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REMOVAL OF CARCINOGENS FROM DRINKING WATER:
A COST-BENEFIT ANALYSIS

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I.   INTRODUCTION

     This study is an analysis of the costs and benefits of
implementing the amendment to the interim primary drinking water
regulations proposed by the Environmental Protection Agency in
January, 1978 (EPA, 1978). Specifically, the study will consider
the benefits and costs involved for those water supply systems
which would be required to remove organic contaminants by installing
granular activated carbon (GAC) as a post-filtration adsorbent.

     This study builds upon and extends the partial analysis of
these regulations conducted by the National Academy of Sciences
(NAS) earlier this year (NAS, 1978). In the words of one of the
panelists, that study "should be viewed as a guide for the method-
ology of benefit-cost analysis and not as a complete and definitive
study of the proposed EPA regulation of trihalomethanes."[1] The
NAS study is insufficient by itself for evaluating the proposed
regulations for the following reasons:

     1. The only drinking water contaminant considered was
         chloroform; the effects of other chlorination by-products
         and synthetic organic chemicals were ignored.

     2. The surface area adjustment method which was used to
         estimate the cancer risk associated with chloroform was
         inconsistent with a previous NAS panel's recommended
         method (NAS, 1975), which yields a risk estimate approxi-
         mately three to 15 times greater than the "upper limit"
         and "most probable" risk estimates, respectively, derived

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[1] Letter from John Cumberland, University of Maryland, to Victor
Kimm, EPA, August 29, 1978.

from the surface-area method.

3. The NAS study made no attempt to integrate evidence from epidemiologic studies in its derivation of risk estimates.

4. The only methods of contaminant removal considered were GAC adsorption and aeration, expensive methods which would not be required for two of the three levels of contamination dealt with in the study.

5. The only benefit considered was reduced cancer death; the prevention of other adverse health effects (mutagenic, teratogenic and fetotoxic), reductions in anxiety about the health risks of contaminated drinking water, and improvements in the taste and odor of tap water were not discussed.

6. Two important factors were excluded from the benefit-cost analysis but must be discussed in evaluating the results: irreversibility and risk aversion.[1]

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[1] In the same letter, op. cit. Cumberland continues: "Since Chapter 8 referred to above was necessarily limited by data, time constraints, and other guidelines established for the considerations of the panel, some important aspects were necessarily omitted, and should be carefully considered in a complete evaluation. Among the additional important considerations which should be evaluated as the basis for any regulation are:
1. benefits of removal of other THMs besides chloroform,
2. benefits of removal of carcinogens which are not THMs,
3. other benefits such as removal of mutagens,
4. consideration of other means of removing THMs such as alternative forms of disinfectants, and
5. inclusion of other means of risk estimation, most importantly inclusion of the epidemiological evidence."

(CWPS) "Analysis of Proposed EPA Drinking Water Regulations" follows in its essential respects the methodology of the NAS study. It furthers the methodology by developing estimates of incremental cost for evaluation of the cost-effectiveness of the regulations as a function of city size and pollution load. While this extension is a contribution, the main intention of the CWPS study was not to develop a methodology but to make realistic estimates of cost per cancer prevented and from these to draw realistic policy recommendations. However, the same six limitations just ennumerated for the NAS study apply as well to the CWPS study, suggesting that CWPS has significantly underestimated the benefits associated with drinking water improvement. Consideration of point two, for example, leads to a "cost per life saved" on order of magnitude lower than estimated by CWPS.

The study presented here will focus on water supply systems with contamination levels which may necessitate the use of GAC, and will be limited to communities large enough to be affected by the proposed regulations. The cancer risks associated with organic drinking water contaminants will be derived from the evidence of animal studies taken together with evidence from epidemiologic studies. Additional benefits will be dealt with to some degree, and it will be explained how the consideration of factors additional to the results of the benefit-cost analysis should enter into the determination of the economic evaluation of GAC.

It is realized that this study will also have short-

comings, particularly with regard to certain simplifying assumptions which were made. As more information is developed regarding the risks of contaminated drinking water, some of those assumptions may need revision. Only a little will be said about irreversibility and social risk aversion. Nevertheless, it is hoped that this study will, at the very least, carry the analysis of the economic evaluation of the proposed regulations an incremental step beyond the NAS and CWPS analyses of the benefits and cost associated with chloroform reduction.

II. BACKGROUND

Under the Safe Drinking Water Act of 1974 (PL 93-523), the Environmental Protection Agency (EPA) is required to prescribe regulations for those contaminants which the Administrator determines may have an adverse effect on human health. Under Section 1412(a)(2), these contaminants are to be controlled "to the extent feasible, . . . (taking costs into consideration) . . . ." The Congressional intent is strongly preventive; conclusive proof of an adverse health effect is not a prerequisite to regulation (United States Congress, 1974).

The Organics Problem

Recent research has demonstrated that organic contaminants potentially harmful to human health are ubiquitous in America's drinking water. Over 700 such contaminants have been identified, yet they represent only about 15 percent by weight of the total organic matter in drinking water (EPA, 1978a; NAS, 1977). Many contaminants cannot be identified and/or quantified given present analytical methodologies.

Only a small fraction (less than 10 percent) of the known contaminants have been adequately tested for adverse health effects. A recent listing by the National Cancer Institute (see Appendix A) identified 23 chemicals as known or suspected carcinogens, 30 chemicals as known or suspected mutagens, while 11 chemicals were identified as tumor promoters (i.e., substances which are not in themselves carcinogens but which interact with carcinogens to enhance the rate of tumor formation). In addition, some drinking

water contaminants are known to be teratogenic (cause birth defects) or fetotoxic (result in fetal deaths or stunted growth) (NAS, 1977).

Although observed concentrations of specific contaminants range from approximately one part per million to five parts per trillion (lowest detectable level)(EPA, 1978 and 1977), concentration levels are meaningless without knowledge of potency. For example, there is more than a 100 millionfold range in the potency of carcinogens in tests on rodents.[1] This means that one part per trillion of one of the most potent carcinogens can cause as much cancer in rodents as one part per million of one of the weakest carcinogens. Most of our knowledge about relative potency comes from animal studies and, while there is much to be learned about interspecies comparisons, it is worth noting that animal studies have shown that chloroform, the most prevalent carcinogen in drinking water, is only moderately potent in rodents, and many of the chemicals present in much lower concentrations are far more potent. Dieldrin, for example, which is generally present in far lower concentrations than chloroform, is 1,500 times more potent than chloroform in mice (NAS, 1977), and when all rodent species are considered the difference is over 3,000 fold.

Furthermore, the total risk associated with exposure to multiple carcinogens may be far greater than the sum of the risks posed by each chemical individually, due to synergistic interactions between carcinogens. Exposure to promoters might also enhance the carcinogenic effect of chemicals in drinking water compared to the effect of single chemicals in rodent studies. A single promoter has been shown to intensify the effects of a particular carcinogen by a factor of 1,000 (Bingham and Falk, 1969). In addition, the effects of drinking water contaminants may be potentiated by other exposures to carcinogens and promoters (e.g., from food, air pollution and smoking), a particular problem for people living in urban areas or exposed to occupational carcinogens.

Thus, organic contaminants pose a potential threat to health today and in the future. The effects of exposure to carcinogens have a typically long latency; the time elapsing between exposure and clinical symptoms of the disease is often as much as twenty to forty years, depending in some occasions upon the level of exposure. Drinking water contamination has been consistently linked to gastrointestinal(GI) and urinary tract (UT) cancer, although associations with other sites (e.g., lung, brain) have been observed.[1] Mutagens are a suspected

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[1] Based on the work of Sawyer, Hooper, Friedman and Ames, in preparation, cited in McCann, 1977.

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[1] Eleven epidemiologic studies have shown significant associations between cancer mortality, principally GI and UT, and chlorinated or surface waters. A twelfth study failed to identify positive associations, but it was beset by methodological problems, primarily population migration. Letter of Dr. Arthur C. Upton, Director, National Cancer Institute, to Dr. Douglas M. Costle, Administrator, Environmental Protection Agency, April 10, 1978. Chlorinated drinking water was associated with elevated rates of lung cancer in Alavanja, et al., 1977; and Cantor, et al., 1977; and with brain cancer in Cantor, et al., 1977.

causal factor in atherosclerosis; and they are capable of causing subtle biochemical changes, some of which may affect health today in unknown ways, and some of which may not be expressed for several generations. Even less is known about the extent to which drinking water contaminants may contribute to fetal deaths, stunted growth and birth defects, although a recent study suggests this may be a potential problem (McKinney, et al., 1976).

While efforts are continually being made to estimate the risks to human health posed by these chemicals, such estimates are highly uncertain. Both means of determining cancer risks-- animal experiments and human epidemiologic studies--have considerable limitations, and the methodolgies used to establish mutagenic and teratogenic effects may be even less applicable to man than animal cancer tests (NAS, 1977).

Sources of Contamination

The major sources of organic contamination are synthetic organic chemicals which enter the water supply via municipal and agricultural runoff, industrial discharges and chemical spills; and natural organic matter resulting from the decomposition of plants and animals. When chlorine disinfectants are added, they interact with the natural organic matter and form a host of chlorinated and brominated compounds. The four trihalomethanes (THMs)-- chloroform, bromoform, dibromochloromethane and bromodichloromethane--are a particular subgroup of these compounds, and they are usually the most prevalent organic contaminants in chlorinated drinking water so far identified.

Organic contamination primarily affects surface water

(as opposed to ground water), which is the source for most urban areas (there are notable exceptions, e.g., Miami). Although the proposed regulations distinguish between THMs and other organics, contaminated surface waters are likely to contain certain amounts of both types of organics.

Regulatory Approach

The proposed regulations consist of two parts, addressing both types of organics:

1. A maximum contaminant level of 100 parts per billion (ppb) was proposed for THMs, since they are present in the greatest concentrations and can be easily quantified.

2. The treatment technique of GAC adsorption, or its equivalent, will be required to reduce synthetic organics as a group. Because they are so numerous and because their concentrations and chemical characteristics make detection difficult, regulation of individual chemicals was considered infeasible.

The THM standard can be met in a variety of ways. Depending on the types and amounts of natural organic matter, THMs may be reduced by using alternative disinfectants; by chlorinating after coagulation, sedimentation, and sand filtration have removed the natural organic precursors; or some combination of these. These solutions are relatively inexpensive compared to

GAC. For water supply systems with 250ppb or more of THMs, however, GAC adsorption may also be necessary. GAC has the additional advantages of removing synthetic organics and natural organics other than THMs, as well as unpleasant tastes and odors resulting from organic contaminants and their interactions with chlorine.

The proposed regulations are to be applied initially to water systems serving populations of 75,000 or greater. Administrative and economic considerations were the primary reasons for this decision. Only 1 percent of public water systems are in this size range (390 out of 40,000), yet they serve approximately 52 percent of the population (EPA, 1978). Lowering the size of communities subject to the regulations would greatly increase the number of systems involved and thus increase the difficulty of monitoring. Also, due to economies of scale, GAC adsorption is relatively more expensive (on a cost-per-gallon treated basis) for smaller systems than for larger systems.

Of the 390 municipal water systems subject to the proposed regulations, an estimated 121 will not meet one or both of the proposed standards (TBS, 1978). Of these systems, 61 will be required to install GAC: 11 to meet the THM standard, 35 to meet the synthetic organics standard, and 15 to meet both standards.

III. BENEFITS OF REMOVING ORGANIC CONTAMINANTS FROM DRINKING WATER

Removal of organic chemical contaminants from drinking water has been associated with the following benefits:

* Prevention of cancer death
* Prevention of non-fatal cancer illness
* Prevention of mutagenic, teratogenic and fetotoxic effects
* Reduction of the anxiety caused by knowing that drinking water poses risks to health[1]
* Improvements in the taste and odor of tap water

While some of these benefits would be expected to accrue immediately after GAC is installed, their full impact would not be realized until some future time. Since an individual exposed to a carcinogen may bear an increased risk of cancer throughout his lifetime, the benefits of GAC may not be fully realized until all persons exposed to drinking water contaminants are no longer living, assumed in this study to be 70 years.

The number of non-fatal cancer illnesses which might be prevented annually will depend upon the extent to which various cancer sites are affected. As discussed below, this number may be as much as one-half the number of deaths which might be prevented annually, although this estimate is highly uncertain. The extent to which mutagenic, teratogenic and fetotoxic effects will be prevented is unknown.

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[1] Anxiety is listed separately because under both the willingness-to-pay and the lost earnings approaches this component of cost is often excluded. See Appendix D.

The Risk of Cancer Posed by Organic Contaminants
In Drinking Water

Cancer causes approximately 365,000 deaths annually in the United States, and about 650,000 new cases of cancer (excluding skin cancer) are detected each year. Gastrointestinal (GI) and urinary tract (UT) cancers, to which organic drinking water contaminants have been most consistently linked, comprise about 30% of total cancer illness and death, or about 200,000 new cases and 115,000 deaths per year.[1] While cancer survival rates vary with site and over time, and data limitations make estimates of survivors imperfect, an estimated 60,000 (30%) of the 200,000 annually detected cases of GI and UT cancers would ultimately be non-fatal,[2] if present survival rates continue (see Appendix C). The annual number of newly detected non-fatal GI and UT cancers thus appears to be about half the annual number of deaths from these cancers.

