

Peer Review for
California Regional Water Quality Control Board
Central Valley Region

Proposed Basin Plan Amendments for New Alamo and Ulatis Creeks in Solano County

I. Action to be Reviewed.

The proposed action requiring peer review was detailed in Attachment 1 to the memorandum dated 29 October, 2009 addressed to Dr. Gerald Bowes, State Water Resources Control Board. This action involves a proposal for site-specific water quality objectives for chloroform, chlorodibromomethane and dichlorobromomethane and permit implementation provisions.

II. Scientific Issues to be Addressed

The specific scientific issues identified for review were to determine whether:

1. the proposed site-specific objectives provide adequate protection of human health
2. the approach to determining “reasonable potential” would be appropriate and reasonable
3. the “attenuation factor” as proposed, is a technically sound approach to derive the effluent limits
4. there are any other “big picture” issues regarding the scientific issues that warrant peer review comment

III. Peer Review Comments on the Scientific Issues to be Addressed

1. Adequate Protection of Human Health

Having reviewed all the documentation provided in the peer review package as well as several other regulatory documents and relevant scientific literature, I am of the view that the human health issue is the dominant issue worthy of detailed comment in terms of scientific validity. Based on the comments provided on this issue, it will be clear that issues 2 and 3 involve relatively minor scientific issues by comparison. Accordingly, discussion of the human health issue will dominate my peer review response.

The contaminants which are the subject of this review chloroform, chloro-dibromomethane (CDBM)¹ and dichlorobromodichloromethane (DCBM)¹ are three of

¹ The terminology used in this documentation, although commonly in use, differs from some common drinking water literature, regulatory documents and the USEPA IRIS database. These reference sources refer to these two contaminants as dibromochloromethane (DBCM) and bromodichloromethane (BDCM). To minimize confusion, I will use the terminology as it has been used in the documentation provided.

the common trihalomethane (THM) disinfection by-products, the other of which is bromoform, a contaminant that is not considered in this proposed action. These disinfection by-products are regulated as a group by a maximum contaminant level (MCL) for total trihalomethanes (TTHM) of 80 µg/L under the Safe Drinking Water Act. It is ironic to be judging the scientific validity of the rationale for setting site-specific water quality objectives to assure adequate protection of human health by classifying the specified water courses for a “MUN” beneficial use (municipal and domestic water supply) when the current Safe Drinking Water Act MCL for TTHM is not “*incorporated by reference in the Basin Plans as a water quality objective for chemical constituents. Thus, the 80 µg/L MCL is not directly applicable as a water quality objective for surface waters.*”² This situation is a logical conundrum given that the entire exercise is aimed at preserving the specified water bodies for potential future use as a source for drinking water supplies yet the most relevant criterion from the Safe Drinking Water Act cannot be applied through the regulatory process.

1(a) Total trihalomethanes (TTHM)

The TTHM were first discovered in drinking water by Dutch chemist Johannes Rook (1974) and independently and almost simultaneously by Bellar, Lichtenberg and Kroner (1974) at the USEPA. The appearance of these trace organics consistently in chlorinated drinking water attracted even greater attention when the National Cancer Institute published results from a rodent cancer bioassay (NCI 1976) showing that chloroform in corn oil dosed by gavage (direct insertion in the stomach) caused a dramatic excess of tumors in exposed animals. Generally, chloroform will comprise the majority of the TTHM in any drinking water system.

Drinking water regulations under the Safe Drinking Water Act were developed by 1979 with a MCL of 100 µg/L for TTHM as a running annual average of quarterly samples. The animal bioassay findings together with a number of ecological and case-control³ epidemiology studies provided some evidence that long term consumption of chlorinated drinking water was associated with increased risk of cancer at various sites and appeared to support a concern that TTHM, or at least chloroform, posed a human cancer risk via drinking water exposure (Orme Zavaleta et al. 1999). The evidence for colon and rectal cancer has been only suggestive of a causal association while the evidence for bladder cancer has been the most consistent association with chlorinated drinking water (Mills et al. 1998). However, an international expert panel for the World Health Organization (ICPS 2000) concluded about THM cancer risk: “*The existing epidemiological data are insufficient to allow a conclusion that the observed associations between bladder cancer*

² quote from Section 3.2, p.11 Draft Staff Report, Regional Water Quality Control Board, Central Valley Region.

