WV-0077 #1

PLAINTIFF'S EXHIBIT

UC-2154

UNION CARBIDE

INTERNAL CORRESPONDENCE

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Date March 8, 1979

Originating Dept.

Subject Ed Light's Comments for Additional Water Pollution Control, Toxics

Gentlemen:

The attached document, by Mr. Ed Light, on the need for additional control of discharges of toxic chemicals to the waterways in the Kanawha Valley is transmitted for your information. This is the information that he presented to the State Water Resources Board during the public hearings for the State NPDES Permitting Program.

Very truly yours,

R. L. Foster

RLF/db

Attachment

*Letter only

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TOWARD A NEW TOXIC WATER POLLUTION CONTROL POLICY: DOCUMENTATION FOR ADDITIONAL STANDARDS

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January 15, 1979

Presented To State Water Resources Board 11/5/79 During Public Hearing For State NPDES Permitting Program.

UCC 006777

INTRODUCTION

Toxic water pollutants are those which, at low concentrations, can either directly cause human health effects, such as cancer, or be poisonous to aquatic life or accumulate to levels in fish which are harmful to humans. This problem has received much attention nationally over the last several years, but current federal policy falls far short of minimizing, to the extent practical, the risk from exposure to toxic water pollutants. The current EPA program basically requires:

A. Best available control technology by 1984 for 129 designated national priority toxic pollutants.

B. Spill prevention measures for some 300 officially designated hazardous pollutants.

The specific problems with this approach include:

1. Pollutants which are known to be toxic, but are only regionally and not nationally significant, are not regulated.

2. Pollutants with inadequate human health and aquatic effects research (which includes most industrial chemicals) are unregulated simply because their effects have not been adequately documented.

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3. Pollutants usually have an additive or even greater effect in the mixtures in which they are discharged, and this is not taken into account for the setting of individual standards.

For these reasons, I propose the following additional standards, based on practical monitoring techniques and control technology, and legally enforceable requirements, to supplement the EPA standards.

A. Installation of best available control technology economically achievable to reduce the discharge of all persistent organics as a general class of pollutants.

B. Aquatic toxicity limits on undiluted effluents requiring they not be lethal to fish in a standard bioassay test.

C. Spill prevention measures required for all industrial chemicals.

D. Water quality standards for specific unregulated carcinogens set no higher than 1/100 the LC_{50} (concentration lethal to 50% of test fish).

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The following report documents the need for this approach focusing on water pollution problems caused by the dozen chemical plants located in the Kanawha Valley, West Virginia. The following issues are discussed:

- Chapter One: Human health problems caused by low concentrations of water pollutants, emphasizing cancer.

- Chapter Two: The impacts of industrial discharges on downstream public water supplies, emphasizing the Kanawha and Ohio Rivers.

- Chapter Three: The magnification of organic water pollutants in fish.

- Chapter Four: Aquatic toxicity standards.

- Chapter Five: Control technology for persistent organics with a discussion of costs.

- Chapter Six: Inventory of Kanawha Valley chemical plant toxic water pollutants.

- Chapter Seven: Federal and state regulatory policies and programs.

- Chapter Eight: Statements from experts supporting the control of persistent organics generally.

- Chapter Nine: Statements from experts documenting the need to expand the scope of spill prevention regulations.

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CHAPTER ONE: INDUSTRIAL WATER POLLUTANTS AND PUBLIC HEALTH

In the U.S., about one out of every four persons get cancer, at an annual cost of about $15 billion. It has been estimated that up to 90% of these cases are caused by exposure to chemicals, some natural, but many synthetic.[11] Some of these synthetic chemicals have also been linked to other chronic health problems including birth defects, gene mutations affecting future generations, vascular disease,[12] liver and kidney damage,[13] and nervous system disorders.[14] However since the most significant (at least best studied) health problems caused by exposure to low concentrations of industrial pollutants is cancer, that will be the focus of this section.

Human exposure to carcinogens discharged into rivers by chemical plants occurs primarily when the compounds persist downstream and are not removed by drinking water treatment plans. Bathing in water contaminated by certain pollutants and ingestion of fish retaining carginogens in their body are also suspected by some scientists of posing a significant cancer risk.[15]

Frightening new evidence further suggests that industrial organics in fish and drinking water concentrate in mothers milk. For example, breast feeding infants consume over 100 times more PCB's in their daily diet (on a body weight basis) than adults. This has led medical experts in certain polluted regions of the country to recommend that women of child bearing age either not consume sport fish or consider bottle feeding their infants.[76f]

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A. Chemical Carcinogenisis: Basic Principals

Cancer produced by chemical exposure is cumulative, shows delayed symptoms, and causes the disease in only a small percent of the affected population. Many carcinogens seem to be more effective with frequent small doses than a few large doses.[16] Cells retain the biological effect of exposure to a carcinogen, and each subsequent incident adds to the chance of this developing into a tumor.[12] The latency period between first exposure and tumor formation is generally 15 to 40 or more years, with the lower the exposure, the longer the time interval.[17] Exposures to small amounts of carcinogens are most likely to effect children because the long latency period is most likely to expire within their lifetime. There is also considerable concern that when mothers are exposed to carcinogens, they can be passed along to their offspring via their womb. [12]

While the concentrationsof certain known carcinogens discharged by chemical plants may be high enough to predict a significant increase in cancer rates on their own, the combined effect of the mixture of hundreds of chemicals in effluents is probably much greater. Experiments show that some chemicals act as "co-carcinogens", having little or no effect by themselves, but considerably augmenting the effect of other carcinogens when present. Phenols, commonly discharged by chemical plants, can act as co-carcinogens.[16] Asbestos may also have this effect, as evidenced by lung cancer rates for asbestos workers which are about normal for nonsmokers, but for smokers are 8 times that of smoking non-asbestos workers, and 92 times that of nonsmoking non-asbestos workers.[17] In many instances, simultaneous exposure to more than one carcinogen has been found to have an additive or even greater (synergistic) effect, although some mixtures can have an inhibitory effect.[12]

Experts generally agree that tests involving the exposure of rodents to high concentrations of a chemical must be relied on to determine whether low concentrations pose a carcinogenic hazard to man,[12,17,18] Several chemicals later proven to be

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human carcinogens (by high cancer rates in exposed workers) were first identified in animal tests.[12] All other proven human carcinogens were later shown to cause cancer in rodents (with the possible exception of arsenic which is still under study).[17] If no tumors are found in a group of 100 mice exposed to a low concentration of a chemical, the only conclusion can be that there is a 95% chance that the actual cancer risk is comething less than 3%,[10] but even a .01% cancer rate can be very significant for a large human population (10^4 cases per 100,000 people). At least 30,000 mice would be needed in a statistically significant sample to detect such a rate, and this is prohibitively expensive.[11] Because cancer rates increase with increasing concentration, researchers have found that exposing rodents to the maximum tolerable dose (not causing acute effects) can provide data to estimate rates at lower concentrations. Experimental work has disproven the claim that injection of large doses of anything in rodents will cause tumors,[12] and of the 6,000 chemicals tested in such a manner, only about 10% were positive.[17]

B. The Ames Test

Thousands of industrial pollutants have not been screened at all for their cancer causing potential, while studies for some chemicals supposedly showing negative results were inadequate (too few animals, or too short a study period) to be conclusive. In order to avoid the extremely long time and high cost needed to test all these chemicals on rodents, the "Ames test" has been developed. The Ames test uses a specially developed bacteria strain, mixed with rat liver (to simulate mammal metabolism) which produces readily observed gene mutations when exposed to certain chemicals.[19] The degree of mutagenic activity in this test correlates very closely with carcinogenic response in mammals. For example, in a screening of 300 chemicals, 90% of the carcinogens were mutagenic, while 90% of the apparent non-carcinogens were not strongly mutogenic.[19] Refinements are being developed to reduce the occurence of false negatives.[20] A number of experts conclude that the Ames test can provide a good preliminary, if not conclusive, screening of many chemicals or mixtures to identify carcinogenic hazards.[16,18,19,20] EPA tests of drinking