While the extent of excess cancer associated with organic contaminants is unknown, considerable evidence is available which suggests the range of possible risk. (See Appendix B for a discussion of the methods and limitations of human risk assess-

ment.) Since this study will consider the expected benefits and costs involved with the reduction in cancer mortality after the removal of THMs and synthetic organics, an attempt was made to select risk estimates reflecting the range suggested by this body of evidence, from animal and short-term tests, as well as epidemiologic studies.
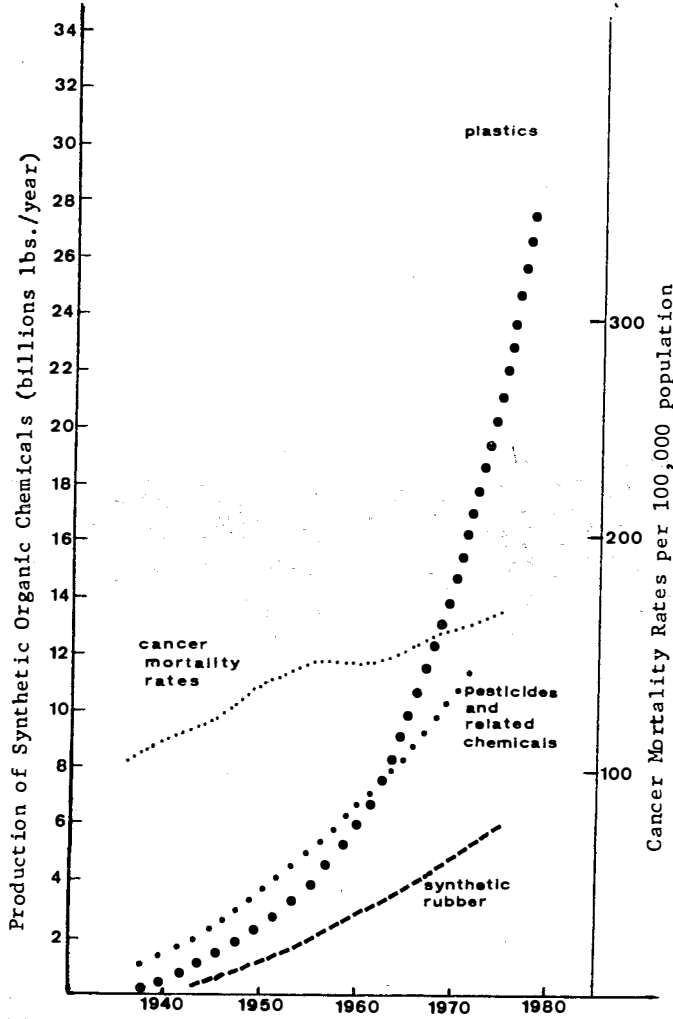
Unfortunately, the epidemiologic studies which relate excess cancer mortality to the sources of drinking water (surface, ground, etc.) do not distinguish the possible effects of THMs from those of synthetic organics which may have been present. While water has been chlorinated since approximately 1910, many synthetic organics are of recent origin. During the "chemical revolution" of the past 30 years, the annual production of synthetic organic chemicals increased from approximately 5 billion to 50 billion pounds per year. Given a probable 20- to 40-year latency for most chemical carcinogens, it is likely that most of the effects of synthetic organics are not yet expressed in total U.S. cancer rates (see Figure 1). Those synthetics which have been present long enough to affect current cancer rates may have had minor impact in comparison to the THMs (Harris, et al., 1977). Hence currently observed excess cancers demonstrated in epidemiologic studies may be primarily the result of chlorination by-products and may not reflect the risk from exposure to current levels of synthetic organics.

Chlorinated and brominated organic compounds, including those which form following chlorination and those

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[1] These figures were based on the following data: (1) estimated resident U.S. population of 217,599,000 as of April 1, 1978 (source: U.S. Bureau of Census, Estimate of Population of the United States to April 1, 1978, Series P-25, No.724); (2) annual incidence rates of 3000 per million population for all cancer, and 929 for GI and UT cancer (source: NCI, 1976); and (3) annual mortality rates of 1673 per million population for all cancer, and 536 for GI and UT cancer (source: NCHS, 1977).

[2] Survival rates tend to stabilize between 5 and 10 years after detection of the disease. For individuals surviving after 10 years, the disease could be considered to have been non-fatal.

Figure 1.

CANCER MORTALITY RATES AND CHEMICAL
PRODUCTION AS A FUNCTION OF TIME

Source: Harris, R.H., T. Page and N.A. Reiches, "Carcinogenic Hazards of Organic Chemicals in Drinking Water," in Origins of Human Cancer, ed. H.D. Hiatt, J.D. Watson and J.A. Winsten, Cold Spring Harbor Laboratory, Cold Spring Harbor, New York, 1977.

which are man-made, are not naturally found in mammalian meta-
bolism. Since their chemical properties make them or their
metabolic intermediates prone to interact with genetic material
(DNA), it is perhaps not surprising that a high percentage
of these compounds have been shown to be carcinogens and/or
mutagens (Ames, 1977).

Chlorination by-products form continuously over time after
chlorination, and it is not possible to make unambiguous state-
ments about the mix of compounds to which the public is exposed.
The concentration and mix of THMs to which the public is
exposed is also dependent upon the manner in which the water is
used and processed in the home (heated or boiled for cooking,
tea, coffee, etc.), as suggested in Table 1. In general,
however, it appears that THMs constitute only 1/10 to 1/5 of
the total chlorinated organic compounds (see Table 2). The
non-THM fraction has yet to be adequately characterized.
Knowledge about chlorination by-products is sparse and may be
briefly summarized as follows:

1. Only one of these compounds, chloroform (a THM), has
   been adequately studied in animal cancer tests, and
   it is a moderately potent carcinogen in rodents (see
   Figure 2).

2. A short-term cancer test (lung adenoma assay) for
   several drinking water contaminants, including all
   four THMs, showed unambiguously positive results only for

Table 1

Percentage Increase or Decrease of Three THMs Above or
Below Levels Present in Cincinnati Cold Tap Water

Compound	Cold Tap	Boiled 5 Sec.	Boiled 30 Min.	Hot Tap
Chloroform	0	(+)96.5	(-)94.0	(+)108.0
Bromodichloromethane	0	(+)72.4	(-)97.2	(+)78.6
Dibromochloromethane	0	(+)121.9	(-)100.0	(+)141.9

Source: Melton, R., et al., "The Analysis of Purgeable Organics
in the Drinking Water of Five U.S. Cities," U.S.E.P.A., June,
1975.

TABLE 2

COMPARISONS BETWEEN TOTAL CHLORINATION BY-PRODUCTS
AND TRIHALOMETHANES (THMs)

Chlorination of Ruhr River Water[1]

Species	Increase (ug/l)
TOCl*	186
Chloroform	6
THM	14

Chlorination of Humic Acid (2 mg/l)[2]

| Species	3.8 mg/l Cl_2 (ug/l)	19.4 mg/l Cl_2 (ug/l)
TOCl*	198	278
Chloroform	39	32

*Total organic chlorine.

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[1]Source: Sontheimer, H., E. Heilker, M. Jekel, H. Nolte and
F.G. Vollmer, "The 'Mulheim Process' -- Experience With a New
Process Scheme for Treating Polluted Surface Water", Journal of
American Water Works Association, in press.

[2]Source: Stevens, A., Environmental Protection Agency,
personal communication, May 26, 1978.

Figure 2

RELATIVE CARCINOGENIC POTENCY
OF CHLOROFORM IN ANIMALS[1]
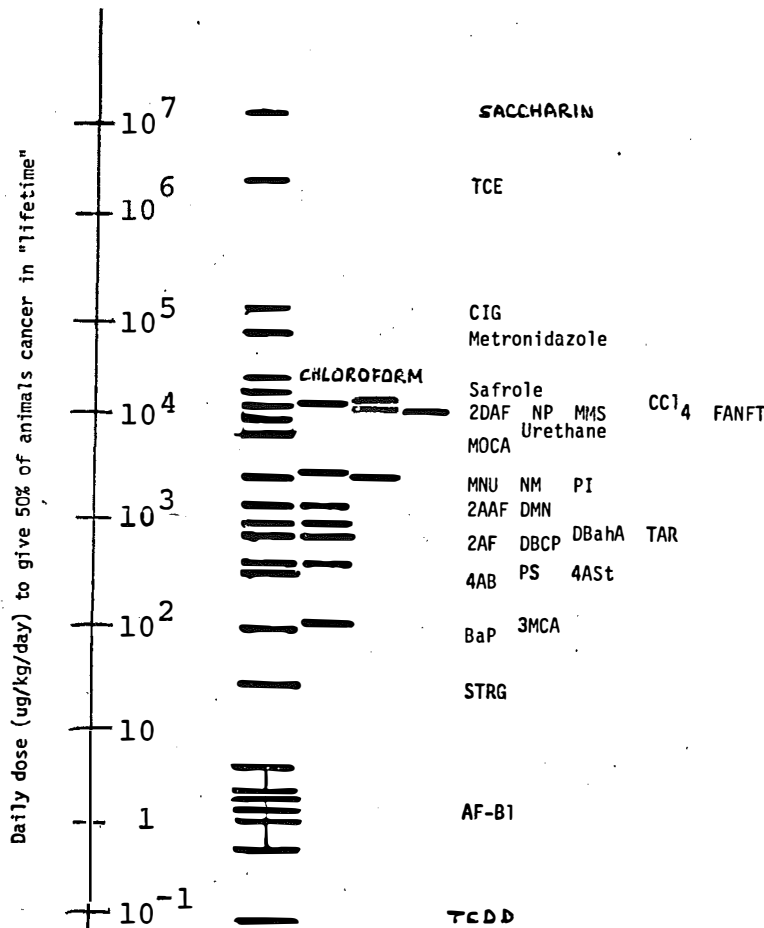
Carcinogenic potency of chemicals in rats and mice. Data calculated
from experiments in the literature by Sawyer, Hooper, Friedman and Ames(unpublished).
(Though it is clear that there is a million-fold range in carcinogenic potency,
the exact location of individual points may change slightly as the calculations
are refined.) AF-B1= aflatoxin B1, STRG= sterigmatocystin, BaP= benzo(a)pyrene,
3MCA= 3-methylcholanthrene, 4AB= 4-aminobiphenyl, PS= propane sultone, 4ASt=
4-aminostilbene, 2AF= 2-aminofluorene, DBCP= dibromochloropropane, DBahA= dibenz-
(a,h)anthracene, TAR= coal tar, 2AAF= 2-acetylaminofluorene, DMN= dimethylnitrosamine,
MNU= methylnitrosourea, NM= nitrogen mustard, PI= propylenimine, MOCA= 4,4'-
methylene-bis-2-chloroaniline, 2DAF=2,7-diaminofluorene, MMN= methyl methanesul-
fonate, CCl4= carbon tetrachloride, FANFT= N-(4-(5-nitro-2-furyl)-thiazolyl)form-
amide, CIG = cigarete smoke, TCE= trichloroethylene.

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[1]Chloroform, saccharin and TCDD (dioxin) added, Robert H. Harris,
unpublished data (1978).

Source: J. McCann, Summary of testimony given before Senator Edward
Kennedy's Health Subcommittee Hearing on saccharin, June 7, 1977.

bromoform, while chloroform yielded negative results
(Theiss, et al., 1977). This is consistent with the
general observation that brominated analogues are
usually more potent carcinogens than the chlorinated species.
The negative result on chloroform cannot be interpreted
to mean that chloroform is not carcinogenic, since the
lung adenoma assay is a relatively insensitive test
for chemical carcinogens.

3. Short-term mutagenicity tests (Ames test) demonstrated
   that all THMs except chloroform were mutagenic and
   therefore probably carcinogenic (Simmon, et al., 1977).[1]

4. Ames tests conducted on the non-THM component demon-
   strated substanial mutagenic activity in comparison
   to the same water before chlorination (Hooper et al.,
   1977).

5. Similar studies have demonstrated mutagenic activity
   of raw surface waters (Pelon, 1976) and of organic
   non-THM concentrates of finished drinking water
   (Loper, et al., 1977). Although precise estimates have
   not been made, the mutagenic activity of the non-THM
   fraction would appear to be in the same range as the
   mutagenic activity of the THM fraction.

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[1] It must be noted that the Ames test does not yield positive
results for several heavily chlorinated compounds shown to be
carcinogenic in animal tests, including chloroform, carbon tetra-
chloride and dieldrin (McCann, et al., 1975).

Therefore, chloroform may be the "tip of the iceberg"
with respect to the cancer hazard posed by chlorination. Chloro-
form appears to be only a moderately potent carcinogen, and if
it is a mutagen, it may be only weakly so in comparison to the
other THMs. It thus appears that the carcinogenic potential
of THMs is likely to be much greater than that of chloroform
alone; indeed, chloroform may be of lesser importance than the
other THMs even though it is usually present in higher concen-
trations. Furthermore, the non-THM, chlorinated component is
clearly mutagenic and probably carcinogenic. This evidence
suggests that the cancer risk resulting from chlorination may
be grossly underestimated if the risk estimate is based on
chloroform alone.

Risk Estimates Based on Animal and Epidemiologic Evidence

As noted above, both animal and epidemiologic studies have
limitations for use in human risk estimates. (These limitations
are discussed more fully in Appendix B.) Animal tests yield
varying results and are beset by uncertainty in converting to
human risk estimates. Epidemiologic studies cannot control
for all possibly confounding factors and, in the case of
drinking water, suffer from the absence of exposure data several
decades ago. Also, only case-control studies, as opposed to
ecologic (whole population) studies, are generally considered
appropriate for yielding precise risk estimates, although
ecologic studies can be interpreted in a manner which sheds

some light on risk. Only one case-control study has so far been completed on the association between organic drinking water contaminants and GI and UT cancer (Alavanja, et al., 1977).

Estimating risk based on animal studies is constrained by the paucity of data on the carcinogenicity of chemicals identified in drinking water; less than 10 percent of these chemicals have been tested for their potential carcinogenicity. Furthermore, there is no generally agreed-upon method for extrapolating risk from animal experiments to humans. Despite these limitations, risk estimates were made for those few carcinogens in Miami and New Orleans drinking water (both highly contaminated) for which animal data and monitoring data were available. Two generally accepted and conservative (yielding higher risk estimates) methods of risk estimation were used: (1) a linear surface-area method used by the NAS drinking water committee (NAS, 1978), and (2) a linear lifetime accumulated-dose method recommended by the NAS pesticide committee (NAS, 1975). This latter method is based on a study of six human carcinogens, while the former is based on theoretical and empirical considerations of chemical carcinogenesis and mammalian metabolism.

