/L.

Alternatively, Bloomfield (population = 2,580) currently treats a surface water (Lake Fisher) with a moderate amount of TOC (4.5 mg/L in the finished water on June 3, 1987) with

chloramines. TTHM in 1993–1998 ranged from 22 to 297 µg/L (25th to 75th percentiles = 45–71 µg/L), with an average of 66 µg/L. In the 1987 Cantor et al. (1998) database, TTHM occurrence (on June 3, 1987) when free chlorine was used was 160 µg/L. The latter value is representative of peak occurrence with the use of free chlorine (similar to that of Mount Ayr). Based on the hit list of surface water utilities, it was found (on average) that annual average occurrence was 80% of the value in the 1987 Cantor et al. (1998) database. For Bloomfield, it was assumed that historical TTHM occurrence with free chlorine on an annual average basis was 128 µg/L ( $80\% \times 160 \mu\text{g/L}$ ) (which is similar to that of Mount Ayr).

Greenfield (population = 2,074) currently treats a blend of surface water (Lake Greenfield) and shallow groundwater. Based on two sample dates in 1994 and 1996, while using chlorine, the average TTHM was 44 µg/L. In the 1987 Cantor et al. (1998) database, TTHM occurrence (on May 20, 1987), when 100% surface water was treated, was 91 µg/L. For this utility, it was assumed that historical TTHM occurrence when treating 100% surface water with free chlorine on an annual average basis was 73 µg/L ( $80\% \times 91 \mu\text{g/L}$ ).

Based on the hit list of alluvial groundwater systems, the average TTHM occurrence for nonsoftening systems was 13 µg/L. In 1992, when Greenfield was using 40% shallow groundwater, it was assumed that the distributed water had a concentration of TTHM based on the surface and groundwater blend:  $60\% \times 73 \mu\text{g/L} + 40\% \times 13 \mu\text{g/L} = 49 \mu\text{g/L}$ . This predicted value is quite similar to the limited 1994–1996 occurrence data.

A number of the small, surface water utilities (e.g., Seymour) were served by Rathbun Rural. In 1976, Rathbun Rural's water was 33% from Centerville, 33% from Chariton, and 34% from deep groundwater. TTHM for Centerville and Chariton were 136 and 122 µg/L, respectively, for water treated in this time period (1962–1989 and 1958–1981, respectively). Based on the hit list of nonalluvial groundwater systems, the median TTHM occurrence was 0.9 µg/L. In 1976, it was assumed that the distributed water for Rathbun Rural had a concentration of TTHM based on the surface and groundwater blend:  $33\% \times 136 \mu\text{g/L} + 33\% \times 122 \mu\text{g/L} + 34\% \times 0.9 \mu\text{g/L} = 85 \mu\text{g/L}$ .

Subsequently (starting in 1977), Rathbun Rural switched to 100% treatment of water from Lake Rathbun Reservoir. In 1989, it started to use chlorine dioxide for predisinfection. TTHM in 1993–1996 ranged from 8 to 100 µg/L (25th to 75th percentiles = 23–48 µg/L), with an average of 41 µg/L. This average THM occurrence was used to predict historical levels before the use of chlorine dioxide. Moving backward in time, in 1986, it did not use chlorine dioxide or any predisinfectant (applying chlorine to the settled water). Based on the hit list of surface water utilities, it was found (on a median basis) that the use of chlorine dioxide reduced THM formation by 37%. Thus, it was assumed that TTHM at Rathbun Rural in 1986 were  $41 \mu\text{g/L} / (1 - 0.37) = 65 \mu\text{g/L}$ . Continuing to move backward in time, in 1979, it used chlorine as a predisinfectant (applying chlorine to the raw water). Based on the hit list of surface water utilities, it was found that moving the point of chlorine addition reduced THM formation by 13%. Thus, it was assumed that TTHM at Rathbun Rural in 1979 was  $65 \mu\text{g/L} / (1 - 0.13) = 74 \mu\text{g/L}$ .

On the hit list, most of the lake and reservoir systems (Osceola, Chariton, Centerville, Creston) produced >100 µg/L TTHM when using chlorine without any alternative disinfectants. However, Spirit Lake in the northwest portion of the state only produced 25 or 46 µg/L TTHM. The four aforementioned high-THM systems are all in the southern portion of the state. Likewise, in the non-hit list utilities, some of the high-THM systems (e.g., Mount Ayr, Bloomfield) are in the southern portion of the state. Similarly, some other low-THM systems (Milford: average = 30 µg/L, Arnolds Park in July 1997 = 39 µg/L) treat water from West Okoboji Lake, which is near

Spirit Lake. This may (in part) reflect a difference in the reactivity of the watershed precursors in the northwest portion of the state.

In the non-hit list utilities, if no data existed for any of the lake or reservoir systems, it was assumed that they produced similar levels of THMs to nearby systems when using the same treatment and disinfection scenario. For example, Okoboji, which treats water from the West Okoboji Reservoir, was assumed to produce similar amounts of THMs as that of Milford. Alternatively, Afton, which previously treated water from Afton Lake with chlorine only, was assumed to produce similar amounts of THMs as that of Creston (12-Mile Reservoir) when Creston used chlorine only (in 1986). Afton subsequently switched to getting its water from Creston (in 1994, when it used chlorine dioxide for predisinfection).

Since 1972 Mount Pleasant has treated a combination of surface water (Skunk River) and deep groundwater. The treatment processes for the two source water types are different, suggesting that they are treated separately, and the treated waters are blended in the distribution system. In the Iowa DNR database, there are multiple sets of THM samples for each sample data. Most of the data are very low (average and maximum = 0.5 and 3  $\mu\text{g/L}$ , respectively) and another portion is quite high (average and maximum = 72 and 124  $\mu\text{g/L}$ , respectively). Most likely Mount Pleasant collects separate samples for the treated surface water and treated groundwater. Although some consumers in Mount Pleasant may receive groundwater and some may receive surface water, while others get a blend of the two, it was assumed that all get blended water (at least on an annual average basis over time). Because surface water only represents a small portion (15%–17%) of the treated water, most of the consumers in this system should have received relatively low THM exposure from the high groundwater usage. It was predicted that the average THM exposure in the distribution system (over time) was 11–13  $\mu\text{g/L}$  based on a blend of treated surface and groundwater.

In terms of THM speciation, it was assumed that the relative site-specific current speciation could be used for predicting historical speciation. For example, in 1993–1998, Bloomfield (with chloramines) had (on average) 86% chloroform and 13% BDCM. In 1987 (with chlorine), it had 88% chloroform and 11% BDCM. Thus, it was assumed that historical THM speciation was 87% chloroform and 12% BDCM. In contrast, Greenfield in 1987 (with surface water only) had 77% chloroform and 20% BDCM. In 1994–1996, when 40% groundwater was treated, the THM speciation of the blended water was 61% chloroform and 31% BDCM. This is probably due to the higher bromine incorporation in most Iowa alluvial groundwaters. For example, on the hit list of alluvial groundwaters with similar treatment (nonsoftening), the median THM speciation was 44% chloroform and 34% BDCM. In 1989, when the percentage of groundwater was lower (i.e., 25%), it was assumed that the surface and groundwater blend had a speciation between that of the surface water only and that of the higher groundwater blend (i.e., 69% chloroform and 25% BDCM).

The median ratio of THMs to HAA5 was 1.7  $\mu\text{g}/\mu\text{g}$  for small, surface water systems in USEPA Regions 5 and 7 (Iowa is in region 7; some of the expanded ICR–Iowa database utilities are in Region 5). These utilities used chlorine only and conventional treatment (median pH = 7.4). These data were obtained from a database used during the negotiation of the U.S. DBP regulation. This TTHM/HAA5 ratio was used for the small, surface water systems not on the Iowa hit list when they used chlorine only. Alternatively, the median ratio of THMs to HAA5 in the expanded ICR–Iowa database was 2.3  $\mu\text{g}/\mu\text{g}$ . Many of the latter systems used alternative disinfectants. Thus, this ratio was used for the small, surface water systems when they switched to alternative disinfectants.

For example, Bloomfield was predicted to have 128  $\mu\text{g/L}$  TTHM when using chlorine and 66  $\mu\text{g/L}$  when using chloramines. Thus, it was predicted that it had 75  $\mu\text{g/L}$  HAA5 (128  $\mu\text{g/L}/1.7$ ) when using chlorine and 29  $\mu\text{g/L}$  HAA5 (66  $\mu\text{g/L}/2.3$ ) when using chloramines. However, for

**Table 7.23**  
**THM levels by type of water source and treatment**

Source	No chlorination	Prefilter-chlorination	Postchlorination only
	(µg/L)		
Surface		73.9	38.1
Well <15 m	0.6	13.8	3.2
Well 15–46 m	0.6	2.0	3.9
Well 46–152 m	0.5	1.8	1.4
Well >152 m	0.5	0.7	0.9

Note: Geometric mean—Iowa, 1987.

Blank cells indicate information was not available.

Milford and other utilities treating water from West Okoboji Lake, it was assumed they had a similar TTHM/HAA5 ratio as that of nearby Spirit Lake (i.e., 2.0 µg/µg).

Next, DCAA and TCAA speciation were predicted based on the central tendency impact of finished water pH on the DCAA/HAA5 and TCAA/HAA5 ratios (from the expanded ICR—Iowa database), which reflect the impact of increasing pH on lowering TCAA formation. For example, in 1986, Rathbun Rural had an average pH of 8.3 and was predicted to produce 28 µg/L HAA5. At pH 8.3, DCAA and TCAA (on a central tendency basis) are 49% and 32% of HAA5, respectively. Thus, it was predicted that Rathbun Rural in 1986 had 14 (49% × 28) and 9 (32% × 28) µg/L of DCAA and TCAA, respectively. In 1997, Rathbun Rural had an average pH of 9.2 and was predicted to produce 19 µg/L HAA5. At pH 9.2, DCAA and TCAA (on a central tendency basis) are 70% and 20% of HAA5, respectively. Thus, it was predicted that Rathbun Rural in 1997 had 13 (70% × 19) and 4 (20% × 19) µg/L of DCAA and TCAA, respectively.

Finally, BCAA was predicted based on the concentration of DCAA and the relative speciation of chloroform and BDCM on a molar basis. For example, in 1994, Bloomfield had 57 and 9 µg/L of chloroform and BDCM, respectively, and it was predicted that DCAA and HAA5 were 14 and 29 µg/L, respectively. From these data, it was predicted that BCAA was 2 µg/L, which results in an HAA6 of 31 µg/L (29 + 2).

## COMPARISON OF THM PREDICTIONS FOR UTILITIES IN IOWA: ORIGINAL VERSUS REANALYSIS

The original THM predictions in Iowa were based on the Cantor et al. (1998) database in which each utility was sampled in the spring or summer for THMs. For each type of source water and treatment, the geometric mean TTHM occurrence was calculated (Table 7.23). These values were used to assign a central tendency prediction for historical TTHM occurrence for each water type and treatment.

In order to improve the exposure assessment for Iowa, various databases were assembled with THM data spanning the years 1975 to 2000 (Table 7.24).

**Table 7.24**  
**THM databases used in Iowa reanalysis**

Database	Dates	Number of Iowa plants
NORS	1975	3
NOMS	1976–1977	2
Cantor et al. (1998) database	1987	356
WIDB (1992)	1990–1991	19
Iowa DNR	1993–1998	811
CHEEC Iowa hit list	1979–2000	34
AWWA WATER:\STATS	1996–1997	12
ICR	1997–1998	3
Project-related survey	1979–2000	22

A case study approach was conducted in which each of the Iowa utilities (Iowa hit list and all of the remaining surface and mixed surface/groundwater utilities) was examined one by one. Site-specific THM predictions were prepared for each of these utilities based on current data and the impact of changes in source water and/or treatment and disinfection processes over time on THM formation.

Table 7.25 shows a comparison of THM predictions (original versus reanalysis) for surface water utilities in Iowa. Historically, some of the surface water utilities used groundwater or initially treated surface water without any disinfectant. The median TTHM predictions for this subgroup of surface water utilities for both the original and the reanalysis were  $\leq 0.5$   $\mu\text{g/L}$ . The only anomalous prediction for this subgroup was for Keosauqua in 1987. According to the CHEEC database, it switched from the treatment of deep well water to surface water (Lake Rathbun Reservoir) sometime in 1987. In May/June 1987, when Iowa utilities were sampled for THMs for the 1987 database in Cantor et al. (1998), Keosauqua was using well water. The reanalysis prediction was 65  $\mu\text{g/L}$  based on treatment of surface water. The original prediction (12  $\mu\text{g/L}$ ) was probably partly based on the use of groundwater for part of 1987. Because the exact date on which treatment or source water changes were made are not available in the CHEEC database, there will be some error in predicting DBPs for the year in which the change was made. However, because this study is based on many decades of exposure, the error in part of a year should not significantly impact the overall exposure assessment.

For the subgroup of surface water utilities that did not use prechlorination, the central tendency original TTHM prediction was 38  $\mu\text{g/L}$ . In the reanalysis, the 25th percentile and median predictions for this subgroup were 31 and 48  $\mu\text{g/L}$ , respectively, which correspond to  $-18\%$  and  $+25\%$  differences to 38  $\mu\text{g/L}$ , respectively. However, the 75th percentile and maximum predictions were 122 and 136  $\mu\text{g/L}$ , respectively, which correspond to  $220\%$  and  $257\%$  differences, respectively. For example, Centerville had TTHM in 1987–1989 of 86–186  $\mu\text{g/L}$  (average = 136  $\mu\text{g/L}$ ) because it postchlorinated a water with a finished water TOC (in July 1987) of 5.1 mg/L, which is relatively high. Likewise, Chariton had TTHM in 1987–1988 of 122–135  $\mu\text{g/L}$  (average = 129  $\mu\text{g/L}$ ), because it postchlorinated a water with a finished water TOC (in June 1987) of



**Table 7.25**  
**Comparison of THM predictions for surface water utilities in Iowa:**  
**Original versus reanalysis**

Statistic	Original Br-THMs	Reanalysis BDCM	Original TTHM	Reanalysis TTHM	Difference in Br-THMs (%)	Difference in TTHM (%)
<b>Primarily wells and/or no disinfectant</b>						
Minimum	0.5	0.0	0.5	0.0		
25th percentile	0.5	0.0	0.5	0.0		
Median	0.5	0.0	0.5	0.0		
Average	0.5	0.6	0.5	3.4		
75th percentile	0.5	0.0	0.5	0.0		
Maximum	0.6	12.1	0.7	64.6	1,917	9,838
<b>No prechlorination</b>						
Minimum	7.4	6.4	38.1	24.9	-13	-35
25th percentile	7.4	8.9	38.1	31.2	21	-18
Median	7.4	10.6	38.1	47.6	44	25
Average	7.4	14.8	38.1	75.6	101	99
75th percentile	7.4	22.7	38.1	122.0	208	220
Maximum	7.4	28.7	38.1	136.0	290	257
<b>Includes prechlorination</b>						
Minimum	13.4	0.0	73.9	2.0	-100	-97
25th percentile	13.4	10.6	73.9	62.4	-21	-16
Median	13.4	12.9	73.9	69.6	-4	-6
Average	13.4	14.3	73.9	86.2	7	17
75th percentile	13.4	17.1	73.9	124.2	27	68
Maximum	13.4	33.0	73.9	219.4	146	197

Note: Blank cells indicate information was not available.

5.3 mg/L. Although postchlorination will minimize THM formation, it will still be high if the finished water TOC is high.

For the subgroup of surface water utilities that use pre- or intermediate (prefiltration) chlorination, the central tendency original TTHM prediction was 74 µg/L. In the reanalysis, the 25th percentile and median predictions for this subgroup were 62 and 70 µg/L, respectively, which correspond to -16% and -6% differences to 74 µg/L, respectively. However, the 75th percentile and maximum predictions were 124 and 219 µg/L, respectively, which correspond to 68% and

197% differences, respectively. For example, Lamoni had TTHM in 1987–1993 of 44–379  $\mu\text{g/L}$  (average = 191  $\mu\text{g/L}$ ) because it intermediate-chlorinated a water with a finished water TOC (in May 1987) of 7.6 mg/L. Note, for Davenport, its average TTHM formation in 1982–1994 with intermediate-chlorination and postchloramination was 71  $\mu\text{g/L}$ . In the May 1987 database in Cantor et al. (1998), its TTHM formation was 75  $\mu\text{g/L}$ . However, these values do not reflect historical THM formation when chlorine only was used, which was predicted to be 116  $\mu\text{g/L}$ .

Table 7.26 shows a comparison of THM predictions (original versus reanalysis) for groundwater utilities in Iowa. Historically, some of the groundwater utilities used surface water. The median TTHM prediction for the subgroup of groundwater utilities that primarily used well water for both the original and the reanalysis was 1.0  $\mu\text{g/L}$ . However, the predictions for some groundwater utilities significantly differed in the two analyses. For example, Sioux City had TTHM in 1987–2000 of 11–53  $\mu\text{g/L}$  (average = 29  $\mu\text{g/L}$ ), due to the treatment of a high blend (48%) of alluvium Dakota groundwater.

For the subgroup of groundwater utilities (i.e., Cedar Rapids) that used surface water, the original and reanalysis predictions for the postchlorination of Cedar River water were 38 and 52  $\mu\text{g/L}$ , respectively. However, when this water was treated with prechloramination (no free chlorine contact time), the predictions were 74 and 3  $\mu\text{g/L}$ , respectively. The original predictions treated prechlorination and prechloramination the same, because on a central tendency basis this was an atypical practice. However, in the case study approach, the control of THM formation with chloramines only was utilized in making a site-specific prediction.

Figure 7.10 and Table 7.27 show a comparison of THM predictions (original versus reanalysis) for mixed surface and groundwater utilities in Iowa. This group of utilities was segmented into three subgroups based on the range of original TTHM predictions (<20  $\mu\text{g/L}$ , 30–40  $\mu\text{g/L}$ , and >50  $\mu\text{g/L}$ ). For example, Burlington in 1987 treated a mixture of Mississippi River and groundwater. The original and reanalysis predictions were 31 and 37  $\mu\text{g/L}$ , respectively. In 1960, Burlington treated Mississippi River water with intermediate chlorination and postchloramination. The original and reanalysis predictions were 74 and 71  $\mu\text{g/L}$ , respectively. In 1920, Burlington treated Mississippi River water with prechlorination and filtration, but no coagulation/sedimentation. Although filtration will remove particulate organic carbon, it will not remove DOC without coagulation. In most surface waters, the DOC is the major portion of the TOC. The original and reanalysis predictions were 74 and 156  $\mu\text{g/L}$ , respectively. Because none of the surface water plants in Iowa in 1987 used filtration only, this lack of physical treatment could not be incorporated into the original central tendency predictions. However, this was handled in the case study approach.

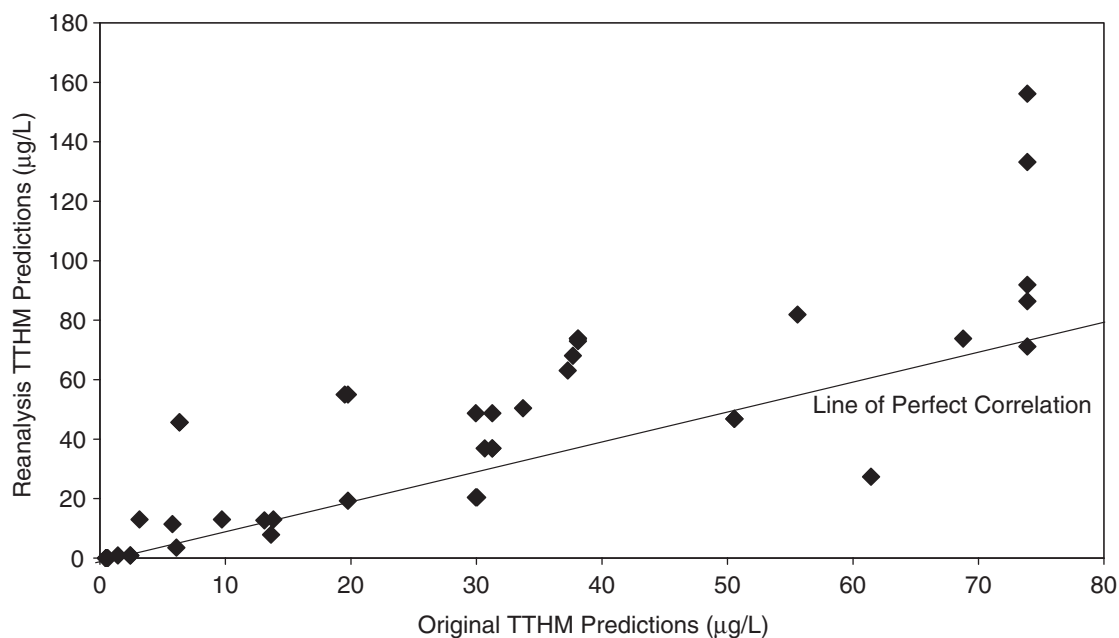
Tables 7.25 through 7.27 also provide comparisons of brominated THM predictions. The original predictions were based on the sum of all three Br-containing THMs (Br-THMs), whereas the reanalysis only focused on BDCM. For the surface waters, most of the sum of the Br-THMs was BDCM, whereas many of the alluvial groundwaters had a greater mixture of other Br-THMs. Nonetheless, these tables show that the original Br-THMs predictions tended to agree with the reanalysis BDCM predictions on a median basis. However, on a site-specific basis, the predictions for the Br-THMs varied in certain cases. For example, for Burlington in 1987 the original prediction for Br-THMs was 6  $\mu\text{g/L}$ , and the reanalysis BDCM prediction was 8  $\mu\text{g/L}$ . However, for Burlington in 1920 and 1960, the original prediction for Br-THMs was 13  $\mu\text{g/L}$ , and the reanalysis predictions were 6 and 18  $\mu\text{g/L}$ , respectively. In the reanalysis, it was predicted that coagulation and postchloramination in 1960 controlled the formation of BDCM (as well as TTHM), whereas prechlorination and no coagulation in 1920 resulted in a significant increase in BDCM (as well as TTHM).



**Table 7.26**  
**Comparison of THM predictions for groundwater utilities in Iowa: Original versus reanalysis**

Statistic	Original Br-THMs	Reanalysis BDCM	Original TTHM	Reanalysis TTHM	Difference in Br-THMs (%)	Difference in (TTHM) (%)
<b>Primarily wells and/or no disinfectant</b>						
Minimum	0.5	0.0	0.5	0.0		
25th percentile	0.5	0.0	0.6	0.0		
Median	0.7	0.5	1.0	1.0	-34	6
Average	1.2	1.6	1.8	5.0	32	176
75th percentile	2.4	1.9	3.2	6.0	-20	90
Maximum	4.4	10.0	7.8	28.9	128	270
<b>Surface water</b>						
Minimum	7.4	0.5	38.1	3.1	-93	-92
Average	10.4	3.2	56.0	27.3	-70	-51
Maximum	13.4	5.8	73.9	51.5	-57	-30

Note: Blank cells indicate information was not available.



**Figure 7.10 Comparison of TTHM predictions for mixed surface/groundwater utilities in Iowa: Original versus reanalysis**

**Table 7.27**  
**Comparison of THM predictions for mixed surface/groundwater**  
**utilities in Iowa: Original versus reanalysis**

	Original Br-THMs	Reanalysis BDCM	Original TTHM	Reanalysis TTHM		
Statistic	(µg/L)				Difference in Br-THMs (%)	Difference in TTHM (%)
Primarily wells and/or no disinfectant						
Minimum	0.5	0.0	0.5	0.0		
25th percentile	0.5	0.0	0.5	0.0		
Median	1.1	0.5	2.4	0.9	−55	−63
Average	1.9	2.7	5.8	11.4	40	98
75th percentile	2.7	3.4	9.7	13.0	25	34
Maximum	8.0	14.6	19.8	55.0	83	178
Primarily no prechlorination						
Minimum	5.7	4.0	30.0	20.4	−30	−32
25th percentile	6.3	7.7	30.7	36.9	22	20
Median	6.4	8.4	31.3	48.7	32	56
Average	6.6	10.9	33.7	50.4	63	50
75th percentile	7.2	15.2	37.7	68.1	112	81
Maximum	7.4	17.6	38.1	73.8	139	94
Includes prechlorination						
Minimum	9.9	6.4	50.6	27.3	−35	−46
25th percentile	10.5	8.4	57.0	52.9	−19	−7
Median	13.0	13.4	71.3	77.9	3	9
Average	12.1	12.8	65.6	81.6	6	24
75th percentile	13.4	17.1	73.9	90.5	27	23
Maximum	13.4	17.7	73.9	156.2	32	111

Note: Blank cells indicate information was not available.

## **CHAPTER 8**

# **REANALYSIS OF ONTARIO CASE-CONTROL STUDY: BLADDER, COLON, AND RECTAL CANCER RISKS ASSOCIATED WITH TTHM AND SPECIFIC CHLORINATION BY-PRODUCT COMPOUNDS**

Epidemiologic studies have examined the association of chlorination by-products and a number of cancer sites. Cancer of the bladder, colon, and rectum have been the focus of the largest number of studies. The sum of evidence from epidemiologic studies conducted to date supports a modest increase in the risk of bladder cancer associated with exposure to chlorination by-products. Studies of colon and rectal cancer are less consistent (Cantor et al. 1998; King, Marrett, and Woolcott 2002).

Recent expert panel reviews in Canada and the United States have identified specific research needs, with improved exposure assessment a priority for epidemiologic studies (Mills et al. 1998). Examination of specific by-products and dose responses are also areas where information is lacking with respect to understanding this relationship (Mills et al. 1998).

The Ontario case-control study observed a dose-response pattern between TTHM exposure and bladder cancer and male colon cancer but none with rectal cancer (King and Marrett 1996; King, Marrett, and Woolcott 2000). Subject exposure was determined by linking residence and water use history to a matrix of TTHM estimates by time and location. The exposure matrix was based on the application of a regression model predicting TTHM level based on historical source characteristics and treatment practices. No estimation was performed for specific THM compounds or other by-product classes.

The basic exposure matrix in the Ontario case-control study was recalculated in order to provide more precise estimates of total TTHM exposure and to provide estimates of exposure to specific THMs and total and specific HAAs. This chapter presents the reanalysis of bladder, colon, and rectal cancer risk in the Ontario case-control study and a comparison to previous results.

## **METHODS**

The methods of the original case-control study have been described in detail elsewhere (King and Marrett 1996; King, Marrett, and Woolcott 2000) and will be summarized here. A population-based case-control study of cancers of the bladder, colon, and rectum was conducted. Individuals in the cases were 30 to 74 years old and residents of Ontario, with a primary cancer of the bladder, colon, or rectum. To identify control subjects, households were randomly selected from telephone listings, and a census of the household led to the identification of an eligible subject. Questionnaires were completed on 927 bladder cases, 971 colon cases, 875 rectal cases, and 2,118 controls. The questionnaire ascertained information on residence and water use history as well as established and suspected risk factors for the cancers of interest.

A database was created that characterized each water supply in the study area according to water source, treatment, and estimated TTHM level in 5-year time intervals. The estimation of TTHM levels was based on a survey that collected information on source and treatment characteristics between 1950 and 1990. Water plant information was collected for an average day in August, which was intended to represent peak levels of TTHM. A model based on monitoring data from the Ontario DWSP for the years 1986–1992 was created, which predicted TTHM levels

based on water source and treatment information. This model was applied to the historical information collected in the survey of Ontario treatment plants in order to estimate TTHM levels for each plant and time period.