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water in 5 cities show that those polluted by industrial wastes cause more mutations in the Ames Test.[76b]

C. Occupational Cancer Studies

Epidemiological studies of chemical plant workers can also be very useful in pinpointing some carcinogens. For example, vinyl chloride, benzidine, and bis chloromethyl ether were proven to be human carcinogens in this manner.[17] A recent study of cancer rates found that in counties with high concentrations of chemical industries, cancer of the liver, bladder, lung, mouth, throat, skin, bone, nasal sinus, and melanoma were significantly higher, apparently due to occupational and environmental exposure.[21] It has also been suggested that Ames tests on workers' body fluids could help identify and monitor their exposure to carcinogens.[16]

Some industrial cancer investigators have obscured problems by improperly comparing worker cancer rates to rates for general populations, diluting highly exposed worker populations with other workers, and not including former workers no longer at the plant.[12] Officials of the National Cancer Institute and National Institute of Occupational Safety and Health (NIOSH) have stated that such surveys, if properly conducted at all plants, would uncover a wealth of valuable new data on carcinogens. This could also provide an early warning system for the general population, who may be exposed to lower concentrations of the same chemicals in water and air pollutants, where the effect is obscured due to longer latency periods, multiplicity of exposure to various carcinogens, and population mobility. It has been recommended that Congress provide the necessary funds and legal authority (i.e. there are currently restrictions on the use of social security data which can provide valuable information on diseases and place of employment) to comprehensively screen chemical plant worker cancer rates.[12]

One such probe was started in 1976 at the DuPont Belle plant when the Union presented a Congressional Committee with a list of cancer victims and pointed out various exposures to liquid and gaseous pollutants. A subsequent Company study showed high rates of kidney, eye, and lung cancer, despite using methods which minimized their significance. NIOSH then found that 13 of the 170 compounds handled

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at the plant were suspect carcinogens, and is now undertaking a full investigation of the plant, which could take several years.[22]

D. Human vs. Animal Cancer Rates

Considerable controversy has occured over the quantitative extrapolation from experimental animal data to humans. Most experts agree that, while the rate of cancer incidence decreases with lower doses, there is no threshold dose below which at least some cases won't occur in a large enough population. Most also agree that at least qualitative reliance on animal data is valid in determining human carcinogens. Substantial disagreement occurs over whether it is possible to determine a safe human dose from animal data.

One such estimation was recently made with the carcinogen chloroform. The lowest dose thought to cause liver tumors in small mice populations is 37 mg/kg body wt./day. This was divided by 14 to give the equivalent dose to man (due to the difference in the surface area to body weight ratio), and found to be 2.7 mg/kg/day. The highest level found in drinking water, 300 parts per billion, is equivalent to an intake of .017 mg/kg/day. The ratio between the two doses, 2.7/.017 or 156, was divided into the rate at which the mouse population got cancer, 15%, and it was concluded that the resulting human cancer rate would be about .1%. This could account for 40% of U.S. liver cancer cases, if everyone drank water with that maximum concentration of chloroform, but would be reduced with lower concentrations.[23] This approach is known as a "linear non-threshold dose-effect model".

Some experts say this model can greatly underestimate the human significance of a carcinogen concentration because:

A. For some chemicals, humans are more sensitive than animals (i.e. birth defects occurred in humans at one hundreth the minimum dose that affected rats).

B. The model ignores the presence of co-carcinogens which can have an additive or synergistic effect on cancer rates.[11]

Thus, it is often recommended that carcinogenic discharges be reduced to the greatest extent practical in order to minimize human risk.

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E. Fish Tumors

Several studies indicate that industrial discharges are causing an increase in tumors in fish and other aquatic life. For example, one Lake Ontario researcher found papillomas in white suckers and gonadal tumors in carp and goldfish with "apparent environmental relevance."[76f] Another research warns:

"As dead canaries warn of potential mining disasters, diseased fish should warn of the potential hazards to fishermen and to the general public who must depend on polluted waters as drinking water supplies and sport fishing areas."[76f]

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CHAPTER TWO: DOWNSTREAM DRINKING WATER SUPPLIES

A. General

In the past several years, studies have been initiated to identify organics present in drinking water supplies, and to determine their health significance. While there are still many unknowns, some experts have reached the conclusion that the adverse impact of such contamination is significant.[24]

Over 300 different organic compounds have been identified in drinking water, in the parts per billion (ppb) range, including an 80 city survey by the EPA in 1975.[24] Some carcinogens, such as chloroform, were found to be primarily produced by chemical reactions which take place when organics from various sources are chlorinated in the disinfection portion of the drinking water treatment plant.[25] Other carginogens, such as carbon tetrachloride, are contributed directly by industrial sources, and escape filtration at most water plants.[24] Only about 15% of the total organic matter in drinking water has been identified. Also, many of the known compounds have not been characterized as far as carcinogenic activity, although their structural similarity to other carginogens suggests they are dangerous.[18] A national academy of Science concluded that of 286 organics found in drinking water, only 22 had sufficient toxicity data to render a hazard evaluation.[76b] Thus, there are probably many more carcinogens present in drinking water than the several already identified. It has been suggested that biological testing of all organic material in drinking water on mammals or bacteria would help to assess the carcinogenic potency of this mixture.[24] Data in a recent evaluation on the toxicity of individual organics in drinking water (based on extrapolation from animal data) suggests that there would be no acute effects at the levels found, that there was no danger of non-carcinogenic chronic effects from organics such as styrene and methyl methacrylate as long as they were below about 40 ppb, but that known carcinogens in drinking water could cause a significant increase in cancer cases of a large population at about 10ppb.[18] Another report suggests that levels below 2ppb

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of carbon tetrachloride were needed to protect the most susceptible people from chronic liver and kidney damage.[13]

Nine epidemeological studies show a significant association between drinking water quality and bladder cancer mortality. Cancer at the other sites, such as stomach, colon, and rectum, have been linked to polluted drinking water in most of these studies.[76f] Epidemeological studies of county cancer rates in Louisiana, Ohio, and New Jersey suggest that there is a 4-20% greater risk of getting cancer by those living where the drinking water source is surface water subject to pollution. These studies were based on 1950-1969 cancer rates, reflecting exposures between about 1930 and 1950 due to the cancer latency period. Since the levels and numbers of carcinogens in water is higher in recent years (due to factors such as rapidly expanding new chemical production), these modest increases in cancer rates are expected to become greater in the future.[24] Moreover, one study shows that organics in drinking water caused reproductive impairment, including birth defects in laboratory animals.[76f]

B. Kanawha Valley Water Supplies

All Kanawha Valley residents living downstream from the chemical plants now obtain their drinking water from the Elk River or wells, often at a higher cost, due to the chemical plant discharges on the Kanawha River. For decades, the Nitro, Dunbar, Institute, Spring Hill and Cross Lanes area was served by a drinking water plant at Nitro contaminated by chemical plant effluents. A 1962 National Cancer Institute (NCI) study produced preliminary evidence that this water was carcinogenic. Skin Application and injection of organic concentrates from this water caused cancer, including leukemia, in some mice.[15] The NCI later found that Kanawha County was a national "hot spot" for leukemia,[26] which may be related. From 1965-1974, activated carbon filters were added to this plant, but a significant

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quantity of organics still leaked through. An expensive pipeline from this area to the Elk River was built, and the supply source was switched in 1974.* The only other downstream water supply on the Kanawha, Winfield, switched to wells in 1973.[1]

A private drinking water supply, serving the employees of the large DuPont Plant at Belle, continued to be polluted by chemical plant wastes. The water intake was located below most of the plant's effluent outfalls. In the spring of 1976, WV-CAG and the union of the plant's laboratory technicians raised questions concerning the safety of this source. The EPA and the State Department of Health then requested the Company to end this practice, and the plant was tapped into the public drinking water from Elk River by the end of the year.