The total risk estimations for New Orleans (Table 3) are approximately 23 and 102 cancers/million population/year; for Miami (Table 4) they are approximately 19 and 34 cancers/million population/year, using the surface-area and lifetime-dose methods, respectively. It must be emphasized, however, that these risk

Table 3

Cancer Risk Estimation for New Orleans Drinking Water for those Carcinogens Identified in EPA Survey[a] Using HEW Surface-Area Method and NAS Lifetime Accumulated Dose Method

Carcinogen	Risk to most Sensitive Animal				Concentration in Drinking Water (ppb)	Risk Estimates CA/million/yr	
	Species	Dose (mg/kg/d)	Exposure (weeks)	Tumor Incidence (%)		Surface Area Method	Accumulated Dose Method
1,2-Dichloroethane	Rats[b]	34	78	30	8	0.3	1.6
Hexachloroethane	Mice[b]	421	78	32	4.3	0.0	0.1
Tetrachloroethylene	Mice[b]	382	78	55	0.2	0.0	0.0
Dieldrin	Rats[c]	0.005	104	36	0.07	13.2	83.8
Chloroform	Mice[d]	51	96	22	200[f]	9.7	15.3
1,1,2-Trichloroethane	Mice[b]	279	78	89	8.5	0.2	0.6
DDE	Mice[b]	15	78	43	0.05	0.0	0.0
Bis(2-chloroethyl)ether	Mice[e]	33	78	72	0.16	0.0	0.1
					Total Risk Estimations:	23.4	101.5

a. Analytical Report: New Orleans Area Water Supply Study, EPA, Dec. 9, 1975.
b. NCI Bioassay Program Report.
c. Stevenson, D. E., et al., Tox. Appl. Pharm., 36, 247 (1976).
d. Roe, F. J. C., Unpublished Report (1976).
e. Innes, J. R. M., et al., JNCI, 42, 1101-1114 (1969).
f. Based on EPA analysis of samples from distribution system, 1978.

Table 4

Cancer Risk Estimation for Miami Drinking Water for those Carcinogens Identified in EPA 80-city Survey[a] Using HEW Surface-Area Method and NAS Lifetime Accumulated Dose Method

Carcinogen	Risk to most Sensitive Animal				Concentration in Drinking Water (ppb)	Risk Estimates CA/million/yr	
	Species	Dose (mg/kg/d)	Exposure (Weeks)	Tumor Incidence (%)		Surface Area Method	Accumulated Dose Method
Vinyl Chloride	Rats[b]	5.4	104	15	5.6	0.6	2.6
Vinylidene Chloride	Rats[d]	1.2	82	18	0.1	0.1	0.6
Hexachloroethane	Mice[c]	421	78	32	0.5	0.0	0.0
Tetrachloroethylene	Mice[c]	382	78	55	0.1	0.0	0.0
Trichloroethylene	Mice[c]	1169	78	47	0.2	0.0	0.0
Dieldrin	Rats[e]	0.005	104	36	0.002	0.4	2.4
Chloroform	Mice[f]	51	96	22	366	17.8	28.4
				Total Risk Estimations:		18.9	34.0

a. Preliminary Assessment of Suspected Carcinogens in Drinking Water: Report to Congress, U.S. EPA, Dec., 1975.
b. Maltoni, C., in Origins of Human Cancer, Cold Spring Harbor, p. 143, 1978.
c. NCI Bioassay Program Report.
d. Maltoni, C., et al., Estratto da La Medicini del Favors, vol. 68, no. 4, 1977.
e. Stevenson, D. E., et al., Tox Appl. Pharm., 36, 247 (1976).
f. Roe, F. J. C., Unpublished Report (1976).

estimates are both incomplete and possibly misleading; the concentration of each carcinogen listed in Tables 3 and 4 is based on limited sampling data and there were present in both waters several additional carcinogens (see Table 5) for which either the exposure or carcinogenicity data were not available.

Based on the lifetime accumulated dose method, which produces the higher risk estimate, it can also be calculated that for a THM level of 250ppb, a THM level above which most utilities would probably require GAC to meet EPA's proposed THM standard of 100ppb, the risk estimate would be approximately 20 cancers/million/year. This contrasts with a risk estimate of 12 cancers/million/year using the estimate of the NAS drinking water committee, and 1.4 cancers/million/year using CWPS's estimate. These all assume that the aggregate THM's are equivalent in potency to chloroform, an assumption which is likely to result in an underestimate of the risk since, as discussed above, the brominated THM's are likely to be more potent carcinogens than chloroform.

Epidemiologic studies suggest substantially higher risks from drinking chlorinated water.[1] In a recent policy statement, the National Cancer Institute (NCI) referred to one ecologic study which implied that reducing chloroform by 250 ppb would result in percentage reductions in combined large intestine and bladder cancer mortality equivalent to approximately 45-110 deaths

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[1] It should also be noted that the epidemiologic studies have focused on risk of mortality while the animal studies have focused more, though not entirely, on incidence. However, the difference between mortality and incidence is relatively small (roughly a factor of two) compared with the range of risk estimate (one or two orders of magnitude). See Appendix C.

TABLE 5

SELECTED CARCINOGENS* AND MUTAGENS FOUND IN 11-CITY SURVEY

Compounds	New Orleans	Miami	Seattle	Ottumwa, Iowa	Phila.	Cincinn.	Tucson	N.Y.C.	Lawrence	Grand F.	Tr. Par.
*BENZENE	X			X	X	X					
*CARBON TETRACHLORIDE	X	X		X	X	X		X	X	X	X
*BIS(2-CHLOROETHYL)ETHER	X				X						
*CHLOROFORM	X	X	X	X	X	X	X	X	X	X	X
*1,2 DICHLOROETHANE	X	X			X	X					
*DIELDRIN	X	X	X	X		X					
*DDT, DDE	X										
*HEPTACHLOR	X										
*HEXACHLOROBENZENE	X										
*HEXACHLOROCYCLOHEXANE						X					
*LINDANE (γ-BHC)						X			X		
*PCB						X					
*TETRACHLOROETHYLENE	X	X		X	X	X	X	X	X	X	
*TRICHLOROETHYLENE	X	X		X	X	X			X		
*VINYL CHLORIDE		X			X						
BROMODICHLOROMETHANE	X	X	X	X	X	X		X	X	X	X
CHLOROBENZENE	X	X	X	X	X	X		X	X	X	X
CHLOROMETHYLETHER	X										
DIBROMOCHLOROMETHANE	X	X	X		X	X	X	X	X	X	X
1,3 DICHLOROBENZENE	X	X			X	X			X		
DICHLOROIODOMETHANE	X	X			X	X		X	X	X	X
METHYLENE CHLORIDE	X	X	X	X	X	X		X	X	X	X
VINYLIDENE CHLORIDE	X	X			-X	X			X		

per million population per year.[1] Another ecologic study using a slightly different data base showed significant associations with additional cancer sites, implying that the excess deaths might be somewhat greater (Cantor, et al., 1977).

Two ecologic studies involving 88 Ohio and 64 Louisiana counties suggested that contaminated surface water was responsible for approximately 8% and 15%, respectively, of the total cancer mortality rate.[2] Given the total United States annual cancer mortality rate of about 1673 per million, these studies imply that contaminated surface water similar to surface water in these two states may be responsible for between 135 and 250 cancer deaths per million annually. Although there is insufficient data on the quality of the water included in these studies to determine the average exposure to the communities involved, it appears that the risk estimates suggested by these studies are not unreasonable for drinking water with 250 ppb of THMs as well as synthetic organic chemicals of industrial origin. Data on

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[1] Letter of A.C. Upton to D.M. Costle, April 10, 1978. As the letter noted, one ecologic study implied that reducing chloroform by 100 ppb would result in the following percentage reduction in cancer mortality: bladder, 1.3%-7.5% for males and 5.3%-10.0% for females; large intestine, 4.0%-8.5% for males and 3.0%-7.5% for females. To calculate the reduction in cancer mortality from a 250 ppb reduction of chloroform, these percentages were multiplied by 2.5 and then applied to the U.S. cancer mortality rates, per million population: bladder, 61.7 for males and 21.0 for females; colon/rectum, 222.0 for males and 231.0 for females (NCHS, 1977).

[2] See Page, et al., 1976, and Harris, et al., 1977. The percentages given in those sources cover a broader range, but they refer to specific sex and ethnic groups.

Cincinnati tap water from the Ohio River (taken prior to the alteration of chlorination procedures) indicate that THM levels ranged from approximately 70-280 over the course of the year, while a one-time reading taken in the fall from the treatment plant for New Orleans indicated THM levels of approximately 140 ppb, and summer readings taken from the distribution system ranged from 170-250 ppb.[1]

A 1977 case control study of cancer rates in seven New York counties (Alavanja, et al., 1977) indicated that urban areas served by chlorinated water supplies have combined GI and UT cancer rates 2.7 times higher than urban areas with non-chlorinated supplies; in rural areas, the cancer rate is 1.8 times higher for chlorinated supplies. (It is not known to what extent these higher rates are associated with the level of chlorination by-products, other drinking water contaminants, or possibly other unforeseen factors.) Using the nationwide combined GI and UT cancer rate of 536 per million as an estimate of the rate for areas served by chlorinated water supplies, the above findings suggest that 238-335 excess deaths per million population annually are associated with chlorinated drinking water.[2] These estimates, together with the estimates derived above are summarized in Table 6.

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[1] Data on Cincinnati tap water were provided by A. Stevens, Environmental Protection Agency, Cincinnati, Ohio, personal communication, July 11, 1978; data on New Orleans were taken from EPA, 1975 (fall reading) and B. Lykins, Environmental Protection Agency, Cincinnati, Ohio, personal communication, July 12, 1978 (summer readings).

[2] The numbers inferred from the above studies are not precise, since the cancer data are based on age-standardized rates.

TABLE 6

SUMMARY OF RISK ESTIMATES

FROM ANIMAL AND EPIDEMIOLOGIC STUDIES

ANIMAL MODELS

| | Cancers/Million/Yr | |
	Surface-Area Method	Lifetime Accumulated Dose Method
New Orleans	23	102
Miami	19	34
Chloroform at 250 ppb	12	20

EPIDEMIOLOGIC MODELS

	Cancer Deaths/Million/Yr
Ohio (Surface vs. Ground)	140
Louisiana (Miss. R. vs Ground)	250
Chloroform 80-City Survey (250 ppb)	45-110
New York Counties (Chlor. vs Non-chlor.)	240-340

Selection of Risk Estimates for This Study

The epidemiologic studies suggest risks between one and two orders of magnitude greater than those suggested by animal studies for chloroform alone. In both the NAS and CWPS studies epidemiologic evidence was omitted. But in a submission to EPA concerning the proposed regulations, the National Cancer Institute incorporated risk estimates based upon an epidemiologic study. To be conservative all the available information must be considered. In fact there are some suggestions that the epidemiologic studies are more reliable than the animal studies:

1. Epidemiologic studies reflect the combined effect of all the chemicals in the water, including promotional and interactive effects; such effects are missed in animal studies based on single chemicals. Epidemiologic studies include the effects of yet unidentified carcinogens, while animal studies deal with a far smaller number of identified chemicals.

2. There is a large disparity between animal doses and human exposures--a 3000 to 4000 fold difference in the case of chloroform. The range of extrapolation for chloroform in water is greater than for a similar extrapolation leading to the risk assessment of chloroform in toothpaste or cough syrup, for example, and makes the risk assessment for the effects of chloroform in drinking water less certain than for extrapolations over a shorter range.

3. Confidence in the risk estimates derived from epidemiologic studies increases when consistent results are obtained from repeated suudies. To date, approximately 12 epidemiologic studies have shown a reasonably consistent pattern of association between GI and UT cancer mortality rates and drinking water contaminants, particularly when the power of the statistical methods used in the epidemiologic studies are considered (Harris et al. 1977).

At the same time there are some suggestions that the epidemiologic studies may be less reliable than the animal studies:

1. Epidemiologic studies, especially ecologic ones, cannot control for all possible confounding variables. This limitation might lead to either too high or too low estimates of risk.

2. The data are likely to incorporate more errors of measurement than in the laboratory conditions of animal studies.

3. Epidemiologic studies are to some extent weakened by migration, which tends to randomize the results (and decrease observed levels of significance).

It is not the purpose of this study to referee between the two sources of information and conclude that one is so much better than the other that one should be used and the other discarded entirely. A conservative approach suggests that a range of risk estimates be used, with the epidemiologic estimates at the higher end and the animal studies at the lower end. As noted, there is about one order of magnitude difference between the risk estimate based on lifetime accumulated dose (20) and the risk estimate based upon the

epidemologic studies of the Mississippi River and chlorinated drinking water in New York State (300). Realistically this is about as narrow a range as could be estimated, given the uncertainties associated with each method of risk assessment discussed above. From this perspective the estimates derived from the two methods (20 and 300) are well within the bounds of uncertainty and the two methods are not incompatible.

It may also be noted that the high estimate may be oriented slightly toward organics besides THMs (including THMs as well) to the extent that the epidemiologic studies have picked up the effects of exposure to other chemicals as well as THMs (as mentioned previously many of the non-THMs are of recent origin and may not be reflected in the epidemiologic studies). And the low end of the estimate may be oriented toward the effects of THMs, as the low estimate is based upon chloroform and the assumption that other THMs are in the aggregate as potent as chloroform. Control for the THM standard is less expensive but does not insure against other organics which might be incorporated in the high risk estimate. On the other hand GAC removes a broad spectrum of organics and includes an insurance aspect, by controlling not only organics existing 20 to 40 years ago and hence included in the high risk estimate, but also newer organics which were not included in the epidemiologic estimates of risk.