³ Ecological and case-control studies, if well done, have only low (ecological) to moderate (case-control) capability to provide evidence in support of disease causation by the exposure variable under study. Apart from specific methodological limitations for these type of studies, effective and accurate exposure assessment (i.e. , How much of the agent under study was an individual exposed to over a matter of decades?) remains a problem for epidemiology studies seeking an association between DBPs and cancer.

or any other cancer and chlorinated drinking water or THMs are causal or provide an accurate estimate of the magnitude of risk.”

1(b) Chloroform

Chloroform was subsequently re-evaluated (after the 1976 NCI bioassay) in a rodent bioassay using oral ingestion of chloroform in drinking water (Jorgenson et al. 1985). This research found increasing kidney tumor incidence with increasing dose level above 200 mg/L relative to the control, but only chloroform at 1,800 mg/L showed a statistically significant increase in tumors relative to the control. Hard et al. (2000) performed a re-evaluation of the Jorgenson et al. (1985) rat bioassay results. They found that all the rats at 1,800 mg/L chloroform and half of those at 900 mg/L exposure showed evidence of cytotoxicity and regenerative cell proliferation which are high dose effects that are not consistent with chloroform acting primarily as a genotoxic, no-threshold carcinogen.

Support for the carcinogenic action of chloroform being a cytotoxic rather than a genotoxic mechanism was provided by additional research. Larson et al. (1994, 1995) demonstrated that the corn oil gavage delivery of chloroform, as used in the 1976 NCI bioassay, induced cytotoxicity and cell proliferation in liver for mice and kidney and liver for rats. These high dose effects were not observed for delivery of similar daily doses of chloroform by oral ingestion of drinking water. A plausible mechanism for chloroform carcinogenicity being a threshold mechanism was supported by extensive evidence showing negligible mutagenic activity for chloroform (Golden et al. 1997). An expert panel, sponsored in part by the USEPA, was convened by the International Life Sciences Institute in 1997. USEPA (1998a) reported that: *“The panel viewed chloroform likely to be a carcinogen above a certain dose range, but considered it unlikely to be a carcinogen below a certain dose range. The panel indicated that: ‘This mechanism is expected to involve a dose-response relationship which is non-linear and probably exhibits an exposure threshold.’ The panel, therefore, recommended the non-linear default or margin of exposure approach as the appropriate one for quantifying cancer risk associated with exposure to chloroform.”*

Taken together, these findings provided the empirical evidence that the USEPA accepted as the basis to propose adopting a non-linear, threshold model of carcinogenesis for chloroform and in so doing, modifying their maximum contaminant level goal (MCLG) for chloroform from zero to 300 µg/L (USEPA 1998a). Because this proposal drew extensive critical commentary, the USEPA chose to delay changing the MCLG for chloroform up from zero (a level indicative of a no threshold, linear model), pending input from its Science Advisory Board and further review of the relative source contribution (RSC) which had been set at 80% to calculate the MCLG of 300 µg/L (USEPA 1998b). In making this decision for further review the USEPA effectively retained a linear, no-threshold model of cancer risk even though they did so while stating: *“EPA believes the non-linear cancer extrapolation is the most appropriate means to establish an MCLG for chloroform based on carcinogenic risk.”*