Exposures were assigned to individuals by linking their residence and water source histories to the relevant treatment plant estimates. Exposures occurring in a 40-year time window beginning 2 years prior to the subject interview were considered in the original analysis. The main analysis included exposure metrics representing duration of exposure to a peak TTHM level over a specified value (e.g., years with a level of 50 µg/L or greater) and cumulative TTHM exposure (e.g., the sum for all residences of peak TTHM level multiplied by years at the residence).

## ANALYSIS

The original and new total THM predictions for each water plant were compared via a Pearson correlation coefficient. This contrast was also made with respect to different types of water supply and population characteristics.

An exposure metric similar to that used in the original study was applied in order to compare risk estimates obtained in the earlier publications from this study. The new exposure estimates were based on yearly average TTHM values, and the original estimates were based on peak values. Thus, it was necessary in the reanalysis of cancer risks to consider the different TTHM exposure estimates. For Ontario, yearly average values for TTHM are approximately 80% of peak values (King and Marrett 1996). To facilitate comparisons of risks with previous exposures the reanalysis considered comparable values. For example, exposures to 50 µg/L TTHM in the original analysis would be equivalent to 40 µg/L in the reanalysis, and the cumulative exposure-risk measure per 1,000 µg/L TTHM-years in the original is equivalent to the measure per 800 µg/L-years in the reanalysis.

The primary reanalysis takes advantage of the longer period of time for which new exposure estimates are available and estimates of specific by-products. Exposures occurring over a 50-year period that ended in the 2 years prior to the subject's interview were considered. To reduce the level of misclassification in exposures, these analyses were restricted to subjects with 35 or more years of known water history (e.g., 70% of the 50-year exposure period). The original analysis considered risks specifically for participants with 75% or more years of known water history.

Subject exposures were represented as the number of years exposed to a water supply over a selected concentration for the parameter of interest. The by-product concentration selected to determine years of exposure was based on criteria of meaningful equal distance categories and sufficient prevalence of high exposure among controls. Average rather than cumulative exposure measures were used in order to provide comparability with the analysis of Iowa data. Average exposure measures were calculated as the sum of the by-product level multiplied by the years at each residence, divided by the number of known exposure years. Categories were based on quartiles of the control distribution, and a high-exposure category was based on the top 10% of exposures. The risk estimates for continuous forms of the average by-product variables were represented per one standard deviation of the control distribution. The combined effects of quantity of tap water ingested and duration of exposure to different DBP exposures were evaluated by inclusion of an interaction term in the regression model.

ORs were used throughout as estimates of relative risk and are presented for each level of exposure in comparison with the lowest category of exposure. OR values greater than unity in the tables represent increased risks, and values less than unity represent decreased risks (a

**Table 8.1**  
**Comparison of new and original TTHM predictions**

	Original (peak TTHM)	New (mean TTHM)
Number of plants predicted	241	235
Mean TTHM for all plants (µg/L)	45.6	27.5
Mean TTHM for surface water plants (µg/L)	73.8	40.7
Correlation for all plants ( $n = 199$ )		0.61
Correlation for surface plants ( $n = 80$ )		0.46
Correlation for ground plants ( $n = 119$ )		0.09
Correlation for large plants ( $n = 28$ )		0.85
Correlation for residence TTHM (17,258)		0.86
Correlation for case residence TTHM ( $n = 9,615$ )		0.87
Correlation for control residence TTHM ( $n = 7,643$ )		0.85

protective effect associated with exposure). For the exposures of primary interest, unconditional logistic regression was used to obtain ORs and 95% CIs adjusted for potential confounders. The analysis controlled for those covariates that were predictive for the cancer sites of interest in the original analysis.

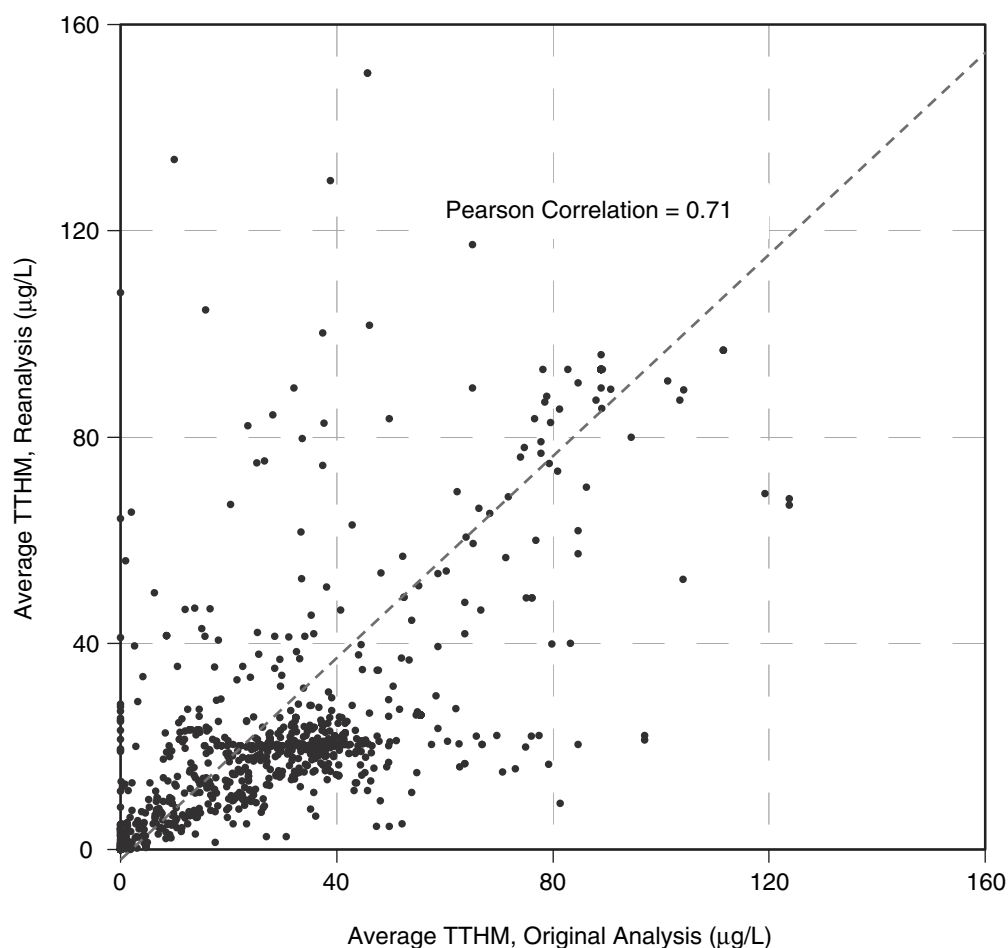
## RESULTS

### Comparison of TTHM Predictions: New Versus Original

Table 8.1 compares the original and new TTHM predictions for each treatment plant. Although a similar number of plants had by-product estimates (241 in the original and 235 in the new predictions), the plants with new predictions did not represent a subset of the original plants. In particular, new by-product predictions were provided for northern Ontario, which were not included in the original. In total, estimates for a subset of 199 plants were provided from both predictions.

TTHM exposure in the original study was based on predicted peak values (August) at each plant by 5-year time periods. The new predictions are based on predicted yearly average values. The mean for all plants is an average peak of 45.6 µg/L in the original and an average mean yearly value of 27.5 µg/L in the new predictions. The mean estimated TTHM value for surface water-based treatment plants is a peak of 74 µg/L in the original study and an average of 41 µg/L in the new predictions. The correlation between new and original values is not affected by use of mean and peak TTHM values in the respective estimates.

Overall, only a moderate correlation is observed between the new and original predictions of total TTHM levels at Ontario plants ( $r = 0.61$ ). A large part of this disagreement is attributable to estimates for chlorinated groundwater supplies ( $r = 0.09$ ) where the original models overestimated exposure to TTHM due to the strong impact of chlorine dose in the prediction model. The



**Figure 8.1 Comparison of subjects' average TTHM levels using original and new TTHM estimates**

correlation is higher for the largest cities ( $r = 0.85$ ), resulting in a high correlation on assignment of exposure to subject residences ( $r = 0.86$ ). The correlation for residence TTHM assignment was similar among cases ( $r = 0.87$ ) and controls ( $r = 0.85$ ).

Figure 8.1 plots the average exposure for control subjects in the 40-year window, for the original and reanalysis exposure estimates. Original exposure estimates are higher, and the majority of scatter points fall below a line representing perfect correspondence, reflecting the use of peak versus average TTHM exposure. The correlation in exposure assignment was 0.71.

### Comparison of TTHM Cancer Risks: New Versus Original

Table 8.2 contrasts results based on new exposure estimates to those obtained in the original study using comparable exposure metrics. Each analysis considers a 40-year exposure window ending 2 years prior to diagnosis, and only subjects with a minimum of 30 years of known exposure information are included. The original exposure variable representing number of years with a (peak) TTHM level  $>50 \mu\text{g/L}$  is contrasted with a new exposure variable of years

**Table 8.2**  
**Comparison of risk estimates based on original and new predictions**

	Original			Reanalysis		
	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
<b>Bladder</b>						
	Peak TTHM >50 µg/L			Mean TTHM >40 µg/L		
<10 years	253	650	1.00 (referent)	593	1,310	1.00 (referent)
10 to 19 years	226	519	1.10 (0.87–1.38)	23	51	1.21 (0.71–2.05)
20 to 34 years	163	297	1.36 (1.05–1.76)	30	68	1.01 (0.63–1.60)
≥35 years	54	79	1.63 (1.08–2.46)	43	65	1.36 (0.90–2.07)
TTHM-years	Quartiles of control distribution					
1	151	387	1.00	150	374	1.00 (referent)
2	160	385	1.20 (0.88–1.64)	150	373	1.12 (0.84–1.50)
3	165	387	1.08 (0.82–1.42)	193	375	1.23 (0.94–1.62)
4	220	386	1.44 (1.10–1.88)	196	375	1.34 (1.02–1.76)
TTHM-years	per 1,000 µg/L-year		1.11 (1.02–1.21)	per 800 µg/L-year		1.11 (1.03–1.21)
<b>Colon (male)</b>						
	Peak TTHM >50 µg/L			Mean TTHM >40 g/L		
<10 years	166	411	1.00 (referent)	365	812	1.00 (referent)
10 to 19 years	138	318	1.13 (0.86–1.50)	13	32	1.13 (0.57–2.22)
20 to 34 years	85	180	1.31 (0.94–1.81)	17	51	1.08 (0.61–1.95)
≥35 years	32	50	1.68 (1.02–2.76)	33	38	1.74 (1.06–2.87)
TTHM-years	Quartiles of control distribution					
1	99	257	1.00 (referent)	93	250	1.00 (referent)
2	96	225	1.30 (0.92–1.84)	102	222	1.45 (1.02–2.04)
3	94	240	1.11 (0.78–1.57)	106	235	1.27 (0.90–1.79)
4	132	237	1.74 (1.25–2.43)	127	227	1.70 (1.21–2.38)
TTHM-years	per 1,000 µg/L-year		1.17 (1.06–1.29)	per 800 µg/L-year		1.16 (1.04–1.28)
<b>Rectum (male)</b>						
	Peak TTHM >50 µg/L			Mean TTHM >40 µg/L		
<10 years	181	411	1.00	348	812	1.00 (referent)
10 to 19 years	118	318	0.94 (0.71–1.25)	16	32	1.47 (0.78–2.76)
20 to 34 years	77	180	1.07 (0.77–1.49)	16	51	0.94 (0.52–1.70)
≥35 years	17	50	0.82 (0.45–1.48)	12	38	0.72 (0.37–1.42)
TTHM-years	Quartiles of control distribution					
1	118	257	1.00 (referent)	116	250	1.00 (referent)
2	99	225	1.20 (0.86–1.67)	92	222	1.10 (0.78–1.54)
3	83	240	0.90 (0.63–1.27)	90	235	0.92 (0.65–1.29)
4	93	237	1.03 (0.73–1.44)	94	227	1.10 (0.78–1.54)
TTHM-years	per 1,000 µg/L-year		0.98 (0.88–1.08)	per 800 µg/L-year		0.97 (0.86–1.09)

\* For bladder cancer, ORs are adjusted for age, sex, highest education level attained, and log pack-years of smoking and current smoking; for colon and rectum cancer, ORs are adjusted for age, sex, highest education level attained, and body mass index.



**Table 8.3**  
**ORs for cancer of the bladder for tap water consumption and duration of TTHM**

Tap water consumption	Original: years of exposure to peak TTHM $\geq 50$ $\mu\text{g/L}$			
	0–9	10–19	20–34	35+
<1.54 $\mu\text{g/L}$ per day	1.0	1.28 (0.83–1.96)	1.70 (1.08–2.68)	1.26 (0.58–2.71)
1.54–2.08	1.35 (0.91–2.01)	1.32 (0.87–1.98)	1.54 (0.95–2.48)	2.58 (1.28–5.21)
>2.08	1.29 (0.87–1.90)	1.37 (0.91–2.06)	1.72 (1.10–2.70)	2.28 (1.12–4.67)
	Reanalysis: years of exposure to average TTHM $\geq 50$ $\mu\text{g/L}$			
	0–9	10–19	20–34	35+
<1.54 $\mu\text{g/L}$ per day	1.00	1.45 (0.52–4.09)	1.51 (0.67–3.43)	1.12 (0.47–2.65)
1.54–2.08	1.15 (0.89–1.49)	1.21 (0.57–2.58)	0.84 (0.39–1.81)	1.75 (0.85–3.61)
>2.08	1.22 (0.94–1.57)	1.64 (0.57–4.70)	1.25 (0.54–2.86)	1.67 (0.87–3.22)

Note: ORs are adjusted for age, gender, education, log pack-years of smoking, and current smoking.

>40  $\mu\text{g/L}$  to reflect the systematic difference between peak and mean TTHM values. The cumulative TTHM-years variable is contrasted using quartiles of the control distribution and a continuous representation. The linkage of subject residence histories to the new exposure predictions resulted in fewer subjects meeting the criteria for inclusion in the main analysis and a smaller proportion of controls in the high-exposure category. The original bladder cancer analysis included 1,545 controls, compared to 1,494 in the reanalysis.

For bladder cancer, the risk estimate in the highest-exposure category for years with a TTHM  $\geq 50$   $\mu\text{g/L}$  and  $\geq 40$   $\mu\text{g/L}$  is similar in the two analyses. However, risk estimates in the reanalysis are lower, had wider CIs, and did not follow a dose-response pattern. OR estimates are similar for the analysis of the cumulative TTHM-years variables. In both analyses, the highest cumulative exposure category is associated with a statistically significant increase in risk.

Colon and rectal cancer results are compared for males to correspond to the analysis demonstrating the highest risk in the original study. The risk estimate for colon cancer with 35 or more years of exposure is 1.68 in the original and 1.74 in the reanalysis. The analysis of cumulative exposure measures also produced very similar results for colon cancer risk. For rectal cancer risk among males, no association with the TTHM exposure measures is observed in either the original or reanalysis.

Table 8.3 replicates the analysis of the interaction between tap water consumption and TTHM exposure from the original study and the reanalysis based on new exposure predictions for bladder cancer. No statistically significant associations are observed in the reanalysis, and risk estimates are, in general, lower than in the reanalysis. In addition, the CIs are wide, reflecting the fact that the large number of categories results in few subjects within each category.

### Analysis Based on New Exposure Estimates

Tables 8.4 to 8.9 present the analysis for bladder, colon, and rectal cancer risk for and exposure to each of the by-product parameters: TTHM, BDCM, HAA5, DCAA, TCAA, and BCAA. A 50-year exposure window is considered and only subjects with 35 or more years of known exposure are included. The analysis of number of years of exposure over a specified cutpoint considers three categories of exposure to ensure adequate sample size in risk estimates. In the analysis of average exposure, categories are determined based on quartiles of the control distribution, and a



**Table 8.4**  
**Reanalysis of bladder cancer risk and exposure to specific chlorination**  
**by-products, years exposed over a specified level during a 50-year exposure**  
**window, for participants with 35 or more years of exposure information**

Exposure	Years (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥40 µg/L	<10	540	1,142	1.00	413	706	1.00	127	436	1.00
	10–29	35	89	0.97 (0.64–1.49)	22	55	0.85 (0.50–1.47)	13	34	1.40 (0.70–2.83)
	30+	57	92	1.28 (0.89–1.84)	38	57	1.15 (0.74–1.81)	19	35	1.65 (0.89–3.08)
BDCM ≥8 µg/L	<10	511	1,126	1.00	378	712	1.00	133	414	1.00
	10–29	47	79	1.40 (0.94–2.08)	36	44	1.63 (1.01–2.64)	11	35	1.02 (0.49–1.61)
	30+	74	118	1.36 (0.98–1.89)	59	62	1.63 (1.10–2.42)	15	56	0.86 (0.46–1.61)
HAA5 ≥30 µg/L	<10	539	1,149	1.00	410	709	1.00	129	440	1.00
	10–29	36	88	1.02 (0.67–1.56)	25	55	1.01 (0.60–1.69)	11	33	1.23 (0.59–2.58)
	30+	57	86	1.42 (0.98–2.05)	38	54	1.24 (0.79–1.95)	19	32	1.93 (1.03–3.64)
DCAA ≥10 µg/L	<10	542	1,154	1.00	411	712	1.00	131	442	1.00
	10–29	35	82	1.07 (0.69–1.65)	24	51	1.02 (0.60–1.73)	11	31	1.35 (0.64–2.85)
	30+	55	87	1.33 (0.92–1.93)	38	55	1.20 (0.77–1.89)	17	32	1.72 (0.90–3.31)
TCAA ≥10 µg/L	<10	531	1,124	1.00	405	693	1.00	126	431	1.00
	10–29	40	97	1.03 (0.69–1.55)	26	60	0.94 (0.57–1.55)	14	37	1.38 (0.70–2.72)
	30+	61	102	1.22 (0.86–1.73)	42	65	1.11 (0.73–1.70)	19	37	1.56 (0.84–2.88)
BCAA ≥4 µg/L	<10	539	1,188	1.00	399	742	1.00	140	446	1.00
	10–29	34	48	1.69 (1.05–2.72)	28	30	2.01 (1.15–3.51)	6	18	1.16 (0.44–3.07)
	30+	59	87	1.53 (1.06–2.21)	46	46	1.69 (1.08–2.65)	13	41	1.13 (0.57–2.23)

\* ORs adjusted for age, gender, education, log pack-years of smoking, and current smoking.

high-exposure category is based on the top 10% of control exposures. ORs for the continuous form of this variable are represented per one standard deviation of the control distribution.

In general, weak positive associations are observed between years of exposure to specified by-product levels and bladder cancer risk for males, females, and both sexes combined (Table 8.4). Among males, the largest risk estimates are observed for exposure to BDCM and BCAA. Among females, the largest risk estimate is for exposure to HAA5.

The analysis according to average by-product exposure and bladder cancer risk is presented in Table 8.5. This analysis is more robust than that described previously with respect to ensuring adequate sample size in each category due to the generation of categories based on the control distribution. For both sexes combined, ORs above 1.4 are observed for TTHM, BDCM, HAA5, TCAA, and BCAA. The largest bladder cancer risk estimates are among males with BDCM and BCAA exposure. Among females, the largest risk estimates are for TTHM, HAA5, and TCAA.

The continuous form of the variable is examined in units representing one standard deviation of the control exposure distribution. This was done to ensure that ORs were estimated

**Table 8.5**  
**Reanalysis of bladder cancer risk and exposure to specific chlorination by-**  
**products, average exposure during a 50-year exposure window, for**  
**participants with 35 or more years of exposure information**

Exposure	Average (µg/L)* (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†
TTHM	<9.2	132	330	1.00	105	217	1.00	27	112	1.00
	9.2–20.1	128	304	1.15 (0.84–1.56)	91	183	1.15 (0.80–1.66)	37	121	1.15 (0.64–2.07)
	20.2–23.6	180	335	1.27 (0.95–1.70)	138	207	1.32 (0.94–1.85)	42	128	1.16 (0.65–2.06)
	23.7–44.2	101	183	1.39 (0.99–1.94)	73	103	1.47 (0.98–2.21)	28	80	1.19 (0.64–2.22)
	>44.2	73	126	1.54 (1.06–2.24)	50	83	1.38 (0.86–2.16)	23	43	2.09 (1.04–4.17)
	Per standard deviation (21.7)			1.14 (1.04–1.24)			1.12 (1.01–1.25)			1.19 (1.01–1.41)
BDCM	<1.9	123	319	1.00	93	211	1.00	30	108	1.00
	1.9–5.6	155	339	1.27 (0.94–1.72)	105	201	1.30 (0.90–1.86)	50	137	1.17 (0.68–2.01)
	5.7–6.9	111	197	1.45 (1.04–2.02)	80	132	1.40 (0.94–2.07)	31	65	1.53 (0.83–2.84)
	7.0–7.9	152	292	1.25 (0.92–1.70)	120	175	1.44 (1.01–2.07)	32	117	0.84 (0.46–1.52)
	>7.9	73	131	1.48 (1.02–2.15)	59	74	1.83 (1.18–2.86)	14	57	0.82 (0.39–1.72)
	Per standard deviation (3.1)			1.14 (1.03–1.25)			1.19 (1.06–1.33)			1.01 (0.83–1.22)
HAA5	<6.2	137	331	1.00	109	215	1.00	28	115	1.00
	6.2–12.3	152	317	1.24 (0.93–1.67)	105	198	1.13 (0.80–1.61)	47	119	1.50 (0.86–2.64)
	12.4–15.9	147	317	1.10 (0.81–1.48)	114	192	1.15 (0.81–1.64)	33	125	0.98 (0.54–1.77)
	16.0–31.6	105	184	1.34 (0.96–1.87)	80	106	1.41 (0.95–2.09)	25	78	1.16 (0.62–2.20)
	>31.6	73	129	1.46 (1.01–2.12)	49	82	1.30 (0.83–2.04)	24	47	1.98 (1.01–3.89)
	Per standard deviation (17.8)			1.11 (1.02–1.21)			1.10 (1.00–1.22)			1.16 (0.98–1.37)
DCAA	<2.0	144	328	1.00	112	217	1.00	32	110	1.00
	2.0–4.2	213	442	1.13 (0.86–1.48)	149	279	1.08 (0.78–1.50)	64	163	1.23 (0.74–2.07)
	4.3–5.9	83	177	1.12 (0.79–1.59)	71	91	1.60 (1.06–2.43)	12	86	0.45 (0.22–0.96)
	6.0–12.8	102	204	1.16 (0.84–1.62)	74	123	1.18 (0.80–1.74)	28	81	1.12 (0.61–2.06)
	>12.8	72	127	1.38 (0.95–2.00)	51	83	1.37 (0.88–2.15)	21	44	1.51 (0.76–3.00)
	Per standard deviation (7.5)			1.11 (1.01–1.21)			1.11 (1.00–1.23)			1.14 (0.96–1.35)
TCAA	<2.0	132	334	1.00	107	218	1.00	25	115	1.00
	2.0–4.0	172	318	1.51 (1.13–2.02)	123	193	1.45 (1.03–2.05)	49	125	1.69 (0.95–2.99)
	4.1–5.1	140	296	1.20 (0.89–1.63)	103	185	1.11 (0.78–1.59)	37	111	1.51 (0.83–2.75)
	5.2–15.9	101	201	1.39 (0.99–1.93)	77	113	1.53 (1.03–2.27)	24	88	1.16 (0.60–2.21)
	>15.9	69	129	1.47 (1.01–2.14)	47	84	1.29 (0.82–2.03)	22	45	2.19 (1.08–4.41)
	Per standard deviation (9.4)			1.11 (1.01–1.21)			1.09 (0.98–1.21)			1.17 (1.00–1.38)
BCAA	<0.9	127	338	1.00	96	218	1.00	31	120	1.00
	0.9–2.2	136	271	1.39 (1.03–1.90)	93	163	1.44 (0.99–2.08)	43	107	1.28 (0.73–2.24)
	2.3–2.6	162	341	1.17 (0.87–1.58)	116	221	1.14 (0.80–1.62)	46	120	1.25 (0.72–2.16)
	2.7–3.2	107	187	1.50 (1.07–2.09)	84	111	1.79 (1.21–2.66)	23	76	0.97 (0.51–1.83)
	>3.2	82	141	1.58 (1.10–2.27)	68	80	1.98 (1.29–3.04)	14	61	0.86 (0.42–1.78)
	Per standard deviation (1.3)			1.16 (1.05–1.28)			1.23 (1.09–1.39)			1.00 (0.83–1.21)

\* Categories are based on control exposure distribution: <25%, 25%–49%, 50%–74%, 75%–89%, >90%.

† ORs are adjusted for age, gender, education, log pack-years of smoking, and current smoking.

**Table 8.6**  
**Reanalysis of colon cancer risk and exposure to specific chlorination**  
**by-products, years exposed over a specified level during a 50-year exposure**  
**window, for participants with 35 or more years of exposure information**

Exposure	Years	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥40 µg/L	<10	584	1,142	1.00	332	706	1.00	252	436	1.00
	10–29	36	89	0.94 (0.62–1.42)	21	55	1.01 (0.59–1.73)	15	34	0.84 (0.44–1.60)
	30+	67	92	1.41 (1.00–1.98)	41	57	1.53 (0.99–2.36)	26	35	1.21 (0.70–2.08)
BDCM ≥8 µg/L	<10	586	1,126	1.00	331	712	1.00	255	414	1.00
	10–29	39	79	1.03 (0.69–1.54)	23	44	1.18 (0.69–2.00)	16	35	0.84 (0.45–1.58)
	30+	62	118	0.96 (0.69–1.34)	40	62	1.29 (0.84–1.99)	22	56	0.64 (0.38–1.08)
HAA5 ≥30 µg/L	<10	591	1,149	1.00	336	709	1.00	255	440	1.00
	10–29	31	88	0.80 (0.52–1.23)	19	55	0.90 (0.52–1.57)	12	33	0.65 (0.32–1.30)
	30+	65	86	1.46 (1.03–2.06)	39	54	1.54 (0.99–2.40)	26	32	1.30 (0.75–2.26)
DCAA ≥10 µg/L	<10	599	1,154	1.00	340	712	1.00	259	442	1.00
	10–29	27	82	0.74 (0.47–1.16)	18	51	0.90 (0.51–1.58)	9	31	0.51 (0.24–1.11)
	30+	61	87	1.34 (0.94–1.90)	36	55	1.38 (0.87–2.17)	25	32	1.24 (0.71–2.17)
TCAA ≥10 µg/L	<10	579	1,124	1.00	328	693	1.00	251	431	1.00
	10–29	37	97	0.87 (0.58–1.30)	22	60	0.94 (0.56–1.59)	15	37	0.75 (0.40–1.42)
	30+	71	102	1.36 (0.98–1.89)	44	65	1.45 (0.95–2.20)	27	37	1.19 (0.70–2.02)
BCAA ≥4 µg/L	<10	623	1,188	1.00	352	742	1.00	271	446	1.00
	10–29	28	48	1.25 (0.77–2.04)	16	30	1.29 (0.68–2.44)	12	18	1.21 (0.56–2.61)
	30+	36	87	0.74 (0.49–1.12)	26	46	1.09 (0.65–1.84)	10	41	0.38 (0.19–0.79)

\* ORs are adjusted for age, sex, current smoking, log pack-years of smoking, and education.

according to a consistently meaningful unit for each by-product. The associated OR can be interpreted as the change in risk associated with the specified magnitude of the standard deviation. For example, for bladder cancer risk in the total group, the OR of 1.14 indicates a 14% increase in bladder cancer risk with each 21.7 µg/L of average TTHM exposure. The analysis of continuous variables in the logistic model assumes that the change in risk is constant across the exposure continuum. That is, the increase in risk associated with a change from 0 to 21.7 µg/L confers the same increase in risk as a change from 60 to 81.7 µg/L. The linear trend observed for most categorical representations of exposure supports this assumption.