Thus neither is the 20 a lower bound nor the 300 an upper bound on the risk assessment. Reliance on the "most probable"

risk based on the surface-area method would have produced an estimate of approximately 1 cancer per million (CWPS's estimate). There appears to be no decisive scientific basis to choose between the two NAS recommended extrapolation techniques, so that the higher estimate (leading to 20) was chosen to be on the conservative side and to reflect the likelihood that the brominated THMs are considerably more potent (on the order of 10-100 times) than chloroform; and that the THMs may represent as little as 10 percent of the total chlorinated organics resulting from chlorination.

Therefore the risk estimates selected for this study are:

Level of Risk	Annual Number of Excess Cancer Deaths Per Million Population
Low	20
Medium	150
High	300

Since the average annual cancer mortality rate is about 1673 per million population for all cancers and 536 for GI and UT cancer, these low, medium and high risk factors would imply that drinking water carciongens are responsible for about 1%, 9% and 18%, respectively, of total cancer mortality, or 4%, 28% and 56% of GI and UT cancer mortality, in those communities whose drinking water has the above characteristics.[1]

The low (20 deaths per million) estimate of risk is based on the experimental animal (mouse) most sensitive to chloroform; the medium (150 deaths per million) estimate of risk is a midpoint between the low and high estimates; and the high (300 cancer

[1] These percentages are based upon current cancer rates. If total, GI, and UT cancer rates double in the next forty years, these estimated percentages would halve.

deaths per million) is based on the epidemiologic studies of chlorination in New York State and of Mississippi River water in Louisiana. In neither of these latter studies was it possible to separate the risk from exposure to THMs from the risk from exposure to synthetic organics. In the New York study, the apparent risk from chlorinated water may be confounded by the association of chlorination with the presence of synthetic organics; in the Louisiana study, both THMs and other synthetic organics were present in the analysis conducted in 1974 (EPA, 1975). The extent to which they were present in the 1930s and 1940s, and thus expressed in the 1950-1969 cancer rates upon which the epidemiologic studies were based, can only be speculated. However, given the recent origin of most synthetic organics, and the 20- to 40-year lag before the effects of newly introduced chemicals would be reflected in cancer mortality rates, it is quite possible that synthetic organics contribute little to the risk estimates derived from the New York, Louisiana and Ohio studies. The calculated benefits of GAC treatment, which are based on these risk estimates, may reflect only the benefits of reducing chlorination by-products; the benefits of removing synthetic organics would be largely excluded. Therefore, to the extent that synthetic organics pose a health hazard, the benefits of GAC treatment calculated from these risk estimates are underestimated (when GAC is required to meet only the THM standard).

In addition to fatal cancer illnesses, drinking water carcinogens would also be expected to cause non-fatal cancer illnesses. For example, about 51% of bladder cancer victims

survive, as compared to 1% for victims of pancreatic cancer (see Appendix C). If all the excess deaths associated with drinking water were due to bladder cancer, the amount of non-fatal illness would be far greater than would be the case if the excess deaths were due solely to pancreatic cancer. As noted earlier, the annual incidence of GI and UT cancer cases which may be considered non-fatal is about half the number of annual deaths from these cancers. If it can be assumed that excess cancers caused by drinking water carcinogens are GI and UT, and that all of these cancers are increased to the same degree, then drinking water carcinogens may be responsible for an annual number of non-fatal illnesses approximately half the number of excess deaths. However, since it is unknown whether or not these assumptions are justified, any estimate of non-fatal illness associated with drinking water contaminants would be uncertain. Although the cost of non-fatal illness is a substantial fraction of the cost of mortality, the difference in the range of risk estimate, between 20 and 300 per million, has almost certainly a larger impact on the estimated benefits of water treatment than does the inclusion of the benefits of morbidity reduction. For this reason we will not attempt to quantify the cost of non-fatal illness, except to note that it is substantial. A more refined cost-benefit analysis should include this benefit, as far as possible.

IV. THE COSTS OF REMOVING ORGANIC CONTAMINANTS FROM DRINKING WATER WITH GAC

The economies of scale associated with GAC will lead to significant differences in costs (on a per gallon basis) for communities of varying sizes. In EPA's economic impact analysis of the proposed regulations (TBS, 1978), communities of 75,000 or greater were divided into three size categories which reflect the differences in costs, i.e. 75,000 - 100,000, 100,000 - 1 million and 1 million or more. The most representative population size in each category was 92,700, 263,200, and 1,193,000, respectively.

Annual costs to the representative communities (see Table 7) include annual operating and maintenance costs, and initial capital expenditures amortized over the life of the adsorption system, based on how capital expenditures are likely to be financed. Standard costs were estimated on the basis of a 60 day regeneration cycle and 9 and 18 minute contact times, longer contact times being necessary for raw water of poorer quality. High cost estimates incorporate 25% extra in capital expenditures which may be necessary for site-specific construction problems.

In this study, the benefits of GAC will be evaluated for the representative populations in each size category, and it is assumed that the results can be generalized to all communities within the same size category. In order to account for the variations in cost in a moderately conservative manner, the standard costs for the 18 minute contact time will be used. For the representative small, medium and large systems, these annual total costs are $1.4 million, $2.6 million and $10.3 million respectively, resulting in residential water bill increases of $11-23 annually.

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TABLE 7

ANNUAL COSTS OF GAC ADSORPTION (1978 Dollars)

	SYSTEM SIZE (POPULATION SERVED)[1]		
	Small: 75,000 - 100,000	Medium: 100,000 - 1 Million	Large: Over 1 Million
Representative Population	92,700	263,200	1,193,000
Increase in Annual Residential Water Bill[2]			
Standard Cost			
9 minute contact time	$16.20	$10.50	$7.10
18 minute contact time	$23.00	$15.00	$11.40
High Cost (25% extra site-specific capital expenditures)			
9 minute contact time	$18.50	$11.90	$7.90
18 minute contact time	$26.10	$17.00	$12.70
Annual Revenue Requirements for Standard Cost, 18 minute Contact Time	$1,418,310	$2,632,000	$10,259,800

[1] Cost estimates were based on plant production on the average day in the month of maximum production. The production figures used for the representative systems are: small - 20 mgd; medium - 60 mgd; and large - 300 mgd.

[2] For a family of three.

Source: Tables II-4 and III-4 of "Revised Economic Impact Analysis of Proposed Regulations on Organic Contaminants in Drinking Water," Temple, Barker and Sloane, Inc., Wellesley, Massachusetts, July, 1978.

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V. METHODS FOR EVALUATING BENEFITS AND COSTS

Sections III and IV indicate that, primarily because of cancer latency, the distribution over time of the benefits of GAC differs markedly from the distribution of costs over time. When such significant distributional impacts occur, the problem of a fair distribution across time, or intertemporal equity, arises. This problem is especially important when the distributional impact spans several generations, as is the case with GAC, and the problem is intensified when human lives are involved. Accounting for distributional or equity effects in a satisfactory way is no easy matter, either in the intratemporal setting or in the intertemporal setting (see Harberger, and Mishan and Page). In the latter case, where there is the problem of determining the fair distribution of costs and benefits across generations, economists have offered several approaches:

1. Use a zero discount rate. This approach has been recommended by Ramsey (1928), Solow (1974a) and discussed by Mishan and Page (1979) Although highly respected economists have favored this approach, it has received little application in actual cost-benefit analysis partly for the reasons discussed in Page (1977).

2. Use a rate of discount lower than the marginal productivity of capital. A 5 percent discount rate was used by d'Arge in his analysis of the ozone depletion problem. The case of ozone deple-

tion is structurally similar to the case of GAC in that the estimated costs of ozone depletion were delayed nearly a century, due to physico-chemical latencies (low diffusion and reaction rates). In order to be "fair" to the future, d'Arge recommended a low (5 percent) discount rate.[1]

3. Compare steady state costs and benefits. Approximately the first 70 years after the installation of GAC can be considered a "transition period" during which the cancer rates are adjusting downward; in this period, the benefits of GAC are growing while the costs remain relatively constant (in real terms). After 70 years, the ratio of benefits to costs will be fairly steady for the forseeable future. As a way of taking into account the future's perspective, which is "permanent" (or at least longer term than the transition period), evaluation can be based upon this comparison of benefits and costs in the steady state. This is another way of being "fair" to the future's interests.

The steady state approach was used in the NAS and CWPS report on the benefits of removing chloroform from drinking water, and it is used by the Council on Wage and Price Stability (CWPS) in its analyses of the inflation impact of other government regulations. This approach is consistent with the policy of the Office of Management and Budget that benefits associated with the future savings of human life not be discounted in benefit-cost analyses (OMB, 1972).[2]

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[1] As expressed by Solow (1974b), "In social decision-making, however, there is no excuse for treating generations unequally and the time-horizon is or should be very long . . . we ought to act as if the social rate of time preference were zero (though we would simultaneously discount future consumption if we expect the future to be richer than the present)."

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[1] Personal communication, July, 1978. (See also Mishan and Page.)

[2] According to one OMB official, discounting the benefits associated with the saving of future lives is "repugnant to some people" and "politically indelicate". One CWPS official noted that discounting these benefits is "politically sensitive," and the steady state method is used to reflect a "conservative" concern for human health.

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4. Use the overtaking principle. The overtaking principle
holds that if, after a certain period of time, all later gener-
ations would prefer the consequences of one decision to another
(the choice being taken by the first generation, e. g., installing
GAC), that decision should be taken in the initial period. A
cardinal version of this approach is discussed by Weizsacker (1965)
and Wan (1971), among others. An ordinal, and independently
derived, version of this principle is developed by Ferejohn and
Page (1978).

As discussed in the paper by Ferejohn and Page (1978),
adjustment of the discount rate is not a satisfactory resolution
of the discount rate problem, but the overtaking principle can
be, when it is appropriate to apply. The appropriate condition
is that a steady state preference ordering be reached soon in
terms of generational time (as is the case for the drinking
water problem), and under this condition the steady state
comparison and the overtaking principle become the same.

Consideration of the overtaking principle lends support to
the steady state approach taken by both NAS and CWPS and this
approach will be used here as well. At the same time calculations
based on varying discount rates will be offered for comparative
purposes.

Quantitative Analysis

For the discounted benefits and costs, a time-horizon of 100
years was selected in order to capture fully the stream of bene-
fits, which was assumed to rise over 70 years and then remain at
the same level for an infinite length of time. (The difference
between discounting over 100 years and an infinite period is
insignificant.) Costs are in 1978 dollars, corrected for
inflation.

The quantitative analysis of benefits will be limited to
prevented cancer deaths. It must be emphasized that this approach
ignores the several additional benefits of GAC, and thus biases
the results of the analysis against GAC. The rationale for
adopting this approach is pragmatic. If GAC appears cost-justi-
fied when it is assumed that the only benefit is prevented cancer
deaths, then it will not be necessary to assess the value of the
additional benefits. The benefits of reduced anxiety and of
improvements of the taste and odor of tap water would be
particularly difficult to measure and the magnitude of the
additional health benefits is uncertain. Such an assessment would
be necessary, however, if GAC does not appear cost-justified
when only prevented cancer deaths are considered.

In evaluating the benefit of prevented cancer deaths, the
saving of a human life will not be assigned a dollar value. In-
stead, for a variety of cancer mortality and discount rates,
the cost of GAC per life saved will be indicated, allocating the
total costs of GAC to saved lives. If this cost is less than
what seems reasonable to spend in saving a human life, then it
can be said that the benefits exceed the costs, without attempting
to quantify the other identified benefits.

Use of the risk estimates of 20 to 300 excess cancer deaths per million population annually implies that the community for which this analysis most directly applies is one which depends for its water supply on moderately polluted surface water, with resulting THM levels in the distribution system of approximately 250 ppb. Communities with lower THM levels could probably meet the EPA standard of 100 μg/l at considerably lower cost by adjustments in the method of disinfection. Although the epidemiologic studies cannot be used to estimate the risk from exposure to current levels of synthetic organics (discussed above), limited risk estimates for New Orleans and Miami (see Tables 3 and 4) suggest that these risks are in the same range as for the THMs, and may be higher considering the presence of numerous carcinogens and suspected carcinogens (mutagens and structural analogs of carcinogens) for which the data are not available in the form appropriate for risk estimating. Furthermore, for communities that are required to install GAC only for synthetic organics, and not for THMs, benefits will accrue not only from reduction of synthetic organics, but also from concurrent reductions of THMs and other products of chlorination.

It was assumed that GAC would reduce the risk of cancer from drinking water by 90 percent. It must be noted that there is some uncertainty about this estimate. It is impossible to determine the reduction in cancer risk that will result from the reduction in organic contaminants. From studies conducted by EPA, it is clear that GAC is at least 60 percent effective in removing THMs, and it is highly effective, in some cases more than 90 percent, in removing industrial chemicals such as polycyclic aromatic hydrocarbons and polychlorinated pesticides (e.g., dieldrin, kepone) Since the latter are likely to be more potent carcinogens and mutagens than the former, it is likely that GAC will reduce the risk of cancer by at least 60 percent and possibly more than 90 percent.

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Method of Calculation

The costs of GAC per life saved were calculated according to the following steps. First, the total number of excess cancer deaths was calculated by applying each risk factor to the total population of each representative community. For example, for the large community with 1.193 million population, the high cancer risk rate of 300 deaths per million annually would yield 300 x 1.193, or 358, excess deaths annually. Second, the number of lives which would be saved annually after the excess cancer rate had ceased declining (after 70 years) was calculated as 90% of the number of excess deaths, since it was assumed that GAC adsorption would eliminate 90% of the excess mortality. For example, 358 x 90% = 322.

To calculate the costs of GAC per life saved in the steady state, the number of lives which would be saved annually after 70 years was divided into the annual cost of GAC, disregarding expenditures in earlier years when fewer lives per year were saved. To calculate the cost per life saved under discounting, three additional steps were taken.