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The USEPA decision to undertake further review rather than adopt a non-zero MCLG for chloroform attracted a legal challenge from the Chlorine Chemistry Council along with some drinking water utilities (Pontius 2000). The Natural Resources Defence Council and Physicians for Social Responsibility intervened to support the USEPA decision to retain an MCLG of zero for chloroform. The legal challenge was based on the requirement of the Safe Drinking Water Act for the USEPA to use the best available science in setting standards. While the case was before the Court, the Chloroform Risk Assessment Review Subcommittee of the USEPA Science Advisory Board released its conclusion that the “*most appropriate way to assess chloroform risks was with a non-linear method.*” Shortly thereafter (Pontius 2000), the USEPA, itself, asked the court to vacate or invalidate the chloroform MCLG of zero that it had issued on December 16, 1998 (USEPA 1998b). On March 31, 2000, the US District of Columbia Circuit issued a decision finding that the USEPA had violated the Safe Drinking Water Act by not using the best available science. On this basis, the Court vacated the MCLG of zero for chloroform and ordered USEPA to set a new MCLG.

Ultimately, the USEPA issued a final MCLG of 70 µg/L for chloroform (USEPA 2006), using the same toxicological information - a reference dose of 0.01 mg/kg/d which incorporates a 1000 fold uncertainty factor from the lowest observed adverse effect level for liver hepatotoxicity in dogs (Heywood et al. 1979) – the reference dose used in previously proposing 300 µg/L (which had been rounded to 1 significant figure from 280 µg/L). However, in consideration of evidence about multiple routes of chloroform exposure, the relative source contribution factor (RSC) was lowered from 80% to 20% - which is the default and lowest allowable RSC under USEPA policy (USEPA 2000). The change in the RSC used (20% vs. 80%) is the sole explanation for the lower final MCLG of 70 µg/L. The floor value of the RSC is presumably intended to reflect the minimum contribution of contaminant worthy of regulatory attention. Use of any lower value of RSC would result in a correspondingly more restrictive water standard for a contaminant that would be primarily (more than 80%) coming from other sources which may not be regulated in any way and which deserve more priority for regulatory attention.

The point of all of this history is that if the best available science is to be used, estimation of cancer risk for chloroform by use of a slope factor as has been done for this proposal is not correct and the more than sufficiently cautious MCLG of 70 µg/L should have been used (except for the various regulatory and/or policy obstacles mitigating against the use of the 70 µg/L MCLG). It is noteworthy that the USEPA toxicology database, IRIS, no longer provides a cancer slope factor for chloroform. Rather, IRIS provides only a reference dose of 0.01 mg/kg/d which is the toxicological criterion that was used to derive the 70 µg/L MCLG along with the minimum allowable RSC of 20%.

In preparing their Final Report on the proposal under review, Robertson-Bryan Inc. (RBI 2007) provided Table 1 which listed relevant criteria for DBCM, DCBM and chloroform. This showed that specifying a value for chloroform was being reserved under the California Toxics Rule “*to allow for reassessment based on new information.*” The cited USEPA recommended criteria provided two values for chloroform 5.7 µg/L and 68 µg/L.

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The first lower value was reported as being taken from the 2006 USEPA National Recommended Water Quality Criteria, but I could find no such 2006 document on the USEPA website. The most recent report before the current 2009 version of this document (USEPA 2009) was issued in 2002. This is a minor point because the 2009 report provides the same value, 5.7 µg/L, for chloroform (USEPA 2009). The 68 µg/L value in Table 1 was cited as coming from a 73 page revised draft report, dated December 2003 titled “*Ambient Water Quality Criterion for the Protection of Human Health – Revised Draft*”. This draft value is only slightly lower than the MCLG of 70 µg/L discussed above because its calculation includes exposure from ingesting drinking water plus ingestion of 17.5 g/d of fish at various trophic levels, which, in total, reduce the criterion by only 2 µg/L below the 70 µg/L MCLG. Considered another way, if the water criterion for human health was based only on fish consumption, this document develops a water criterion of 2,400 µg/L, showing that the fish consumption route alone contributes very little to lowering the allowable water concentration of chloroform for protecting human health.