The change in risk for other units or exposure contrasts can be estimated by representing the change in terms of standard deviation units (e.g., dividing by the standard deviation) and exponentiation of the product of the log of the OR multiplied by this unit change. For example, a risk associated with an average total TTHM exposure of 100 µg/L versus 40 µg/L is estimated as the risk associated with a 60 µg/L change and can be calculated as:

$$OR_{60 \mu g/L} = \exp [ \log(1.14) * (60/ 21.7) ] = 1.44$$

Tables 8.4 and 8.5 are repeated for colon cancer (Tables 8.6 and 8.7). In the analysis of years exposed over specified by-product levels, few exposures are related to colon cancer risk. Among males, the category representing 30 or more years of exposure is suggestive of excess colon cancer risk for TTHM ≥40 µg/L and HAA5 ≥30 µg/L. In the analysis of average exposure (Table 8.7) the

**Table 8.7**  
**Reanalysis of colon cancer risk and exposure to specific chlorination by-products,**  
**average exposure during a 50-year exposure window, for participants with**  
**35 or more years of exposure information**

Exposure	Average (µg/L)* (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†
TTHM	<9.2	181	330	1.00	82	217	1.00	99	112	1.00
	9.2–20.1	146	304	1.03 (0.78–1.36)	94	183	1.56 (1.08–2.26)	52	121	0.58 (0.37–0.89)
	20.2–23.6	183	335	1.10 (0.85–1.44)	104	207	1.43 (1.00–2.06)	79	128	0.79 (0.53–1.18)
	23.7–44.2	86	183	0.94 (0.68–1.30)	58	103	1.60 (1.05–2.45)	28	80	0.44 (0.26–0.74)
	>44.2	78	126	1.32 (0.93–1.87)	49	83	1.83 (1.16–2.89)	29	43	0.82 (0.47–1.43)
	Per standard deviation (21.7)			1.09 (0.99–1.19)			1.16 (1.04–1.30)			0.96 (0.83–1.11)
BDCM	<1.9	175	319	1.00	77	211	1.00	98	108	1.00
	1.9–5.6	167	339	1.06 (0.81–1.40)	110	201	1.76 (1.23–2.54)	57	137	0.53 (0.34–0.81)
	5.7–6.9	116	197	1.23 (0.91–1.67)	76	132	1.74 (1.17–2.59)	40	65	0.79 (0.48–1.29)
	7.0–7.9	158	292	1.08 (0.82–1.43)	84	175	1.40 (0.95–2.06)	74	117	0.79 (0.52–1.19)
	>7.9	58	131	0.88 (0.60–1.27)	40	74	1.55 (0.96–2.52)	18	57	0.40 (0.22–0.73)
	Per standard deviation (3.1)			1.01 (0.92–1.12)			1.13 (1.00–1.28)			0.87 (0.75–1.01)
HAA5	<6.2	180	331	1.00	83	215	1.00	97	115	1.00
	6.2–12.3	162	317	1.12 (0.85–1.47)	105	198	1.59 (1.11–2.28)	57	119	0.68 (0.44–1.04)
	12.4–15.9	167	317	1.07 (0.82–1.41)	90	192	1.31 (0.90–1.91)	77	125	0.84 (0.56–1.26)
	16.0–31.6	89	184	0.98 (0.71–1.35)	60	106	1.58 (1.04–2.40)	29	78	0.49 (0.29–0.82)
	>31.6	76	129	1.26 (0.89–1.79)	49	82	1.80 (1.14–2.84)	27	47	0.75 (0.43–1.31)
	Per standard deviation (17.8)			1.07 (0.98–1.17)			1.13 (1.01–1.27)			0.96 (0.83–1.11)
DCAA	<2.0	187	328	1.00	89	217	1.00	98	110	1.00
	2.0–4.2	230	442	1.06 (0.82–1.36)	133	279	1.34 (0.96–1.87)	97	163	0.78 (0.53–1.14)
	4.3–5.9	86	177	0.96 (0.69–1.33)	49	91	1.54 (0.98–2.40)	37	86	0.54 (0.33–0.87)
	6.0–12.8	96	204	0.91 (0.67–1.24)	68	123	1.47 (0.99–2.20)	28	81	0.43 (0.25–0.72)
	>12.8	75	127	1.23 (0.87–1.75)	48	83	1.69 (1.07–2.66)	27	44	0.75 (0.43–1.33)
	Per standard deviation (7.5)			1.07 (0.98–1.17)			1.12 (1.00–1.26)			0.97 (0.83–1.12)
TCAA	<2.0	183	334	1.00	89	218	1.00	94	115	1.00
	2.0–4.0	175	318	1.17 (0.89–1.53)	110	193	1.61 (1.13–2.30)	65	125	0.74 (0.48–1.12)
	4.1–5.1	140	296	0.96 (0.73–1.28)	72	185	1.02 (0.69–1.49)	68	111	0.87 (0.57–1.33)
	5.2–15.9	95	201	1.00 (0.73–1.36)	66	113	1.68 (1.12–2.53)	29	88	0.45 (0.27–0.75)
	>15.9	81	129	1.33 (0.94–1.88)	50	84	1.69 (1.08–2.66)	31	45	0.91 (0.53–1.58)
	Per standard deviation (9.4)			1.08 (0.99–1.17)			1.12 (1.00–1.25)			1.00 (0.87–1.15)
BCAA	<0.9	187	338	1.00	85	218	1.00	102	120	1.00
	0.9–2.2	131	271	1.03 (0.78–1.37)	90	163	1.64 (1.13–2.38)	41	107	0.52 (0.33–0.83)
	2.3–2.6	171	341	1.01 (0.77–1.32)	92	221	1.13 (0.79–1.64)	79	120	0.89 (0.60–1.34)
	2.7–3.2	123	187	1.27 (0.94–1.71)	80	111	1.90 (1.28–2.82)	43	76	0.73 (0.46–1.17)
	>3.2	62	141	0.88 (0.61–1.26)	40	80	1.38 (0.86–2.22)	22	61	0.47 (0.27–0.83)
	Per standard deviation (1.3)			1.00 (0.91–1.10)			1.13 (1.00–1.29)			0.84 (0.72–0.97)

\* Categories are based on control exposure distribution: <25%, 25%–49%, 50%–74%, 75%–89%, >90%.

† ORs are adjusted for age, sex, education, and body mass index.

**Table 8.8**  
**Reanalysis of rectal cancer risk and exposure to specific chlorination by-products, years exposed over a specified level during a 50-year exposure window, for participants with 35 or more years of exposure information**

Exposure	Years (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥40 µg/L	<10	517	1,142	1.00	307	706	1.00	210	436	1.00
	10–29	35	89	1.05 (0.70–1.60)	25	55	1.32 (0.80–2.20)	10	34	0.69 (0.33–1.45)
	30+	29	92	0.70 (0.45–1.08)	19	57	0.82 (0.47–1.41)	10	35	0.53 (0.25–1.10)
BDCM ≥8 µg/L	<10	502	1,126	1.00	298	712	1.00	204	414	1.00
	10–29	36	79	1.17 (0.77–1.77)	24	44	1.49 (0.88–2.53)	12	35	0.78 (0.39–1.56)
	30+	43	118	0.82 (0.57–1.19)	29	62	1.16 (0.73–1.86)	14	56	0.49 (0.26–0.90)
HAA5 ≥30 µg/L	<10	519	1,149	1.00	311	709	1.00	208	440	1.00
	10–29	34	88	1.02 (0.67–1.55)	22	55	1.17 (0.69–1.98)	12	33	0.81 (0.40–1.64)
	30+	28	86	0.72 (0.46–1.13)	18	54	0.82 (0.47–1.44)	10	32	0.57 (0.27–1.20)
DCAA ≥10 µg/L	<10	525	1,154	1.00	313	712	1.00	212	442	1.00
	10–29	31	82	0.97 (0.63–1.50)	21	51	1.17 (0.68–2.01)	10	31	0.70 (0.33–1.49)
	30+	25	87	0.64 (0.40–1.02)	17	55	0.76 (0.43–1.35)	8	32	0.46 (0.20–1.02)
TCAA ≥10 µg/L	<10	512	1,124	1.00	306	693	1.00	206	431	1.00
	10–29	37	97	1.01 (0.68–1.51)	25	60	1.20 (0.73–1.98)	12	37	0.76 (0.38–1.51)
	30+	32	102	0.70 (0.46–1.07)	20	65	0.76 (0.45–1.29)	12	37	0.61 (0.31–1.20)
BCAA ≥4 µg/L	<10	529	1,188	1.00	316	742	1.00	213	446	1.00
	10–29	26	48	1.49 (0.90–2.45)	16	30	1.60 (0.84–3.02)	10	18	1.27 (0.56–2.87)
	30+	26	87	0.67 (0.42–1.06)	19	46	1.01 (0.58–1.77)	7	41	0.33 (0.14–0.75)

\* ORs are adjusted for age, sex, education, and body mass index.

highest-exposure category is associated with a relative risk approaching 2 for TTHM, HAA5, DCAA, TCAA, and BCAA. Relative risks are consistently <1 for high exposure among females. For BDCM and BCAA, these protective effects are statistically significant.

Tables 8.8 and 8.9 present the analysis of rectal cancer risk using the new exposure estimates. No statistically significant findings are found for the high-exposure categories in any of the rectal cancer analyses.

### Analysis Based on New Exposure Estimates: Risk Associated With High Exposure

Tables 8.10, 8.11, and 8.12 examine risk associated with high exposures to each of the by-products. For each by-product, a level of exposure was selected such that it represented a meaningful definition of high exposure and a reasonable number of subjects were exposed. Years of exposure above that level were then considered.

**Table 8.9**  
**Reanalysis of rectal cancer risk and exposure to specific chlorination**  
**by-products, average exposure during a 50-year exposure window, for**  
**participants with 35 or more years of exposure information**

Exposure	Average (µg/L)* (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†
TTHM	<9.2	171	330	1.00	108	217	1.00	63	112	1.00
	9.2–20.1	126	304	0.96 (0.72–1.28)	81	183	1.10 (0.77–1.59)	45	121	0.76 (0.47–1.23)
	20.2–23.6	151	335	0.96 (0.73–1.27)	76	207	0.84 (0.59–1.21)	75	128	1.09 (0.71–1.68)
	23.7–44.2	75	183	0.90 (0.64–1.25)	49	103	1.16 (0.75–1.77)	26	80	0.59 (0.34–1.03)
	>44.2	44	126	0.81 (0.54–1.20)	28	83	0.87 (0.53–1.44)	16	43	0.68 (0.35–1.32)
	Per standard deviation (21.7)			0.94 (0.85–1.05)			0.98 (0.86–1.12)			0.88 (0.74–1.05)
BDCM	<1.9	159	319	1.00	100	211	1.00	59	108	1.00
	1.9–5.6	132	339	0.93 (0.70–1.23)	85	201	1.08 (0.75–1.55)	47	137	0.71 (0.44–1.15)
	5.7–6.9	93	197	1.19 (0.81–1.54)	60	132	1.11 (0.75–1.66)	33	65	1.12 (0.65–1.93)
	7.0–7.9	136	292	1.00 (0.75–1.33)	66	175	0.88 (0.60–1.29)	70	117	1.12 (0.72–1.75)
	>7.9	47	131	0.82 (0.56–1.22)	31	74	1.05 (0.64–1.72)	16	57	0.55 (0.29–1.06)
	Per standard deviation (3.1)			1.00 (0.91–1.11)			1.00 (0.88–1.13)			1.01 (0.86–1.19)
HAA5	<6.2	171	331	1.00	107	215	1.00	64	115	1.00
	6.2–12.3	120	317	0.88 (0.66–1.18)	77	198	0.97 (0.67–1.40)	43	119	0.74 (0.46–1.19)
	12.4–15.9	147	317	1.02 (0.77–1.34)	72	192	0.89 (0.61–1.29)	75	125	1.15 (0.75–1.78)
	16.0–31.6	82	184	0.95 (0.69–1.32)	55	106	1.20 (0.80–1.82)	27	78	0.64 (0.37–1.10)
	>31.6	47	129	0.85 (0.57–1.26)	31	82	0.98 (0.60–1.61)	16	47	0.63 (0.33–1.22)
	Per standard deviation (17.8)			0.94 (0.84–1.04)			0.98 (0.86–1.12)			0.86 (0.71–1.03)
DCAA	<2.0	171	328	1.00	107	217	1.00	64	110	1.00
	2.0–4.2	186	442	0.95 (0.73–1.23)	103	279	0.90 (0.64–1.26)	83	163	0.99 (0.65–1.51)
	4.3–5.9	72	177	0.90 (0.64–1.26)	42	91	1.14 (0.73–1.78)	30	86	0.65 (0.38–1.10)
	6.0–12.8	92	204	0.96 (0.70–1.32)	57	123	1.08 (0.72–1.61)	35	81	0.79 (0.47–1.32)
	>12.8	46	127	0.86 (0.58–1.28)	33	83	1.06 (0.65–1.73)	13	44	0.54 (0.27–1.10)
	Per standard deviation (7.5)			0.96 (0.86–1.06)			1.01 (0.89–1.15)			0.85 (0.70–1.03)
TCAA	<2.0	168	334	1.00	107	218	1.00	61	115	1.00
	2.0–4.0	146	318	1.08 (0.81–1.42)	93	193	1.17 (0.82–1.66)	53	125	0.93 (0.58–1.47)
	4.1–5.1	121	296	0.91 (0.68–1.22)	62	185	0.79 (0.54–1.16)	59	111	1.08 (0.68–1.70)
	5.2–15.9	891	201	1.03 (0.75–1.42)	52	113	1.17 (0.77–1.78)	37	88	0.83 (0.50–1.38)
	>15.9	43	129	0.78 (0.52–1.17)	28	84	0.86 (0.52–1.42)	15	45	0.64 (0.32–1.26)
	Per standard deviation (9.4)			0.94 (0.85–1.04)			0.98 (0.86–1.11)			0.87 (0.72–1.04)
BCAA	< 0.9	169	338	1.00	103	218	1.00	66	120	1.00
	0.9–2.2	118	271	1.02 (0.76–1.37)	79	163	1.21 (0.84–1.75)	39	107	0.78 (0.48–1.27)
	2.3–2.6	154	341	1.03 (0.78–1.36)	86	221	0.95 (0.66–1.36)	68	120	1.14 (0.74–1.77)
	2.7–3.2	82	187	0.94 (0.68–1.31)	45	111	0.95 (0.62–1.45)	37	76	0.93 (0.56–1.54)
	>3.2	44	141	0.72 (0.49–1.07)	29	80	0.95 (0.57–1.56)	15	61	0.46 (0.24–0.89)
	Per standard deviation (1.3)			0.97 (0.88–1.08)			1.00 (0.88–1.14)			0.93 (0.80–1.09)

\* Categories are based on control exposure distribution: <25%, 25%–49%, 50%–74%, 75%–89%, >90%.

† ORs are adjusted for age, sex, education, and body mass index.

**Table 8.10**  
**Reanalysis of bladder cancer risk and exposure to specific chlorination**  
**by-products, average exposure during a 50-year exposure window, for**  
**participants with 35 or more years of exposure information**

Exposure	Average (µg/L) (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM	<10.0	134	347	1.00	107	225	1.00	27	121	1.00
	10.0–39.9	403	790	1.33 (1.04–1.71)	298	477	1.33 (0.99–1.78)	105	313	1.34 (0.82–2.18)
	40.0–59.9	19	48	1.10 (0.61–1.99)	12	31	1.05 (0.50–2.19)	7	17	1.47 (0.53–4.08)
	60.0–79.9	13	30	1.32 (0.64–2.73)	9	22	1.05 (0.45–2.48)	4	8	2.57 (0.67–9.94)
	≥80.0	45	63	1.82 (1.16–2.88)	31	38	1.69 (0.97–2.95)	14	25	2.26 (1.00–5.11)
BDCM	<2.0	143	357	1.00	109	231	1.00	34	126	1.00
	2.0–3.9	68	153	1.14 (0.79–1.64)	47	94	1.09 (0.70–1.69)	21	58	1.20 (0.62–2.32)
	4.0–6.4	111	226	1.33 (0.97–1.82)	72	140	1.18 (0.80–1.74)	39	86	1.64 (0.94–2.88)
	6.5–8.9	272	496	1.31 (1.01–1.70)	211	301	1.43 (1.05–1.95)	61	195	1.03 (0.62–1.69)
	≥9.0	20	46	1.04 (0.58–1.87)	18	27	1.32 (0.68–2.58)	2	19	0.43 (0.09–2.00)
HAA5	<7.5	167	395	1.00	130	257	1.00	37	137	1.00
	7.5–19.9	332	677	1.19 (0.93–1.51)	247	407	1.23 (0.93–1.64)	85	270	1.11 (0.70–1.76)
	20.0–39.9	55	97	1.35 (0.90–2.01)	38	57	1.36 (0.84–2.21)	17	40	1.35 (0.67–2.74)
	40.0–59.9	16	39	1.03 (0.54–1.97)	10	28	0.82 (0.37–1.83)	6	11	1.97 (0.64–6.06)
	≥60.0	44	70	1.50 (0.97–2.34)	32	44	1.48 (0.87–2.52)	12	26	1.62 (0.72–3.66)
DCAA	<5.0	392	854	1.00	291	540	1.00	101	313	1.00
	5.0–9.9	147	272	1.19 (0.93–1.52)	114	156	1.32 (0.99–1.78)	33	116	0.89 (0.56–1.43)
	10.0–19.9	20	64	0.76 (0.44–1.30)	12	40	0.71 (0.36–1.42)	8	24	0.90 (0.38–2.15)
	20.0–29.9	21	44	1.19 (0.68–2.08)	14	30	0.97 (0.49–1.93)	7	14	1.89 (0.71–5.02)
	≥30.0	34	44	1.58 (0.97–2.58)	26	27	1.73 (0.97–3.11)	8	17	1.40 (0.56–3.51)
TCAA	<5.0	433	932	1.00	326	588	1.00	107	343	1.00
	5.0–9.9	90	179	1.13 (0.84–1.51)	71	101	1.30 (0.92–1.85)	19	78	0.78 (0.44–1.37)
	10.0–19.9	27	60	1.02 (0.62–1.67)	16	38	0.89 (0.47–1.67)	11	22	1.41 (0.64–3.12)
	20.0–29.9	24	45	1.38 (0.81–2.36)	13	29	1.13 (0.56–2.27)	11	16	1.95 (0.84–4.52)
	≥30.0	40	62	1.36 (0.88–2.10)	31	37	1.44 (0.85–2.42)	9	25	1.24 (0.54–2.85)
BCAA	<1.0	132	345	1.00	99	221	1.00	33	124	1.00
	1.0–1.9	105	210	1.33 (0.96–1.84)	69	129	1.24 (0.84–1.85)	36	80	1.44 (0.81–2.58)
	2.0–4.9	365	715	1.30 (1.01–1.68)	279	438	1.44 (1.06–1.94)	86	277	1.02 (0.63–1.64)
	≥5.0	12	8	3.27 (1.26–8.47)	10	5	3.71 (1.19–11.55)	2	3	2.56 (0.39–16.68)

\* ORs are adjusted for age, gender, education, log pack-years of smoking, and current smoking.

**Table 8.11**  
**Reanalysis of colon cancer risk and exposure to specific chlorination**  
**by-products, average exposure during a 50-year exposure window, for**  
**participants with 35 or more years of exposure information**

Exposure	Average (µg/L) (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM	<10.0	185	347	1.00	83	225	1.00	102	121	1.00
	10.0–39.9	407	790	1.08 (0.87–1.36)	251	477	1.56 (1.15–2.13)	156	313	0.67 (0.48–0.94)
	40.0–59.9	19	48	0.93 (0.52–1.64)	14	31	1.51 (0.75–3.05)	5	17	0.39 (0.14–1.13)
	60.0–79.9	15	30	1.25 (0.64–2.44)	9	22	1.54 (0.66–3.58)	6	8	1.01 (0.33–3.08)
	≥80.0	48	63	1.51 (0.98–2.33)	30	38	2.26 (1.28–3.97)	18	25	0.87 (0.44–1.71)
BDCM	<2.0	199	357	1.00	94	231	1.00	105	126	1.00
	2.0–3.9	66	153	0.94 (0.66–1.33)	43	94	1.33 (0.85–2.08)	23	58	0.57 (0.32–1.00)
	4.0–6.4	111	226	1.04 (0.77–1.39)	73	140	1.49 (1.01–2.18)	38	86	0.61 (0.38–0.99)
	6.5–8.9	279	496	1.10 (0.87–1.39)	163	301	1.40 (1.02–1.93)	116	195	0.81 (0.56–1.16)
	≥9.0	19	46	0.82 (0.46–1.47)	14	27	1.32 (0.64–2.72)	5	19	0.36 (0.13–1.01)
HAA5	<7.5	205	395	1.00	205	257	1.00	107	137	1.00
	7.5–19.9	352	677	1.12 (0.90–1.40)	352	407	1.54 (1.14–2.08)	137	270	0.73 (0.52–1.02)
	20.0–39.9	51	97	1.10 (0.75–1.63)	51	57	1.61 (0.97–2.67)	19	40	0.64 (0.34–1.18)
	40.0–59.9	15	39	1.02 (0.54–1.93)	15	28	1.40 (0.65–2.99)	4	11	0.61 (0.19–2.03)
	≥60.0	51	70	1.50 (0.99–2.26)	51	44	2.00 (1.17–3.42)	20	26	0.98 (0.51–1.88)
DCAA	<5.0	467	854	1.00	250	540	1.00	217	313	1.00
	5.0–9.9	127	272	0.89 (0.70–1.14)	85	156	1.21 (0.89–1.65)	42	116	0.55 (0.37–0.82)
	10.0–19.9	18	64	0.56 (0.33–0.97)	15	40	0.89 (0.48–1.67)	3	24	0.18 (0.05–0.62)
	20.0–29.9	32	44	1.57 (0.97–2.55)	17	30	1.46 (0.78–2.75)	15	14	1.64 (0.75–3.56)
	≥30.0	30	44	1.19 (0.73–1.95)	20	27	1.53 (0.82–2.85)	10	17	0.78 (0.35–1.77)
TCAA	<5.0	492	932	1.00	267	588	1.00	225	343	1.00
	5.0–9.9	92	179	1.02 (0.77–1.35)	64	101	1.46 (1.03–2.09)	28	78	0.57 (0.35–0.91)
	10.0–19.9	20	60	0.72 (0.43–1.23)	11	38	0.79 (0.39–1.59)	9	22	0.60 (0.27–1.35)
	20.0–29.9	28	45	1.35 (0.82–2.22)	19	29	1.56 (0.85–2.88)	9	16	1.02 (0.43–2.40)
	≥30	42	62	1.28 (0.84–1.94)	26	37	1.59 (0.93–2.74)	16	25	0.88 (0.45–1.72)
BCAA	<1.0	190	345	1.00	87	221	1.00	103	124	1.00
	1.0–1.9	105	210	1.09 (0.81–1.48)	72	129	1.64 (1.11–2.43)	33	80	0.61 (0.37–1.00)
	2.0–4.9	372	715	1.05 (0.83–1.31)	223	438	1.40 (1.02–1.91)	149	277	0.72 (0.51–1.02)
	≥5.0	7	8	1.41 (0.47–4.22)	5	5	1.87 (0.48–7.37)	2	3	0.86 (0.14–5.44)

\* ORs are adjusted for age, gender, education, log pack-years of smoking, and current smoking.



**Table 8.12**  
**Reanalysis of rectal cancer risk and exposure to specific chlorination by-**  
**products, average exposure during a 50-year exposure window, for**  
**participants with 35 or more years of exposure information**

Exposure	Average (µg/L) (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM	<10.0	173	347	1.00	110	225	1.00	63	121	1.00
	10.0–39.9	346	790	1.00 (0.80–1.26)	201	477	1.02 (0.76–1.37)	145	313	0.96 (0.66–1.40)
	40.0–59.9	19	48	1.01 (0.57–1.80)	12	31	1.02 (0.50–2.10)	7	17	0.96 (0.37–2.52)
	60.0–79.9	7	30	0.61 (0.26–1.44)	6	22	0.76 (0.29–1.97)	1	8	0.27 (0.03–2.26)
	≥80.0	22	63	0.77 (0.46–1.31)	13	38	0.84 (0.43–1.67)	9	25	0.63 (0.27–1.46)
BDCM	<2.0	170	357	1.00	107	231	1.00	63	126	1.00
	2.0–3.9	48	153	0.78 (0.53–1.14)	35	94	0.93 (0.59–1.48)	13	58	0.53 (0.27–1.06)
	4.0–6.4	117	226	1.30 (0.96–1.76)	77	140	1.42 (0.97–2.07)	40	86	1.12 (0.68–1.85)
	6.5–8.9	216	496	1.00 (0.78–1.29)	115	301	0.92 (0.66–1.27)	101	195	1.10 (0.74–1.65)
	≥9.0	16	46	0.82 (0.45–1.51)	8	27	0.75 (0.33–1.73)	8	19	0.91 (0.37–2.25)
HAA5	<7.5	198	395	1.00	126	257	1.00	72	137	1.00
	7.5–19.9	286	677	0.96 (0.77–1.21)	164	407	0.97 (0.73–1.30)	122	270	0.93 (0.64–1.35)
	20.0–39.9	51	97	1.12 (0.76–1.65)	30	57	1.21 (0.73–2.00)	21	40	0.97 (0.52–1.79)
	40.0–59.9	14	39	0.95 (0.50–1.82)	11	28	1.09 (0.51–2.31)	3	11	0.64 (1.67–2.44)
	≥60	18	70	0.58 (0.33–1.00)	11	44	0.63 (0.31–1.27)	7	26	0.48 (0.19–1.17)
DCAA	<5.0	390	854	1.00	229	540	1.00	161	313	1.00
	5.0–9.9	123	272	1.02 (0.80–1.31)	75	156	1.18 (0.86–1.63)	48	116	0.82 (0.55–1.23)
	10.0–19.9	27	64	1.04 (0.65–1.68)	19	40	1.30 (0.73–2.32)	8	24	0.69 (0.30–1.60)
	20.0–29.9	14	44	0.82 (0.44–1.52)	11	30	1.06 (0.51–2.18)	3	14	0.42 (0.12–1.53)
	≥30.0	13	44	0.66 (0.35–1.25)	8	27	0.77 (0.34–1.75)	5	17	0.50 (0.18–1.41)
TCAA	<5.0	427	932	1.00	256	588	1.00	171	343	1.00
	5.0–9.9	79	179	1.00 (0.74–1.34)	44	101	1.09 (0.74–1.61)	35	78	0.87 (0.56–1.37)
	10.0–19.9	26	60	1.14 (0.70–1.85)	19	38	1.44 (0.80–2.58)	7	22	0.67 (0.27–1.62)
	20.0–29.9	22	45	1.22 (0.71–2.08)	16	29	1.49 (0.78–2.83)	6	16	0.80 (0.30–2.16)
	≥30.0	13	62	0.46 (0.25–0.85)	7	37	0.48 (0.21–1.09)	6	25	0.42 (0.17–1.06)
BCAA	<1.0	170	345	1.00	103	221	1.00	67	124	1.00
	1.0–1.9	75	210	0.85 (0.61–1.18)	53	129	1.02 (0.68–1.54)	22	80	0.62 (0.35–1.10)
	2.0–4.9	317	715	1.02 (0.81–1.29)	183	438	1.05 (0.77–1.42)	134	277	0.96 (0.66–1.40)
	≥5.0	5	8	1.13 (0.36–3.56)	3	5	1.17 (0.27–5.09)	2	3	1.12 (0.18–7.20)

\* ORs are adjusted for age, gender, education, log pack-years of smoking, and current smoking.