First, for years 71-100, the number of lives saved per year was the same as under the steady state. For years 1-70, it was assumed that the number of lives saved each year would initially be small, and would rise in the manner described in Appendix E. For the large community, assuming the high rate of cancer risk, the number of lives saved would be 0 in year 5, 51 in year 25, 271 in year 45, and 322 in year 65. Second, the cost of GAC for

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each representative community was discounted using the formula:

$$\sum_{y=1}^{100} \frac{c_y}{(1+r)^y}$$

where c is the annual cost of GAC, r is the discount rate, and y
is the year of the time-horizon over which benefits and costs
were discounted. For example, for the large community the dis-
counted cost of $10,259,800 per year for 100 years, using a
10% discount rate, was $102,590,550. Third, a cost per life
saved was chosen so that when discounted back (at r) and summed, the
total would be equal to the discounted costs of GAC.[1] For the
large community, and assuming the high risk of cancer and
a 10% discount rate, this figure was $545,000.

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[1] This approach incorporates one definition, but not the only
definition, of the intertemporal opportunity cost per life saved
(See Mishan and Page, 1979).
   The calculated sum will differ under various discount rates
due to the differing profiles of costs and benefits.

VI. RESULTS

The results of the quantitative analyses for large, medium
and small communities are presented in Table 8 . The cost per life
saved ranges from about $32,000 to $17.4 million. In 16 out of 36
cases, the cost per life saved is less than $500,000, and it is
about $100,000 or less in 8 cases. In 12 cases the cost per life
exceeds $1 million.

Due to economies of scale, the cost per life saved for the
small community is 1.5 to 2 times that for the large community,
while the cost for the medium community is about 1.1 times the
cost for the large community. The cost per life saved is affected
by the discount rate more than by the assumption of excess can-
cer mortality. For any assumed level of cancer mortality, applying
the various discount rates causes the cost per life saved to vary
by a factor of 17 to 25. For any selected discount rate, or in the
steady state, the assumptions of cancer mortality cause the cost
per life saved to vary by only a factor of 12 to 17.

Large Communities - The cost per life saved ranges from $32,000
to $8.8 million. It ranges from $32,000 to $489,000 in the steady
state and from about $545,000 to $8.8 million under 10% discounting,
depending on which rate of excess cancer mortality is assumed. For
the high rate of excess cancer mortality (300 deaths annually per
million population), the cost per life saved ranges from $32,000
to $545,000, depending on the discount rate; and for the low rate
of excess cancer mortality (20 per million) it ranges from $489,000
to $8.8 million, depending on the method of discounting.

TABLE 8

ALLOCATION OF COSTS OF GAC TO REDUCED CANCER MORTALITY ALONE

System Size and Population[1]	Excess Cancer Mortality Rate (Annual Deaths Per Million)[2]	Cost Per Life Saved (Thousands of $)			
		Steady State	Discount Rate Applied Over 100 Years		
			3%	7%	10%
Large: More than 1,000,000	300	$32	$93	$269	$545
	150	$64	$189	$526	$1,088
	20	$489	$1,434	$4,147	$8,789
Medium: 100,000 – 1,000,000	300	$37	$108	$308	$639
	150	$73	$214	$609	$1,268
	20	$526	$1,552	$4,620	$10,197
Small: 75,000 – 100,000	300	$57	$166	$476	$1,000
	150	$109	$320	$932	$1,994
	20	$709	$2,132	$7,206	$17,474

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[1] Calculations were based on the following representative systems in each size category: Large - population 1,193,000, annual GAC costs $10.3 million; Medium - population 263,200, annual GAC costs $2.6 million; Small - population 92,700, annual GAC costs $1.4 million.

[2] Based on THM concentrations of 250 ppb. It is assumed that treatment by GAC adsorption will reduce the excess cancer mortality rate by 90%.

Medium Communities - The cost per life saved ranges from $37,000 to $10 million. It ranges from $32,000 to $526,000 in the steady state and from $639,000 to $10 million under 10% discounting, depending on the assumption of excess cancer mortality. For the high rate of excess cancer mortality, the cost per life saved ranges from $32,000 to $639,000, depending on the discount rate, while for the low rate of excess cancer mortality, the range is from $526,000 to $10 million.

Small Communities - The cost per life saved ranges from $57,000 to $17.4 million. It ranges from $57,000 to $709,000 in the steady state and from $1 million to $17.4 million under 10% discounting, varying with the assumption of excess cancer mortality. Assuming the high rate of excess cancer mortality, the cost per life saved varies from $57,000 to $1 million, depending on the discount rate, while it varies from $709,000 to $17.4 million for the low assumption of excess cancer mortality.

These results are depicted graphically in Figures 3 through 5.

Figure 3

Communities with Populations of More than 1,000,000:
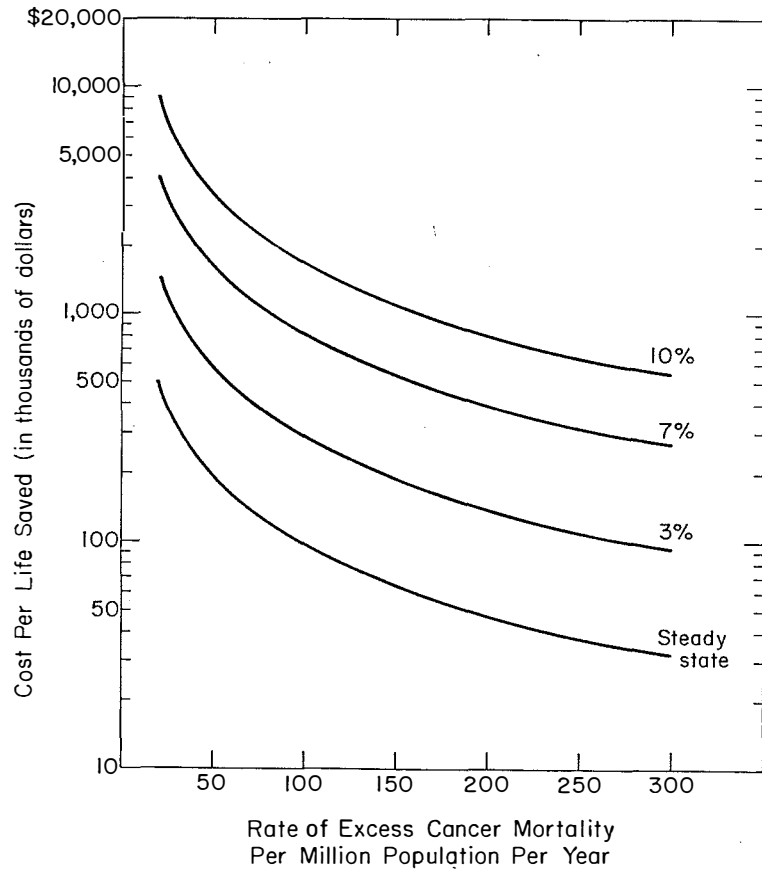### Allocation of Cost of GAC to Reduction in Cancer Mortality Alone

Figure 4

Communities with Populations of 100,000 to 1,000,000:
Allocation of Cost of GAC to Reduction in Cancer Mortality Alone

Figure 5

Communities with Populations of 75,000 to 100,000:
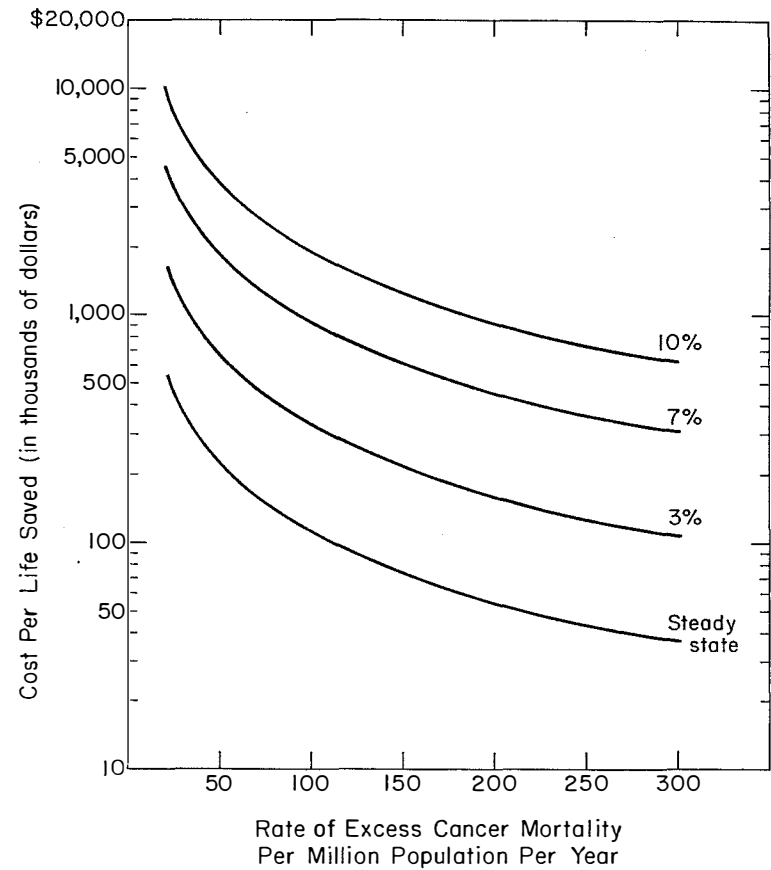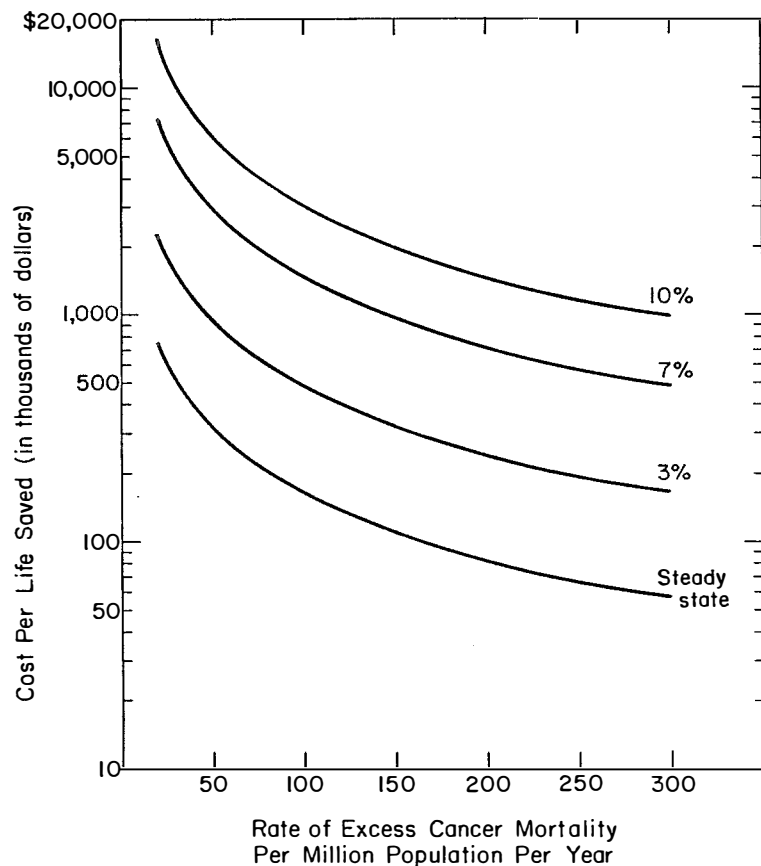Allocation of Cost of GAC to Reduction in Cancer Mortality Alone

Rate of Excess Cancer Mortality
Per Million Population Per Year

VII. DISCUSSION

Setting aside for the moment consideration of the additional
benefits of GAC, it can first be considered whether or not GAC is
cost-justified when it is assumed that the only benefit associated
with the cost of GAC is reduction in cancer deaths. The cost of
GAC per life saved is an amount dependent upon a subjective judg-
ment of the cancer risk associated with organic drinking water
contaminants, and upon social judgments regarding how the interests
of future generations should be weighed relative to the interests
of the present generation. In addition to considering these issues,
decision makers must consider how much should be spent to save a
human life.

It is not possible, of course, satisfactorily to value the
saving of a human life in dollars, hence it is difficult to determine
how much is reasonable to spend in saving a life. However, because
policy decisions frequently involve the saving of lives, several
efforts have been made to determine valuations of human life
which may serve as a practical guide to decision makers. Three
principal methods have been employed to determine such valuations.
(These approaches and their results are more fully elaborated on in
Appendix D.)

The willingness-to-pay approach is most consistent with the
principle that benefits should be valued by the beneficiary himself,
and is hence the approach favored by economists. Following
this approach economists have estimated the extra amount in
wages paid to workers who accept jobs which increase their risk of
death, taken to be the amount workers would be willing to pay to

avoid the risk. This "willingness to pay" for reduced risk is then used to determine how much workers collectively would be willing to pay to avoid one death among them. Two studies utilizing this approach indicated that workers value their lives at $200,000 and $1.5 million, respectively. A similar study indicated that the lives of Air Force pilots were valued at $135,000-$980,000. The figures resulting from these studies can be taken as under-estimates of the cost of cancer mortality because (1) They do not take into account the amount that the individual's family, friends, and society at large would be collectively willing to pay to prevent the individual's death. And (2) people are concerned about the manner of death as well as the time of death. Cancer is now the most dread disease and the willingness to pay to decrease the risk of accidental death understates the willingness to pay to lower the risk of death by cancer (see note 1 on page 11).

The lost earnings approach measures how much a death costs society in lost resources, such as future earning, medical care, family expenses, and property and tax effects. In terms of medical care and lost earnings alone, a cancer death costs society an estimated $70,000; another estimate, which includes additional costs, is approximately $200,000. Highway and air traffic fatalities have been estimated as costing society about $300,000. A major objec-tion to this approach is that it reflects the value of an individual's contribution to the Gross National Product, not the value of his life to himself and others. Surprisingly, the lost earnings approach and the willingness-to-pay one are not greatly different in their estimates.