Robertson-Bryan Inc. concluded that because the 68 µg/L recommendation was in a draft report, they were obliged to use the previous value of 5.7 µg/L, for chloroform. That decision was consistent with the lack of any change in the 2009 National Recommended Water Quality Criteria (USEPA 2009), but the reason for having no update six years after the draft chloroform risk assessment (USEPA 2003) is difficult to understand.

From the perspective of the best available scientific evidence, it is difficult to rationalize any scientific sense to referring to a limit of 5.7 µg/L to protect human health from drinking water ingestion using ambient water that is based on an out-dated understanding of the mode of chloroform carcinogenicity when the drinking water MCLG for chloroform has been set at 70 µg/L in the Safe Drinking Water Act following a protracted and exhaustive review of the scientific evidence. The MCLG is by definition intended to mean that there is no cancer risk at exposure levels below the MCLG.

The evaluations in the report under review for human health concerns of all options for a site-specific water quality criterion for chloroform were all done using a linear, no threshold cancer slope factor that has been abandoned under the Safe Drinking Water Act and by IRIS, the primary toxicological reference base for the USEPA.

For all of the foregoing reasons, I find that there is no scientifically credible basis for any human health concerns with the extremely cautious recommended limit for chloroform of 45.5 µg/L.

1(c) Dichlorobromomethane (DCBM)

DCBM (also referred to in some USEPA and international literature as BDCM) has a less detailed regulatory history than chloroform, but this disinfection by-product came under increasing scrutiny after some epidemiological study results suggested that it may be important for adverse reproductive outcomes (Waller et al. 1998, Dodds et al. 2004).

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Epidemiological studies of mixed exposures such as disinfection by-products are rarely robust enough to allow quantitative determination of a health-based criterion. In any case, a major study (Savitz et al. 2005, 2006) undertaken to evaluate the apparent association of spontaneous abortion with DCBM previously reported by Waller et al. (1998) found that this could not be replicated using a study design which had substantially improved exposure assessment compared with Waller et al. (1998). A recent expert panel for Health Canada called to review its drinking water guideline for DCBM concluded on the question of epidemiological evidence for reproductive effects that: *“Overall, the evidence from epidemiological studies is inconsistent and by international standards, the current weight of evidence is not sufficient to support an association between adverse reproductive and developmental effects in humans and environmental exposures to BDCM.”* [DCBM in the current report] (Health Canada 2008).

On the question of toxicological evidence of adverse reproductive effects with DCBM, the expert panel concluded: *“Although BDCM has been shown to cause adverse reproductive effects in animals, these have only been observed at maternally-toxic doses which are 5000 to 15000 times higher than levels found in drinking water. Overall, the current weight of evidence from toxicological studies does not support an association between adverse reproductive/developmental effects and exposure to BDCM at concentrations that occur in chlorinated drinking water.”* [DCBM in the current report] (Health Canada 2008).

The Health Canada expert panel was called because Canada had set a drinking water guideline for DCBM in 2006 of 16 µg/L, which was based on a 10⁻⁵ lifetime cancer risk using a cancer slope factor derived from linearized multistage (i.e. linear, no threshold) modeling of 1987 rodent bioassay results (NTP 1987). These bioassay results, like those for chloroform in 1976, had been obtained using high bolus gavage doses of DCBM in corn oil. When a bioassay was done using oral ingestion dosing of DCBM in drinking water to male rats and female mice (NTP 2006), no evidence of any carcinogenic activity in either species at target concentrations up to 700 mg/L of DCBM was found. This finding taken together with the Savitz et al. (2005, 2006) negative findings for adverse reproductive effects led Health Canada to review the DCBM guideline which had been set. Given the new evidence, the expert panel found no need to keep the guideline for DCBM and it was rescinded by the Canadian Federal / Provincial / Territorial Committee on Drinking Water in April 2009 leaving Canada with a limit only on TTHM. This regulatory position is consistent with the Safe Drinking Water Act which has a MCL only for TTHM (80 µg/L).