For bladder cancer risk, TTHM exposure  $>80 \mu\text{g/L}$  is associated with an 80% increase in risk ( $\text{OR} = 1.82$ ,  $95\% \text{ CI} = 1.16\text{--}2.88$ ). High BDCM exposure is not associated with an increase in risk. HAA5 and each of the specific HAA by-products are associated with an increase in risk, with the largest risk estimate observed for BCAA. The OR estimates for sex-specific analysis have wide CIs for the highest exposure category and therefore need to be interpreted cautiously.

For colon cancer (Table 8.11), as in previous analyses, an increase in risk is only observed among males. ORs are  $>1.8$  for male colon cancer for TTHM exposure of  $\geq 80 \mu\text{g/L}$  and for HAA5 of  $\geq 60 \mu\text{g/L}$ . Rectal cancer is not associated with by-product exposure in the analysis of higher exposures (Table 8.12).

### Joint Effects of Chlorination By-Products and Tap Water Consumption

Tables 8.13 and 8.14 present the risk estimates associated with exposure to each of three chlorination by-products (TTHM, BDCM, and HAA5) and volume of tap water consumed for bladder and male colon cancer. The analysis was restricted to the by-product exposures suggestive of a relationship in previous analyses. The referent category (TTHM quartile 1, volume tertile 1) for comparisons are those subjects with less than 10 years of exposure to a high level of chlorination by-products and in the lowest third of tap water consumption. The contrast of most relevance to the relationship is for those with the highest level of chlorination by-product exposure and the highest volume of tap water consumption. In addition, a pattern of increasing risks for TTHM quartile 4 (e.g., increasing water consumption among those with high by-product exposure) and across the bottom row (e.g., increasing by-product level among those consuming the highest volume of water) are relevant to interpreting the joint effects of by-product and water consumption. CIs are wide in these comparisons, reflecting the number of categories of exposure examined and subsequent small number of subjects in some categories.

For bladder cancer, statistically significant relative risks of  $\geq 2$  are found in the highest-exposure category for TTHM, BDCA, HAA5, and BCAA exposures (Table 8.13). Relative risks also increase with water consumption among those with high by-product exposure (TTHM quartile 4) and with by-product exposure among those with the high volume of water consumed (volume tertile 3). Also of note is a pattern of increasing risk with volume of water consumed for those with low by-product exposure (TTHM quartile 1), as this result supports an independent effect of water consumption.

For colon cancer, those with high by-product exposure and with high tap water consumption had statistically significant relative risks  $>2$  for each exposure (Table 8.14). The highest relative risks are for HAA5 and BCAA exposure metrics. The pattern of risk across exposure categories is inconsistent.

### Restricted to Those Served by Surface Water

A concern in the analysis presented above is that subjects whose water source has primarily been groundwater are placed in the referent category for all analyses; thus observed effects may be due to another ground-surface water quality difference apart from chlorination by-products. Selected analyses are replicated for a subset of subjects served by a surface water source for a minimum of 30 years (of the 50-year exposure window) (Tables 8.15 and 8.16). Relative risk estimates are similar, but in general lower for the restricted analysis.

**Table 8.13**  
**New exposures, bladder cancer: ORs for joint effect exposure and tap water consumption**

Volume of tap water (tertiles)	Quartiles			
	1	2	3	4
<b>TTHM</b>				
Low	1.00	1.40 (0.77–2.56)	1.96 (1.11–3.47)	1.73 (0.99–3.05)
Medium	1.62 (0.93–2.85)	1.79 (1.02–3.16)	1.64 (0.95–2.83)	1.61 (0.93–2.80)
High	1.46 (0.84–2.53)	1.53 (0.87–2.71)	1.71 (1.00–2.94)	2.81 (1.62–4.89)
<b>BDCM</b>				
Low	1.00	1.66 (0.92–3.00)	1.80 (0.96–3.39)	2.04 (1.17–3.57)
Medium	1.79 (1.00–3.12)	1.78 (1.01–3.13)	2.15 (1.17–3.95)	1.63 (0.95–2.80)
High	1.51 (0.85–2.66)	2.04 (1.16–3.59)	2.34 (1.25–4.37)	2.06 (1.21–3.50)
<b>HAA5</b>				
Low	1.00	1.83 (1.03–3.26)	1.73 (0.97–3.10)	1.51 (0.86–2.68)
Medium	1.62 (0.93–2.83)	1.49 (0.84–2.64)	1.65 (0.95–2.87)	1.81 (1.05–3.13)
High	1.54 (0.89–2.67)	1.93 (1.12–3.33)	1.29 (0.73–2.28)	2.61 (1.51–4.51)

Note: ORs are adjusted for age, sex, current smoking, log pack-years of smoking, and education.

**Table 8.14**  
**New exposures, colon cancer (males): ORs for joint effect exposure and tap water consumption**

Volume of tap water (tertiles)	Quartiles			
	1	2	3	4
<b>TTHM</b>				
Low	1.00	1.67 (0.85–3.29)	1.88 (0.95–3.71)	2.07 (1.09–3.92)
Medium	1.72 (0.91–3.26)	1.80 (0.91–3.56)	1.48 (0.77–2.83)	1.95 (1.02–3.72)
High	1.13 (0.57–2.23)	2.52 (1.32–4.80)	2.18 (1.16–4.09)	2.71 (1.36–5.42)
<b>BDCM</b>				
Low	1.00	2.13 (1.08–4.19)	2.12 (1.02–4.40)	2.25 (1.16–4.34)
Medium	1.90 (0.96–3.73)	2.43 (1.25–4.73)	2.13 (1.03–4.43)	1.58 (0.82–3.06)
High	1.34 (0.66–2.70)	2.93 (1.49–5.74)	3.30 (1.57–6.95)	2.33 (1.22–4.44)
<b>HAA5</b>				
Low	1.00	1.56 (0.78–3.12)	1.93 (0.97–3.81)	2.31 (1.21–4.40)
Medium	1.85 (0.97–3.52)	1.98 (1.01–3.86)	1.48 (0.75–2.90)	1.83 (0.95–3.55)
High	1.19 (0.59–2.37)	2.82 (1.50–5.32)	1.90 (0.97–3.73)	2.73 (1.38–5.40)

Note: ORs are adjusted for age, sex, current smoking, log pack-years of smoking, and education.

**Table 8.15**  
**Relative risk for bladder cancer among surface water users for selected exposures\***

Bladder	Years (50-year exposure window)	Years	30+ surface water years
		OR (95% CI) <sup>†</sup>	OR (95% CI) <sup>†</sup>
TTHM ≥40 µg/L	<10	1.00	1.00
	10–29	1.05 (0.71–1.55)	1.07 (0.70–1.65)
	30+	1.23 (0.87–1.73)	1.15 (0.81–1.63)
Average TTHM per standard deviation (21.7)		1.12 (1.03–1.22)	1.10 (1.00–1.20)
BDCM ≥8 µg/L	<10	1.00	1.00
	10–29	1.40 (0.96–2.05)	1.19 (0.79–1.80)
	30+	1.28 (0.93–1.76)	1.07 (0.79–1.46)
Average BDCM per standard deviation (3.1)		1.13 (1.03–1.24)	1.04 (0.93–1.16)
HAA5 ≥30 µg/L	<10	1.00	1.00
	10–29	1.04 (0.70–1.54)	1.08 (0.71–1.66)
	30+	1.35 (0.95–1.91)	1.27 (0.89–1.81)
Average HAA5 per standard deviation (17.8)		1.10 (1.01–1.19)	1.08 (0.99–1.18)
BCAA ≥4 µg/L	<10	1.00	1.00
	10–29	1.69 (1.07–2.65)	1.39 (0.83–2.31)
	30+	1.43 (1.00–2.04)	1.32 (0.93–1.87)
Average BCAA per standard deviation (1.3)		1.16 (1.05–1.27)	1.07 (0.96–1.20)

\* 575 cases, 1,153 controls.

<sup>†</sup> ORs are adjusted for age, sex, current smoking, log pack-years of smoking, and education.

## DISCUSSION

The reestimation of chlorination by-product levels in Ontario and linkage to the Ontario case-control study resulted in a more robust dataset. The assignment of TTHM exposure is similar to the original study, and therefore the risk estimates associated with TTHM exposure are similar for bladder, colon, and rectal cancers. A smaller number of subjects is assigned high exposure in the reanalysis, resulting in wide CIs in some analyses. Perhaps the most important contribution of the new exposure database is the opportunity to explore relationships with other by-product representations, including BDCM and total and specific HAAs that were not available in the original study.

One of the objectives of this study was to reduce misclassification of subject exposure. Although the correlation between new and original predictions at the plant level was only moderate, the correlation was higher among large treatment plants, resulting in a high correlation for assignment of TTHM exposure to subject residence ( $r = 0.86$ ). In addition, the correlation between new and original exposure assignment is similar for cases and controls, indicating that misclassification in the original study was likely nondifferential. Nondifferential misclassification

**Table 8.16**  
**Relative risk for colon cancer among surface water users for selected exposures\***

Colon	Years (50-year exposure window)	Years	30+ surface water years
		OR (95% CI) <sup>†</sup>	OR (95% CI) <sup>†</sup>
TTHM ≥40 µg/L	<10	1.00	1.00
	10–29	1.04 (0.71–1.52)	1.00 (0.57–1.73)
	30+	1.37 (0.99–1.89)	1.36 (0.89–2.08)
Average TTHM per standard deviation (21.7)		1.16 (1.04–1.29)	1.12 (0.99–1.25)
BDCM ≥8 µg/L	<10	1.00	1.00
	10–29	1.08 (0.73–1.58)	1.17 (0.68–2.01)
	30+	0.93 (0.67–1.28)	1.13 (0.74–1.73)
Average BDCM per standard deviation (3.1)		1.12 (1.00–1.26)	0.98 (0.85–1.13)
HAA5 ≥30 µg/L	<10	1.00	1.00
	10–29	0.90 (0.61–1.33)	0.92 (0.53–1.61)
	30+	1.41 (1.02–1.97)	1.38 (0.89–2.13)
Average HAA5 per standard deviation (17.8)		1.13 (1.02–1.26)	1.10 (0.98–1.23)
BCAA ≥4 µg/L	<10	1.00	1.00
	10–29	1.27 (0.80–2.02)	1.32 (0.69–2.51)
	30+	0.73 (0.49–1.09)	0.94 (0.57–1.55)
Average BCAA per standard deviation (1.3)		1.14 (1.01–1.29)	1.01 (0.87–1.17)

\* 356 cases, 710 controls.

† ORs are adjusted for age, sex, education, and body mass index.

of exposure tends to make cases and controls more similar with respect to exposure assignment and therefore to bias risk estimates toward observing no effect.

The Ontario monitoring data for the period 1986–1993, used in the original study, report peak TTHM values. The peak value is on average 80% of the yearly mean value for each water plant. Analyses presented here, using the original (peak) and new (mean) exposure assignment, consider TTHM categories reflecting this contrast. However, the ratio of TTHM estimates by plant in the new (mean) and original (peak) estimates is only 55%. This resulted in fewer exposed subjects in analysis using new estimates compared to the original estimates. Categories in the analysis of cumulative TTHM-years were determined by the quartiles of the distribution among controls and are therefore not influenced by systematic differences between peak and mean estimates.

The relative risks associated with TTHM exposure obtained in the original study are compared with similar metrics based on the new exposure estimates. In general, results from the reanalysis are consistent with the original study and do not alter the conclusions or interpretation of the original papers.

The new by-product estimates were used for the analysis of several by-product parameters over a 50-year exposure window for the three cancer sites. The analysis of average exposure is the most robust with respect to ensuring adequate sample size in each exposure category. Relative risks for bladder cancer >1.4 are associated with high average exposure to TTHM, BDCM, HAA5, TCAA, and BCAA. Of note, the largest bladder cancer risks are found for the brominated by-product exposures (BDCM and BCAA). For colon cancer, excess risk is only observed among males. Relative risks for colon cancer approaching 2.0 are observed for TTHM and all of the HAA5 exposures. Risk estimates are lower and not statistically significant for high BDCM exposure. Risk estimates for rectal cancer are consistently close to the null value.

In addition to the results presented here, the new exposure estimates provided by this project result in numerous other opportunities for analysis. Other analyses currently being explored include risk among subgroups (i.e., bladder risk among smokers and nonsmokers), simultaneous modeling of risk associated with different by-products, examination of different exposure windows, analysis restricted to those with homogeneous exposures, examination of colon and rectal risk according to cancer location (i.e., distal colon, proximal colon, sigmoid junction, rectum), and pooling of the Ontario and Iowa data.

This analysis based on new exposure estimates confirms our previously reported results and provides some additional insights with respect to a relationship between specific by-products and cancer risk. In particular, the relationship between bladder cancer and brominated by-products and the relationship between male colon cancer with TTHM and HAAs are noteworthy.

## **CHAPTER 9**

# **REANALYSIS OF IOWA CASE-CONTROL STUDY: BLADDER, COLON, AND RECTAL CANCER RISKS ASSOCIATED WITH THMs AND OTHER CHLORINATION BY-PRODUCT COMPOUNDS**

## **INTRODUCTION**

The disinfection of drinking water supplies with chlorine generates a mixture of many by-products, including THMs and HAAs. The health effects of exposure to this mixture of chlorination by-products through ingestion or dermal absorption have been studied epidemiologically for the past 30 years. The focus has been on cancer and reproductive outcomes. With some consistency, the studies of bladder cancer have observed elevated risk. Evidence for a link with colon and rectal cancers is less consistent. The more recent studies (among them Cantor et al. [1998] and King and Marrett [1996]) have improved on the limitations of earlier studies by gathering residential and water usage data for individuals over an extended period of time rather than using recent cross-sectional information, but the estimate of past DBP levels has remained a challenge. It is not clear which component or components of the mixture may be responsible for increased cancer risks or other adverse health outcomes.

Most epidemiologic studies have examined chlorinated surface water as a marker for the DBP mix since by-product levels are known to be higher in treated surface water sources than in groundwater sources, due to higher levels of organic matter present in surface waters. Some studies examined risk associated with specific by-products, primarily chloroform and TTHM. However, little or no quantitative data existed for these compounds before the mid-1970s. In view of the typically long latency for most cancers, past levels of specific DBPs before the 1970s must be estimated. Statistical models to predict DBP levels have been developed using current DBP and water quality data (King and Marrett 1996), but their use in the estimate of past exposure is often limited by the availability of historical water quality and treatment data to develop the models. Data on TOC concentrations and chlorine dose, for example, are often difficult to obtain or not available historically.

In the 1987 Iowa case-control analysis, a semi-quantitative model for estimating past DBP exposure was developed. Mean concentrations of 1987 TTHM levels were used to estimate past levels for communities using a similar source and treatment. Since this method assigns the same THM level to communities with similar water source and treatment practices, it may not accurately represent the variation between communities, especially for those that use surface water sources which have varying amounts of organic matter. To improve the precision of exposure assessment in our study, the authors participated in this collaborative effort to reestimate historical THM and other DBP levels for Iowa water utilities by examining each community separately instead of grouping them. Quantitative DBP and water quality data from 1987 until the present were used from multiple sources in conjunction with historical water source and treatment data to develop new estimates of past TTHM levels for Iowa water utilities (chapter 7). Estimates of HAA levels were also made to allow for an analysis of the potential associations between HAAs and cancer risk.

In this chapter, we present the results from a reanalysis of the Iowa case-control data from 1987 using exposure indices calculated from the new THM and HAA estimates. Risk estimates



for bladder, colon, and rectal cancer are presented for some specific THMs and HAAs, TTHM, HAA5, and HAA6, in addition to a comparison of cancer risks between the original and new exposure assessment methods for TTHM.

## **MATERIALS AND METHODS**

### **Study Population**

The study population used in the original analyses of the 1987 case-control data has been described in detail elsewhere (Cantor et al. 1998). In brief, study subjects were chosen for analysis if their water usage could be established for at least 70% of their lifetime. This selection criterion resulted in an analysis subset of 1,983 controls, 1,123 bladder cancer cases, 560 colon cancer cases, and 537 rectal cancer cases and accounted for 80.6% of total study person-years of exposure. In the reanalysis of the original exposure measurements, which were calculated for exposure occurring over the subjects' lifetime, we assigned DBP exposure for 97.1% of person-years that were spent on Iowa public water in the original analysis. After applying the restriction that at least 70% of a person's lifetime have known exposure, the resulting subset used in the reanalysis contained 1,972 controls and 1,114, 559, and 535 cases, respectively, for cancers of the bladder, colon, and rectum, and accounted for 80.1% of total study person-years. To facilitate comparisons of cancer risks with the Ontario case-control study, the exposure period of 50 years for each participant up to 2 years before his or her entry into the study was analyzed. Subjects in these analyses had to have measurable exposure for at least 70% of the time period (35 of those 50 years). This resulted in a study subset of 2,046 controls, 1,187 bladder cancer cases, 579 colon cancer cases, and 553 rectal cancer cases. The person-years in this 50-year subset accounted for 61.3% of total lifetime person-years. A description of the study populations by number of subjects and person-years is presented in [Table 9.1](#).

### **Exposure Assessment**

In both the original analysis and the current reanalysis, residential histories collected from subjects were linked with historical water source and treatment data for those years during which the subjects reported drinking public water. In the 1987 analysis, geometric mean TTHM levels from 1987 water samples were used as estimates of past exposure for communities with similar water sources and chlorination practices. For the calculation of means, communities were grouped according to the water source (surface or ground with average depth <15, 15–45, 45–150, or >150 m (<51, 51–150, 151–500, or >500 ft) and point of chlorination (no chlorination, pre- and postchlorination, or postchlorination only) used by the community's water treatment utility.

A different approach was used to assess historical exposure for the current analysis. Instead of grouping together communities with similar water sources and treatments, each community receiving surface water, mixed surface and groundwater, or shallow groundwater (served by alluvial wells <15 m [<50 ft] deep) was examined separately. Multiple sources of DBP data were used in conjunction with treatment data and water quality measurements, such as pH and TOC concentrations, to develop estimates for each of eight DBPs or combinations (CHCl<sub>3</sub>, BDCM, TTHM, DCAA, TCAA, HAA5, BCAA, and HAA6). Community-specific DBP levels based on current DBP measurements were used as estimates of past exposure if the water source and treatment serving that same community were similar to those used when the water sample



**Table 9.1**  
**Summary of study populations used in analyses: Iowa case-control study of**  
**bladder, colon, and rectal cancers**

Study population	Number of subjects				Person-years	
	Controls	Bladder cancer	Colon cancer	Rectal cancer	Count	%
Total subjects eligible for participation in study	2,434	1,452	685	655	361,317	100.0
Subjects with known exposure for 70% of lifetime, original exposure assessment only (1987 analyses)	1,983	1,123	560	537	291,251	80.6
Subjects with known exposure for 70% of lifetime, old and new exposure assessment (2003 comparison with 1987)	1,972	1,114	559	535	289,510	80.1
Subjects with known exposure for 70% of lifetime, new exposure assessment only (not yet analyzed)	2,012	1,152	564	545	295,985	81.9
Subjects with known exposure for 70% of 50-year window, new exposure assessment only (2003 analysis of new exposures)	2,046	1,187	579	553	221,414	61.3

was taken. If there were differences in source or treatment, appropriate adjustments to DBP levels were made based on what is known about the chemistry of water chlorination. These estimates used a case study approach. For communities with low DBP levels, primarily those served by deeper ground or nonalluvial waters, a central tendency model was developed to assign an average level as historic exposure based on well depth and whether or not the community implemented softening or coagulation treatments. A more detailed description of this new exposure assessment method is found in chapter 7.

## Analysis

For use in statistical analyses of cancer risk in our study population, various exposure metrics were calculated for each of the estimated DBPs. In this report, average TTHM concentration was examined for the subjects' lifetime to allow for direct comparisons with results published previously. For the 50-year period up to 2 years before study entry, both average concentration and the duration at moderate (e.g., 40 µg/L for TTHM, 30 µg/L for HAA6) or high (e.g., 80 µg/L

or 100 µg/L for TTHM, 60 µg/L for HAA6) concentrations were analyzed to facilitate comparisons with the reanalysis of the Ontario study (chapter 8). Average concentration was defined as the sum of the DBP concentration, multiplied by years at that level, divided by the number of years with available exposure data.

OR estimates of cancer risk due to DBP exposure were generated using unconditional logistic regression. The OR is an estimate of the relative risk associated with an estimated high exposure to DBPs compared to a referent lower exposure. For example, an OR of 1.5 can be interpreted as an increased risk of 50%. Variables included in the models as potential confounders were the same as those used in the original analyses of these data. These include age, sex, study group, education, cigarette smoking, and high-risk occupation for bladder cancer models; age and sex for colon cancer models; and age, sex, and average population size for rectal cancer models. Tests for trends were performed by entering the exposure measure as a continuous variable. For the analysis of new average DBP exposures, the continuous form of the exposure was examined per one standard deviation of the distribution among controls. This allowed for an interpretation of the OR associated with the continuous form of the variable in terms of a meaningful unit for each DBP.

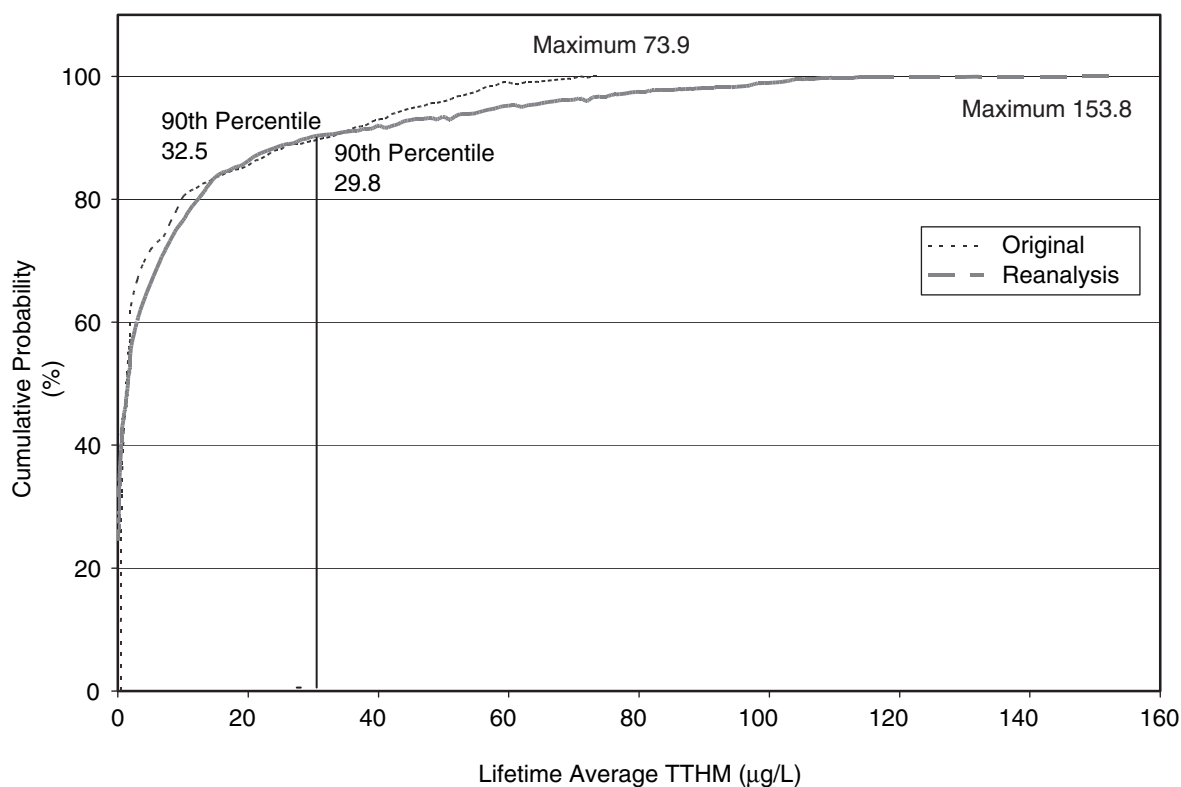
Comparisons between old and new average TTHM concentrations among the control population were made using the Pearson correlation coefficient.

## RESULTS

### Comparison of Old and New Estimates of Lifetime Average THM

Using our new estimates of historic THM levels, lifetime average TTHM was recalculated for our study subjects and compared with the measurement of lifetime average TTHM concentration used in the original analysis. [Figure 9.1](#) shows the cumulative probability distribution for each of the two variables among our control population ( $n = 1,972$ ). The distributions are similar for concentrations up to the 90th percentile, or about 30 µg/L. For the 10% of controls with the highest concentrations of average TTHM, concentrations of the new lifetime average variable ranged from 29.8 to 153.8 µg/L, whereas the concentrations using our previous estimates ranged from 32.5 to 73.9 µg/L. [Figure 9.2](#) shows a scatterplot among all controls of the old lifetime average variable by the new variable. When the control population is considered as a whole, these measures are highly correlated (Pearson  $r = 0.84$ ,  $p < 0.0001$ ); however the magnitude of the correlation decreases when subpopulations with higher average TTHM concentrations are considered. The correlation is 0.67 among subjects with average TTHM  $> 10$  µg/L ( $n = 523$ ), and 0.42 when that level is increased to 25 µg/L ( $n = 295$ ). The correlation decreases to 0.22 ( $p < 0.002$ ) when controls with average exposure of at least 40 µg/L ( $n = 209$ ) are examined. Comparable correlations were observed among cases between the old and new measures of average TTHM concentration.