C. Ohio River Water Supplies

The drinking water plant on the Ohio River located closest to the mouth of the Kanawha is Huntington. This plant is one of the few to employ activated carbon filters which, if changed frequently enough (approximately monthly)[25], can effectively remove many carcinogenic organics. The Huntington filters are changed annually, with 1/3 being regenerated every four months.[28] As a result of this insufficient frequency, about 50% of the carbon tetrachloride leaked through this system during the recent spill incidents.[29]

The President of the Cabell County Commission has stated that Huntington residents are extremely unset due to the carbon tet incident and repeated problems of bad testing water from industrial discharges. As a result, sales of home water purifiers and bottled water have boomed. He suggested at a Congressional hearing that alternative water sources be explored and, if feasible, provided by the Federal Government, but that it would be far more practical to protect the present water supply.[76f]

Hamilton County, Ohio which includes Cincinnati and whose residents drink mostly Ohio River water, has a higher overall cancer rate than over 98% of U.S.

* The Elk River water plant, which now serves 200,000 customers from Belle to Nitro, has an intake located 1/4 mile above the point where the Elk joins the Kanawha. During periods of lower flow on the Elk, Kanawha River backwater containing DuPont effluent, can reach this intake.

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Counties. It has the highest nonwhite female cancer rate among major U.S. Counties. The City's Health Commissioner states that he cannot rule out the possibility that this excess cancer is due to water pollution, but that the exact relationship can never be absolutely proven. He also notes that studies of organic mixtures taken from Cincinnatti drinking water produce positive Ames Test results, indicating they are carcinogenic.[76e] Table V lists public water supplies affected by Kanawha chemical plants:

TABLE V

WATER TREATMENT FACILITIES SUPPLIED BY THE OHIO RIVER IN THE KANAWHA TO MISSISSIPPI RIVER REACH

Municipal water supply	Intake location (river mile)	Population supplied
West Virginia		
Huntington Water Corp	304.2	110,000
Huntington Water Corp	306.3	
Total		110,000
Ohio		
Ironton	327.8	15,840
Portsmouth	356.7	49,000
Cincinnati (river plant only)	462.8	850,000
Total		914,840
Indiana		
New Albany	608.6	38,000
Evansville	791.6	125,000
Mount Vernon	829.5	7,000
Total		170,000
Illinois		
Rosiclare	851.4	1,421
Golconda	902.4	922
Cave	977.3	6,277
Total		8,620
Kentucky		
Ashland	319.6	53,500
	408.4	8,360
Maysville	453.8	20,850
Newport	462.9	81,650
Covington	608.8	716,500
Louisville	851.5	45,000
Henderson	842.4	9,450
Morganfield	842.9	1,300
Uniontown	934.5	53,650
Paducah (influenced by Tennessee River)		
Total		1,081,260
Grand total		2,284,720

from ref. 76.

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CHAPTER THREE: BIOACCUMULATION

Chemical plant effluents at sublethal concentrations can cause a buildup of dangerous compounds in fish flesh. There have been only very limited attempts to evaluate the Kanawha River for these effects. In 1971, an EPA study noted that tainted flesh in catfish indicated some type of chemical contamination.[1] Subsequently, there has been a survey for the presence of polychlorinated biphenyls in fish flesh (they are not discharged by Kanawha Valley plants consequently the amounts found were not significant). Bioaccumulation should be of increasing concern on the Kanawha River as fish are returning due to higher oxygen levels. However EPA and DNR have apparently closed their eyes to the problem. A recent DNR press release boasted: "Sportsmen have noticed the improvement in the Kanawha. Only a few years ago, no one would have expected to catch any game fish from this river," says Callaghan. "Today, however, bass, walleye and channel catfish have returned. Test nettings by DNR fishery biologists indicate thriving fish populations throughout the course of the Kanawha."[80]

Bioaccumulation is the buildup of pollutants in an organism. For example, chlorinated phenols can bioaccumulate up to 10,000 times in fish. This condition is often aggravated when the pollutant concentration increases as one goes up the food chain, a condition known as biomagnification.

It would be very complex and expensive to analyze Kanawha River fish for all of the hundreds of hazardous organics and metals present. Methods for screening these compounds to pinpoint those with the greatest potential to bioaccumulate would be very helpful in conducting studies and setting standards. Since the bioaccumulation potential is usually unknown, the best way to accomplish this is to focus on those compounds with high octanol/water partition coefficients.[30]

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The octanol/water partition coefficient measures the relative amount of a compound which will concentrate in the two liquids when present in separate layers. The higher the coeffient, the greater the chemical's ability to adhere to fat, thus the potential to bioaccumulate. The coefficient is generally a good predictor when compared to actual measured buildups in fish (in a few cases factors such as metabolism change the outcome).[30]

Although octanol/water partition coefficients have been measured for only some compounds, mathematical formulas based on chemical structure have been developed to accurately calculate the coefficient. These formulas indicate that the coefficient is generally much higher for aromatic and halogenated organics (containing a benzene ring or chlorine) than most other categories.[30] For example, the coefficient is high (greater than 2) for dichlorobenzene, toluene, xylene, carbon tetrachloride, and dimethylaniline, and low (less than 1) for allyl alcohol, acrilonitrile, formic acid, and dimethyl amine.[5]

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CHAPTER FOUR: AQUATIC TOXICITY STANDARDS

Some treated Kanawha Valley chemical plant effluents meeting permit limits are still lethal to fish. Quarterly bioassay reports to EPA showed the following LC_{50} values (% dilution of pure effluent which killed half of the test fish sample):

Plant	Quarter	LC_{50}
Monsanto	3Q77	18
	4Q77	22
	1Q78	5.4
	3Q77	.75
Fike	1Q78	36
Union Carbide, Institute	3Q77	58
Dupont, Belle		

Dr. Donald Mount, Director of EPA's Duluth Environmental Research Laboratory, has stated:

> "There are tens of tousands of chemicals now in production and use. Discharges from industrial complexes contain almost an infinite array of mixtures that change with time, so that most of the toxic materials in effluents must be identified and controlled in a manner other than on a chemical-by-chemical basis. Single pollutant control is still needed for persistent pollutants that are highly toxic and that emanate from multiple sources so as to increase the concentration in receiving waters as a result of their persistence and common discharge...
>
> "For nearly all effluents, mixtures are the rule rather than the exception and toxicity tests which use organism response as a measure of effect can and should be used to characterize these effluents and limit their discharge accordingly. Detailed chemical analyses will not reveal the true toxicity of such mixtures in many instances...
>
> "In any event, it is clear that a chemical-by-chemical approach is never going to tell us whether or not the treatment given to complex wastes is adequate and whether or not there are going to be problems from particular constituents perhaps at some other time or place..."
>
> "We should control the "discharge of toxicity" by using effluent biossays in the same way that we limit the discharge of biological oxygen demand (BOD)."[76c]

Union Carbide has stated:

> "Aquatic toxicity limits should be considered when practical test standards become available."[78]

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A DuPont spokesman testified to Congress:

> "In my opinion EPA's approach will not solve the aquatic toxicity problems...Stream standards specifying a toxicity limit could be established under Section 303. In conjunction with this, the Section 402 NPDES permit program could be used to require that bioassays be performed periodically on effluent streams..."[76]

Several EPA regional offices routinely require bioassays to help identify toxicity problems in industrial effluents. Using their typical "snail's pace method" of setting standards, EPA has not yet set aquatic toxicity standards.

However, several states, including California, Washington, South Carolina (and perhaps Florida and North Carolina) regulate aquatic toxicity on effluent permits. California uses a system of "toxicity units", giving some credit to dilution effects of the receiving water. The State of Washington requires on a typical permit that a 65% concentration of wastewater be non-toxic to any salmonoid fish for 96 hours.