The U.S. regulatory history on DCBM is less explicit. There is no separate drinking water MCL for DCBM and the MCLG has been kept at zero despite the recent negative findings on carcinogenicity (NTP 2006). The USEPA IRIS toxicological database for DCBM was last revised March 7, 2005, so the data there do not reflect the latest rodent carcinogen bioassays results. Even so, DCBM was listed, as of February 5, 2003, as *“being reassessed under the IRIS program”*. Given the delays in processing carcinogenicity evidence for this chemical, the Board is faced with judging DCBM with

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only the available regulatory reference material. However, the Board can be reassured that current scientific evidence is pointing towards less, not more, cancer risk associated with DCBM.

Within that context, the question of judging the cancer risks cited for various water quality objective options requires consideration of the basis for those cancer risk predictions made without the benefit of recent and better scientific evidence.

Accepting it at face value, the regulatory model used for calculating cancer risks based on a linear, low dose extrapolation provides an upper bound estimate of cancer risk, not an expected cancer risk. This was most clearly explained in the 1986 Cancer Risk Assessment Guidelines (USEPA 1986) which remain posted on the USEPA website and state: *“It should be emphasized that the linearized multistage procedure leads to a plausible upper limit to the risk that is consistent with some proposed mechanisms of carcinogenesis. Such an estimate, however does not necessarily give a realistic prediction of the risk. The true value of the risk is unknown, and may be as low as zero. The range of risks, defined by the upper limit given by the chosen model and the lower limit which may be as low as zero, should be explicitly stated.”* When it comes to judging, from a scientific perspective, whether there is a substantial risk to human health from the water quality objectives proposed, the range of estimated risks must be considered to provide that perspective.

The proposal of a site-specific water quality objective for DCBM of 15.5 µg/L provides a cancer risk estimate ranging from $10^{-4.6}$ (i.e., 0.000025) to zero lifetime cancer risk, which means that if 40,000 consumers each ingested 2L per day of this concentration of DCBM continuously for all of 70 years, the predicted range of cancer outcome would be between zero and possibly 1 case cancer attributable to DCBM over a 70 year period.

For all of the foregoing reasons, I find that there is a negligible scientific basis to justify any credible human health concerns with the recommended site-specific water quality objective for DCBM of 15.5 µg/L.

1(d) Dibromochloromethane (DBCM)

DBCM has an even less extensive evidentiary base for carcinogenesis than the two other THMs considered. Only weak evidence for carcinogenicity was obtained in a rodent bioassay performed with high doses by corn oil gavage (NTP 1985). The USEPA IRIS toxicology data base reports that DBCM is classified as “C – possible human carcinogen”, the lowest classification to be treated as a carcinogen. This classification is based on: *“inadequate human data and limited evidence of carcinogenicity in animals; namely, positive carcinogenic evidence in B6C3F1 mice (males and females), together with positive mutagenicity data, and structural similarity to other trihalomethanes, which are known animal carcinogens.”* The latter point about structural similarity to other THMs which are described as “known animal carcinogens”, given current scientific

evidence of limited carcinogenicity for the other THMs, provides relatively weak support to the otherwise limited basis for classifying DBCM as a carcinogen.

The proposal of a site-specific water quality objective for DBCM of 4.5 µg/L provides a cancer risk estimate ranging from $10^{-4.9}$ (i.e., 0.0000126) to zero lifetime cancer risk, which means that if 80,000 consumers each ingested 2L per day of this concentration of DBCM continuously for all of 70 years, the predicted range of cancer outcome would be between zero and possibly 1 case of cancer attributable to DBCM over a 70 year period.

For all of the foregoing reasons, I find that there is a negligible scientific basis to justify any credible human health concerns with the recommended site-specific water quality objective for DBCM of 4.5 µg/L.

2. Determination of “Reasonable Potential”

The recommended option 4b and its associated determination of “*reasonable potential*” to cause or contribute to an excursion above the site-specific THM objectives within the defined segments of the watershed are not scientifically challenging, if somewhat administratively complex. The fact that a short term excursion of any of the parameters above the site-specific THM objectives at any location in the defined segments would have no health-related consequences to any transient or incidental drinking water user provides a substantial buffer for any unforeseen failures in the implementation of this amendment.