OR estimates of cancer risk for bladder, colon, and rectal cancers are presented in Tables 9.2, 9.3, and 9.4 for both the new and old estimated levels of lifetime average TTHM exposure. Both estimates of TTHM were categorized using the 35th, 60th, 75th, 90th and 95th percentiles from the control distribution of the original variable, which resulted in a high to low comparison of  $\geq 46.4$  µg/L to  $\leq 0.7$  µg/L. Results using the recalculated exposure index are very similar to the risk estimates from the original analysis. Elevated risks were observed for bladder cancer among males (OR = 1.46, 95% CI = 1.03–2.10, for highest level compared to lowest level, [Table 9.2](#)), and rectal cancer among males and females (OR = 1.51, 95% CI = 1.01–2.30, for highest level

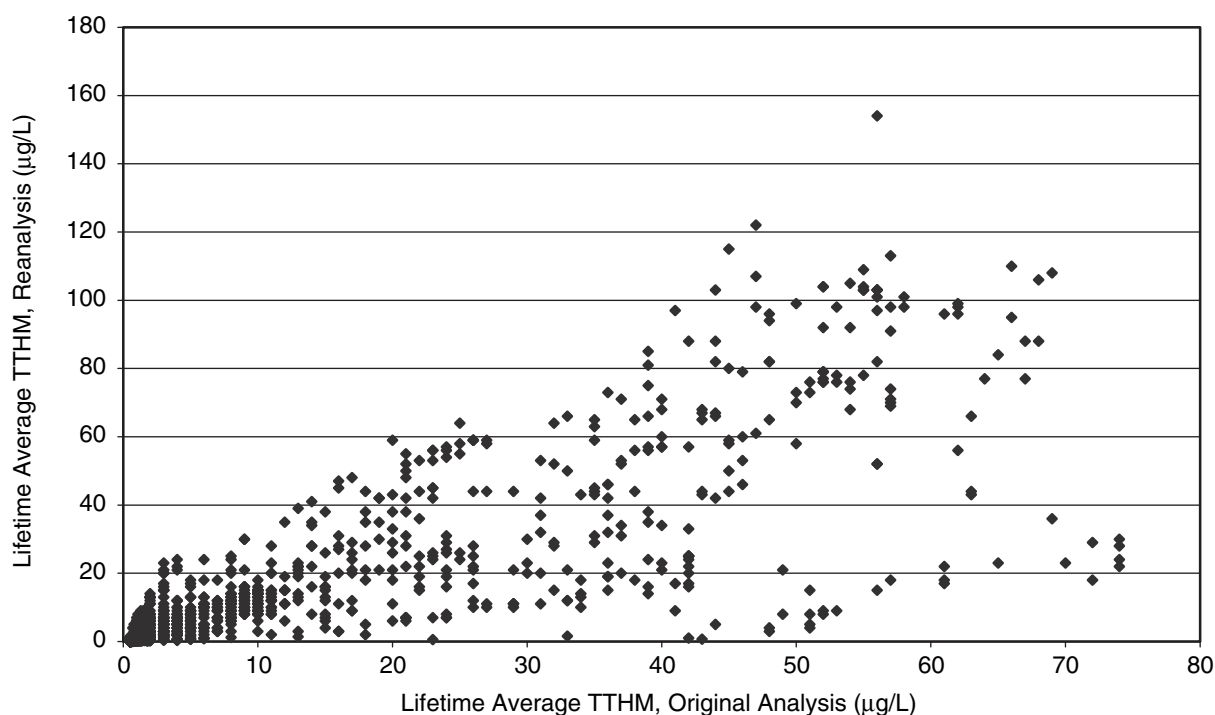


**Figure 9.1 Cumulative probability distributions for lifetime average TTHM calculated using two exposure methods among controls, Iowa 1987**

compared to lowest level, [Table 9.4](#)). Also observed was a modest but statistically nonsignificant increase in risk for colon cancer among males; ORs were 1.51 (95% CI = 0.7–3.3, [Table 9.3](#)) and 1.35 (95% CI = 0.8–2.2, [Table 9.3](#)) for the highest two exposure levels compared to the lowest level. As reported originally, no increase in colon cancer risk was observed for males and females combined.

### Analyses of New Exposure Estimates

Since the new average TTHM variable differed from the original measurement at the upper end of the exposure distribution, the new average TTHM concentration was categorized using ranges of exposure that would better allow the examination of potential risk associated with higher exposures. These results are presented for each by-product or by-product group (TTHM, BDCM, DCAA, TCAA, BCAA, and HAA6) in [Tables 9.5](#), [9.6](#), and [9.7](#) for bladder, colon, and rectal cancers, respectively. The 35th, 70th, 90th, 93.75th and 97.5th percentiles among the controls were used to categorize subjects into exposure groups. For TTHM, this categorization resulted in a high-to-low comparison of  $\geq 96.1$  µg/L to  $\leq 0.5$  µg/L. Subjects with identifiable exposure for at least 35 of the 50 years up to 2 years before study entry (i.e., 70% of the time period) were used in these analyses to facilitate comparisons with the Ontario findings presented in chapter 8.



**Figure 9.2 Comparison of lifetime average TTHM calculated using two exposure methods among controls, Iowa 1987**

For TTHM, DCAA, TCAA, and HAA6, elevated risks were observed for the highest-exposure category compared with the lowest-exposure category among males for all three cancer sites. Exposure to average TTHM levels  $>96.1 \mu\text{g/L}$  compared with levels  $\leq 0.5 \mu\text{g/L}$  was associated with an 80% increased risk of bladder cancer (95% CI = 1.02–3.2, [Table 9.5](#)), a 64% increased risk of colon cancer (95% CI = 0.8–3.5, [Table 9.6](#)), and a 166% increased risk of rectal cancer (95% CI = 1.3–5.5, [Table 9.7](#)). For males at the highest average total HAA exposure (HAA6  $>52.6 \mu\text{g/L}$ ), the estimate of risk for rectal cancer (OR = 2.67, 95% CI = 1.3–5.4, [Table 9.7](#)) was slightly higher than colon cancer (OR 1.92, 95% CI = 0.95–3.9, [Table 9.6](#)) and bladder cancer (OR = 2.00, 95% CI = 1.2–3.4, [Table 9.5](#)). Among females, risk increases in rectal cancer for TTHM, DCAA, TCAA, and total HAAs were inconsistent and nonsignificant; for bladder and colon cancer, no increases in risk were observed.

Risk estimates for the two brominated by-products, BDCM and BCAA, were not as consistent among males as those observed for TTHM, DCAA, TCAA, and HAA6. For BDCM, high average exposure is suggestive of an increased risk of bladder, colon, and rectal cancers among males, but results did not reach statistical significance and no linear trends were observed. No consistent increases in risk estimates were observed for elevated BCAA exposure; in fact, ORs were  $<1.0$  for the highest-exposure category for each site. Among females, no significant trends or elevations in risk were observed for these compounds.

In addition to examining average DBP exposures, the risk associated with duration of exposure at specified by-product concentrations was also examined. The results from these analyses for moderate and high levels are presented in [Tables 9.8](#) and [9.9](#) for bladder cancer, in [Tables 9.10](#) and [9.11](#) for colon cancer, and in [Tables 9.12](#) and [9.13](#) for rectal cancer.

**Table 9.2**  
**Comparison of risk estimates of bladder cancer for two methods of**  
**estimating lifetime TTHM exposure**

Lifetime average TTHM*	Original (1987) analysis			Reanalysis		
	Controls	Cases	OR <sup>†</sup> (95% CI)	Controls	Cases	OR <sup>†</sup> (95% CI)
<b>Males</b>						
≤0.7	501 (38.3)	269 (30.7)	1.00	592 (45.4)	347 (40.0)	1.00
0.8–2.2	314 (24.0)	244 (27.9)	1.27 (1.00–1.60)	157 (12.0)	107 (12.3)	1.02 (0.80–1.40)
2.3–8.0	188 (14.4)	123 (14.1)	1.14 (0.85–1.50)	212 (16.3)	156 (18.0)	1.09 (0.80–1.40)
8.1–32.5	194 (14.8)	133 (15.2)	1.11 (0.80–1.50)	230 (17.6)	150 (17.3)	1.02 (0.80–1.30)
32.6–46.3	54 (4.1)	53 (6.1)	1.67 (1.10–2.60)	28 (2.2)	24 (2.8)	1.32 (0.70–2.40)
≥46.4	57 (4.4)	53 (6.1)	1.53 (1.00–2.40)	85 (6.5)	83 (9.6)	1.46 (1.03–2.10)
P (trend)			0.03			0.06
<b>Females</b>						
≤0.7	194 (28.7)	71 (28.6)	1.00	249 (37.3)	93 (37.7)	1.00
0.8–2.2	181 (26.8)	68 (27.4)	0.85 (0.60–1.30)	85 (12.7)	33 (13.4)	0.97 (0.60–1.60)
2.3–8.0	110 (16.3)	42 (16.9)	0.76 (0.50–1.30)	128 (19.2)	44 (17.8)	0.75 (0.50–1.20)
8.1–32.5	103 (15.3)	44 (17.7)	0.91 (0.60–1.50)	132 (19.8)	57 (23.1)	0.92 (0.60–1.40)
32.6–46.3	45 (6.7)	11 (4.4)	0.58 (0.30–1.30)	22 (3.3)	5 (2.0)	0.52 (0.20–1.50)
≥46.4	42 (6.2)	12 (4.8)	0.60 (0.30–1.30)	52 (7.8)	15 (6.1)	0.75 (0.40–1.50)
P (trend)			0.07			0.30
<b>Total</b>						
≤0.7	695 (35.1)	340 (30.3)	1.00	841 (42.7)	440 (39.5)	1.00
0.8–2.2	495 (25.0)	312 (27.8)	1.18 (0.96–1.50)	242 (12.3)	140 (12.6)	1.02 (0.80–1.30)
2.3–8.0	298 (15.0)	165 (14.7)	1.07 (0.80–1.40)	340 (17.2)	200 (18.0)	1.01 (0.80–1.30)
8.1–32.5	297 (15.0)	177 (15.8)	1.07 (0.80–1.40)	362 (18.4)	207 (18.6)	1.00 (0.80–1.30)
32.6–46.3	99 (5.0)	64 (5.7)	1.26 (0.87–1.80)	50 (2.5)	29 (2.6)	1.04 (0.60–1.70)
≥46.4	99 (5.0)	65 (5.8)	1.22 (0.85–1.80)	137 (7.0)	98 (8.8)	1.23 (0.91–1.70)
P (trend)			0.40			0.30

\* Exposure categories based on 35th, 60th, 75th, 90th, and 95th percentiles in distribution among controls of lifetime average TTHM as estimated in the original analysis.

† Adjusted for age (four groups), sex (where appropriate), study group, high-risk occupation, cigarette smoking, and education.

**Table 9.3**  
**Comparison of risk estimates of colon cancer for two methods of**  
**estimating lifetime THM exposure**

Lifetime average TTHM*	Original (1987) analysis			Reanalysis		
	Controls	Cases	OR <sup>†</sup> (95% CI)	Controls	Cases	OR <sup>†</sup> (95% CI)
<b>Males</b>						
≤0.7	501 (38.3)	100 (35.3)	1.00	592 (45.4)	129 (45.6)	1.00
0.8–2.2	314 (24.0)	72 (25.4)	1.17 (0.80–1.60)	157 (12.0)	28 (9.9)	0.83 (0.50–1.30)
2.3–8.0	188 (14.4)	39 (13.8)	1.04 (0.70–1.60)	212 (16.3)	44 (15.6)	0.96 (0.70–1.40)
8.1–32.5	194 (14.8)	40 (14.1)	1.05 (0.70–1.60)	230 (17.6)	48 (17.0)	0.97 (0.70–1.40)
32.6–46.3	54 (4.1)	16 (5.7)	1.54 (0.80–2.80)	28 (2.2)	9 (3.2)	1.51 (0.70–3.30)
≥46.4	57 (4.4)	16 (5.7)	1.39 (0.80–2.50)	85 (6.5)	25 (8.8)	1.35 (0.80–2.20)
P (trend)			0.2			0.2
<b>Females</b>						
≤0.7	194 (28.7)	91 (32.9)	1.00	249 (37.3)	118 (42.8)	1.00
0.8–2.2	181 (26.8)	70 (25.3)	0.84 (0.60–1.20)	85 (12.7)	21 (7.6)	0.52 (0.30–0.87)
2.3–8.0	110 (16.3)	41 (14.8)	0.80 (0.50–1.20)	128 (19.2)	58 (21.0)	0.96 (0.70–1.40)
8.1–32.5	103 (15.3)	49 (17.7)	1.05 (0.70–1.60)	132 (19.8)	55 (19.9)	0.89 (0.60–1.30)
32.6–46.3	45 (6.7)	11 (4.0)	0.52 (0.30–1.10)	22 (3.3)	6 (2.2)	0.58 (0.20–1.50)
≥46.4	42 (6.2)	15 (5.4)	0.77 (0.40–1.50)	52 (7.8)	18 (6.5)	0.73 (0.40–1.30)
P (trend)			0.30			0.50
<b>Total</b>						
≤0.7	695 (35.1)	191 (34.1)	1.00	841 (42.7)	247 (44.2)	1.00
0.8–2.2	495 (25.0)	142 (25.4)	1.01 (0.80–1.30)	242 (12.3)	49 (8.8)	0.68 (0.50–0.95)
2.3–8.0	298 (15.0)	80 (14.3)	0.93 (0.70–1.30)	340 (17.2)	102 (18.3)	0.97 (0.70–1.30)
8.1–32.5	297 (15.0)	89 (15.9)	1.07 (0.80–1.40)	362 (18.4)	103 (18.4)	0.94 (0.70–1.20)
32.6–46.3	99 (5.0)	27 (4.8)	0.93 (0.60–1.50)	50 (2.5)	15 (2.7)	0.97 (0.50–1.80)
≥46.4	99 (5.0)	31 (5.5)	1.06 (0.70–1.60)	137 (7.0)	43 (7.7)	1.03 (0.70–1.50)
P (trend)			0.90			0.60

\* Exposure categories based on 35th, 60th, 75th, 90th, and 95th percentiles in distribution among controls of lifetime average TTHM as estimated in the original analysis.

† Adjusted for age (four groups) and sex.

**Table 9.4**  
**Comparison of risk estimates of rectal cancer for two methods of**  
**estimating lifetime THM exposure**

Lifetime average TTHM*	Original (1987) analysis			Reanalysis		
	Controls	Cases	OR <sup>†</sup> (95% CI)	Controls	Cases	OR <sup>†</sup> (95% CI)
<b>Males</b>						
≤0.7	501 (38.3)	115 (36.2)	1.00	592 (45.4)	144 (45.3)	1.00
0.8–2.2	314 (24.0)	70 (22.0)	1.03 (0.70–1.50)	157 (12.0)	36 (11.3)	1.01 (0.70–1.60)
2.3–8.0	188 (14.4)	53 (16.7)	1.34 (0.90–2.00)	212 (16.3)	40 (12.6)	0.83 (0.60–1.30)
8.1–32.5	194 (14.8)	39 (12.3)	1.02 (0.70–1.60)	230 (17.6)	66 (20.8)	1.36 (0.93–2.00)
32.6–46.3	54 (4.1)	23 (7.2)	2.19 (1.20–3.90)	28 (2.2)	5 (1.6)	0.75 (0.30–2.00)
≥ 46.4	57 (4.4)	18 (5.7)	1.69 (0.91–3.20)	85 (6.5)	27 (8.5)	1.54 (0.91–2.60)
P (trend)			0.02			0.10
<b>Females</b>						
≤0.7	194 (28.7)	58 (26.5)	1.00	249 (37.3)	78 (35.9)	1.0
0.8–2.2	181 (26.8)	53 (24.2)	1.04 (0.70–1.60)	85 (12.7)	26 (12.0)	1.05 (0.60–1.80)
2.3–8.0	110 (16.3)	33 (15.1)	1.10 (0.70–1.90)	128 (19.2)	34 (15.7)	0.92 (0.60–1.50)
8.1–32.5	103 (15.3)	44 (20.1)	1.53 (0.91–2.60)	132 (19.8)	47 (21.7)	1.20 (0.70–2.00)
32.6–46.3	45 (6.7)	13 (5.9)	1.11 (0.50–2.30)	22 (3.3)	10 (4.6)	1.64 (0.70–3.80)
≥46.4	42 (6.2)	18 (8.2)	1.68 (0.80–3.30)	52 (7.8)	22 (10.1)	1.53 (0.80–2.90)
P (trend)			0.20			0.07
<b>Total</b>						
≤0.7	695 (35.1)	173 (32.2)	1.00	841 (42.7)	222 (41.5)	1.00
0.8–2.2	495 (25.0)	123 (22.9)	1.05 (0.80–1.40)	242 (12.3)	62 (11.6)	1.02 (0.70–1.40)
2.3–8.0	298 (15.0)	86 (16.0)	1.24 (0.91–1.70)	340 (17.2)	74 (13.8)	0.87 (0.60–1.20)
8.1–32.5	297 (15.0)	83 (15.5)	1.23 (0.88–1.70)	362 (18.4)	113 (21.1)	1.29 (0.96–1.70)
32.6–46.3	99 (5.0)	36 (6.7)	1.66 (1.06–2.60)	50 (2.5)	15 (2.8)	1.15 (0.60–2.10)
≥46.4	99 (5.0)	36 (6.7)	1.66 (1.05–2.60)	137 (7.0)	49 (9.2)	1.51 (1.01–2.30)
P (trend)			0.01			0.03

\* Exposure categories based on 35th, 60th, 75th, 90th, and 95th percentiles in distribution among controls of lifetime average TTHM as estimated in the original analysis.

† Adjusted for age (four groups), sex (where appropriate), and average population size of lifetime residences.

**Table 9.5**  
**Analysis of bladder cancer risk and average exposure to specific chlorination by-products**

Exposure	Average* (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI) <sup>†</sup>	Cases	Controls	OR (95% CI) <sup>†</sup>	Cases	Controls	OR (95% CI) <sup>†</sup>
Average	≤0.5	370	717	1.00	294	510	1.00	76	207	1.00
TTHM	0.6–8.4	442	716	1.06 (0.88–1.30)	341	442	1.13 (0.91–1.40)	101	274	0.82 (0.60–1.20)
	8.4–36.1	238	409	0.98 (0.80–1.20)	179	264	1.04 (0.80–1.30)	59	145	0.77 (0.50–1.20)
	36.2–58.0	46	77	0.97 (0.60–1.50)	38	47	1.15 (0.70–1.80)	8	30	0.63 (0.30–1.60)
	58.1–96.1	55	76	1.17 (0.80–1.80)	46	51	1.31 (0.80–2.10)	9	25	0.80 (0.30–2.00)
	>96.1	36	51	1.30 (0.80–2.10)	30	26	1.80 (1.02–3.20)	6	25	0.52 (0.20–1.40)
	Average standard deviation (23.5)			1.03 (0.95–1.10)			1.08 (0.98–1.20)			0.91 (0.80–1.10)
Average	≤0.1	367	717	1.00	294	508	1.00	73	209	1.00
BDCM	0.2–2.1	435	716	1.04 (0.86–1.30)	336	442	1.09 (0.88–1.40)	99	274	0.86 (0.60–1.30)
	2.2–6.7	259	410	1.09 (0.87–1.40)	194	270	1.08 (0.80–1.40)	65	140	1.06 (0.70–1.70)
	6.8–9.6	36	76	0.81 (0.50–1.30)	26	45	0.91 (0.50–1.60)	10	31	0.61 (0.30–1.50)
	9.7–12.0	60	76	1.32 (0.90–2.00)	52	46	1.58 (1.01–2.50)	8	30	0.84 (0.40–2.00)
	>12.0	30	51	1.04 (0.60–1.70)	26	29	1.25 (0.70–2.30)	4	22	0.52 (0.20–1.70)
	Average standard deviation (3.9)			1.01 (0.93–1.10)			1.06 (0.96–1.20)			0.90 (0.80–1.06)
Average	≤0.6	346	717	1.00	272	514	1.00	74	203	1.00
DCAA	0.7–2.6	488	717	1.33 (1.10–1.60)	386	449	1.49 (1.20–1.90)	102	268	0.91 (0.60–1.30)
	2.7–10.3	218	409	0.99 (0.80–1.20)	158	260	1.03 (0.80–1.40)	60	149	0.76 (0.50–1.20)
	10.4–16.1	52	76	1.24 (0.80–1.90)	42	46	1.51 (0.94–2.40)	10	30	0.72 (0.30–1.70)
	16.2–19.2	55	77	1.35 (0.90–2.00)	46	46	1.67 (1.10–2.70)	9	31	0.73 (0.30–1.70)
	>19.2	28	50	1.16 (0.70–1.90)	24	25	1.67 (0.91–3.10)	4	25	0.40 (0.10–1.20)
	Average standard deviation (5.5)			1.04 (0.97–1.10)			1.11 (1.01–1.20)			0.89 (0.80–1.04)
Average	≤0.1	362	717	1.00	285	510	1.00	77	207	1.00
TCAA	0.2–0.9	447	716	1.08 (0.90–1.30)	352	450	1.19 (0.95–1.50)	95	266	0.76 (0.5–1.1)
	1.0–6.9	241	409	1.07 (0.86–1.30)	177	255	1.09 (0.80–1.40)	64	154	0.98 (0.6–1.5)
	7.0–14.4	47	77	0.96 (0.60–1.50)	39	49	1.11 (0.70–1.80)	8	28	0.63 (0.3–1.6)
	14.5–27.7	54	76	1.27 (0.85–1.90)	47	44	1.62 (1.02–2.60)	7	32	0.59 (0.2–1.5)
	>27.7	36	51	1.30 (0.80–2.10)	28	32	1.47 (0.80–2.60)	8	19	0.85 (0.3–2.2)
	Average standard deviation (6.6)			1.03 (0.95–1.10)			1.07 (0.98–1.20)			0.92 (0.8–1.1)
Average	0.0	484	866	1.00	385	603	1.00	99	263	1.00
BCAA	0.1–0.9	316	567	0.87 (0.70–1.10)	240	360	0.86 (0.70–1.10)	76	207	0.85 (0.6–1.3)
	1.0–3.1	251	409	0.98 (0.80–1.20)	196	262	1.07 (0.80–1.04)	55	147	0.76 (0.5–1.2)
	3.2–3.9	54	77	1.10 (0.70–1.60)	45	43	1.43 (0.90–2.30)	9	34	0.51 (0.2–1.2)
	4.0–5.2	53	76	1.20 (0.80–1.80)	41	41	1.32 (0.80–2.10)	12	35	0.93 (0.4–2.0)
	>5.2	29	51	0.84 (0.50–1.40)	21	31	0.83 (0.50–1.50)	8	20	0.81 (0.3–2.0)
	Average standard deviation (1.5)			1.01 (0.94–1.10)			1.05 (0.96–1.10)			0.93 (0.8–1.1)

(Continued)



**Table 9.5 (Continued)**

Exposure	Average* (50-year window)	Total			Males			Females		
		Cases	Controls	OR (95% CI) <sup>†</sup>	Cases	Controls	OR (95% CI) <sup>†</sup>	Cases	Controls	OR (95% CI) <sup>†</sup>
Average	≤0.9	366	717	1.00	290	508	1.00	76	209	1.00
HAA6	1.0–5.0	456	716	1.11 (0.92–1.30)	362	461	1.18 (0.95–1.50)	94	255	0.85 (0.60–1.30)
	5.1–22.1	228	409	0.97 (0.80–1.20)	161	248	1.00 (0.80–1.30)	67	161	0.82 (0.50–1.30)
	22.2–34.1	47	77	1.00 (0.70–1.50)	40	47	1.25 (0.80–2.00)	7	30	0.52 (0.20–1.30)
	34.3–52.6	48	77	1.06 (0.70–1.60)	39	48	1.19 (0.70–1.90)	9	29	0.74 (0.30–1.80)
	>52.6	42	50	1.52 (0.96–2.40)	36	28	2.00 (1.20–3.40)	6	22	0.64 (0.20–1.80)
Average standard deviation (14.0)				1.03 (0.96–1.10)			1.08 (0.99–1.20)			0.89 (0.80–1.10)

\* Exposure categories based on 35th, 70th, 90th, 93.75th, and 97.5th percentiles in distribution among controls.

† Adjusted for age (four groups), sex (where appropriate), study group, high-risk occupation, cigarette smoking, and education.

**Table 9.6**  
**Analysis of colon cancer risk and average exposure to specific chlorination by-products**

Exposure	Average* (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI) <sup>†</sup>	Cases	Controls	OR (95% CI) <sup>†</sup>	Cases	Controls	OR (95% CI) <sup>†</sup>
Average	≤0.5	223	717	1.00	118	510	1.00	105	207	1.00
TTHM	0.6–8.4	184	716	0.79 (0.60–0.99)	91	442	0.90 (0.70–1.20)	93	274	0.68 (0.50–0.94)
	8.4–36.1	110	409	0.83 (0.60–1.10)	51	264	0.84 (0.60–1.20)	59	145	0.81 (0.60–1.20)
	36.2–58.0	16	77	0.64 (0.40–1.10)	9	47	0.83 (0.40–1.80)	7	30	0.46 (0.20–1.10)
	58.1–96.1	27	76	1.12 (0.70–1.80)	15	51	1.26 (0.70–2.30)	12	25	0.95 (0.50–2.00)
	>96.1	19	51	1.08 (0.60–1.90)	10	26	1.64 (0.80–3.50)	9	25	0.71 (0.30–1.60)
Average standard deviation (23.5)				1.02 (0.93–1.10)			1.08 (0.96–1.20)			0.95 (0.80–1.10)
Average	≤0.1	209	717	1.00	107	508	1.00	102	209	1.00
BDCM	0.2–2.1	183	716	0.85 (0.70–1.10)	94	442	1.02 (0.80–1.40)	89	274	0.67 (0.50–0.94)
	2.2–6.7	125	410	1.02 (0.80–1.30)	60	270	1.05 (0.70–1.50)	65	140	0.95 (0.70–1.40)
	6.8–9.6	20	76	0.86 (0.50–1.40)	11	45	1.16 (0.60–2.30)	9	31	0.60 (0.30–1.30)
	9.7–12.0	29	76	1.25 (0.80–2.00)	15	46	1.55 (0.80–2.90)	14	30	0.95 (0.50–1.90)
	>12.0	13	51	0.80 (0.40–1.50)	7	29	1.13 (0.50–2.60)	6	22	0.55 (0.20–1.40)
Average standard deviation (3.9)				0.98 (0.90–1.10)			1.04 (0.91–1.20)			0.93 (0.80–1.10)
Average	≤0.6	203	717	1.00	101	514	1.00	102	203	1.00
DCAA	0.7–2.6	218	717	1.04 (0.80–1.30)	113	449	1.28 (0.95–1.70)	105	268	0.79 (0.60–1.10)
	2.7–10.3	96	409	0.80 (0.60–1.10)	47	260	0.94 (0.60–1.40)	49	149	0.66 (0.40–0.98)
	10.4–16.1	24	76	1.08 (0.70–1.80)	14	46	1.56 (0.80–3.00)	10	30	0.67 (0.30–1.40)
	16.2–19.2	24	77	1.04 (0.60–1.70)	11	46	1.22 (0.60–2.40)	13	31	0.84 (0.40–1.70)
	>19.2	14	50	0.88 (0.50–1.60)	8	25	1.60 (0.70–3.70)	6	25	0.47 (0.20–1.20)
Average standard deviation (5.5)				0.99 (0.91–1.10)			1.09 (0.97–1.20)			0.90 (0.80–1.03)

(Continued)

**Table 9.6 (Continued)**

Exposure	Average* (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†
Average	≤0.1	208	717	1.00	108	510	1.00	100	207	1.00
TCAA	0.2–0.9	197	716	0.91 (0.70–1.10)	100	450	1.06 (0.80–1.40)	97	266	0.76 (0.50–1.10)
	1.0–6.9	113	409	0.91 (0.70–1.20)	51	255	0.96 (0.70–1.40)	62	154	0.83 (0.60–1.20)
	7.0–14.4	13	77	0.58 (0.30–1.10)	8	49	0.79 (0.40–1.70)	5	28	0.38 (0.10–1.01)
	14.5–27.7	28	76	1.19 (0.80–1.90)	16	44	1.71 (0.93–3.10)	12	32	0.76 (0.40–1.60)
	>27.7	20	51	1.30 (0.80–2.20)	11	32	1.59 (0.80–3.30)	9	19	0.98 (0.40–2.20)
Average standard deviation (6.6)				1.03 (0.94–1.10)			1.09 (0.93–1.20)			0.96 (0.80–1.10)
Average	0.0	264	866	1.00	141	603	1.00	123	263	1.00
BCAA	0.1–0.9	124	567	0.70 (0.60–0.89)	56	360	0.67 (0.50–0.94)	68	207	0.71 (0.50–1.01)
	1.0–3.1	137	409	1.07 (0.80–1.40)	67	262	1.10 (0.80–1.50)	70	147	1.02 (0.70–1.50)
	3.2–3.9	20	77	0.81 (0.50–1.40)	12	43	1.21 (0.60–2.40)	8	34	0.51 (0.20–1.10)
	4.0–5.2	23	76	0.91 (0.60–1.50)	13	41	1.35 (0.70–2.60)	10	35	0.61 (0.30–1.30)
	>5.2	11	51	0.66 (0.30–1.30)	5	31	0.67 (0.30–1.80)	6	20	0.63 (0.30–1.60)
Average standard deviation (1.5)				0.99 (0.90–1.1)			1.06 (0.96–1.20)			0.92 (0.80–1.10)
Average	≤0.9	215	717	1.00	111	508	1.00	104	209	1.00
HAA6	1.0–5.0	185	716	0.84 (0.70–1.10)	95	461	0.95 (0.70–1.30)	90	255	0.71 (0.50–1.00)
	5.1–22.1	118	409	0.92 (0.70–1.20)	54	248	1.01 (0.70–1.50)	64	161	0.80 (0.60–1.20)
	22.2–34.1	15	77	0.63 (0.40–1.10)	8	47	0.79 (0.40–1.70)	7	30	0.47 (0.20–1.10)
	34.3–52.6	23	77	0.95 (0.60–1.60)	14	48	1.32 (0.70–2.50)	9	29	0.61 (0.30–1.30)
	>52.6	23	50	1.42 (0.80–2.40)	12	28	1.92 (0.95–3.90)	11	22	1.00 (0.50–2.20)
Average standard deviation (14.0)				1.01 (0.92–1.10)			1.09 (0.97–1.20)			0.93 (0.80–1.10)

\* Exposure categories based on 35th, 70th, 90th, 93.75th, and 97.5th percentiles in distribution among controls.