Since 1973 ORSANCO has encouraged Ohio Basin industries to conduct bioassays with goldfish and calculate whether there is sufficient dilution water in the river to eventually dilute the effluent to 1/20 the lethal concentration. However they state that this standard may not be adequate to protect the most sensitive Ohio River fish.[79]

A more stringent standard, giving no credit for dilution, is justified because:

1. Even if a representative local species is used, it is impractical to use or even determine the most sensitive organism in the aquatic food chain.
2. Dilution factors from lethal concentrations do not necessarily protect against chronic effects such as reproductive failure and growth impairment.

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3. Aquatic life comes into contact with concentrated effluent in the river mixing zone.

4. Reducing concentrations of chemical mixtures which are biologically active in fish will help reduce health effects in humans exposed to the same water through injestion or body contact.

5. Effluents from several plants interact in a river such as the Kanawha and can cause additive or synergistic toxic effects.

If an effluent violates the above standard, the burden of proof is placed on the polluter to do the necessary research to determine and control the combination of toxic compounds. This is very practical, if a reasonable compliance schedule is given to the polluter. The only responsibility for the regulatory agency is to determine if the test fish survive.

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CHAPTER FIVE: CONTROL OF PERSISTENT ORGANICS

July 1, 1977 EPA effluent limits for organics from chemical plants were based on "best practicable technology" (BPT) which has been defined to be a well operated biological treatment plan, where bacteria digest biodegradable organics. Kanawha Valley chemical plants have generally complied with these limits by upgrading their biological treatment plants and directing various sources of plant leaks to the treatment plant. Although this has been expensive, process changes in the manufacturing units which reduce the total amount of oxygen demanding wastes which the treatment plants must degrade, have probably reduced this cost significantly.* This is due to the recovered value of chemicals which were previously wasted, and lower costs for operating the required treatment plant. For example, in 1973 Dow Chemical found that expenditures of $37 million for all pollution control projects at their various plants actually resulted in a net savings of $10 million in the recovery of valuable chemicals.[2]

These controls result in about 95% reduction of easily biodegraded organics (as measured by the BOD_5 or five day biological oxygen demand test). However, the bulk of organics are generally not easily biodegraded, thus pass through this type of treatment and are discharged. Reference 4 presents data for a number of model chemical plants which allows this to be quantified. The BOD_5 generally makes up only about 25-35% of the total oxygen demand of the wastes or COD. The relative amount of persistent organics can be estimated by subtracting the BOD_5 from the COD, showing they make up 65-75% of the raw wasteload. The treatment plans which reduce 90-98% of the biodegradable organics, only reduce the persistent organics 0-25%. Raw and treated wasteloads for an example plant (#16 in reference 4) are as follows:

*i.e. Process changes reduced the raw BOD wasteload of Union Carbide Institute by 50% and Monsanto by 30%.[3]

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	Persistent (lbs./day COD-BOD_5)	Biodegradable (lbs./day BOD_5)
Raw Waste Before Treatment	209,000	80,000
Discharged After Best Practicable Treatment:	176,000	3,300

Many of the most hazardous organics make up this persistent fraction. For example chloroform uses up only 4% of its total chemical oxygen demand in a five day BOD test.[5] Halogenated organics tend to be more persistent and more toxic than non-halogenated organics.[76b] Toxic organics which are handled by Kanawha Valley plants and are very difficult or impossible to biodegrade (from data in refs. 5 and 6) include: dinitrotoluene, tetrachloroethylene, phthalates, formaldehyde, ethylene glycol, acrolein, crotonaldehyde, dichlorobenzene, allyl alcohol, and dichloro ethyl ether.

A treatment method which can effectively reduce most persistent organics is adsorption on activated carbon filters.[4] Chlorinated and aromatic organics, which are classes of organics most likely to be persistent and contain carcinogens and compounds that build up in fish, appear to also be the classes which are generally the most effectively reduced by activated carbon.[7] EPA at one time set its 1983 "best available technology" (BAT) effluent limits for organic chemical plants based on activated carbon adsorption and filtration of wastes discharged from a biological treatment plant. Such a system produces a 60-85% reduction in persistent organics.[4] The example chemical plant cited above showed the following discharges of persistent and biodegradable organics (lbs./day) with and without this activated carbon treatment (BAT and BPT respectively):

	Persistent	Biodegradable
BPT:	176,000	3,300
BAT:	34,000	1,210

There have been 2 estimates of the price of achieving BAT for seven Kanawha Valley chemical plants. The National Commission on Water Quality concluded the cost would be about $56 million initial, and $18 million annual operating cost. This cost was concluded to be affordable, amounting to less than 1% of the value of the

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chemicals manufactured and resulting in no significant change in employment.[8] The regional 208 study consultant did a more detailed study and concluded the cost would be even less: $35 million initial and $44 million present worth of 20 year operation cost.[78] Process changes focused on cutting down the total amount of persistent organics in the plant's most concentrated waste streams will lower this cost by recycling additional chemicals and reducing the amount of activated carbon needed.

EPA has subsequently dropped this requirement imposing BAT for organics generally (see pp 40-42) EPA replaced it with BAT only for individual designated "toxics". The added cost of imposing a BAT standard for persistent organics as a group would be less than cited above because the treatment would overlap somewhat with that already required to remove designated toxics on the EPA list by 1984. The economic health of the U.S. organic chemical industry as a whole is very good with a projected annual growth of 8% for the next several years despite government regulation.[77]

Activated carbon treatment is already being used on a limited scale in West Virginia.[9] The Mobay New Martinsville plant treats all its process effluent and the DuPont Belle, and FMC Nitro and South Charleston plants each treat one of their many waste streams with activated carbon.[3]

While activated carbon treatment of chemical plant wastes will result in a sizeable and beneficial reduction of persistent organics, a significant amount will still be left in the effluent. A goal of the 1972 Federal Water Pollution Control Act was to achieve "zero discharge of pollutants" when consistent with best available technology for an industry. Dow Chemical has achieved this in a few plants, including one at Hanging Rock, Ohio.[2] In 54 inorganic chemical manufacturing processes it is economically possible to achieve a purity of wastewater that can be recycled back to the process and not discharged.[10] Large organic chemical manufacturing complexes, such as in the Kanawha Valley, will have a more difficult time meeting the zero discharge goal, but the possibility should not be discounted.

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CHAPTER SIX: KANAWHA VALLEY CHEMICAL PLANT DISCHARGES

The FMC carbon tetrachloride incidents of 1977 may represent the largest discharges of a single known carcinogen into the Kanawha/Ohio Rivers. However, the combined impact of other carcinogens still remaining in the Kanawha effluents is much greater. Unfortunately, most of the compounds discharged have yet to be publically indentified, while many of those known to be present have not been tested for carcinogenic activity. Thus, it is much more difficult to get a handle on this problem. This section will summarize what is publically known about the Kanawha effluents relating to the toxicity issue.

A. Persistent Organic Wasteloads

The total loads of organics resistant to biodegradation are of interest for each plant because they contain those pollutants most likely to be inefficiently removed by current treatment, and remain in the river long enough to contaminate downstream fish and water supplies. Many carcinogenic and other hazardous organics tend to be persistent.

Chemical plants in the Kanawha Valley are allowed by their latest Federal permits to discharge an average of almost 50,000 lbs./day of persistent organics, with the major contributors being:

Union Carbide, South Charleston:	36%
Union Carbide, Institute:	30%
Monsanto, Nitro:	12%
Avtex, Nitro:	12%
DuPont, Belle:	9%

These calculations are based on the measurement of "Resistent Organic Carbon" (ROC) which is estimated from the Total Organic Carbon (TOC) and five day Biological Oxygen Demand (BOD) values listed on the permits. ROC is estimated by the formula $TOC - \frac{BOD}{3}$ (One third the BOD is the approximate weight of the carbon atoms contributing to the oxygen demand). Table I lists the pollution loads of the major dischargers, including ROC, TOC, COD, and BOD values or estimates.