Some observers have interpreted government safety and health

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regulations as indications of how much society is collectively willing to pay to save a human life, although the cost of saving a life may not have been explicit in the decision making process. Judgments in court cases have also been used as measures of the cost of death and injury. Such evaluations, which vary a great deal, are often considerably higher than the estimates derived from willingness-to-pay or lost earnings approaches.

In assessing the reasonableness of the cost of GAC per life saved, one standard of comparison might be the value which indivi-duals implicitly attach to their lives when undertaking jobs which increase their risk of death, i.e., $200,000 to $1.5 million. In the steady state, GAC would be justified for a risk of 20 deaths or more. Twenty per million is the risk estimate calculated from the accumulated lifetime dose method and thus GAC can be justified without recourse to the epidemiologic data.

Clearly the steady state approach, taking intertemporal equity into account, is more "favorable" to the future, as can be seen in Figures 3, 4, and 5. The highest curves in the figures show net benefits discounted at 10 percent under the assumption that cancer prevention is the only benefit. These curves can be interpreted as calculations omitting consideration of intertemporal equity. The curves labled 3 and 7 percent represent intermediate steps between the two approaches.

Figures 3, 4, and 5 indicate that if three non-conservative assumptions are taken together GAC will not be justified. The non-conservative assumptions are: little or no weight be given

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to the epidemiologic evidence, so that the risk of cancer is assumed low, less than 20 per million; little or no benefit to be assigned to prevention of mutagenic disease, birth defects, and improvements in taste and odor, so that the cost per life saved is totally allocated to cancer prevention; and no consideration be given to intertemporal equity, so that cancer deaths should be discounted over periods of 40 years and more. CWPS and NAS (chapter 8) employ the first two but not the last of the non-conservative assumptions.

An important issue in the evaluation of GAC is how conservative toward the risk of cancer should society be. We believe that a prudent measure of conservatism toward risk would place some weight on the epidemiologic evidence and on other evidence of the mutagenicity and carcinogenicity of non-chloroform, and non-THM organics generally. Nevertheless, as just stated above, an important conclusion to emerge from the calculations of cost per life saved, under varying assumptions, is that justification of GAC need not be based on the epidemiologic evidence at all, nor on the inclusion of other benefits besides cancer prevention. Application of the accumulated lifetime dose instead of surface area adjustment is a relatively small step toward a conservative attitude toward cancer risk, and yet this step is enough to justify GAC.

But benefits other than cancer prevention should be included to the extent they can be quantified, or at least qualitatively considered. For example, some sense of the value of benefits of taste and odor control is indicated by the costs of bottled water and home filtration devices, which are used primarily to produce good tasting drinking water, and which would no longer be necessary if GAC adsorption is installed. Compared to the annual household costs of GAC of $11-23, regular household use of bottled water costs about $65 to $115 per year, depending on geographic region.[1] Home filtration devices cost about $5-12 per year for filter replacement, after initial costs of $20-40.[2] About 40 communities seek good tasting drinking water at lower household costs by using GAC in sand filter beds; the cost of using GAC for this purpose is about 15-20 percent of the cost of using GAC in contactors as a post-filtration adsorbent.[3] Communities which do not already use GAC for improvements in taste and odor may not value these improvements highly enough to justify this cost, but probably value these improvements at somewhere between zero and this cost.

Altogether, these additional benefits may have substantial value, and some portion of the total costs of GAC should be allocated to securing them. Put differently, the cost of life-saving benefits alone will comprise only part of the cost of GAC. For this reason, the cost of saving a life will be less than the costs per life saved discussed above.

[1] Based on consumption of 5 gallons every two weeks (a common minimum order for home delivery), and per gallon costs of 50-90 cents.

[2] Based on recommended retail prices of 3 major brands.

[3] For the three sizes of water treatment plants upon which this analysis is based, the annual costs of using GAC in filter beds for taste and odor control would be: large system (300 mgd) $2,342,800; medium system (60 mgd) $574,000; and small system (20 mgd) $219,500. These costs include the construction of a filter shell, and were based on a 3 year carbon replacement cycle. Source: Environmental Protection Agency, Municipal Environmental Research Laboratory, Cincinnati, Ohio, personal communication, August 23, 1978.

If the value of the additional benefits can be assessed relative to the value of the total benefits, the cost per life saved could be adjusted downward proportionally to determine the cost of saving a life by itself. For example, if the additional benefits were valued at one third of the total benefits, then the cost of saving a life would be one third less than the costs per life saved reported above. This type of adjustment will provide a more appropriate basis for evaluating the cost of saving of life with GAC.

As discussed above, one of the major problems in evaluating GAC is the large range of uncertainty of the cancer risk. As information concerning the nature of the risk is developing, it may at first seem sensible to recommend delay until more is known about the risk, in order to decrease the chance that GAC filtration plants will not be unnecessarily constructed. If the risk of cancer later turns out to be less than 1 or 5 per million, society would have to live with this mistake for the life of the plant (about forty years). However, against this irreversibility one must balance the irreversibilities associated with cancer, mutagenic disease, and birth defects. The costs associated with these diseases are also largely irreversible. The length of irreversibility for cancer is forty or more years, due to its latency period; for mutagenic disease the period of irreversibility is in terms of generations. Thus if the true risk turns out to be 20 or more per million and GAC plants are delayed, society will have to live with this mistake, (the unnecessarily caused diseases generated during this period of delay) for a considerably longer period than the one associated with the irreversibility of GAC plant construction.

In the first few years after a decision to build GAC plants, in the planning and permit stage, the decision to build is reversible at low cost. Once the physical plant is constructed, perhaps five or more years after the decision to implement GAC, the original decision becomes in part irreversible, due to sunk capital costs. Operating and maintence costs, however, remain reversible. In contrast, every year of exposure to carcinogens and mutagens is largely irreversible in ultimate effect. Consideration of irreversibility usually suggests delay until more is known lest highly valued options be irreversibly foreclosed. In the case of drinking water treatment, preservation of option suggests earlier introduction of preventative measures because the potentially more severe irreversibilities are on the side of cancer and mutation. That is, as more information becomes known it is less costly to rectify an irreversible mistake of over protection than one of under-protection.

Taking into account the relative irreversibilities of GAC implementation, in terms of planning, operation and maintenance, and capital construction, and of cancer and other genetic disease, in terms of cure possibilities, Table 8 suggests that a false negative is a more costly mistake than a false positive.[1] Social risk aversion implies a greater avoidance of the potentially more costly mistake than indicated by expected value calculations. Because the calculations of cost per life saved are based on expected values, in this paper[2], consideration of risk aversion adds an additional factor in favor of preventative treatment.

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[1] A false negative would be to delay treatment when cancer and other health costs are very high. A false positive would be to impement GAC when cancer and other health costs are very low.

[2] Expected value calculations are also used in CWPS (1978) and NAS (1978).

VIII. SUMMARY AND CONCLUSIONS

The objective of this analysis was to evaluate the bene-
fits of removing organic contaminants from drinking water by
GAC adsorption. These benefits include:

(1) prevention of cancer deaths,

(2) prevention of non-fatal cancer illnesses,

(3) prevention of mutations and birth defects,

(4) reduction in the anxiety about the health risks of
contaminated drinking water, and

(5) improvements in the taste and odor of tap water.

In evaluating these benefits, particular emphasis was placed on
the risk of cancer, the delayed impact of GAC, the prospect of
uncertain and irreversible consequences, and the value of saving
human lives. In part, because of the paucity of data, the only
benefit of GAC quantitatively considered in the analysis was
the reduction in cancer deaths.

In assessing the cancer risk posed by drinking water con-
taminants, careful thought should be given to the weights placed
on risk assessments from animal and epidemiologic studies.
Extrapolation from animal models are limited by the fact that
only a small fraction of the organic chemicals in drinking
water have been characterized toxicologically. Despite this
limitation, risk estimations using animal data were made for
three cases: (1) the concentration of carcinogens reported
by the EPA to be present in New Orleans Drinking Water

in a 1974 survey of the lower Mississippi, (2) the concentra-
tion of those carcinogens reported by EPA in 1975 to be present
in Miami drinking water, and (3) a chloroform concentration of
250 ppb. The risk estimates for these three cases were approxi-
mately 23-102, 19-34, and 12-20 cancers per million per year,
respectively. The risk estimation for 250 ppb of chloroform
(20 cancer/million/yr.) was taken as the lower bound on the
risk since it was assumed that in the absence of carcinogens
of pollution origin, GAC use under the conditions assumed in
this study (18 min. contact time, two month regeneration frequency)
would probably only be necessary to meet EPA's proposed THM
standard of 100 ppb if chloroform levels were higher than the
200-300 ppb range.

On the other hand, well-conducted epidemiologic studies offer the
potential of comprehensive estimates of risk, since they
measure the human condition, which includes the heterogeneity
in response and exposure to all chemicals; animal models must
be based on only a few of the myriad of organic chemicals
found in drinking water for which data are available. Based on
the epidemiologic evidence, the cancer risk posed by organic
chemicals in drinking water ranged up to approximately 300
cancer deaths per million per year, which was taken as the
upper bound on the risk in the benefit-cost analysis. It is
recognized, however, that epidemiologic studies indicate risks
for exposures 20 to 40 years (the latency period) prior to
the study. Hence, the risk from current levels of contamina-
tion may be underestimated by existing studies. But there are
limitations to the existing epidemiologic studies, as discussed

above, and it is difficult to know how much weight to place
on these higher estimates of risk.

It is also unclear to what extent removing organic
contaminants from drinking water may reduce non-fatal cancer
illness and prevent mutations and birth defects. It is
difficult to attach monetary values to these potential benefits,
as well as to the benefit of reduced anxiety about the health
risks of contaminated drinking water and the benefit of improved taste
and odor of tap water. The difficulties of dealing with these
benefits prompted the pragmatic approach of first examining
the cost-justification of GAC under the assumption that the
only benefit is reduced cancer mortality.

Since the cost of GAC (on a per-gallon basis) will differ
significantly for communities of varying sizes the quantitative
analysis of benefits and costs (assuming the only benefit was
reduced cancer mortality) was based on representative communi-
ties in three population size categories: 75,000-100,000;
100,000-1,000,000; and more than 1,000,000. The cost of GAC
per life saved was determined under three assumptions of the
cancer risk (i.e. 20,150 and 300 deaths per million population
per year) for the steady state and, by way of comparison, for three
discount rates (3,7 and 10%). The resulting cost per life saved
ranged from about $32,000 to $709,000 on a steady state basis.
The costs for medium and small communities were 1.1 and 1.5-2 times
as great, respectively, as the cost for large communities. For
a particular assumption of the cancer risk, the cost varied by
12-17 fold over the range of discounting approaches; for a given

discounting approach, the cost varied by 17-25 fold over the
range of risk assumptions.

To evaluate the cost-justification of GAC when it is
assumed that the only benefit is reduced cancer mortality,
benchmarks must be sought regarding how much is reasonable to
spend in saving a human life. Three possible standards are:
(1) the $200,000-$1.5 million range in value of a human life
implicit in the willingness of workers to accept the risk of
death in hazardous jobs; (2) the $200,000 that a cancer death
costs society in productive resources, and (3) the $1 million
and larger amounts implicit in other government programs and
explicit in court settlements. Each has shortcomings. The
willingness-to-pay figures do not incorporate the value of the
individual to his family, friends and society at large; the
cost in productive resources does not include the individual's own
evaluation of the risk of early death by cancer; and the cost of
government regulations may not have been explicitly taken into
account when the regulations were adopted. In the absence of
other criteria, however, these figures have some usefulness.

Conclusions

1. Using the steady state method of analysis, the
$32,000 to $709,000 cost of GAC per life saved is considerably
less than that of similar health programs undertaken by the
federal government, also evaluated on a steady state basis, and
is thus a relative bargain, especially for the larger cities
and for more polluted water sources, where the cost of GAC per
life saved is lowest.

2. Imprecise knowledge about the health risks of organic contaminants in drinking water and about the effectiveness of GAC in reducing these risks creates uncertainties which weigh both for and against GAC. On the one hand, the effectiveness of GAC in reducing the health risks posed by organic contaminants may not be as great as the 90% assumed in this study, although it is unlikely to be less than 60% under the operating assumptions used in this analysis. On the other hand the attractiveness of GAC is enhanced by the protection it provides against the worst possible hazards of organic contaminants; this is particularly so with respect to irreversible damage to the gene pool, which, if it were to occur, would impose an additional cost on a decision not to install GAC today.

3. While it is difficult to know how much weight to place on each of the two basic approaches to risk assessment, extrapolations from animal studies and human epidemiology, a decision in favor of GAC and other means of protection against toxic chemicals in drinking water does not depend on the resolution of this issue. Even if the epidemiologic evidence were set aside completely, GAC is justified on the basis of animal studies using the accumulated lifetime dose technique. A conservative approach toward cancer risk strongly suggests placing some weight on the epidemiologic studies. This strengthens the decision in favor of GAC and other preventive measures.

CARCINOGENS, MUTAGENS AND PROMOTERS FOUND IN UNITED STATES DRINKING WATER

Carcinogens and Suspect Carcinogens

Benzo(a)pyrene	Vinylidene chloride
Carbon tetrachloride	Heptachlor
Chloroform	1,1,2-Trichloroethane
Vinyl chloride	1,1,2-Trichloroethylene
1,4-Dioxane	Bis(2-chloroethyl)ether
Methyl iodide	Simazine
DDE	Tetrachloroethylene
DDT	Heptachlor expoxide
Chlordane	Acrylonitrile
Lindane	Aldrin
Dieldrin	Butyl bromide
Benzene	

Mutagens and Suspect Mutagens

1,1,1-Trichloroethane	Dichloroacetonitrile
Bromomethane (methyl bromide)	Methylene bromide
Methyl chloride	Chlordane
Bromochloromethane	Vinylidene chloride
Methylene chloride	n-Butylbromide
Bromoform	Bis(2-chloroethyl)ether
Bromodichloromethane	Acrylonitrile
2-Chloropropane	Benzo(a)pyrene
1,2-Dichloropropane	Methyl iodide
1-Chloropropene	Vinyl chloride
1,2-Dichloroethane (recently shown to be carcinogenic by NCI)	1,3-Butadiene
Bis(2-chloroisopropyl)ether	1,2-Bis(chloroethoxy)ethane
Chlorodibromomethane	Pyrene
1,3-Dichloropropene	1,1,2-trichloroethylene
2,6-Dinitrotoluene	Tetrachloroethylene (Perchloroethylene)

List of Promoters

Ortho-Cresol	n-Decane
2,4-Dimethylphenol	Limonene
Phenol	Octadecane
n-Dodecane	n-Tetradecane
Eicosane	n-Undecane
2,4-Dichlorophenol	

Source: National Cancer Institute, May 1978.