† Adjusted for age (four groups) and sex (where appropriate).

**Table 9.7**  
**Analysis of rectal cancer risk and average exposure to specific chlorination by-products**

Exposure	Average* (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†	Cases	Controls	OR (95% CI)†
Average	≤0.5	190	717	1.00	122	510	1.00	68	207	1.00
TTHM	0.6–8.4	179	716	0.97 (0.80–1.30)	103	442	1.02 (0.70–1.40)	76	274	0.88 (0.60–1.30)
	8.4–36.1	114	409	1.10 (0.80–1.50)	65	264	1.14 (0.80–1.70)	49	145	1.06 (0.60–1.70)
	36.2–58.0	25	77	1.25 (0.80–2.10)	8	47	0.73 (0.30–1.60)	17	30	1.86 (0.92–3.70)
	58.1–96.1	21	76	1.13 (0.70–1.90)	16	51	1.45 (0.80–2.80)	5	25	0.67 (0.20–1.90)
	>96.1	24	51	1.98 (1.20–3.40)	13	26	2.66 (1.30–5.50)	11	25	1.49 (0.70–3.40)
Average standard deviation (23.5)				1.11 (1.01–1.20)			1.13 (1.00–1.30)			1.09 (0.95–1.30)

(Continued)

**Table 9.7 (Continued)**

Exposure	Average* (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI) <sup>†</sup>	Cases	Controls	OR (95% CI) <sup>†</sup>	Cases	Controls	OR (95% CI) <sup>†</sup>
Average BDCM	≤0.1	188	717	1.00	121	508	1.00	67	209	1.00
	0.2–2.1	183	716	1.01 (0.80–1.30)	105	442	1.05 (0.80–1.50)	78	274	0.95 (0.60–1.50)
	2.2–6.7	120	410	1.20 (0.89–1.60)	68	270	1.17 (0.80–1.70)	52	140	1.24 (0.80–2.00)
	6.8–9.6	20	76	1.04 (0.60–1.80)	9	45	0.87 (0.40–1.90)	11	31	1.23 (0.60–2.70)
	9.7–12.0	28	76	1.56 (0.94–2.60)	13	46	1.40 (0.70–2.80)	15	30	1.75 (0.80–3.70)
	>12.0	14	51	1.14 (0.60–2.10)	11	29	1.76 (0.80–3.70)	3	22	0.49 (0.10–1.70)
Average standard deviation (3.9)				1.05 (0.96–1.20)			1.08 (0.95–1.20)			1.02 (0.88–1.20)
Average DCAA	≤0.6	182	717	1.00	117	514	1.00	65	203	1.00
	0.7–2.6	209	717	1.18 (0.92–1.50)	124	449	1.28 (0.94–1.80)	85	268	1.02 (0.70–1.60)
	2.7–10.3	101	409	1.00 (0.70–1.40)	52	260	0.93 (0.60–1.40)	49	149	1.08 (0.70–1.80)
	10.4–16.1	20	76	1.06 (0.60–1.80)	10	46	1.03 (0.50–2.20)	10	30	1.06 (0.50–2.40)
	16.2–19.2	20	77	1.09 (0.60–1.90)	12	46	1.32 (0.60–2.70)	8	31	0.82 (0.30–2.00)
	>19.2	21	50	1.81 (1.03–3.20)	12	25	2.42 (1.10–5.10)	9	25	1.31 (0.60–3.10)
Average standard deviation (5.5)				1.10 (1.00–1.20)			1.15 (1.02–1.30)			1.04 (0.89–1.20)
Average TCAA	≤0.1	193	717	1.00	125	510	1.00	68	207	1.00
	0.2–0.9	184	716	0.98 (0.80–1.30)	105	450	1.02 (0.70–1.40)	79	266	0.92 (0.60–1.40)
	1.0–6.9	102	409	0.94 (0.70–1.30)	57	255	0.97 (0.70–1.40)	45	154	0.91 (0.60–1.50)
	7.0–14.4	27	77	1.29 (0.80–2.10)	11	49	0.92 (0.50–1.90)	16	28	1.81 (0.89–3.70)
	14.5–27.7	26	76	1.34 (0.80–2.20)	18	44	1.86 (0.99–3.50)	8	32	0.85 (0.40–2.00)
	>27.7	21	51	1.63 (0.93–2.90)	11	32	1.63 (0.80–3.50)	10	19	1.66 (0.70–3.90)
Average standard deviation (6.6)				1.11 (1.02–1.20)			1.12 (1.00–1.30)			1.10 (0.97–1.30)
Average BCAA	0.0	234	866	1.00	150	603	1.00	84	263	1.00
	0.1–0.9	134	567	0.87 (0.70–1.10)	75	360	0.86 (0.60–1.20)	59	207	0.89 (0.60–1.30)
	1.0–3.1	140	409	1.32 (1.00–1.70)	79	262	1.33 (0.92–1.90)	61	147	1.35 (0.87–2.10)
	3.2–3.9	23	77	1.14 (0.70–1.90)	12	43	1.22 (0.60–2.50)	11	34	1.04 (0.50–2.30)
	4.0–5.2	16	76	0.80 (0.50–1.40)	6	41	0.63 (0.30–1.60)	10	35	0.96 (0.40–2.10)
	>5.2	6	51	0.49 (0.20–1.20)	5	31	0.77 (0.30–2.00)	1	20	0.17 (0.02–1.30)
Average standard deviation (1.5)				1.00 (0.90–1.10)			1.02 (0.89–1.20)			0.97 (0.80–1.10)
Average HAA6	≤0.9	192	717	1.00	123	508	1.00	69	209	1.00
	1.0–5.0	183	716	0.98 (0.80–1.30)	111	461	1.04 (0.80–1.40)	72	255	0.88 (0.60–1.40)
	5.1–22.1	107	409	0.99 (0.70–1.40)	55	248	0.97 (0.70–1.50)	52	161	1.00 (0.60–1.70)
	22.2–34.1	27	77	1.30 (0.80–2.10)	10	47	0.88 (0.40–1.90)	17	30	1.80 (0.89–3.60)
	34.3–52.6	18	77	0.93 (0.50–1.70)	13	48	1.21 (0.60–2.40)	5	29	0.57 (0.20–1.60)
	>52.6	26	50	2.12 (1.30–3.60)	15	28	2.67 (1.30–5.40)	11	22	1.66 (0.70–3.80)
Average standard deviation (14.0)				1.10 (1.01–1.20)			1.14 (1.00–1.30)			1.07 (0.93–1.20)

\* Exposure categories based on 35th, 70th, 90th, 93.75th, and 97.5th percentiles in distribution among controls.

† Adjusted for age (four groups), sex (where appropriate), and average population size.

**Table 9.8**  
**Analysis of bladder cancer risk and length of exposure to moderate levels of specific chlorination by-products among subjects with 30 years of known exposure**

Exposure	Years (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥40 µg/L	<10	978	1,720	1.00	766	1,150	1.00	212	570	1.00
	10–29	100	153	1.08 (0.80–1.40)	72	90	1.08 (0.80–1.50)	28	63	1.04 (0.60–1.80)
	30+	109	173	1.06 (0.80–1.40)	90	100	1.23 (0.90–1.70)	19	73	0.73 (0.40–1.30)
P (trend)				0.50			0.10			0.30
BDCM ≥8 µg/L	<10	1,017	1,771	1.00	792	1,172	1.00	225	599	1.00
	10–29	84	131	1.02 (0.80–1.40)	63	87	1.03 (0.7–1.5)	21	44	1.04 (0.60–1.90)
	30+	86	144	0.98 (0.70–1.30)	73	81	1.17 (0.8–1.7)	13	63	0.59 (0.30–1.10)
P (trend)				0.70			0.50			0.10
DCAA ≥10 µg/L	<10	949	1,683	1.00	744	1,130	1.00	205	553	1.00
	10–29	96	151	1.03 (0.80–1.40)	70	94	0.99 (0.7–1.4)	26	57	1.04 (0.60–1.80)
	30+	142	212	1.17 (0.91–1.50)	114	116	1.40 (1.1–1.9)	28	96	0.77 (0.50–1.30)
P (trend)				0.30			0.03			0.20
TCAA ≥10 µg/L	<10	1,050	1,827	1.00	816	1,205	1.00	234	622	1.00
	10–29	63	103	1.00 (0.70–1.40)	50	70	0.97 (0.70–1.40)	13	33	1.08 (0.50–2.20)
	30+	74	116	1.08 (0.80–1.50)	62	65	1.33 (0.91–1.90)	12	51	0.61 (0.30–1.20)
P (trend)				0.70			0.20			0.20
BCAA ≥4 µg/L	<10	996	1,746	1.00	778	1,169	1.00	218	577	1.00
	10–29	109	166	1.11 (0.85–1.50)	90	93	1.40 (1.01–1.90)	19	73	0.64 (0.40–1.10)
	30+	82	134	1.02 (0.80–1.40)	60	78	1.02 (0.70–1.50)	22	56	1.01 (0.60–1.80)
P (trend)				0.90			0.60			0.50
HAA6 ≥30 µg/L	<10	1,024	1,778	1.00	795	1,175	1.00	229	603	1.00
	10–29	80	140	0.88 (0.70–1.20)	65	91	0.91 (0.60–1.30)	15	49	0.73 (0.40–1.40)
	30+	83	128	1.09 (0.80–1.50)	68	74	1.26 (0.88–1.80)	15	54	0.75 (0.40–1.40)
P (trend)				0.80			0.20			0.20

\* Adjusted for age (four groups), sex (where appropriate), study group, high-risk occupation, cigarette smoking, and education.

In general, time spent on water containing high DBP levels was more strongly associated with cancer risk than time spent on water containing moderate DBP levels, but the fewer number of subjects exposed at the higher levels resulted in wider CIs. Exposure to 30 or more years to TTHM levels >80 µg/L or >100 µg/L was associated with a 43% increase in risk for bladder cancer in males (95% CI = 0.9–2.4, [Table 9.9](#)) compared to a 23% increase for 30 or more years spent on TTHM levels >40 µg/L (95% CI = 0.9–1.7, [Table 9.8](#)). For male colon cancer, the OR for 30 or more years ≥80 µg/L was 1.69 (95% CI = 0.9–3.2, [Table 9.11](#)), 2.00 (95% CI = 0.9–4.6,

**Table 9.9**  
**Analysis of bladder cancer risk and length of exposure to high levels of specific chlorination**  
**by-products among subjects with 35 years of known exposure**

Exposure	Years (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥80 µg/L	<10	1,072	1,884	1.00	834	1,238	1.00	238	646	1.00
	10–29	73	99	1.19 (0.85–1.70)	59	67	1.17 (0.80–1.70)	14	32	1.28 (0.60–2.70)
	30+	42	63	1.14 (0.70–1.70)	35	35	1.43 (0.87–2.40)	7	28	0.62 (0.30–1.50)
	P (trend)			0.30			0.10			0.50
TTHM ≥100 µg/L	<10	1,105	1,920	1.00	862	1,262	1.00	243	658	1.00
	10–29	59	89	1.08 (0.80–1.60)	48	60	1.13 (0.70–1.70)	11	29	0.92 (0.40–2.10)
	30+	23	37	1.14 (0.70–2.00)	18	18	1.43 (0.70–2.80)	5	19	0.70 (0.30–2.00)
	P (trend)			0.60			0.20			0.30
BDCM ≥10 µg/L	<10	1,049	1,816	1.00	816	1,198	1.00	233	618	1.00
	10–29	86	147	0.92 (0.70–1.20)	68	95	0.97 (0.70–1.40)	18	52	0.83 (0.50–1.50)
	30+	52	83	1.03 (0.70–1.50)	44	47	1.20 (0.80–1.90)	8	36	0.66 (0.30–1.50)
	P (trend)			0.90			0.40			0.20
DCAA ≥20 µg/L	<10	1,091	1,906	1.00	850	1,252	1.00	241	654	1.00
	10–29	68	90	1.21 (0.86–1.70)	55	63	1.21 (0.80–1.80)	13	27	1.26 (0.60–2.70)
	30+	28	50	0.99 (0.60–1.60)	23	25	1.28 (0.70–2.30)	5	25	0.55 (0.20–1.50)
	P (trend)			0.50			0.20			0.40
TCAA ≥20 µg/L	<10	1,077	1,869	1.00	837	1,231	1.00	240	638	1.00
	10–29	62	111	0.86 (0.60–1.20)	51	72	0.92 (0.60–1.40)	11	39	0.73 (0.30–1.60)
	30+	48	66	1.23 (0.80–1.90)	40	37	1.48 (0.91–2.40)	8	29	0.74 (0.30–1.70)
	P (trend)			0.70			0.20			0.30
BCAA ≥5 µg/L	<10	1,074	1,857	1.00	842	1,227	1.00	232	630	1.00
	10–29	46	72	1.08 (0.80–1.60)	34	47	0.98 (0.60–1.60)	12	25	1.60 (0.70–3.50)
	30+	67	117	0.93 (0.70–1.30)	52	66	0.99 (0.70–1.50)	15	51	0.77 (0.40–1.50)
	P (trend)			0.40			0.60			0.50
HAA6 ≥60 µg/L	<10	1,105	1,919	1.00	862	1,262	1.00	243	657	1.00
	10–29	59	90	1.07 (0.70–1.50)	48	60	1.13 (0.80–1.70)	11	30	0.90 (0.40–2.00)
	30+	23	37	1.14 (0.70–2.00)	18	18	1.43 (0.70–2.80)	5	19	0.70 (0.30–2.00)
	P (trend)			0.60			0.20			0.30

\* Adjusted for age (four groups), sex (where appropriate), study group, high-risk occupation, cigarette smoking, and education.

**Table 9.10**  
**Analysis of colon cancer risk and length of exposure to moderate levels of specific chlorination by-products among subjects with 30 years of known exposure**

Exposure	Years (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥40 µg/L	<10	486	1,720	1.00	247	1,150	1.00	239	570	1.00
	10–29	41	153	0.91 (0.60–1.30)	19	90	0.99 (0.60–1.70)	22	63	0.83 (0.50–1.40)
	30+	52	173	1.01 (0.70–1.40)	28	100	1.29 (0.80–2.00)	24	73	0.78 (0.50–1.30)
P (trend)				0.90			0.30			0.30
BDCM ≥8 µg/L	<10	502	1,771	1.00	253	1,172	1.00	249	599	1.00
	10–29	39	131	1.08 (0.70–1.60)	23	87	1.24 (0.80–2.00)	16	44	0.88 (0.50–1.60)
	30+	38	144	0.86 (0.60–1.30)	18	81	1.00 (0.60–1.70)	20	63	0.75 (0.40–1.30)
P (trend)				0.40			0.90			0.20
DCAA ≥10 µg/L	<10	471	1,683	1.00	240	1,130	1.00	231	553	1.00
	10–29	46	151	1.08 (0.80–1.50)	21	94	1.09 (0.70–1.80)	25	57	1.06 (0.70–1.70)
	30+	62	212	0.98 (0.70–1.30)	33	116	1.32 (0.88–2.00)	29	96	0.72 (0.50–1.10)
P (trend)				0.80			0.10			0.20
TCAA ≥10 µg/L	<10	517	1,827	1.00	258	1,205	1.00	259	622	1.00
	10–29	25	103	0.89 (0.60–1.40)	15	70	1.02 (0.60–1.80)	10	33	0.74 (0.40–1.50)
	30+	37	116	1.06 (0.70–1.60)	21	65	1.46 (0.88–2.40)	16	51	0.74 (0.40–1.30)
P (trend)				0.60			0.10			0.40
BCAA ≥4 µg/L	<10	493	1,746	1.0	251	1,169	1.00	242	577	1.00
	10–29	46	166	0.95 (0.70–1.30)	24	93	1.26 (0.80–2.00)	22	73	0.73 (0.40–1.20)
	30+	40	134	0.99 (0.70–1.40)	19	78	1.11 (0.70–1.90)	21	56	0.89 (0.50–1.50)
P (trend)				0.70			0.40			0.20
HAA6 ≥30 µg/L	<10	504	1,778	1.00	255	1,175	1.00	249	603	1.00
	10–29	31	140	0.77 (0.50–1.20)	13	91	0.67 (0.40–1.20)	18	49	0.89 (0.50–1.60)
	30+	44	128	1.16 (0.80–1.70)	26	74	1.59 (0.99–2.50)	18	54	0.79 (0.50–1.40)
P (trend)				0.60			0.10			0.50

\* Adjusted for age (four groups) and sex (where appropriate).

**Table 9.11**  
**Analysis of colon cancer risk and length of exposure to high levels of specific chlorination by-products among subjects with 35 years of known exposure**

Exposure	Years (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥80 µg/L	<10	524	1,884	1.00	264	1,238	1.00	260	646	1.00
	10–29	30	99	1.12 (0.70–1.70)	17	67	1.21 (0.70–2.10)	13	32	1.01 (0.50–2.0)
	30+	25	63	1.34 (0.80–2.20)	13	35	1.69 (0.88–3.20)	12	28	1.05 (0.50–2.10)
P (trend)				0.20			0.03			0.90
TTHM ≥100 µg/L	<10	537	1,920	1.00	271	1,262	1.00	266	658	1.0
	10–29	25	89	1.03 (0.70–1.60)	15	60	1.15 (0.70–2.10)	10	29	0.86 (0.40–1.80)
	30+	17	37	1.48 (0.80–2.70)	8	18	2.00 (0.86–4.70)	9	19	1.16 (0.50–2.60)
P (trend)				0.40			0.20			0.90
BDCM ≥10 µg/L	<10	512	1,816	1.00	257	1,198	1.00	255	618	1.00
	10–29	37	147	0.91 (0.60–1.30)	22	95	1.09 (0.70–1.80)	15	52	0.70 (0.40–1.30)
	30+	30	83	1.19 (0.80–1.80)	15	47	1.44 (0.80–2.60)	15	36	0.99 (0.50–1.80)
P (trend)				0.80			0.30			0.50
DCAA ≥20 µg/L	<10	530	1,906	1.00	267	1,252	1.00	263	654	1.00
	10–29	29	90	1.21 (0.80–1.90)	17	63	1.28 (0.70–2.20)	12	27	1.11 (0.60–2.20)
	30+	20	50	1.31 (0.80–2.20)	10	25	1.81 (0.80–3.70)	10	25	0.98 (0.50–2.10)
P (trend)				0.40			0.10			0.90
TCAA ≥20 µg/L	<10	526	1,869	1.00	264	1,231	1.00	262	638	1.00
	10–29	26	111	0.85 (0.50–1.30)	16	72	1.03 (0.60–1.80)	10	39	0.63 (0.30–1.30)
	30+	27	66	1.36 (0.86–2.20)	14	37	1.71 (0.91–3.20)	13	29	1.08 (0.60–2.10)
P (trend)				0.50			0.20			0.70
BCAA ≥5 µg/L	<10	528	1,857	1.00	269	1,227	1.00	259	630	1.00
	10–29	17	72	0.85 (0.50–1.50)	8	47	0.83 (0.40–1.80)	9	25	0.89 (0.40–1.90)
	30+	34	117	0.95 (0.60–1.40)	17	66	1.14 (0.70–2.00)	17	51	0.80 (0.40–1.40)
P (trend)				0.4			0.9			0.20
HAA6 ≥60 µg/L	<10	537	1,919	1.00	271	1,262	1.00	266	657	1.00
	10–29	25	90	1.01 (0.60–1.60)	15	60	1.15 (0.70–2.10)	10	30	0.83 (0.40–1.70)
	30+	17	37	1.48 (0.80–2.70)	8	18	2.00 (0.86–4.70)	9	19	1.16 (0.50–2.60)
P (trend)				0.50			0.20			0.90

\* Adjusted for age (four groups), sex (where appropriate), and average population size.

**Table 9.12**  
**Analysis of rectal cancer risk and length of exposure to moderate levels of specific chlorination by-products among subjects with 30 years of known exposure**

Exposure	Years (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥40 µg/L	<10	446	1,720	1.00	273	1,150	1.00	173	570	1.00
	10–29	51	153	1.29 (0.92–1.80)	24	90	1.17 (0.70–1.90)	27	63	1.44 (0.88–2.40)
	30+	56	173	1.30 (0.94–1.80)	30	100	1.36 (0.87–2.10)	26	73	1.27 (0.80–2.10)
P (trend)				0.04			0.09			0.20
BDCM ≥8 µg/L	<10	460	1,771	1.00	281	1,172	1.00	179	599	1.00
	10–29	48	131	1.39 (0.98–2.00)	23	87	1.09 (0.70–1.80)	25	44	1.95 (1.20–3.30)
	30+	45	144	1.26 (0.88–1.80)	23	81	1.25 (0.80–2.10)	22	63	1.27 (0.80–2.20)
P (trend)				0.20			0.50			0.20
DCAA ≥10 µg/L	<10	436	1,683	1.00	265	1,130	1.00	171	553	1.00
	10–29	48	151	1.22 (0.86–1.70)	24	94	1.10 (0.70–1.80)	24	57	1.42 (0.80–2.40)
	30+	69	212	1.31 (0.96–1.80)	38	116	1.56 (1.03–2.40)	31	96	1.09 (0.70–1.70)
P (trend)				0.04			0.03			0.50
TCAA ≥10 µg/L	<10	476	1,827	1.00	286	1,205	1.00	190	622	1.00
	10–29	33	103	1.23 (0.80–1.90)	14	70	0.83 (0.5–1.5)	19	33	1.96 (1.10–3.50)
	30+	44	116	1.54 (1.10–2.20)	27	65	1.96 (1.2–3.2)	17	51	1.19 (0.70–2.10)
P (trend)				0.02			0.04			0.20
BCAA ≥4 µg/L	<10	467	1,746	1.00	282	1,169	1.00	185	577	1.00
	10–29	56	166	1.24 (0.89–1.70)	31	93	1.42 (0.91–2.20)	25	73	1.08 (0.70–1.80)
	30+	30	134	0.86 (0.60–1.30)	14	78	0.78 (0.40–1.40)	16	56	0.92 (0.50–1.70)
P (trend)				0.40			0.50			0.60
HAA6 ≥30 µg/L	<10	466	1,778	1.00	280	1,175	1.00	186	603	1.00
	10–29	40	140	1.10 (0.80–1.60)	18	91	0.84 (0.50–1.40)	22	49	1.49 (0.87–2.00)
	30+	47	128	1.48 (1.03–2.10)	29	74	1.81 (1.10–2.90)	18	54	1.18 (0.70–2.10)
P (trend)				0.01			0.02			0.20

\* Adjusted for age (four groups) and sex (where appropriate).



**Table 9.13**  
**Analysis of rectal cancer risk and length of exposure to high levels of specific chlorination by-products among subjects with 35 years of known exposure**

Exposure	Years (50-year exposure window)	Total			Males			Females		
		Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*	Cases	Controls	OR (95% CI)*
TTHM ≥80 µg/L	<10	491	1,884	1.00	294	1,238	1.00	197	646	1.00
	10–29	33	99	1.30 (0.86–2.00)	16	67	1.01 (0.60–1.80)	17	32	1.88 (1.00–3.50)
	30+	29	63	1.92 (1.20–3.10)	17	35	2.38 (1.30–4.40)	12	28	1.54 (0.80–3.10)
P (trend)				0.01			0.06			0.07
TTHM ≥100 µg/L	<10	506	1,920	1.00	302	1,262	1.00	204	658	1.00
	10–29	26	89	1.12 (0.70–1.80)	13	60	0.95 (0.50–1.80)	13	29	1.51 (0.80–3.00)
	30+	21	37	2.33 (1.30–4.10)	12	18	3.23 (1.50–6.90)	9	19	1.67 (0.70–3.80)
P (trend)				0.03			0.08			0.20
BDCM ≥10 µg/L	<10	478	1,816	1.00	287	1,198	1.00	191	618	1.00
	10–29	40	147	1.02 (0.70–1.50)	19	95	0.83 (0.50–1.40)	21	52	1.33 (0.80–2.30)
	30+	35	83	1.73 (1.10–2.60)	21	47	2.07 (1.20–3.60)	14	36	1.40 (0.70–2.70)
0.2P (trend)				0.04			0.1			0.20
DCAA ≥20 µg/L	<10	501	1,906	1.00	299	1,252	1.00	202	654	1.00
	10–29	26	90	1.10 (0.70–1.70)	13	63	0.87 (0.50–1.60)	13	27	1.62 (0.80–3.20)
	30+	26	50	2.16 (1.30–3.50)	15	25	2.95 (1.50–5.80)	11	25	1.57 (0.70–3.30)
P (trend)				0.02			0.07			0.10
TCAA ≥20 µg/L	<10	488	1,869	1.00	294	1,231	1.00	194	638	1.00
	10–29	34	111	1.18 (0.80–1.80)	13	72	0.78 (0.40–1.40)	21	39	1.88 (1.10–3.30)
	30+	31	66	1.91 (1.20–3.00)	20	37	2.52 (1.40–4.50)	11	29	1.35 (0.70–2.80)
P (trend)				0.02			0.04			0.20
BCAA ≥5 µg/L	<10	512	1,857	1.00	311	1,227	1.00	201	630	1.00
	10–29	17	72	0.82 (0.50–1.40)	5	47	0.41 (0.20–1.05)	12	25	1.54 (0.80–3.10)
	30+	24	117	0.75 (0.50–1.20)	11	66	0.68 (0.40–1.30)	13	51	0.82 (0.40–1.60)
P (trend)				0.04			0.04			0.40
HAA6 ≥60 µg/L	<10	506	1,919	1.00	302	1,262	1.00	204	657	1.00
	10–29	26	90	1.11 (0.70–1.80)	13	60	0.95 (0.50–1.80)	13	30	1.45 (0.70–2.90)
	30+	21	37	2.33 (1.30–4.10)	12	18	3.23 (1.50–6.90)	9	19	1.67 (0.70–3.80)
P (trend)				0.03			0.08			0.20

\* Adjusted for age (four groups), sex (where appropriate), and average population size.