Preliminary estimates by EPA (included as a research provision in the NPDES permits) indicate these ROC loads could be reduced between 43% and 72% by applying BAT to the entire effluent.

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TABLE I: MAJOR KANAWHA EFFLUENT LOADS

	1977 Permit Limit (Ave. lbs./day)					Preliminary 1983 Permit Limits[F]			
	ROC[A]	BOD_5	TOC	COD	Organic N	ROC[A]	BOD_5	TOC	Organic N
DuPont	4,000	21,000	11,000	33,000	2,000	2,300	2,000	3,000[E]	125
Union Carbide, S. Chas.	16,500	8,400	19,300[B]	58,000	2,500	6,500	3,500	10,000[B]	900[C]
FMC, S. Chas.					4,000[C]				1,025
Union Carbide, Institute	14,200	8,500	17,000		1,800	4,000	1,000	7,000	900[C]
Monsanto	5,800	2,200	6,500		750	3,200	600	3,400	
Avtex	5,700	3,125	6,700[B]	20,000	275	2,800	1,500[D]	3,300	
TOTAL	46,000	43,225	60,500		11,325	18,800	10,600	26,700	2,950

A. "Resistant Organic Carbon" = TOC - $\frac{BOD_5}{3}$

B. Total Organic Carbon estimated by $\frac{COD}{3}$

C. Organic Nitrogen estimated as $\frac{TKN}{2}$

D. Estimated by 50% of 1977 permit limit.

E. Estimated.

F. Estimated by EPA to be result of applying BAT to entire effluent.

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B. Toxics Inventory

Table II is a partial list of the pollutants known to be continuously or occasionally present in Kanawha chemical plant effluents, which appear to present the greatest potential hazards. They were compiled from several sources including:

- The 1975 partial analysis by EPA of organics in Kanawha effluents.
- Recent spills reported to the State of West Virginia
- 1977 West Virginia discharge permit application
- Federal Discharge Permit final limits

Amounts discharged are not known in many cases and for some compounds are estimated from concentration on the one day sampled.

Carcinogens include chloroform (3000 gallons spilled by Union Carbide Institute on November 1976), formaldehyde, methylene dianiline, dimethyl nitrosamine, sevin, bis (2 chloroethyl) ether, arsenic, hexavalent chromium, carbon, tetrachloride, phenol, dimethyl phenol, vinyl chloride, dichloro ethyl ether, asbestos, acrilonitrile, and zinc sulfate.

Several other compounds were listed as suspected carcinogens by various sources, and should be investigated further. Teratogens (birth defect producing) include phthalates, sevin, cresol, vinyl chloride, and acrilonitrile. Mutagens include cyanide, dimethylamine, ethylene oxide, vinyl chloride and carbon tetrachloride.

Chemicals known to bioconcentrate include copper, phthalates, hexavalent chromium, antimony, and zinc. In addition, some 30 organics containing chlorine or a benzene ring are suspected of concentrating in fish flesh. (See Chapter IV)

In regard to acute mammaliam toxicity some 30 compounds are listed which may exceed the State criteria to be included on the discharge application (LD_{50} less than .5 gm/kg), yet were not included on the Company's application.

Table III lists a number of compounds which have recently been reported to be handled in Kanawha Valley plants and appear to be carcinogenic or highly toxic. Most of these are probably at least occasionally discharged, and this should be investigated.

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TABLE II: TOXICS IDENTIFIED IN KANAWHA EFFLUENTS

Chemical	Ref.	Amount (lbs./day)[I]	Chronic	Bioconc	LD_{50} (gm/kg)[F]	1980 STD.
DuPont, Belle						
copper	NPDES	9		U		+
diethyl phthalate	A		TER[L]	C		+
acetone cyanhydrin	E	int.			.003	
aluminum sulfate	E	int.			.27	
aniline	E		SCARC[M]	J	.064	
antimony	NPDES	6		V		+
dimethylamine	E		MUT[M]		.24	
formaldehyde	E,H		CARC[G]		.26	
methylene dianiline	H	int.	CARC[G]		.2	
methyl mercaptan	E	int.			.002	
styrene	E			J	.316	
triethylamine	E	int.			.46	
trimethylamine	E				.09	
vanadium pentoxide	E	int.			.023	
methacrylic acid	H	int.			.048	
formic acid	H	int.			.145	
ethylene glycol	H	int.	CARC[G]			
methylformamide	H	int.	TER[G]			
dimethyl nitrosamine	K	.1	CARC[G]		.028	+
hydrogen cyanide	E	int.	MUT[N]		.001	+
ammonia	NPDES	13056				
Methyl methacrylate	H	int.	SCARC[R]			
Carbide Institute						
ammonia	NPDES	1500				
acrolein	TOX	int.				
crotonaldehyde	TOX	int.	SCARC[R]			+
dicyclopentadiene	TOX	int.				
ethylene oxide	TOX	int.	SCARC[M],MUT[G]			
sodium flouride	TOX	1664				
sevin	TOX	int.	CARC[G],TER[G]			
chloroform	H	int.	CARC[G]	J		
mesityl oxide	H	int.			.354	+
2 nitro 4 aminotoluene	A	10		J		
alkyl benzene	H	int.		J		
diphenyl oxide	H	int.		J		
bis (2 chloroethyl) ether	A	2	CARC[G]		.075	+
p dichlorobenzene	A	2	SCARC[M]	J		+
o dichlorobenzene	A	9	SCARC[M]	J	.3	+
dimethylaniline	A	11			.467	

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Chemical	Ref.	Amount (lbs/day)[I]	Chronic	Bioconc	LD_{50} (gm/kg)[F]	1983 STD.
2,6 dinitrotoluent*	A	14			.177	+
3 phenyl 3 ethyl pentane	A			J		
3 phenyl 3 methyl pentane	A			J		
3 phenyldodecane	A			J		
toluene	H	int.	SCARC[M]	J		+
phenolics	NPDES	70	SCARC[G]	J		+
FMC Nitro						
arsenic	NPDES	.4	CARC[D]			+
hexavalent chromium	NPDES	.4	CARC[O]	U		+
o - ethyl phenol	A	1		J		
toluene	A	4	SCARC[M]	J		+
phenol	NPDES	4	CARC[Q]	J		+
2,6 diisopropylphenol	A	18		J		
p - cresol	A	3	TER[Q]	J	.207x	
o - isopropyphenol	A	104		J		
2,5 dimethyl phenol	A	18	CARC[Q]	J	.444x	
2,6 dimethyl phenol	A	1	CARC[Q]	J	.150x	
Monsanto						
tetrachloroethylene	A	.5		J		+
2,6 dimethyl phanol	A	.5	CARC[G]	J	.150	
benzothiazole	A	.5		J	.1	
diisobutyl phthalate	A	1	TER[Q]	C		+
n dibutyl phthalate	A	.5	TER[B]	C		+
5 tert butyl m cresol	A	1		J		
2 methylthiobenzothiazole		8		J		
2,2,4 trimethyl 6 ethoxy quinolene diphenylamine	A			J		
t butylbenzene	A			J		
dimethylbenzylamine	A			J		
ammonia	NPDES	760			.265	
hexavalent chromium	NPDES	1	CARC[G]	U		+
mesityl oxide	O				.354	
aniline	O	750	SCARC[M]		.064	
xylene	O			J		
toluene	O		SCARC[M]	J		+
cyanide	NPDES	1	MUT[N]			+
octylamine	A				.1	
phthalates	O	835	TER[B]	C		+