APPENDIX B

METHODS OF ESTIMATING CARCINOGENIC RISKS TO HUMANS

The extent to which cancer is caused by organic contaminants
in drinking water is extremely difficult, if not impossible, to
determine. The three principal means of assessing cancer risks
all have considerable limitations.

Animal Cancer Tests

Suspected carcinogens are administered to animals, usually
rodents, over a period of two to three years. Based on the number
of tumors resulting and the dose levels, estimates of human risk
are made. For statistical reasons, higher doses are often re-
quired in animal studies than humans experience. Therefore, vari-
ous models have been developed for extrapolation of animal risk to
lower doses, and then estimating from this the risk to humans. Con-
sidering the differences in these methods, in addition to inter-
species susceptibilities to a carcinogen, human risk estimates
may vary by a factor of 10 or 100; there is no certainty that the
true risk does not lie outside this range.

Short-Term Tests

A number of short-term tests are used to detect a chemical's
mutagenicity. These methods usually examine the effects on animal
cells, fruit flies or bacteria. Comparison of bacterial tests to
animal cancer tests shows that, with few exceptions, the detected
mutagens are also carcinogens. Although the potency exhibited in
both tests appears to be related (Ames, 1977), short-term tests are
presently used for qualitative, but not quantitative, purposes.

Epidemiologic Studies

Epidemiologic studies relate the incidence of human disease to
known or suspected causal factors. There are two types of studies.
Analytic (case control or cohort) studies compare a group of indi-
viduals with the disease being studied to a group of similar indi-
viduals without the disease, matching individuals with similar
characteristics on a case by case basis. Ecologic (whole population)
studies compare population groups without matching on an indivi-
dual basis.

Ecologic studies are primarily used for the generation of hypo-
theses concerning the association of drinking water contaminants and
cancer. Although relevant factors such as occupation, age, sex
and ethnic group can be entered into the calculus, these determi-
nations are not made at the individual level, and quantification
of the excess risk due to drinking water remains imprecise.
Quantifying excess cancer rates on the basis of ecologic studies
is generally not an accepted practice. In the absence of more
precise data, however, such calculations have some utility.

Excess cancer risks can be quantified from analytic studies,
since individual determinations of relevant factors are made. Only
one such study has been completed which associates drinking water
contaminants with GI and UT cancer (Alvanja, et al., 1977). Even
these types of studies, however, cannot control for all the poten-
tially relevant factors and must be interpreted with caution.

It is difficult to know from epidemiologic studies whether the
observed excess cancer is solely attributable to drinking water

contaminants, or whether the effects of these contaminants are potentiated by other types of exposures; e.g., from food, air pollution or smoking. Even if other exposures do have influence, it may still be the case that removing carcinogens from water will also eliminate the excess cancer risk.

Another difficulty in quantifying excess cancer risks from epidemiologic studies is that cancer is a disease with a typically long latency period, generally twenty to thirty years. Any cancer observed now may be caused by unknown exposures occurring decades prior, and current cancer rates may not reveal the effects of changes in exposures to carcinogens that occurred in recent decades. Relating currently observed cancers to current water quality data can involve considerable distortions.

Confidence in risk estimates based on epidemiologic studies usually increases the more extensive the analysis, or the greater the number of studies conducted. In the case of drinking water, the approximately 12 epidemiologic studies are likely to provide a more precise estimate of the risk from THMs than would the models of extrapolating from animal studies, since only one (chloroform) of the THMs has been tested in animals. Furthermore, synergistic and promotional effects are not evident from animal cancer tests, unlike the results from epidemiologic studies.

Appendix C

ESTIMATION OF NONFATAL CANCER ILLNESS

The extent to which cancer illness is nonfatal is reflected by the percentage of cancer victims who are still alive ten years after detection of the disease, since survival rates tend to stabilize between five and ten years after detection. The extent of nonfatal gastrointestinal and urinary tract cancer may be estimated by applying first incidence rates and then ten-year survival rates to the population at risk. Such estimates are necessarily imprecise, due to differences in survey methods, determinations of the causes of illness, variations in classifications, and changes over time in incidence rates and in the survival of treated patients.

Recognizing these limitations, and assuming that recent incidence and survival trends continue, it appears that approximately 30% of combined GI and UT cancer is nonfatal, or about 60,000 of the 200,000 cases which are newly detected each year. On an annual basis, the number of newly detected nonfatal cases is about 50% of the number of deaths from these cancers, which is about 115,000. In the long run, however, nonfatal illness would be about 40% of fatal illness (i.e., 60,000/140,000). The difference between annual and long run percentages may be attributed to the limitations of the data described above.

(See table on next page)

TABLE 9

	Annual Incidence per 100,000 Population, 1969-71[1]			Ten-Year Survival Rate (Percentage), 1960-73[2]		
	Total	White	Black	Total	White	Black
Gastrointestinal	74.3	73.0	84.0	–	–	–
Esophagus	3.4	2.8	9.7	3	3	2
Stomach	10.5	10.0	15.2	11	11	11
Small Intestine	1.0	1.0	1.1	NA	NA	NA
Colon	30.9	31.0	27.9	37	40	34
Rectum	13.6	13.7	11.4	35	36	23
Liver	2.2	2.1	3.4	3	3	(3)[3]
Gallbladder	3.0	3.0	2.2	6	6	(6)[3]
Pancreas	9.7	9.4	13.1	1	1	2
Urinary Tract	18.6	19.1	13.0	–	–	–
Bladder	13.2	13.6	7.9	51	53	24
Kidney	5.4	5.5	5.1	35	35	33
Total Gastrointestinal and Urinary Tract	92.9	92.1	97.0	–	–	–
Excluding Small Intestine	91.9	91.1	95.9	29	30	19

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[1] Age-standardized to 1970 population. Source: National Cancer Institute, The Third National Cancer Survey Advanced Three Year Report 1969-71 Incidence, 1974.

[2] These are "relative" survival rates, defined as "the ratio of observed survival rate for the patient group to the rate expected of patients in the general population similar with respect to age, sex, race and calendar year of observation". Source: National Cancer Institute, Cancer Patient Survival, Report #5, 1976. Survival rates by ethnic group and specific site were provided in that document. Survival rates for all ethnic groups by site, and for total GI and UT cancer (excluding small intestine) were calculated by applying the site-specific incidence rates for whites and blacks to the 1970 U.S. population: white 178,098,000, and non-white 25,138,000 (source: U.S. Bureau of Census, Statistical Abstract of the United States, 1977), and then applying the relevant survival rates.

[3] Not available. Assumed to be the same as for whites.

Appendix D

METHODS FOR VALUING HUMAN LIFE

This appendix discusses three principal methods which have
been used to value human life: willingness-to-pay, lost
earnings, and valuations implicit in government decisions.[1]

A major difficulty in applying any of these approaches is
that there is a distinction between "known" and "statistical"
lives. The benefit of a life-saving project is that it reduces
everyone's chance of death; without the project a certain number
of unidentified individuals will die. The valuation placed on
the certain death of unknown individuals is far lower than valua-
tion placed on life once the victim is known. The difference in
these values is most evident in the legal system (Zamolo, 1977),
under which enormous sums of money have been awarded as compen-
sation after the death has occurred.

Willingness-to-Pay

Numerous attempts have been made to apply the "willingness-
to-pay" concept. A direct attempt is to ask individuals directly
how much they would be willing to pay for reductions in the risk of
early death, or a particularly dreaded manner of death. While
this direct approach has been discussed (Schelling, 1968), a major
obstacle is that individuals cannot conceptualize the small risk
reductions involved in life-saving projects; e.g. from 2 in
100,000 to 1 in 100,000. Another obstacle arises in the
hypothetical nature of the question.

[1]A good survey can be found in Linnerooth(1975).

Another approach has been to study the wage differential
between risky and nonrisky jobs to determine how a specific reduc-
tion in the risk of death might be valued. The problems here are,
first, that individuals taking such jobs may be less averse to
risks than the general population and, second, knowing how an
individual values a risk reduction from 16 to 15 in 100,000 yields
no information about how the individual might value a reduction
from 3 to 2 in 100,000. These values may not be the same or
even close. A further complication is that even if the
reduced risk of future cancer were the same as the reduced risk
of dying in an industrial accident in a particular year, the fact
that in the first case the chance of death is far in the future
makes comparisons to the latter valuation problematical (Freeman, in
press).

The value of risk reduction to the individual worker, as
reflected in wage differentials, has been used to determine how
much workers collectively would be willing to pay to avoid the
death of one among them (in the statistical sense). The best
example of such a study, that of Thaler and Rosen in 1973, deter-
mined that individuals value a risk reduction of .001 at $200,
thus implying that life is worth about $200,000 (Rhoads, 1978).
A similar study found that a risk reduction from 16 to 8 in
100,000 was valued at $120, implying a valuation of $1.5 million
per life (Smith, 1976). In a 1963 study of flight pay for U.S.
Air Force captains, Carlson determined that the implied valuation
of a pilot's life was between $135,000 and $980,000, depending on
the type of plane flown (Zeckhauser, 1975).

Fewer attempts have been made to assess how individuals value life itself. In an informal survey, Schelling noted that certain professionals value their lives at 10 to 100 times their annual salary, or at approximately $200,000 to $3,000,000 (Schelling, 1968). In a study of the psychosocial costs of cancer, Abt considered the valuation of life in terms of the willingness to pay to keep people alive in nursing homes after their economic value to society has been exhausted (Abt, 1975). This cost is about $10,000 a year for public nursing homes, which Abt considered to be the amount society is willing to pay to preserve the life of unknown individuals. The cost is about $20,000 a year for private nursing homes which Abt considered reflective of the family's willingness to pay. Abt considered the value of preventing a cancer death to be the collective annual willingness to pay of society, friends and family, multiplied by the average reduction in life expectancy resulting from a cancer death. Abt calculated this figure to be about $400,000, based on a reduced life expectancy of 10 years. The weaknesses of this approach are that the valuation is dependent upon the age of the victim and excludes the victim's own willingness to pay.

Lost Earnings[1]

The lost earnings approach attempts to assess the cost to society in productive resources when one of its members dies. These calculations include medical costs, lost earnings and insurance payouts. A fundamental objection to this approach is its

[1] The term "societal cost" was used by the National Highway Traffic Safety Administration, for this approach.

close link to the assumption that the value of an individual is his contribution to the Gross National Product. Of the various government agencies which utilize these figures in determining the benefits of safety projects, at least two emphatically assert that these figures are not construed as the value of a human life.[1] Nevertheless, the association is difficult to avoid. It is perhaps reasonable to consider these figures a lower bound on the financial benefits to society of saving a life, realizing that they exclude the valuation placed on life itself.

The lost productivity of the individual is measured by the present discounted value of future earnings which will be lost when the individual dies. One objection to this approach is that the resulting valuations of life vary with age, sex, occupation, and ethnic group. For example, the figures for housewives are much lower than for young executives.

In applying the lost earnings approach, Cooper and Rice concluded that the cost of cancer illness and death, comprised of medical costs and lost earnings, was $15-$17 billion in 1972 dollars (Cooper and Rice, 1976), or $65,000 - $76,000 per death in 1978 dollars.[2] The cost per death includes the treatment costs of cancer survivors, and the higher and lower figures reflect the use of different discount rates. In a recent report by the

[1] Mahesh Podar, National Highway Traffic Safety Administration, personal communication, May 11, 1978; Steve Zaidman, Federal Aviation Administration, personal communication, May 11, 1978.

[2] 1978 costs were derived by adjusting for the 59% increase in medical costs and the 51% increase in wages from 1972-1978. Source: U.S. Department of Commerce, Bureau of Economic Analysis, Business Statistics, 1975 (publ. 1976), and Survey of Current Business 58 (4) 1978.

National Academy of Sciences (NAS, 1978), the Cooper and Rice data were adjusted in a manner which combines the cost of death and willingness-to-pay approaches. Indirect costs (business taxes, effects of property income) were included, and compensation for being exposed to the risk of cancer was added. The resulting figure was approximately $200,000 ($150,000 when the willingness-to-pay approach risk compensations were excluded).[1]

The National Highway Traffic Safety Administration places the cost of a traffic fatality at $287,000 (1975 dollars), which includes medical costs, lost earnings, insurance payouts, traffic delay and other miscellaneous costs.[2]

The Federal Aviation Administration measures the cost of an air traffic fatality in terms of the insurance payouts resulting from court settlements with the families of victims, rather than lost earnings. The figure for 1977 is $300,000.[3]

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[1]John Cumberland, University of Maryland, personal communication, May 25, 1978.

[2]U.S. Department of Transportation, National Highway Traffic Safety Administration, 1975 Societal Costs of Motor Vehicle Accidents, 1976.

[3]Walter Faison, Federal Aviation Administration, personal communication, May 11, 1978.

Valuations Implicit in Government Decisions

A third approach to discovering society's valuation of human life is to determine the valuations implicit in government health and safety decisions and regulations. The essential weakness of this approach is that such decisions are subject to the vagaries of the political process, resulting in sometimes bizarre implications, e.g., Bailey noted that safety decisions in the military implied valuations of human life ranging from several million dollars to a negative value (Bailey, 1968).