Table 9.11) for  $\geq 100$   $\mu\text{g/L}$ , and 1.29 (95% CI = 0.8–2.0, Table 9.10) for exposure to levels  $\geq 40$   $\mu\text{g/L}$ . For male rectal cancer, the OR for 30 or more years for  $\geq 80$   $\mu\text{g/L}$  was 2.38 (95% CI = 1.3–4.4, Table 9.13), 3.23 (95% CI = 1.5–6.9, Table 9.13) for  $\geq 100$   $\mu\text{g/L}$  and 1.36 (95% CI = 0.9–2.1, Table 9.12) for exposure to levels  $\geq 40$   $\mu\text{g/L}$ . Similar differences between exposure to high and moderate levels were observed for total HAAs among males for all three cancer sites.

For BDCM, exposure of 30 or more years to a concentration of 10  $\mu\text{g/L}$  or greater was associated with a risk increase for male rectal cancer (OR = 2.07, 95% CI = 1.2–3.6, Table 9.13); risks for male bladder and male colon cancer were slightly elevated but not statistically significant. ORs for extended exposure to the more moderate BDCM concentration of 8  $\mu\text{g/L}$  were smaller and nonsignificant for all three sites. No consistent elevations in risk were observed for extended BCAA exposure at either the moderate or high levels for any of the three cancer sites.

### Joint Effects of DBPs and Tap Water Consumption

Tables 9.14, 9.15, and 9.16 present the results from analyses examining the joint effect of average TTHM and HAA6 concentrations and usual daily tap water consumption for male bladder, colon, and rectal cancers. The referent group for all comparisons contains subjects with low tap water consumption ( $\leq 1.7$  L/day) and low average DBP levels ( $\leq 0.5$   $\mu\text{g/L}$  for TTHM;  $\leq 0.9$   $\mu\text{g/L}$  for HAA6). For male bladder cancer (Table 9.14), the comparison of subjects with high DBP and high tap water consumption to the referent group yielded an OR of 1.65 (95% CI = 0.99–2.8) for average THM  $> 36.1$   $\mu\text{g/L}$ , and 1.85 (95% CI = 1.1–3.1) for average HAA6  $> 22.1$   $\mu\text{g/L}$ . Reading down the columns defined by DBP exposure, risk estimates rise with increasing water consumption for all but one of the DBP exposure categories, which is suggestive of an independent effect of tap water consumption.

Results for male rectal cancer (Table 9.16) are inconsistent across exposure categories and are difficult to interpret. Among subjects with low water consumption, there is an apparent effect of increasing DBP exposure, with ORs exceeding 2.5 for exposure to high levels of DBPs compared to those exposed to low water and low DBPs. Males exposed to high levels of DBPs and tap water are not at increased risk of cancer. Reading down the columns defined by DBP exposure groups, risk estimates rise with increasing water consumption for low DBP exposure. This pattern of increasing risk is reversed for subjects exposed to higher DBP levels, with subjects drinking large volumes of water at no increased risk of rectal cancer.

For male colon cancer (Table 9.15), there were no consistent patterns of risk across either the tap water or DBP exposure categories.

## DISCUSSION

In the late 1980s the authors conducted a case control study in Iowa to analyze the association between DBPs and cancer risk. Lifetime residential histories were collected from study subjects and linked with historical source and treatment data available for Iowa water treatment utilities in order to develop individual indices of lifetime exposure to DBPs. Evaluated was risk with duration of time spent on chlorinated surface water supplies. In addition, average TTHM exposure was calculated, which was based on crude estimates of past THM levels in Iowa drinking water supplies. In an effort to reduce misclassification in the original TTHM exposure

**Table 9.14**  
**Joint effect of tap water with average THM and HAA6 on bladder cancer risk among males**

Usual tap water consumption (L/day)	Average THM (µg/L <sup>*†</sup> )			
	≤0.5 (95% CI)	0.6–8.4 (95% CI)	8.5–36.1 (95% CI)	>36.1 (95% CI)
≤1.7	1.0 [83, 172]	0.92 (0.6–1.4) [77, 143]	1.25 (0.8–2.0) [52, 79]	1.23 (0.7–2.3) [27, 37]
1.8–2.5	1.19 (0.8–1.7) [107, 183]	1.31 (0.89–1.9) [108, 139]	1.17 (0.7–1.9) [51, 80]	1.37 (0.8–2.4) [31, 38]
>2.5	1.28 (0.87–1.9) [97, 145]	1.59 (1.1–2.3) [146, 153]	1.05 (0.7–1.6) [68, 101]	1.65 (0.99–2.8) [47, 48]
	Average HAA6 (µg/L <sup>*†</sup> )			
	≤0.9 (95% CI)	1.0–5.0 (95% CI)	5.1–22.1 (95% CI)	>22.1 (95% CI)
≤1.7	1.0 [83, 173]	1.03 (0.7–1.5) [79, 141]	1.13 (0.7–1.8) [49, 80]	1.33 (0.7–2.4) [28, 37]
1.8–2.5	1.26 (0.87–1.8) [107, 178]	1.36 (0.93–2.0) [117, 152]	1.13 (0.7–1.8) [43, 70]	1.28 (0.7–2.3) [30, 40]
> 2.5	1.23 (0.8–1.8) [93, 147]	1.63 (1.1–2.4) [153, 159]	1.10 (0.7–1.7) [64, 96]	1.85 (1.1–3.1) [48, 45]

\* Exposure categories based on 35th, 70th and 90th percentiles in distribution among controls.

† All analyses adjusted for age, sex (where appropriate), study group, high-risk occupation, cigarette smoking (where appropriate), and education.

**Table 9.15**  
**Joint effect of tap water with average THM and HAA6 on colon cancer risk among males**

Usual tap water consumption (L/day)	Average THM (µg/L <sup>*†</sup> )			
	≤0.5 (95% CI)	0.6–8.4 (95% CI)	8.5–36.1 (95% CI)	>36.1 (95% CI)
≤1.7	1.0 [41, 172]	0.75 (0.4–1.3) [25, 143]	0.80 (0.4–1.5) [15, 79]	1.02 (0.5–2.3) [9, 37]
1.8–2.5	0.77 (0.5–1.3) [34, 183]	1.05 (0.6–1.7) [35, 139]	0.79 (0.4–1.5) [15, 80]	1.30 (0.6–2.7) [12, 38]
>2.5	0.81 (0.5–1.4) [28, 145]	0.77 (0.5–1.3) [28, 153]	0.65 (0.4–1.2) [16, 101]	0.87 (0.4–1.9) [10, 48]
	Average HAA6 (µg/L <sup>*†</sup> )			
	≤0.9 (95% CI)	1.0–5.0 (95% CI)	5.1–22.1 (95% CI)	>22.1 (95% CI)
≤1.7	1.0 [36, 173]	1.01 (0.6–1.7) [29, 141]	0.97 (0.5–1.9) [16, 80]	1.16 (0.5–2.6) [9, 37]
1.8–2.5	0.90 (0.5–1.5) [34, 178]	1.06 (0.6–1.8) [34, 152]	1.13 (0.6–2.2) [16, 70]	1.41 (0.7–3.0) [12, 40]
>2.5	0.85 (0.5–1.5) [26, 147]	0.84 (0.5–1.4) [28, 159]	0.90 (0.5–1.7) [18, 96]	1.07 (0.5–2.3) [10, 45]

\* Exposure categories based on 35th, 70th, and 90th percentiles in distribution among controls.

† All analyses adjusted for age and sex (where appropriate).

**Table 9.16**  
**Joint effect of tap water with average THM and HAA6 on rectal cancer risk among males**

Usual tap water consumption (L/day)	Average THM ( $\mu\text{g/L}^{*†}$ )			
	$\leq 0.5$ (95% CI)	0.6–8.4 (95% CI)	8.5–36.1 (95% CI)	$> 36.1$ (95% CI)
$\leq 1.7$	1.0 [29, 172]	1.29 (0.7–2.3) [30, 143]	1.83 (0.95–3.5) [22, 79]	2.68 (1.3–5.7) [15, 37]
1.8–2.5	1.29 (0.8–2.2) [41, 183]	1.35 (0.8–2.4) [31, 139]	1.42 (0.7–2.8) [17, 80]	1.73 (0.8–3.9) [10, 38]
$> 2.5$	1.79 (1.1–3.0) [46, 145]	1.37 (0.8–2.4) [34, 153]	1.52 (0.8–2.8) [24, 101]	1.08 (0.4–2.6) [8, 48]
	Average HAA6 ( $\mu\text{g/L}^{*†}$ )			
	$\leq 0.9$ (95% CI)	1.0–5.0 (95% CI)	5.1–22.1 (95% CI)	$> 22.1$ (95% CI)
$\leq 1.7$	1.0 [30, 173]	1.21 (0.7–2.2) [29, 141]	1.68 (0.88–3.2) [22, 80]	2.58 (1.2–5.5) [15, 37]
1.8–2.5	1.33 (0.8–2.2) [43, 178]	1.19 (0.7–2.1) [30, 152]	1.30 (0.6–2.7) [15, 70]	1.69 (0.8–3.8) [11, 40]
$> 2.5$	1.60 (0.95–2.7) [43, 147]	1.67 (0.98–2.9) [45, 159]	1.01 (0.5–2.0) [16, 96]	1.10 (0.5–2.6) [8, 45]

\* Exposure categories based on 35th, 70th, and 90th percentiles in distribution among controls.

† All analyses adjusted for age, sex (where appropriate), and average population size.

estimates, the current study was undertaken to improve estimates of past DBP levels in Iowa waters and to reevaluate their associations with cancer risk. Results from the reanalysis, using the current estimates of exposure, are presented in this chapter.

In the earlier analysis, increased risks for bladder and rectal cancer were reported, but no significant increases were observed for colon cancer. Risk estimates were generally stronger for time spent on chlorinated surface water than for average TTHM level. When the original analysis of average TTHM was reproduced using the newly estimated average TTHM and the original categories for classification of exposures, risk estimates were obtained for all three sites comparable to those presented originally. The new exposure data permitted the establishment of strata of more highly exposed persons. In addition, the new estimates of exposure afforded an opportunity to examine risks associated with HAAs as well as THMs. The strongest and most consistent increases in cancer risk were observed among males for all three sites for the top exposure categories for the nonbrominated THMs and HAAs. Compared to subjects exposed to TTHM  $\leq 0.5$   $\mu\text{g/L}$ , males exposed to TTHM  $> 96.1$   $\mu\text{g/L}$  were at an 80% increased risk of bladder cancer, a 64% increased risk of colon cancer, and a 166% increased risk of rectal cancer. Comparable or higher risks were observed for average HAA6 levels of  $> 52.6$   $\mu\text{g/L}$  compared to very low levels. Risk estimates were not elevated among females for cancers of the colon and bladder. Some increases were observed for rectal cancer among females, with inconsistencies. Increases in risk were observed when duration of exposure at high DBP levels were examined as well as average DBP exposure and seem to be the most prominent for subjects exposed at the very highest levels ( $\geq 100$   $\mu\text{g/L}$  TTHM or  $\geq 60$   $\mu\text{g/L}$  HAA6).

The current estimate of historic DBP levels allowed better examination of the very highest-exposed subjects in the study by increasing the ability to detect the known variability in

surface water DBP levels. In the earlier analysis, surface water treatment utilities were assigned one of two possible THM levels depending on the point of chlorination in the treatment chain. The reassessment of historic exposure for the current reanalysis involved the consideration of each utility receiving surface or mixed surface and groundwater on a case-by-case basis. Some utilities that used shallow groundwater were also considered individually. Public water systems with deeper groundwater sources were assigned contaminant levels using a central tendency approach, except for those with elevated levels of brominated DBPs. Multiple treatments and water quality parameters were considered in conjunction with actual DBP measurements to develop estimates of past levels. As seen in [Figure 9.1](#), the highest 10% of exposed controls had average TTHM levels ranging from 30 to 154 µg/L for the recalculated lifetime average variable to 33 to 74 µg/L for the original measurement of average TTHM. This greater variability in values allowed examination of subjects exposed at much higher levels than was previously possible, and it is in these upper strata where the most interesting results were observed.

The improvement of exposure assessment among subjects and the resulting reduction in exposure misclassification was one of the primary goals of the current undertaking. The estimate of TTHM and HAA levels on a utility-by-utility basis, using a wide range of information, is an important step forward in advancing our knowledge of historical exposures to DBPs. Using these methods created a better way to capture the variability in DBP levels in surface and mixed surface and groundwater sources. Although the correlation between old and new average TTHM levels is quite high when the control group is considered as a whole ( $r = 0.84$ ), the correlation between the two measures decreases at higher TTHM levels ([Figure 9.2](#)). Subjects formerly classified at relatively high TTHM levels (>40 µg/L) in the current study are less likely to have a comparable exposure than subjects exposed at lower TTHM levels. Correlations between the old and new TTHM exposures were similar for cases and for controls, providing evidence that misclassification of the original exposure measure was likely nondifferential. Nondifferential misclassification almost always results in a risk estimate that is biased toward the null, and evidence for this type of bias in the current analysis are the risks observed among the most highly exposed subjects. The previous TTHM estimates did not allow the estimation of risks among subgroups of subjects with similar high-TTHM exposures, and a direct comparison of these risks could not be made. When the previous exposure categories for lifetime average TTHM estimates are used to present a direct comparison of the cancer risks in the previous and current analyses ([Tables 9.2 to 9.4](#)), the effects of the misclassification of exposure are not apparent, as the cancer risks are generally similar among subjects with similar estimated exposures. This indicates little improvement in exposure definition when all subjects above about 40 µg/L TTHM as a group are considered. However, the increases in risk that were observed when higher ranges of exposure were considered suggests that the refined estimates of exposure at the high end of the exposure range did result in more precise classifications. There is no doubt that a substantial level of misclassification still remains, implying that risks at the high end of the exposure range are likely greater than those that we have calculated.

One of the issues in the reanalysis of the Iowa data was the choice of analysis subset. Since the earlier analyses of these data examined lifetime exposure, exposure over each subject's lifetime was likewise assessed for comparisons with earlier results. To facilitate comparisons with results from the Ontario reanalysis (chapter 8), exposures that occurred during the 50-year period up to 2 years before study entry were also considered. The 50-year window yielded a slightly higher number of eligible subjects than those entering the lifetime analyses, since more subjects had identifiable exposure for at least 70% (35 years in this case) of the exposure period under consideration. Fewer subjects met this criterion for the analyses of lifetime exposures due at least

partially to a greater level of missing residential histories and more limited information about water source and treatment in the distant past. This uncertainty in exposure occurring in the distant past may yield worse estimates of DBP levels than those based on more recent time periods. When risk estimates associated with lifetime and 50-year exposures were compared, we found slightly higher ORs associated with DBP exposures occurring during the 50-year time period. A more extensive time-window analysis will be completed in the future.

Another topic for future analyses is the examination of several by-products in a single multiple regression logistic model. In contrast to the original analysis, where only past levels of TTHM (the sum of the four THMs) were estimated, the current study involved the estimation of three HAAs (DCAA, TCAA, and BCAA) and two HAA groups (HAA5 and HAA6) in addition to two of the THMs ( $\text{CHCl}_3$  and BDCM) and TTHM. Analyses of each by-product or by-product group separately, presented in this chapter, yielded consistent increases in risk for the highest-exposure categories of the nonbrominated THMs and HAA exposure measurements for bladder, colon, and rectal cancers among males. Risk increases were moderately higher for HAA compounds than for THMs for cancers of the bladder and colon, but not for rectal cancer, making it difficult to comment on the relative importance of either chemical group. No consistent risk increases were noted for either of the brominated compounds that were evaluated: BDCM or BCAA. It will be of interest to examine the correlations between the individual DBPs and to explore the possibilities of examining multiple by-products in one statistical model.

Accurate assessment of exposure is essential in estimating cancer risk that may be linked with occupational and environmental carcinogens. The current results confirm the authors' past findings and suggest some specificity for effects linked with particular classes of by-products or their correlates in the mixture. In particular, when compared with the earlier findings, the higher estimates of risk here for the highest-exposure categories of TTHM and HAA for male bladder, colon, and rectal cancers are of interest. In addition, the risks seen for the 50-year time window are comparable, and in many instances higher, than risks based on lifetime exposures, suggesting the adequacy of the 50-year estimates.



## CHAPTER 10

### SUMMARY AND RECOMMENDATIONS

The objective of this study was to reanalyze two well-conducted, peer-reviewed epidemiology studies in Iowa and Ontario that have reported an increased risk of bladder cancer associated with chlorinated drinking water and THMs; in this current study, other cancer sites (i.e., colon and rectal cancers) were considered in the reanalysis. The motivation for such a reanalysis is application of an improved exposure assessment embodying more complete information on classes and species of DBPs formed during chlorination and chloramination. Given the long latency period for cancer, historical databases contain only limited DBP data, while more recent monitoring efforts (e.g., the U.S. ICR program) are more comprehensive. Through modeling and correlation analysis, it is possible to estimate past DBP levels based on present trends and knowledge about changes in source water(s) and treatment practices, both present and past. Such estimates can permit an improved DBP exposure assessment.

### DISCUSSION OF RESULTS FROM DBP MODELING STUDIES

A key aspect of the improved exposure assessment was the development of DBP prediction models. This effort began with the acquisition and assembly of national (U.S. and Canadian) DBP databases and was augmented by site-specific databases for the province of Ontario and the state of Iowa. These various DBP databases were analyzed and compared with the goal of using them as a basis for national and site-specific DBP prediction models.

#### Databases

##### *National DBP Databases for the United States and Canada*

In the United States, DBPs were first detected in drinking water systems in the early 1970s and have been regulated and monitored since. National U.S. databases have been developed since 1975 and include the NORS, NOMS, AwwaRF THM Survey, 35-Utility Study, AWWA WATER\STATS, AWWA WIDB, and ICR databases. Canadian regulations concerning DBPs were first enacted in 1993 as a guideline. National Canadian databases over the last decade include Health Canada's 53-Utility Survey and One-Year Survey of Halogenated DBPs in the Distribution System of Treatment Plants Using Three Different Disinfection Processes (Health Canada 1995, 1996).

A comparison of the databases reveals that, compared to U.S. drinking waters, Canadian drinking waters tend to differ in two ways. Canadian waters generally have lower levels of influent bromide, resulting in lower levels of brominated DBPs, and reflect natural organic matter properties that promote the formation of HAAs and species over TTHM and species. Both of the Canadian databases showed significantly less TTHM formation than the U.S. surveys. However, the Canadian databases also indicate similar amounts of HAAs compared to TTHM, a result not observed in the U.S. databases.

Some of the earlier surveys only reported THMs as TTHM, with later surveys reporting THM species (chloroform, BDCM, DBCM, and bromoform). Only more recent surveys have reported HAAs, including HAA5, HAA6, and HAA9. Some of the surveys contain DBP

precursor information (e.g., TOC or DOC and  $\text{Br}^-$ ), water quality information (e.g., pH and temperature), and disinfection information (e.g., chlorine dose).

### ***Site-Specific Databases for Ontario and Iowa***

The only Ontario database available was the DWSP database, a very robust database whose only deficiency is a lack of  $\text{Br}^-$  data. A comparison between Ontario and Canada DBP trends is difficult to make because the only national Canadian database is the Health Canada survey conducted in 1993. However, from these data, it is apparent that Ontario DBP formation is very similar to national Canadian trends. Some relevant ICR data were also assembled corresponding to U.S. utilities using Lake Ontario water, the major drinking water source in Ontario.

The Iowa databases available included the 1987 database in Cantor et al. (1998) used in the original Iowa epidemiology study, the CHEEC database, the Iowa DNR database, Iowa entries in WATER\STATS, Iowa entries in the ICR database, and the expanded ICR database including utilities proximate to Iowa and situated in similar watersheds. Iowa waters tend to form less DBPs than are found for the U.S. national levels. The TTHM and HAA5 for Iowa fall slightly below the median values for all of the national databases.

When comparing Iowa with Ontario, it is evident that the Ontario waters tended to form less TTHM than the Iowa waters. The Ontario waters also tended to form higher levels of HAAs and HAA species, presumably because the Ontario waters contain natural organic matter that promotes HAA formation over TTHM formation. The Ontario waters also contained less bromide (inferred from analysis of THM and HAA species data), resulting in lower formation of brominated DBPs and thus higher formation of chlorinated DBPs. This is evident in the observation that the overall Ontario databases exhibited lower BDCM, DBCM, and  $\text{CHBr}_3$  concentrations compared to the overall Iowa values. The trend of Ontario waters in forming HAAs preferentially over THMs may also be attributed to factors other than natural organic matter, including pH and temperature.

### **DBP Prediction Models**

The major objective of the modeling effort was to provide estimates of chlorination and chloramination DBPs (TTHM, individual THM species, HAA5, HAA6, HAA9, and individual HAA species) and DBP-related parameters (e.g., DBP-Br) over an approximately 50-year period of record. More specifically, the objective was to estimate missing values within the Iowa and Ontario databases. The approach was to chronologically progress backward along a time line from the present (robust database with few missing values) to the past (sparse database with many more missing values), recognizing historical milestones of significance (e.g., promulgation of the 1979 THM Rule and subsequent compliance over the promulgation period for Iowa and the 1993 THM guideline for Ontario).

Statistically based models were developed through multiple regression analysis using the various databases. In applying these models to making predictions in Ontario and Iowa, it became apparent that central tendency models work well for utilities with near-median levels of DBP precursors, water quality conditions, and disinfection conditions that lead to the formation of near-median DBP levels. However, they are poor in predicting more extreme (e.g., 10th or 90th percentile) conditions. A decision was made, midway through the project, to develop a second, complementary modeling approach, involving case study models based on “an expert system” in



the form of the project's DBP expert, Stuart Krasner. In the case of Ontario, the two modeling approaches were truly complementary and both approaches were used, whereas, in the case of Iowa, the case study approach was used almost exclusively because of data limitations in developing robust central tendency models.

Once the historical timeline was created, models were utilized to fill in any missing gaps of data (e.g., projecting past HAA occurrence). Missing data gaps occurred because the historical data tended to be extremely fragmented or did not exist at all, especially regarding HAAs, due to the lack of regulations or monitoring requirements present at that time. It is noteworthy that THM occurrence in chlorinated drinking water was only discovered in the early 1970s.

### ***Central Tendency Models***

Both national and site-specific multiple regression models were developed to predict individual DBPs from information on DBP precursors, water quality, and disinfection conditions, or to predict certain DBPs (e.g., HAA species) from other DBPs (e.g., THM species). In some cases, simple adjustment factors were applied to predictions where data were unavailable for model development (approximating  $\text{Br}^-$  from DBP-Br, the sum of the  $\text{Br}^-$  present in THM and HAA species). After its development, each model was validated in terms of an error analysis. As expected, relative error was greatest for data corresponding to more extreme conditions in DBP precursors, water quality, and disinfection conditions.

### ***Case Study Models***

The case study approach involved careful examination and analysis of data associated with each utility and ascertaining simple site-specific correlations and relationships, taking into account chronological changes in treatment and disinfection practice. All such DBP predictions have been well-documented along with requisite assumptions. A secondary modeling effort was taken for groups of plants that treated water from the same or similar watersheds and whose current treatment and disinfection scheme was the same as another plant's historical treatment and disinfection scheme, permitting predictions of historical DBP exposure for the latter plant.

## **DISCUSSION OF RESULTS FROM EPIDEMIOLOGIC STUDIES**

### **Introduction**

Exposure assessment issues have caused and will likely continue to cause uncertainties about observed epidemiologic associations, and methods such as those employed in this project should be used to better estimate exposures in future epidemiologic studies. Improved modeling of historical levels of DBPs using current measures of DBPs and other water quality parameters, historical water quality, and water system operational information can help reduce uncertainty when evaluating epidemiologic results. The long latency of most cancers requires that exposures be evaluated over a relatively lengthy time period when water quality measures of DBPs may not be available. Thus, improved modeling of historical DBP levels is especially important for assessing cancer risks.

To improve exposure assessment requires not only better measures of water contaminant levels but also a person's likely exposure to them. The intensity and duration of exposure are both

important measures for assessing epidemiologic associations. Currently, exposure assessment is one of environmental epidemiology's weakest links. Among 60 recently published case-control studies of occupational and environmental exposures, only 20 (32%) considered quantitative data about the intensity and duration of exposure.

The association of cancer risks with disinfected water could be considerably strengthened if exposures considered quantitative measures of at least the major DBPs or groups of by-products formed rather than simply considering chlorinated and unchlorinated water of TTHM. Exposures can be estimated from water quality records, current analyses of specific by-products (e.g., chloroform, dichloroacetate, dibromoacetonitrile, DCAA, or TCAA) and source water quality (e.g., bromide, TOC, pH, treatment modalities). To better address the relationship, individual estimates of specific DBPs are required.

In this project, the modeling of historical water quality was specifically tailored to areas (Iowa and Ontario) where two epidemiologic studies had previously been conducted to evaluate cancer risks that may be associated with chlorination and TTHM. In both studies, sufficient information was collected for residential histories and water consumption patterns of the cases and controls so that the intensity and duration of exposure could be considered using the results of more intensive modeling of the historical water quality for DBPs.

The value of a more rigorous assessment of exposure to DBPs is twofold. Most importantly, an analysis is now available in Iowa and Ontario to evaluate the bladder, colon, and rectal cancer risks that may be associated with several specific by-products. In addition, researchers can compare the newly reported exposure and risk estimates for TTHM in Iowa and Ontario with previously reported exposures and risks.

### **Differential and Nondifferential Exposure Misclassification**

The improvement of exposure assessment among subjects and the resulting reduction in exposure misclassification were among the primary goals of the project. Exposure misclassification can occur due to errors of study participants in recalling residential histories and water consumption over long periods of time and the investigator's inadequate characterization of historical levels of DBPs over the study period. Epidemiologic studies may under- or overestimate the cancer risks due to either differential or nondifferential exposure misclassification. Nondifferential or randomly distributed misclassification bias almost always prejudices study results in the direction of not observing an effect (or observing a smaller risk than may actually be present). Differential misclassification bias can result in either higher or lower estimates of risk, depending on how the misclassification is distributed. Epidemiologists often acknowledge the possibility of exposure misclassification bias when interpreting their results. Many environmental epidemiologists assume that the misclassification is nondifferential and conclude that the risks observed are smaller than the actual risk.

Few studies have evaluated the effect of exposure misclassification in water disinfection studies. Lynch et al. (1989) examined the effects of misclassification of exposure using empirical data from an interview-based case-control study of bladder cancer in Iowa. They found bladder cancer risk estimates were higher when more information was known about the study participants' residential histories and their possible exposures to chlorinated water sources. This suggests misclassification bias in epidemiologic studies of chlorinated water may be nondifferential, but in studies where residential mobility is different than Iowa, the direction of the bias may be different. As part of this project, the authors assessed the effects of a more rigorous modeling

of TTHM levels and resulting exposures in two previously reported case-control studies in Iowa and Ontario. Analyses compared previously and newly reported TTHM levels or exposures and previously and newly reported cancer risks using these exposure estimates.