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Chemical	Ref.	Amount (lbs/day)[I]	Chronic	Bioconc	LD_{50} (gm/kg)[F]	1983 STD.
Carbide - South Charleston						
vinyl chloride	P	1	$CARC^{G}, TER^{Q}, MUT^{M}$	J		+
phenol	NPDES	3	$CARC^{G}$	J		+
dichloro ethyl ether	TOX	int.	$CARC^{G}$	J		+
ethylene chlorohydrin	TOX	int.		J		
acrilonitrile	TOX	int.	$CARC^{S}, TER^{T}$	J		+
allyl alcohol	TOX	int.				
crotonaldehyde	TOX	int.	$SCARC^{R}$			
ethyl acrylate	TOX	int.				
FMC - South Charleston						
asbestos	TOX	100	$CARC^{G}$			+
carbon disulfide	TOX	130	$SCARC^{R}$			
carbon tetrachloride	TOX	150	$CARC^{G}, MUT^{M}$	J		+
chloromethylbutene	A			J		
2,3 dichloro - 2 methylbutane	A			J		
arsenic	NPDES	2	$CARC^{D}$			+
ammonia	NPDES	3472				
mercury	TOX	.015		C		+
chlorinated hydrocarbons	TOX	400		J		
2 chloromethyl 1 butane	A			J		
1 or 3 chloro 2 methylbut 1 ene	A			J		
Avtex						
zinc	NPDES	336	$CARC^{L}$	U		+

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TABLE III: OTHER TOXICS HANDLED AT KANAWHA PLANTS

DuPont, Belle[1]		
dimethyl sulfate		methylene chloride
dimethyl carbamate	methylamine	phenylene diamine
dimethyl formamide	methyl chloroformate	
lannate	methyl formate	
Union Carbide, So. Charleston		
acetaldehyde[1]	benzene[2]	glycolether[2]
butyl carbitol[1]	dioxane[2]	ethanol[2]
propylene oxide[1]	methanol[1]	methyl isobutyl ketone[2]
morpholine[1]	methyl amyl alcohol[1]	mesityl oxide[2]
ethylene oxide[2]	ethylene glycol[2]	
Union Carbide, Institute		
acetaldehyde[1]	carbowax[2]	
vinyl cyclohexene[1]	ethyl acrylate[1]	
ethylene oxide[1]	picoline[1]	
polyethylene glycol[1]	toluene diisocyanate[1]	
polyurethane[1]	temik[1]	
FMC, So. Charleston		
carbonyl sulfide	hydrogen sulfide	dichlorocyanurate
cyanuric acid	napthalene	trichlorocyanurate
Monsanto, Nitro[1]		
formalin	trichloroethylene	methyl mercaptan
hydroquinone	acrolein	toluene sulfonic acid
kerosene	carbon disulfide	trichloropropene
morpholine	cyclohylamine hexyl	
para phenetidine	hydrogen cyanide	
FMC, Nitro[2]		
phosphorous oxychloride	xylenols	
Avtex, Nitro[2]		
carbon disulfide		

1. Light. "Unregulated Air Pollutants: the Chemical Industry". 1976.
2. Montgomery Engineers, "Report on Wasteloads . . . of Major Industrial Dischargers".

note: Possible carcinogens are underlined.

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K E Y

A. EPA-NFIC, Denver. Results of Kanawha Industrial Samples. August and September, 1975.

B. Science Advisory Board. A Report, Assessment of Health Risk from Organics in Drinking Water. EPA. 1975.

C. tends to bioconcentrate according to EPA. Quality Criteria for Water. 1976.

D. EPA. Water Quality Criteria Data Book Vol.2: Inorganic Chemical Pollution of Freshwater. 1971.

E. Winterkamp. Letter to EPA. Dupont. 7/23/76.

F. acute toxicity to mammals listed as gm/kg if less than .5 and not included on State application. LD_{50} taken from reference G.

G. NIOSH. Registry of Toxic Effects of Chemical Substances. 1975.

H. WV Division of Water Resources. Industrial Spill Files. 1977.

I. From references A, O, and P amount estimated from concentration. From NPDES permit amount is the final average limit.

J. in general class of compounds which tend to biocentrate according to: Neely et al. "Predicting Bioconcentration Potential." Env. Science and Technology. Dec., 1974.

K. DuPont. "Dimethylnitrosamine Sampling Program 12/1-5/75."

L. NIOSH. Registry of Toxic Effects of Chemical Substances. 1976.

M. Dorigan et al. Preliminary Scoring of Selected Organic Air Pollutants. EPA. 1976.

N. Epstein and Legator. The Mutagenicity of Pesticides. 1971.

O. Monsanto. Plant Wastewater Pollution Analysis. 4/1/76.

P. EPA. Discharge Monitoring Reports. 1976.

Q. EPA. STAR Report on Vinyl Chloride. 1975.

R. Harris et al. "Carcinogenic Hazards of Organic Chemicals in Drinking Water." Environmental Defense Fund. 1976.

S. NRDC Newsletter. "The Plastic Bottle Hits a Snag." Vol. 6 #1. Jan., 1977.

T. Federal Register. 3/11/77. p. 13564.

U. tends to bioconcentrate according to: Mathias "Selected Metals in Sediments, Water and Biota in the Illinois River" J. Water Pollution Control Fed. Vol. 45. 7/73.

V. tends to bioconcentrate according to: EPA. "Physical, Chemical, and Biological Considerations with respect to the DuPont Permit for Ocean Dumping". 1974.

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K E Y (Cont'd.)

CARC: Produces tumors in humans or test animals.

TER: Causes birth defects in test animals.

SCARC: Suspected of being a carcinogen in at least one reference.

MUT: Produces gene mutations in test organisms.

* may be 2, 3 dinitro toluene

** may be m dichlorobenzene, LD_{50} .5 gm/kg

\+ effluent standards based on best available technology to be set by 1983 under NRDC vs. Train Court settlement.

NPDES: limited on Federal permit.

TOX: listed as being highly toxic to mammals on application for West Virginia Permit.

x not reported by FMC to DNR because considered to be included in "phenolies" reported.

int.: compound discharged intermittently during spill situations.

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C. The Carbon Tetrachloride Incident in Perspective

The FMC South Charleston Plant has made carbon tetrachloride (CCl_4) for fifty years and has at least from 1975-1977 lost large quantities in its effluent. Initiation of monitoring for the chemical in downstream water supplies finally convinced Federal officials to take the precedent setting step of limiting it as a carcinogenic water pollutant.

The plant discharges CCl_4 continuously (from the decanter and sulfur wash units) and also intermittently loses larger quantities during leaks and spills.[49] FMC reported to EPA in 1971 that there was 70 lbs./day in its effluent.[50] In 1974, EPA ignored a request by Campaign Clean Water to limit CCl_4 on FMC's new Federal discharge permit. In 1975, EPA found that the Huntington drinking water had the highest level of CCl_4 in the U.S. (3ppb) and subsequently traced the major source to an FMC outfall.[75] FMC reported in October of that year it was discharging more than 200 lbs/day. EPA again turned down requests to regulate it as a carcinogen. Surveys in 1976 found even higher levels in downstream drinking water, 10 ppb in September at Huntington,[50] and about 10 ppb in Cincinatti beginning in February.[51] EPA then initiated a major study of all potential dischargers. In January 1977 FMC reported discharges of 4000 lbs./day. In early February, there was 86 ppb in the Cincinnatti drinking water.[50]

The public did not find out about all this until mid February, when EPA detected 340 ppb in the Kanawha and 240 ppb at Huntington. They stated that a 70 ton slug was moving down the Ohio River, and warned downstream residents to boil water to evaporate CCl_4. Unfortunately, delays in analyzing the samples and an underestimate of river flow resulted in EPA issuing the warnings to cities after the major slug had passed. In late February, FMC announced a spill of 5300 lbs./ CCl_4 and this time, downstream water suppliers were able to either temporarily close their intakes or urge people to boil their water. EPA then inspected the FMC plant, finding corroded tanks and pipes, and discovering that FMC had reported 20 spills of CCl_4 to the State since 1972.[50]

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In early March, EPA got a restraining order to close the plant under the imminent hazard provisions of the Safe Drinking Water Act and Federal Water Pollution Control Act. Within a week, a consent agreement was worked out and no employees were ever layed off.[50] The agreement required installation of facilities to catch and hold any spill, activated carbon treatment of the decanter unit effluent, and upgrading of tanks and pipes. CCl_4 must be monitored hourly, with an interim limit of 150 lbs./day. By 1978, there must be activated carbon treatment on the sulfur wash unit, and the discharge will be limited to about 25 lbs./day.[49] In April, 1977, EPA charged FMC with violating the agreement by discharging excess CCl_4 through prohibited outfalls.[52] Cincinatti sued FMC for over $6 million in damages,[53] and Evansville, Indiana initiated a similar damage suit.[54] FMC has announced a phasing out of the CCl_4 unit in 1979, with no plant layoffs forecast. This appears to be more related to the reduced fluorocarbon market (CCl_4 is a raw material) than water pollution control.