Valuations implicit in government regulations are analyzed by the Council on Wage and Price Stability (CWPS) while the regulations are in proposed form. The CWPS method of determining these valuations when the reduction in deaths will occur over time is to determine the cost per life saved in the "steady state" which will be reached after the transition period during which the reduction occurs. For two Occupational Health and Safety Administration regulations which control exposure to carcinogens, the CWPS has estimated the following implicit valuations of human life: (1) inorganic arsenic: $1 million - $9 million, and (2) coke oven emissions: $4.5 million - $158 million.[1]

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[1]Council on Wage and Price Stability, "Exposure to Coke Oven Emissions Proposed Regulation", May 11, 1976, and "Exposure to Inorganic Arsenic Proposed Standard", September 14, 1976.

Appendix E

METHODOLOGICAL APPENDIX

1. Determining How Cancer Mortality Would Decline Over Time,
   Following Installation of GAC Absorption

In order to determine the cost of GAC per life saved under
discounting, it is necessary to determine the number of lives which
would be saved in each year following the installation of GAC.
Unfortunately, too little is known about the causal processes of
cancer to make certain statements about how cancer mortality would
decrease over time following the removal of organic contaminants
from drinking water. Numerous assumptions had to be made; it must
be realized that these assumptions are speculative.

It was first assumed that cancer rates would continuously decline
for a period of 70 years, after which persons currently exposed
to drinking water carcinogens would no longer be living, and
thus no one would bear an increased risk of cancer. This future
time during which the excess cancer risk would be fully reduced
is termed the "steady state". Seventy years was selected as the
time necessary to reach the steady state because it is consistent
with the average life expectancy (about 70).

It was then assumed that the excess death rate would not de-
crease for several years, which is consistent with the evidence
that it takes several years after the cessation of cigarette smo-
king for the risk of lung cancer to cease increasing (Peto, 1977).

Finally, it was assumed that the excess cancer rate would
decrease slowly, then rapidly, then slowly. This pattern is con-
sistent with the evidence that cancer risks often appear to
increase with exposure slowly, then rapidly, then slowly (Peto, 1977).

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To translate this pattern into numerical terms, this pattern was
assumed to approximate the cumulative frequency of a normal curve
with a midpoint of 35 years and a standard deviation of 10 years.
By using a standard deviation of ten, the first reductions occur-
red after five years, approximately the lag in experiencing a
change in the risk of lung cancer after stopping smoking.

Alternative scenarios were considered, but too little infor-
mation exists to put them in quantitative terms. For example,
if THM levels have increased in recent years such that the effect
is not yet evident in current cancer rates, it might the the case
that not removing THMs would cause an increase in the cancer rate,
and removing them now would not decrease the cancer rate as soon
as assumed above. However, the number of lives saved would be
weighed against the increase in cancer mortality which did not
occur, and this number would ultimately rise above the current
level of excess cancer deaths. The benefits estimated in this study
would be underestimated.

Similarly, in the case of synthetic organics, removing them
now might not reduce cancer rates, but prevent an increase in these
rates. The number of lives saved would be the increase in cancer
deaths which did not occur.

2. Calculating the Number of Lives Saved for the Representative
   Populations

Steady State

The number of lives saved annually in the steady state was
assumed to be 90% of the excess cancer deaths which were assumed
to be caused by drinking water contaminants, since it was assumed

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that GAC would reduce both contaminants and the risk of cancer by 90%. The number of excess deaths were determined by multiplying each population size (1,193,000, 263,200 and 92,700) by each estimate of the excess mortality rate per million population per year (20,150 and 300); the number of lives saved in the steady state was calculated by multiplying the number of excess deaths by 90%. The following results were obtained for the representative systems upon which this analysis was based:

System Size	Population	Excess Cancer Mortality Rate	Number of Lives Saved
Large	1,193,000	300	322
		150	161
		20	21
Medium	263,200	300	71
		150	36
		20	5
Small	92,700	300	25
		150	13
		20	2

Over 100 Years

During the last 30 years of the 100 year time horizon, the steady state would have been reached and the number of lives saved in each representative population per year would be the same as indicated above. For the first 70 years, however, the number of lives saved would be increasing from none to the number reached in the steady state, in the pattern described in the previous section. To determine the number of lives saved in each of those years, each year was converted to its standard deviation from the mean of a normal

curve with a midpoint of 35 and a standard deviation of ten. For example, year 25 was -1.0 standard deviations from the mean.

For each year's standard deviation, the cumulative frequency from the normal table was multiplied by the number of lives which would be saved in the steady state. For example, for year 25 (standard deviation of -1.0), the cumulative frequency would be 0.1587. For the large population, assuming the high (300) rate of excess mortality, the number of lives saved in year 25 would be 0.1587 x 322 = 51.

Presented below are the number of lives saved at ten year intervals for the large system, using the high (300) and low (20) risk estimates:

			Number of Lives Saved	
Year	Standard Deviation	Cumulative Frequency	(High-Risk Rate) 322	(Low-Risk Rate) 21
5	-3.0	0.0013	0	0
15	-2.0	0.0228	7	0
25	-1.0	0.1587	51	3
35	0.0	0.5000	161	11
45	1.0	0.8413	271	18
55	2.0	0.9772	315	21
65	3.0	0.9987	322	21
71-100	-	-	322	21

The number of lives saved over the total 100 year period were as follows:

System Size	Population	Excess Cancer Mortality Rate	Total Number of Lives Saved
Large	1,193,000	300	20,931
		150	10,465
		20	1,367
Medium	263,200	300	4,613
		150	2,340
		20	325
Small	97,200	300	1,624
		150	845
		20	131

REFERENCES

Abt, C.C., 1975. "The Social Costs of Cancer," Social Indicators Research, 2:175-190.

Alavanja, M., I. Goldstein and M. Susser, 1977. "Case Control Study of Gastrointestinal Cancer Mortality in Seven Selected New York Counties in Relation to Drinking Water Cholorination," Columbia University, New York, New York.

Ames, B.N., 1977. "Environmental Chemicals Causing Cancer and Genetic Birth Defects: Developing a Strategy for Minimizing Human Exposure," California Policy Seminar.

Bailey, M.J., 1968. "Comment on 'The Life You Save May Be Your Own,'" in Problems of Public Expenditure Analysis, ed. S. Chase, Brookings Institution, Washington, D.C.

Bingham, E., and H.L. Falk, 1969. "Environmental Carcinogens-- Modifying Effects of Carcinogens on the Threshold Response," Archives of Environmental Health, 19:779-785.

Cantor, K.P., R. Hoover, T.J. Mason and L.J. McCabe, 1977. "Association of Halomethanes in Drinking Water With Cancer Mortality" (draft), Environmental Epidemiology Branch, National Cancer Institute.

Cooper, B.S., and D.P. Rice, 1976. "The Economics of Cost of Illness Revisited," Social Security Bulletin, 39(2):21-36.

Council on Wage and Price Stability (Ivy Broder) 1978. "Analysis of Proposed EPA Drinking Water Regulations," Washington, D.C. (Sept.)

d'Arge, R.,1975. U.S. Department of Transportation, Climatic Impact Assessment Program.

Environmental Protection Agency (EPA), 1975. "New Orleans Area Water Supply Study," Environmental Protection Agency, Dallas, Texas.

_____, 1977. "Statement of Basis and Purpose for an Amendment to the National Interim Primary Drinking Water Regulations on a Treatment Technique for Synthetic Organic Chemicals," Office of Water Supply, Environmental Protection Agency, Washington, D.C.

_____, 1978. "Interim Primary Drinking Water Regulations: Control of Organic Chemical Contaminants in Drinking Water," Environmental Protection Agency, Washington, D.C.

Ferejohn, J., and T. Page, 1978. "On the Foundations of Intertemporal Choice", Proc. Am. J. Agr. Econ., May, 1978, pp.269-275.

Freeman, A.M.. Measuring Benefits: Theory and Practice (in press), Resources for the Future, Washington, D.C.

Harberger, A.C., 1971. "Three Basic Postulates for Applied Welfare Economics", Journal of Economic Literature (9)3:785-97.

Harris, R.H., T. Page and N.A. Reiches, 1977. "Carcinogenic Hazards of Organic Chemicals in Drinking Water," in Origins of Human Cancer, ed. H.D. Hiatt, J.D. Watson and J.A. Winsten, Cold Spring Harbor Laboratory, Cold Spring Harbor, New York.

Hooper, K., C. Gold and B. Ames, 1977. "Development of Methods for Mutagenicity Testing of Wastewater and Drinking Water Samples by the Ames Salmonella Test," Report to the Water Resources Board, State of California.

Linerooth, Joanne, 1975. "The Evaluation of Life-Saving: International Institute for Applied Systems Analysis, Laxenburg, Austria.

Loper, J.C., et al., 1977. "In Vitro Mutagenic and Carcinogenic Testing of Residual Organics in Drinking Water" (unpublished), University of Cincinnati.

McCann, J., E. Choi, E. Yamasaki and B. Ames, 1975. "Detection of Carcinogens as Mutagens in the Salmonella-microsome Test: Assay of 300 Chemicals," Proceedings of the National Academy of Sciences, 72:5131.

McCann, J., 1977. "Summary of Testimony Given Before Senator Kennedy's Health Subcommittee Hearing on Saccharin".

McKinney, J.D., R.R. Maurer, J.R. Hass and R.D. Thomas, 1976. "Possible Factors in the Drinking Water of Laboratory Animals Causing Reproductive Failure," in Identification and Analysis of Organic Pollutants in Waters, ed. L.H. Keith, Ann Arbor Science Publishers, Inc., Ann Arbor, Michigan.

Mishan, E., and T. Page. "The Methodology of Cost-Benefit Analysis-With Particular Reference to the Ozone Problem." BACER Program (James Hibbs, director), EPA, Washington, D.C. 1978. Also California Institute of Technology Social Science Working Paper (1979).

National Academy of Sciences (NAS), 1975. Contemporary Practices and Prospects: The Report of the Executive Committee, Vol. I, National Research Council (NRC), NAS, Washington, D.C.

_____, 1977. "Summary Report: Drinking Water and Health," Safe Drinking Water Committee, NRC, NAS, Washington, D.C.

_____, 1978. Nonfluorinated Halomethanes in the Environment, Panel on Low Molecular Weight Halogenated Hydrocarbons of the Coordinating Committee for Scientific and Technical Assessments of Environmental Pollutants, NRC, NAS, Washington, D.C.

National Cancer Institute (NCI), 1974. The Third National Cancer Survey Advanced Three Year Report 1969-71 Incidence, U.S. Department of Health, Education and Welfare, DHEW Publication No. (NIH) 74-637.

National Center for Health Statistics (NCHS), 1977. Vital Statistics of the United States, 1973, Vol. II, Part A, U.S. Department of Health, Education and Welfare.

Office of Management and Budget (OMB), 1972. "Discount Rates to be Used in Evaluating Time-Distributed Costs and Benefits," Circular No. A-94 Revised.

Page, T., 1977. "Intertemporal Equity," in Conservation and Economic Efficiency, Johns Hopkins Press, Baltimore, Maryland.

Page, T., R.H. Harris and S.S. Epstein, 1976. "Drinking Water and Cancer Mortality in Louisiana," Science, 193:55-57.

Pelon, W., B.F. Whitman, and T.W. Beasley, "Reversion of Histidine - Dependent Mutant Strains of Salmonella Typhimurium by Mississippi River Water Samples." Envir. Sci. and Tech., 11, 619(1977).

Peto, R., 1977. "Epidemiology, Multistage Models, and Short-Term Mutagencitiy Tests", in Origins of Human Cancer, ed. H.D. Hiatt, J.D. Watson and J.A. Winsten, Cold Spring Harbor Laboratory, Cold Spring Harbor, New York.

Ramsey, F., 1928. "A Mathematical Theory of Savings", Eco. J., 38:543-9.

Rhoads, S.E., 1978. "How Much Should We Spend to Save a Life?" The Public Interest, 51:74-92.

Schelling, T.C., 1968. "The Life You Save May Be Your Own," in Problems of Public Expenditure Analysis, ed. S. Chase, Brookings Institution, Washington, D.C.

Simmon, V.F., K. Auhaner and R.D. Tardiff, 1977. "Mutagenic Activity of Chemicals Identified in Drinking Water," in Progress in Genetic Toxicology, ed. D. Scott, B.A. Bridges and F.H. Sobels, Elsevier-North Holland, Biomedical Press, New York.

Smith, R., 1976. The Occupational Safety and Health Act, American Enterprise Institute, Washington, D.C.

Solow, R., 1974a. "Intergenerational Equity", Rev. of Eco. Studies, 41:29-45.

_____, 1974b. "The Economics of Resources or the Resources of Economics," Proc. Am. Econ. Rev. 64:9.

Temple, Barker and Sloane, Inc., 1978. "Revised Economic Impact Analysis of Proposed Regulations on Organic Contaminants in Drinking Water", Temple, Barker and Sloane, Inc., Wellesley, Massachusetts.

Theiss, J.C., G.D. Stoner, M.B. Shimkin and E.K. Weisburger, 1977. "Test for Carcinogenicity of Organic Contaminants of United States Drinking Waters by Pulmonary Tumor Response in Strain A Mice", Cancer Research, 37(8):2717-2720.

United States Congress, 1974. House of Representatives, Report No. 93-1185, U.S. Government Printing Office, Washington, D.C.

Wan, H., Optimal Growth, Harcourt-Brace Jovanovich, New York, 1971.

Weizsacker, C.C. von, 1965. "Existence of Optimal Program of Accumulation for an Infinite Time Horizon," Rev. of Eco. Studies, 32:85-104.

Zamolo, P., 1977. "Valuing Human Life: The Economic and Legal Measures," University of California Berkeley (unpublished manuscript).

Zeckhauser, R., 1975. "Procedures for Valuing Lives," Public Policy 23(4): 419-464.