I find that the determination of “reasonable potential” to cause or contribute to an excursion above the site-specific THM objectives within the defined segments of the watershed for recommended option 4b poses no scientific problems with its derivation.

3. The Proposed “Attenuation Factor”

The proposed “*attenuation factor*” for recommended option 4b is derived from a basic mass balance calculation which is mathematically and conceptually correct for determining the appropriate values to define a maximum effluent concentration. As such, there are no underlying scientific issues concerning this proposal.

I find that the proposed determination of the “attenuation factor” for recommended option 4b to determine the appropriate values to define a maximum effluent concentration poses no scientific problems with its derivation.

4. Other “Big Picture” Scientific Issues Warranting Comment

4(a) Use of significant figures in final documentation of rationale.

The Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health - 2000 (USEPA 2000) provides some valuable advice, as follows: “*When developing criteria, EPA recommends rounding the number of significant figures at the end of the criterion calculation to the same number of significant figures in the least precise parameter.*” (p. 2-11) This is a scientific issue which has a bearing on the communication and credibility of the recommendations being proposed. There are countless examples in the documentation provided where an excessive number of significant figures have been carried over to final results, the most notable being the recommended site-specific water quality criteria themselves, two of which are expressed to 3 significant figures. The cancer risk predictions which underlie all of these site-specific water quality recommendations are based on using cancer slope factors with all of their attendant uncertainty. The guidance which was given on presenting numerical cancer risk estimates was (USEPA 1986): “*Irrespective of the options chosen, the degree of precision and accuracy in the numerical risk estimates currently do not permit more than one significant figure to be presented.*” Other limitations such as sampling and analytical accuracy will often limit estimates to no more than two significant figures.

If more significant figures are used than are warranted, the message conveyed is that the values are known with greater precision and possibly accuracy than they really are known.

4(b) Delays in adopting new chloroform evidence.

Although the authors of the recommendations that were submitted for peer review are captive to the regulatory system which authorizes their actions, the enormous delays that are evident in translating the best available scientific evidence into practice should be a concern for all who must work with the criteria. In the case of chloroform, there has clearly been a seriously inconsistent adoption of the best available evidence across the various elements of the USEPA which left the authors of the current work obliged to use evidence which is clearly outdated and scientifically inaccurate on the face of it.

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4(c) Beware of spurious data in monitoring low levels of brominated THMs.

From personal experience with my own research lab, I can caution that there is a potential difficulty with assuring accurate quantitative results for routine (gas chromatographic) monitoring of brominated THMs (in this case DCBM and DBCM) at levels approaching the detection limits, which is a range in which the recommended site specific water quality criteria will require monitoring results to be meaningful. In particular, the implementation of this program should allow a sensible and pragmatic approach calling for immediate re-testing if any exceedance values of either compound are apparently detected.

4(d) Rationale for protecting future MUN use.

The physical and chemical properties of the THMs are such that they represent transient, not persistent, nor substantially bioaccumulative pollutants. Likewise, any aquatic ecological concerns associated with these contaminants would have given rise to much higher (less restrictive) water quality objectives. Given these scientific realities, it would have been worthwhile to consider whether the protection of future MUN water use required imposition of stringent water quality controls at present to protect uses which may never occur rather than imposing such controls in the future if a need arose to protect actual MUN beneficial water uses.

4(e) Economic analysis of UV disinfection vs. long term monitoring.

The City of Vacaville may want to consider performing an economic analysis of the long term monitoring costs under the recommended program, the current status of its disinfection facilities (including dechlorination if that is practiced) and the economic viability of implementing UV disinfection for its wastewater effluent to avoid TTHM formation altogether. Ongoing monitoring costs can become substantial and UV disinfection is becoming more cost effective.

Submitted in accordance with the review request of 30 December, 2009



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