CCl_4 has been known to induce liver cancer in mice for over thirty years.[16] A recent study based on animal data indicated that the presence of 10 ppb in drinking water could cause 2 cancer cases per 100,000 population.[18] An EPA expert witness in the recent legal proceeding stated that the level of CCl_4 found in drinking water posed a serious carcinogenic hazard, and there was no known method for setting a safe exposure level.[55]

In full page newspaper advertisements, FMC claimed that a person would have to drink 3000 gallons of water to injest the lowest amount of CCl_4 affecting experimental animals, and that there was no evidence that their spills damaged human health.[56] However, FMC's independent toxicologist who developed this information later admitted it was based on only acute liver damage, not long term effects.[57]

D. Nitrosamines

Nitrosamines are an extremely potent class of carcinogens which are present in trace amounts in certain Kanawha effluents as a by product of the manufacture of various chemicals. The plants also discharge large quantities of amines, which may lead to nitrosamine formation downstream or in the digestive tract.

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At the lowest dose tested, .075 mg/kg/day, injestion of dimethyl nitrosamine (DMN) caused tumors in small test populations of rats. Nitrosamines are also potent teratogens (causing birth defects).[44] EPA extrapolated from the rat data that a small continuous emission of the substance into the air by the DuPont Belle Plant caused excess cancer cases at a rate of about 30 per 100,000 people in the surrounding area. Pollution control was subsequently ordered for the air emission source.[45] The carcinogenic effects of nitrosamines can be enhanced greatly by the presence of other chemicals. For example, a mixture of DMN and carbon tetrachloride had a synergestic effect on rats.[48]
The presence of DMN is suspected by NIOSH as contributing to the DuPont Plant's high kidney cancer rate.[22]

DuPont's treatment plant effluent was found in December 1975 to contain up to 8 ppb of DMN,[46] amounting to a discharge of about .1 lb./day. In the summer of 1976, the most concentrated source of DMN to the treatment plant (the "small lots" scrubber wastewater) was being at least temporarily stored, but other plant units are apparently also contributing to the effluent DMN. At a hearing on the DMN air control plan, DuPont offered no comparable specific program to eliminate DMN in its effluent.[45] DMN has also been found at the 3 ppb level in the Fike Chemicals effluent in Nitro, W.Va.,[44] and as a contaminant in a hydraulic fluid made at the Union Carbide South Charleston Plant.[47] The production of amines at the Union Carbide South Charleston and Institute Plants also makes their effluent suspect of containing nitrosamines.[44]

DMN has been found to form in lake water from a reaction between secondary amines and nitrate. Natural secondary amine concentrations are low, and an accumulation from other sources could result in spontaneous nitroamine formation.[44] The Kanawha River generally contains about 3 ppm nitrate (mostly from natural and municipal sources). Federal effluent limits allow the Kanawha plants to discharge an average of 11,000 lbs./day organic nitrogen, much of this being various amines. The major contributors are FMC South Charleston, Union Carbide South Charleston, DuPont Belle, Union Carbide Institute, and Monsanto (see Table I). With the only river sampling for DMN thus far being a few grab

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samples in Charleston and Chelyan, it is not clear whether downstream DNR is significant.

Likely to be much more significant is the contamination of Ohio River water supplies with these amines, and the subsequent internal formation of nitrosamines. Saliva contains nitrite which can react in the acidic conditions of the stomach to form nitrosamines when certain amines are injested.[18] This reaction of amines and oxides of nitrogen in the digestive tract has been demonstrated in animals and shown to lead to tumor formation.[48]

For the many years that the FMC South Charleston plant has made chlorine using the diaphragm process, they discharged asbestos. This material is used as a filter in the manufacturing unit, with a significant amount being lost in the effluent.[34] FMC's State Permit Application estimated 100 lbs./day were discharged. Filtration may remove about 99% of this material,[34] and the Company apparently initiated such a control process recently.[36]

Asbestos is well established to be carcinogenic in exposed workers. As a result, a Federal Judge ruled in 1974 that the discharge of a large quantity of asbestos by Reserve Mining "substantially endangered" the public health in cities drinking Lake Superior water. A pollution elimination schedule was ordered, and additional filtration of Duluth drinking water was initiated.[36]

F. Vinyl Chloride

According to 1976 monitoring reports submitted to the EPA under their Federal permit, the Union Carbide South Charleston Plant discharges about 1 lb./day of vinyl chloride. No standard has been set for this water pollutant.

Vinyl chloride is a proven carcinogen. For example, at the Union Carbide Plant where it is used to make plastic, at least 5 workers have died from angiosarcoma, a rare form of liver cancer.[37] An EPA model based on animal data estimated that air pollution outside all U.S. polyvinyl chloride plants would cause 1-10 cases/yr. of angiosarcoma and an equal number of cases of cancer of other organs.[38] More recent animal test data found that vinyl chloride was even

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a more potent carcinogen, producing tumors at 1/50 the concentration of the lowest previous study.[39] Vinyl chloride is also believed to cause mutations in exposed workers' chromosomes and cause a significant increase in fetal death of their offspring.[40] Occupational and air pollution standards have been set based on best available technology to reduce vinyl chloride air emissions.

Regarding water pollution, an EPA report states that the risk of injesting vinyl chloride is unknown, but it is prudent to assume it will have the same effect as inhalation. Bioaccumulation may also be possible.[41] However, EPA has not set an effluent standard because they feel that vinyl chloride tends to quickly leave a solution and escape into the air, posing no downstream danger.[42] However, it should be noted that vinyl chloride has a virtually identical evaporation rate from solution as carbon tetrachloride,[43] which has been found to persist down the Ohio River from upstream discharges.

G. Chlorinated Ethers

EPA sampling indicated several years ago that bis (2 - chloroisopropyl) ether, a probable carcinogen, is discharged in significant quantitites to the Kanawha, and contaminates Ohio River water supplies. In November, 1971, none was detected above the Kanawha Chemical plants, while 12 ppb was found at the Kanawha's mouth. .4 ppb was found in the Ohio River upstream of the Kanawha, while 1.3 ppb was found at Huntington and .8 ppb at Cincinatti.[33] The Union Carbide South Charleston and Institute Plants are suspected sources since they handle related compounds.

An EPA assessment of bis (2 - chloroisopropyl) ether said in 1975 that it is a highly persistant compound which, although not yet tested, is expected to be a potent carcinogen due to its close structure-activity relationship to bis (2 chloroethyl) ether.[23] It was recently estimated from animal data that each part per billion of bis (2 - chloroethyl) ether in drinking water causes 1 additional cancer case per 100,000 people.[18]

The 1975 EPA Kanawha effluent organics survey noted that the Union Carbide Institute Plant discharged bis (2 - chloroethyl) ether at a concentration which amounts to about 2 lbs./day. The Union Carbide South Charleston Plant noted they handled dichloro ethyl ether in its application for a State Discharge Permit.