### ***Misclassification in Previously Conducted Iowa Study***

The current prediction of historical DBP levels allowed better examination of the very highest-exposed subjects in Iowa by increasing the ability to detect the known variability in surface water DBP levels. The reassessment of historical exposure for the current reanalysis involved the case-by-case consideration of each water utility that received surface or mixed surface and groundwater. Some utilities that used shallow groundwater were also considered on a case-by-case basis. Public water systems with deeper groundwater sources were assigned DBP levels using a central tendency approach, except for those with elevated levels of brominated DBP.

Our analyses suggest that misclassification of the exposure measure used in the original Iowa study (Cantor et al. 1998) was probably nondifferential. Newly estimated TTHM exposures (lifetime average) were compared with exposures used in the original analysis. Although the correlation between the original and new estimates is quite good when the control group is considered as a whole, the correlation between the two estimates decreased at the higher TTHM levels. At estimated lifetime average TTHM levels of  $>40$   $\mu\text{g/L}$ , controls were less likely to have a comparable estimated exposure. Similar correlations between previous and current TTHM exposure estimates were observed among cases and controls.

We assessed the effects of misclassification bias in the original study by comparing risk estimates that used previous and current TTHM exposures. In the current analysis, the highest 10% of exposed controls had lifetime average TTHM levels ranging from 30 to 154  $\mu\text{g/L}$  compared to 33 to 74  $\mu\text{g/L}$  for the original estimate of average TTHM. This greater variability in values allowed examination of subjects exposed at much higher levels than was previously possible, and it is in these upper strata where the most interesting results were observed. Using the current modeling results, cancer risks were able to be evaluated among three subgroups of subjects with the very highest estimated TTHM exposures (36.2–58.0, 58.1–96.1, and  $>96.1$   $\mu\text{g/L}$ ). In the original analysis, fewer subjects were classified in the highest lifetime average TTHM exposure category ( $>46.3$   $\mu\text{g/L}$ ), and they were all considered in this single grouping. Thus, a direct comparison of risks could not be made for the newly estimated exposure categories. However, a direct comparison of the cancer risks in the previous and current analysis was available for the previously used exposure categories for lifetime average TTHM estimates. The effects of the exposure misclassification are not readily apparent, as the comparison indicates little improvement in exposure definition when all subjects above 40  $\mu\text{g/L}$  TTHM were considered as a group. However, the increases in risk observed when higher ranges of exposure were considered suggests that the refined estimates of exposure at the high end of the range did result in more precise classifications. It also appears that the risk estimates in Iowa are reduced at lower exposures, suggesting that persons with some exposure who were erroneously classified as low previously are now more correctly classified as having a higher exposure.

### ***Misclassification in Previously Conducted Ontario Study***

Our analysis suggests that misclassification of the exposure measure used in the original Ontario study also was probably nondifferential. Although the correlation between new and original

TTHM predictions at the water plant level was only moderate, a high correlation was observed among the large water treatment plants. This resulted in a high correlation with respect to assignment of TTHM exposure based on the subject's residence. The correlation between new and original exposure assignment is similar for cases and controls.

A direct comparison of the cancer risks in the previous and current analyses was also available. In this comparison, the effects of exposure misclassification are not readily apparent, as the cancer risks are generally similar when all subjects are considered. This may be due to the high correlation of original and current TTHM estimates for Great Lakes surface waters that served the vast majority of persons included in the study. The greatest differences between the original and current TTHM predictions were for chlorinated groundwater utilities. In these water utilities the current TTHM predictions were lower than the previous predictions, but, because of the small number of study participants served by the utilities, the effect on risks estimates was small.

### **Reanalysis Using Current Estimates of TTHM and Other DBPs**

One criticism of previously conducted epidemiologic studies is the lack of risk information for the various by-products that may be formed during disinfection. Previous studies have primarily considered exposures to chlorinated, unchlorinated, and chloraminated water, and some studies have considered current and recent historical levels of TTHM. The analyses in this project are the first to consider long-term predictions of historical water quality levels of TTHM, HAA5, HAA6, and specific species of THMs and HAAs for use in estimating exposures and assessing cancer risks.

#### ***Cancer Risk Analyses in Iowa***

In the original analysis, increased risks were observed for bladder and rectal cancer, but no significant increases were observed for colon cancer. Risk estimates were generally stronger for time spent on chlorinated surface water than for average TTHM level. When the original analysis was reproduced using the newly estimated average TTHM and the original categories for classification of exposures, risk estimates were obtained for all three cancer sites comparable to those presented originally. As noted earlier, the new exposure data permitted establishing strata of more highly exposed persons. The strongest and most consistent increases in cancer risk were observed among males for all three sites for the highest-exposure categories for the nonbrominated THMs and HAAs. Compared to subjects exposed to TTHM  $\leq 0.5$   $\mu\text{g/L}$ , males exposed to TTHM  $> 96.1$   $\mu\text{g/L}$  were at an 80% increased risk of bladder cancer, a 64% increased risk of colon cancer, and a 166% increased risk of rectal cancer. Comparable or higher risks were observed for average HAA6 levels of  $> 52.6$   $\mu\text{g/L}$  compared to very low HAA6 levels. For females, risk estimates were not elevated for cancers of the colon and bladder, and inconsistent increases in rectal cancer risks were observed.

#### ***Cancer Risk Analyses in Ontario***

The reestimation of chlorination by-product levels in Ontario and linkage to the Ontario case-control study also resulted in a more robust dataset. Since the assignment of TTHM exposure is similar to the original study (King and Marrett 1996), the risk estimates associated with TTHM exposure are similar for bladder, colon, and rectal cancers. A smaller number of subjects are

assigned high exposure in the reanalysis, resulting in wide CIs in some analyses. The most important contribution of the new exposure database is the opportunity to explore relationships with other by-products, including BDCM and HAAs. The new by-product estimates were used to evaluate risks of exposure over a 50-year exposure period. Relative risks for bladder cancer >1.4 (40% excess risk) are associated with high average exposure to TTHM, BDCM, HAA5, TCAA, and BCAA. The largest bladder cancer risks are found for the brominated by-product exposures (BDCM and BCAA). Excess risk of colon cancer was observed only among males. Relative risks for colon cancer approaching 2.0 (a doubling of risk) were observed for TTHM and HAA5 exposures. Risk estimates were lower and not statistically significant for high-BDCM exposure. Risk estimates for rectal cancer are consistently low and close to the null value. In general, the analyses based on new exposure estimates confirm previously reported results and provide additional insights with respect to a relationship between specific by-products and cancer risk. In particular, the relationship between bladder cancer and brominated by-products and the relationship between male colon cancer with TTHM and HAAs are noteworthy.

### **Analysis of Populations Using Only Surface Water**

An incomplete exposure assessment for specific DBPs is not the only source of uncertainty when evaluating results of previous epidemiologic studies. The observed associations may be confounded because exposure to chlorinated water and DBPs may also result in exposure to other water contaminants. Water sources may be contaminated by naturally occurring and synthetic water-soluble, nonvolatile chemicals, which also may be associated with increased cancer risks. Organic compounds are more frequently identified in surface waters than in groundwaters. Because microbial contaminants are often not found in groundwater sources, these sources are less frequently disinfected, and thus, in almost all of the previous studies, cancer risks in populations exposed to chlorinated surface water were compared with populations exposed to unchlorinated groundwater. In these studies, persons using groundwater may have been the only ones who were not exposed to DBPs or exposed to very low levels of DBPs. All public surface waters are disinfected. Although some surface waters have a low DBP-formation potential, many chlorinated surface waters will have moderate to high levels of TTHM.

In Ontario, a significant population is exposed to surface waters with relatively low levels of DBPs. This allowed a separate analysis to be conducted of the cancer risks for populations exposed only to surface water sources. The analyses in Ontario considered a subset of study participants served by a surface water source for a minimum of 30 years of the 50-year exposure period. In these analyses, bladder and colon cancer risks were considered for exposure to selected DBPs (TTHM, HAA5, BDCM, and BCAA) among surface water users. The relative risk estimates (ORs) are similar to results reported when groundwater users were included, but, in general, the risks are lower for surface water users. Insufficient numbers of subjects did not allow for similar analyses in Iowa, but the effects of DBPs for subjects with at least 15 years on surface water for the 50-year window were assessed. The Iowa results were similar to those found in Ontario. The risk estimates were elevated but slightly lower than those reported when groundwater-only users were included. This suggests that other surface water contaminants may confound associations observed. However, it should be remembered that the effect of including a groundwater population in a study is likely to vary depending upon the levels and types of contaminants in both the surface water and groundwater systems being studied.



## Comparison of Ontario and Iowa Results

[Table 10.1](#) provides a summary of results of the Iowa and Ontario analyses for TTHM (>80 versus <10 µg/L), BDCM (>9 versus <2 µg/L), and HAA5 (>60 versus 7.5 µg/L) and is the basis for the discussion of similarities and differences in cancer risks observed in Iowa and Ontario. Detailed analyses are presented in chapters 8 and 9, and readers who are interested in a more thorough comparison of the results should evaluate the analyses presented there. To allow such a comparison, the analyses in chapters 8 and 9 were conducted with similar exposure ranges.

### Similarities in Relative Risk

Many similarities in risk were observed in Iowa and Ontario. For bladder cancer, the relative risk observed among males in Ontario and Iowa was similar for TTHM exposures >80 µg/L (OR in Iowa = 1.28; 95% CI = 0.81–2.02 versus OR in Ontario = 1.69; 95% CI = 0.97–2.95), BDCM exposures >9 µg/L (OR in Iowa = 1.39; 95% CI = 1.00–1.94 versus OR in Ontario = 1.32; 95% CI = 0.68–2.58), and HAA5 exposures >60 µg/L (OR in Iowa = 1.05; 95% CI = 0.38–2.94 versus OR in Ontario = 1.48; 95% CI = 0.87–2.52) compared to TTHM exposures <10 µg/L, BDCM exposures >2 µg/L, and HAA5 exposures >7.5 µg/L. Risks were moderately elevated for TTHM but only slightly elevated for BDCM and HAA5 with slightly higher estimates for Ontario. The risks were not statistically significant for BDCM in Ontario and HAA5 in either Iowa or Ontario. For bladder cancer among females, similar risks were found for BDCM exposures; no increased risk was observed (OR in Iowa = 0.75; 95% CI = 0.40–1.40 versus OR in Ontario = 0.43; 95% CI = 0.09–2.00).

For colon cancer among females, no increased risks were observed in either Iowa or Ontario. For colon cancer among males, a moderate increased risk was observed in Iowa and Ontario for TTHM (OR = 1.68; 95% CI = 0.92–2.94 in Iowa and OR = 2.26; 95% CI = 1.28–3.97 in Ontario); risks for BDCM were slightly increased (30%) in both studies, but the results were not statistically significant. For rectal cancer among females, no increased risks were found in either Iowa or Ontario.

### Differences in Relative Risk

Several important differences in risk were observed in the Iowa and Ontario populations and are illustrated in [Table 10.1](#). Readers who wish to directly compare results for different exposure ranges are referred to chapters 8 and 9 where the more detailed analyses are presented.

For bladder cancer, the increased relative risk observed among females in Ontario was more than doubled (OR = 2.26; 95% CI = 1.00–5.11) for TTHM exposures >80 µg/L, whereas in Iowa no increased risk (OR = 0.83; 95% CI = 0.38–1.80) was observed for these exposures. A similar difference in bladder cancer risk was found for HAA5 exposures. An increased relative risk of bladder cancer (62%) was observed for females in Ontario (OR = 1.62; 95% CI = 0.72–3.66) for HAA5 exposures >60 µg/L, whereas in Iowa no increased risk (OR = 0.35; 95% CI = 0.07–1.67) was observed for these exposures.

For colon cancer, the increased relative risk observed for males in Ontario was doubled (OR = 2.00; 95% CI = 1.17–3.42) for HAA5 exposures >60 µg/L. In Iowa, no increased risk (OR = 0.49; 95% CI = 0.06–3.91) was observed for HAA5 exposures >60 µg/L.

**Table 10.1**  
**Comparison of Iowa and Ontario results—selected analyses**

Cancer sites/selected DBPs DBP values (µg/L)	Iowa OR (95% CI)	Ontario OR (95% CI)	Test for heterogeneity (p-value)
<b>Bladder</b>			
<i>All</i>			
TTHM >80 versus <10	1.14 (0.77–1.67)	1.82 (1.16–2.88)	0.12
BDCM >9 versus <2	1.17 (0.88–1.56)	1.04 (0.58–1.87)	0.72
HAA5 >60 versus <7.5	0.75 (0.33–0.72)	1.50 (0.97–2.34)	0.15
<i>Male</i>			
TTHM >80 versus <10	1.28 (0.81–2.02)	1.69 (0.97–2.95)	0.45
BDCM >9 versus <2	1.39 (1.00–1.94)	1.32 (0.68–2.58)	0.89
HAA5 >60 versus <7.5	1.05 (0.38–2.94)	1.48 (0.87–2.52)	0.56
<i>Female</i>			
TTHM >80 versus <10	0.83 (0.38–1.80)	2.26 (1.00–5.11)	0.08
BDCM >9 versus <2	0.75 (0.40–1.40)	0.43 (0.09–2.00)	0.51
HAA5 >60 versus <7.5	0.35 (0.07–1.67)	1.62 (0.72–3.66)	0.09
<b>Colon</b>			
<i>All</i>			
TTHM >80 versus <10	1.32 (0.86–2.04)	1.51 (0.98–2.33)	0.67
BDCM >9 versus <2	1.09 (0.77–1.55)	0.82 (0.46–1.47)	0.41
HAA5 >60 versus <7.5	0.45 (0.13–1.52)	1.50 (0.99–2.26)	0.07
<i>Male</i>			
TTHM >80 versus <10	1.68 (0.92–2.94)	2.26 (1.28–3.97)	0.47
BDCM >9 versus <2	1.30 (0.81–2.10)	1.32 (0.64–2.72)	0.98
HAA5 >60 versus <7.5	0.49 (0.06–3.91)	2.00 (1.17–3.42)	0.20
<i>Female</i>			
TTHM >80 versus <10	1.00 (0.53–1.91)	0.87 (0.44–1.71)	0.77
BDCM >9 versus <2	0.91 (0.55–1.51)	0.36 (0.13–1.01)	0.11
HAA5 >60 versus <7.5	0.42 (0.09–1.91)	0.98 (0.51–1.88)	0.32
<b>Rectum</b>			
<i>All</i>			
TTHM >80 versus <10	1.84 (1.19–2.84)	0.77 (0.46–1.31)	0.01
BDCM >9 versus <2	1.28 (0.89–1.84)	0.82 (0.45–1.51)	0.22
HAA5 >60 versus <7.5	1.00 (0.37–2.71)	0.58 (0.33–1.00)	0.34
<i>Male</i>			
TTHM >80 versus <10	2.08 (1.17–3.72)	0.84 (0.43–1.67)	0.05
BDCM >9 versus <2	1.41 (0.87–2.29)	0.75 (0.33–1.73)	0.20
HAA5 >60 versus <7.5	1.55 (0.41–5.84)	0.63 (0.31–1.27)	0.24
<i>Female</i>			
TTHM >80 versus <10	1.66 (0.86–3.20)	0.63 (0.27–1.46)	0.08
BDCM >9 versus <2	1.18 (0.68–2.05)	0.91 (0.37–2.25)	0.63
HAA5 >60 versus <7.5	0.68 (0.15–3.12)	0.48 (0.19–1.17)	0.70

For rectal cancer, TTHM risks differed for both males and females with the higher risks being found in Iowa. In Iowa, an increased relative risk was observed among females (OR = 1.66; 95% CI = 0.86–3.20) for TTHM exposures >80 µg/L, whereas in Ontario no increased risk (OR = 0.63; 95% CI = 0.27–1.46) was observed. A similar difference in rectal cancer risk was found for males. The increased relative risk observed among males in Iowa was doubled (OR = 2.08; 95% CI = 1.17–3.72) for high TTHM exposures, whereas in Ontario no increased risk (OR = 0.84; 95% CI = 0.43–1.67) was observed.

### ***Joint Effects of Exposure to DBPs and Water Consumption***

The joint effect of exposure to DBPs and water consumption was also evaluated in both Iowa and Ontario. The analyses suggest an independent effect of tap water consumption for some cancer risks.

In Iowa, the joint effect of average TTHM and HAA6 concentrations and the usual daily tap water consumption for male bladder, colon, and rectal cancers was examined. The referent group for all comparisons contained subjects with low tap water consumption ( $\leq 1.7$  L/day) and low average DBP levels ( $\leq 0.5$  µg/L for TTHM;  $\leq 0.9$  µg/L for HAA6). For bladder cancer, males with the highest TTHM and HAA5 exposure and the highest tap water consumption had a 65%–85% increased relative risk. The risk estimates rose with increasing water consumption for all except one of the TTHM and HAA5 exposure categories. Results for male rectal cancer were inconsistent across exposure categories and difficult to interpret. Risk estimates rose with increasing water consumption for low TTHM and HAA5 exposures. The pattern of increasing risk is reversed for males exposed to higher levels; men drinking large volumes of water were at no increased risk of rectal cancer. For male colon cancer, there were no consistent patterns of risk across either the tap water or DBP exposure categories.

The joint effect of water consumption and DBP exposure was also evaluated in Ontario, but the DBP exposure and water consumption categories may differ from Iowa. In addition, the Ontario analysis included both males and females and evaluated BDCM exposures. Similar to Iowa, subjects in Ontario with the highest exposures and highest water consumption had the highest bladder cancer risks. Bladder cancer risks of 2 or greater were found in this category for TTHM, BDCM, and HAA5 exposures. Relative risks for TTHM and HAA5 exposures rose with increasing water consumption only for subjects in the highest-exposure category. For subjects with the highest water consumption, bladder cancer risks generally rose with increasing concentrations of TTHM, BDCM, and HAA5 exposure. For colon cancer, subjects with high by-product exposure and high tap water consumption had statistically significant relative risks greater than 2 for TTHM, BDCM, and HAA5 exposures, but the pattern of risk across exposure and water consumption categories was generally inconsistent.

### **Future Analyses of the Epidemiologic Data**

In addition to the results presented in this report, the new exposure estimates result in numerous opportunities for other analyses. Other analyses currently being explored include risk among subgroups (i.e., bladder risk among smokers and nonsmokers), simultaneous modeling of risk associated with different by-products, examination of different exposure windows, analysis restricted to those with homogeneous exposures, examination of colon and rectal risk according to cancer location (i.e., distal colon, proximal colon, sigmoid junction, rectum), and pooling of



the Ontario and Iowa data. Current funding is insufficient to conduct these analyses as part of this project.

### ***Simultaneous Modeling of Risk Associated With Different By-Products***

An important topic for future analyses is the examination of several DBPs in one multiple regression logistic model. The current study involved the estimation of three HAAs (DCAA, TCAA, and BCAA), two HAA groups (HAA5 and HAA6), and two THMs (chloroform and BDCM) in addition to TTHM. The analysis of each by-product or by-product group separately for Iowa yielded consistent increases in risk for the highest-exposure categories of the nonbrominated THMs and HAAs for bladder, colon, and rectal cancers among males. Risk increases were moderately higher for HAA compounds than for THMs for cancers of the bladder and colon, but not for rectal cancer, making it difficult to comment on the relative importance of either chemical group. No consistent risk increases were noted for either of the brominated compounds, BDCM or BCAA, that were evaluated. It will be of interest to further examine the correlations between the individual DBPs in both Iowa and Ontario and to explore the possibilities of examining multiple by-products in one statistical model. The authors plan to first look at the correlation between the various DBPs that have been modeled before including them in a multiple regression equation. If the correlation between them is strong, then there is little possibility of separating out their potential effects.

### ***Pooled Analysis of the Ontario and Iowa Data***

Because of the similar study design and similar quality of historical DBP predictions, a pooled analysis of the Iowa and Ontario data should be considered. The pooled analysis would offer several benefits. The larger population available for analysis should provide a more precise estimate of risk for a larger number of DBP exposure categories. In addition, an analysis restricted to surface water users in both Ontario and Iowa could be conducted using the pooled data. Before a pooled analysis can be considered, a test for heterogeneity should be conducted. A requirement for pooled analysis is that the study results be homogeneous. A preliminary analysis ([Table 10.1](#)) shows that, with the exception of findings for TTHM and rectal cancer among males and females and for bladder cancer among females, most of the relative risk results are homogeneous and could be considered in a pooled analysis of the Iowa and Ontario data. We emphasize the preliminary nature of our heterogeneity analysis. For example, the HAA5 results for Iowa presented in [Table 10.1](#) are statistically unstable (few subjects were exposed to HAA5 levels >60 µg/L) and not very representative of the results that were presented in chapter 9 for HAA exposure. The authors plan to conduct additional comparisons using the Q statistic before making a decision on pooling.

## **CONCLUSIONS**

The improvement of exposure assessment among subjects and the resulting reduction in exposure misclassification was one of the primary goals of the current undertaking. The estimation of TTHM and HAA levels on a utility-by-utility basis, using a wide range of information, is an important step in advancing our knowledge of historical exposures to DBPs. The more rigorous

modeling of historical DBP levels suggested that exposure was misclassified in the two previously conducted epidemiologic studies (Cantor et al. 1998; King and Marrett 1996). Using these modeling methods to estimate historical DBP levels, investigators should be better able to capture the variability in DBP levels in surface and mixed surface and groundwater sources.

The TTHM cancer risks were similar when the exposure ranges from the authors' earlier studies were used to define exposure. This is reflected in the high correlation coefficient observed when comparing old to new TTHM estimates, overall. However, there was important variability in TTHM estimates when old TTHM were compared with new TTHM in the more highly exposed group, and the correlation coefficient in these cases became much smaller (e.g.,  $>40 \mu\text{g/L}$ ). This indicates that the bulk of the misclassification was among high-exposure people. In risk analyses, using the new data, when the exposure ranges were changed to include the highest-exposed people in separate strata, a more uniform trend and higher estimates of risk were observed at the very high end of exposure, especially among men. This represents an important change in the risk estimates and justifies the current, more rigorous effort to model historical DBP levels. It appears that the risk estimates improved.

The authors believe the improved water quality modeling efforts have considerable benefits and should be applied in future epidemiologic studies of DBPs and cancer and other health risks. The modeling not only adds considerable transparency to the exposure assessment but also additional information about the various species of DBPs. The minimal costs associated with this modeling effort allowed for a more precise estimate of exposure and a more robust data set for the evaluation of DBP risk, both important for interpreting epidemiologic results.

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## ABBREVIATIONS

AOC	assimilable organic carbon
AWWA	American Water Works Association
AwwaRF	Awwa Research Foundation
BCAA	bromochloroacetic acid
BDCAA	bromodichloroacetic acid
BDCM	bromodichloromethane
BDL	below detection limit
Br <sup>-</sup>	bromide
Br-THMs	bromine-containing THMs
°C	degree Celcius
CaCO <sub>3</sub>	calcium carbonate
CH	chloral hydrate
CHBr <sub>3</sub>	bromoform
CHCl <sub>3</sub>	chloroform
CHEEC	Center for Health Effects of Environmental Contamination
CI	confidence interval
Cl <sup>-</sup>	chloride ion
Cl <sub>2</sub>	chlorine
Cl <sub>2</sub> /Cl <sub>2</sub>	prechlorination and postchlorination
Cl <sub>2</sub> /CLM	prechlorination and postchloramination
Cl <sub>2</sub> /NH <sub>3</sub>	postchloramination
Cl <sub>2</sub> / & CLM/CLM	prechlorination followed with ammonia and postchloramination
ClO <sub>2</sub>	chlorine dioxide
CLM/CLM	prechloramination and postchloramination
CLX/Cl <sub>2</sub>	prechlorine dioxide and postchlorination
CLX/CLM	prechlorine dioxide and postchloramination
CNCl	cyanogen chloride
CP	chloropicrin
CPD	cumulative probability distribution
cu	color unit
DAI	direct aqueous injection
DBAA	dibromoacetic acid
DBCAA	dibromochloroacetic acid
DBCM	dibromochloromethane
DBP	disinfection by-product
DBP-Br	sum of THM-Br and HAA-Br
DCAA	dichloroacetic acid
D/DBP Rule	Disinfectants/Disinfection By-Products Rule
DNR	Department of Natural Resources (Iowa)
DOC	dissolved organic carbon
DS	distribution system

DSE	distribution system equivalent
DWSP	Drinking Water Surveillance Program (Ontario)
DXAA	total sum of the dihaloacetic acids
ft	foot
g	gram
GAC	granular activated carbon
GW	groundwater
HAA	haloacetic acid
HAA3	sum of three HAA species beyond HAA6
HAA5	sum of five regulated HAA species
HAA6	HAA5 + BCAA
HAA9	sum of all nine HAA species
HAN	haloacetonitrile
HK	haloketone
ICR	Information Collection Rule
L	liter
L/cm-mg	liters per centimeter-milligram
L/m-mg	liters per meter-milligram
m	meter
MBAA	monobromoacetic acid
MCAA	monochloroacetic acid
MCL	maximum contaminant level
mg/L	milligram per liter
µg/L	microgram per liter
NAGW/HiBr	nonalluvial groundwater systems with high-brominated THM concentrations
NAGW/LoBr	nonalluvial groundwater systems with low-brominated THM concentrations
NH <sub>2</sub> Cl	monochloramine
NH <sub>3</sub>	ammonia
NH <sub>3</sub> -N	ammonia-nitrogen
NOMS	National Organics Monitoring Survey
NORS	National Organics Reconnaissance Survey
ntu	nephelometric turbidity unit
OCl <sup>-</sup>	hypochlorite
O <sub>3</sub> /CL <sub>2</sub>	preozonation and postchlorination
O <sub>3</sub> /CLM	preozonation and postchloramination
OR	odds ratio



PAC	powdered activated carbon, Project Advisory Committee
POC	particulate organic carbon
ppb	parts per billion
ppm	parts per million
PY	person-years
QA/QC	quality assurance/quality control
RNDB	regulation negotiation (reg neg) database
SDWA	Safe Drinking Water Act
ShGW/HiBr	shallow groundwater systems with high-brominated THM concentrations
ShGW/LoBr	shallow groundwater systems with low-brominated THM concentrations
SUVA	specific ultraviolet absorbance
SW	surface water
SW/GW	mixed systems in which utilities utilize both surface water and groundwater
TBAA	tribromoacetic acid
TCAA	trichloroacetic acid
TDS	total dissolved solids
THM	trihalomethane
THM-Br	calculated bromide incorporation associated with measured THM species
TOC	total organic carbon
TOX	total organic halides
TTHM	total THMs (sum of four species)
TXAA	total sum of trihaloacetic acids
U.S.	United States
USEPA	U.S. Environmental Protection Agency
USGS	U.S. Geological Survey
UV	ultraviolet
UVA	ultraviolet absorbance
UVA <sub>254</sub>	UV absorbance at 254 nanometers
WIDB	Water Industry Data Base
WSS	water supply system
WTP	water treatment plant





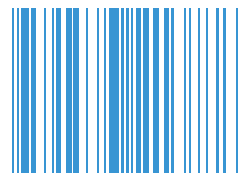


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