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CHAPTER SEVEN: REGULATORY POLICIES

A. State Program

The W. Va. Division of Water Resources approach to toxic pollutant control has been very limited and can generally be described by the attitude, "What we don't know can't hurt us." The former Chief of DWR was quoted in the press as claiming that there are no hazardous materials in the Kanawha River present in sufficient quantities to harm fish or humans, "How many people have died of carbon tetrachloride poisoning. If they have, I haven't heard of them."[58] When a major slug of carbon tetrachloride had passed, but there was still 4ppb measured in the Ohio River, the Chief stated, "For all practical purposes there is none there. . .There is no problem in the Ohio River,"[59] These quotes indicate a lack of appreciation for the potential long-term hazards of having low levels of carcinogens present in drinking water and fish.

Even though DWR has the responsibility for overseeing the discharges of some two dozen chemical plants, its personnel apparently do not have any references summarizing the known toxic effects and possible carcinogenicity of most of the pollutants involved.

The Division apparently does not have the NIOSH Registry of Toxic Effects of Chemical Substances (the most up-to-date and comprehensive listing of chemical health effects) and appears to depend only on the EPA and California water quality criteria books (which are both very incomplete and outdated in the area of chronic toxicity).

For example, the FMC, South Charleston application listing asbestos had been submitted for over a year without DWR apparently realizing it was carcinogenic. Two months after WV-CAG pointed this out to DWR, they had not yet determined if recently installed pollution controls had taken care of the problem.

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The only W. Va. water quality standards actually violated by the Kanawha Chemical effluents are dissolved oxygen (only during low flow conditions) and phenol. The Kanawha River phenol standard of 1ppb was set for taste and odor control and has been frequently violated for years. However since phenol appears to be at least a co-carcinogen, it is imperative that this standard be met. 1977 phenol limits on the plant discharge permits may not be stringent enough to achieve this. EPA proposed, but the W. Va. Water Resources Board failed to adopt, in 1977, water quality standards for ammonia (20 ppb) and phthalates (3 ppb) to protect aquatic life. The chemical plant effluents would probably violate those standards as well.

Regarding fish toxicity, the W. Va. standards require only that an individual pollutant not exceed 1/10 the concentration lethal to 50% of test fish (Tlm) after full dilution with the river. A stricter standard was deleted by the W. Va. Water Resources Board in 1977, requiring any combination of pollutants not to exceed 1/10 the Tlm within the effluent mixing zone.

DWR's policy for regulating pollutants not listed in the West Virginia standards is vague and basically depends on the Companies' good faith. A DWR official recently said that the State has not found anything to indicate that unreported toxics are being discharged, and that if the chemical companies had correctly answered the questions on their permit applications, there was no reason to be concerned with toxic discharges.[60] Question B(2) of the W. Va. Industrial Waste Disposal Application requires each company to list any compound in its effluent which has a high acute toxicity to rats (LD_{50} of .5 gm/kg or less). This definition ignores many carcinogens which do not have a high acute toxicity. Moreover, none of the Kanawha plants appear to have fully answered even that limited question. Table II lists the following number of toxic compounds at each plant with a high acute toxicity to mammals thought to be at least occasionally discharged, which the companies failed to include in their applications:

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DuPont, Belle	(15)
Union Carbide, Institute	(5)
Monsanto, Nitro	(5)

Each of these unreported pollutants is signified in Appendix III by the presence of a value in the column headed "LD_{50}". Although the toxicity listed is for any one of several species of mammals, most of these compounds probably violate the specified level of .5 gm/kg for rats, thus represent an apparent "failure to report violation." There are many more compounds actually discharged into the Kanawha than were idenitifed in the references used to develop the list in Appendix III. Thus these apparent violations represent just the tip of an iceberg.

The State permits generally require that "efforts shall be put forth to considerably reduce all compounds with a rat oral single dose LD_{50} of .5 mg/kg or less". Most of the compounds listed by the companies in this category are described as being present only in trace amounts or during spill situations and few, if any, toxic pollutant effluent limits have actually been set by DWR using this approach.

One example of an enforcement action regarding toxic pollutants took place in 1977 when DWR finally cracked down on a small Nitro pesticides manufacturing plant which had discharged toxic wastes without a permit for 22 years.[61] DWR sampling showed the Chemical Formulators effluent had a high level of phenol (450 ppm[61]) and methoxychlor (5 ppm [62]). The sample was

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taken from the final treatment pond which was discharged later that day. DWR rejected an application by the Company for a permit because the methoxychlor can be toxic to fish at only a few ppb, and the phenol could violate the river standard for that pollutant.[62]

Many spill incidents have been reported to DWR caused by leaks at the Kanawha chemical plants, yet only a few have resulted in fines. The most frequent recent offender appears to be the DuPont Belle Plant. That plant has apparently not been fined for a spill since 1968.[31] Yet a DWR letter to the Company called the 16 spills reported in 1976 and 1977 "excessive" and stated that some were "inexcusable." Six were caused by operating errors, six by valve and pump failure, and four by cooler leaks.[32] Some of the compounds involved in recent spills are listed in Table II. The state spill enforcement does have one advantage over the federal in that all industrial chemical plants are regulated, subject to fines and a spill prevention plan if an incident occurs.

B. Kanawha Chemical Plant NPDES Permits.

The first permits for the Kanawha chemical plants under the Federal Water Pollution Control Act were drafted in 1974, and basically required a reduction in oxygen demanding wastes, heavy metals, and phenols. At a public hearing on the draft permits, Campaign Clean Water pointed out that a number of carcinogens were being handled at the plants (including carbon tetrachloride and asbestos at FMC) and should be limited on the permits. EPA responded that further study was needed of the toxic pollutant question. The only carcinogen added was vinyl chloride, to the Union Carbide, So. Charleston

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Plant permit, and that was only a monitoring requirement. All plants were to complete by 1978 an inventory of organics in their discharges. Also added at CCW's request was a prohibition against any combination of pollutants lethal to fish, where each plant would have to show it was meeting the W.Va. mixing zone standard by conducting effluent bioassays.

All the major Kanawha chemical firms appealed their Federal permits, winning significant concessions in an out of court settlement with EPA. Among the changes in the permits was replacement of the bioassay standard with just a monitoring requirement, deletion of the organics monitoring for DuPont and FMC, and increases in the permissible levels of oxygen demanding wastes at each plant (to a point where the River dissolved oxygen standard will probably be violated during low flow conditions).

Since issuance of those permits, EPA made an additional chemical inventory requirement of the DuPont Plant, (in the form of an information "subpoena" under section 308 of the Act). In response to repeated spills at the plant in 1976, DuPont was asked to identify which of an EPA list of 400 potentially hazardous compounds were handled there (33 were), which were discharged continuously(17 were listed as being sent to the waste treatment plant but discharge amounts were not given for most) and spill prevention measures being taken. The DuPont response added, "It should be recognized that within a large chemical complex such as the Belle plant, there are compounds present which have not been identified."[65]

The EPA plan to regulate organics at the Kanawha plants includes some or all of the following:[66]

- Identification of organics in each effluent as required either by the permit or (in the case of FMC and DuPont) by a "308 letter".

- An inventory and accounting for hazardous compounds handled at each plant.

- Additional organics surveys by EPA (they did a partial analysis of some effluents in 1975).

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In developing these monitoring and inventory requirements, EPA is focusing primarily on their list of 129 toxic pollutants, which do not necessarily include all the problem pollutants of the Kanawha plants. Data in Table II indicates that only about half of the known toxic pollutants identified thus far at Kanawha plants are actually included on the EPA list of 129. EPA recognizes that there are carcinogens and other dangerous chemicals discharged into the Kanawha which were not included on their lists.[67]

The Regional EPA office has denied a WV-CAG request to include Ames Tests as a means of estimating the carcinogenic potential of the unique mixtures of compounds present in each Kanawha effluent. In refusing to use this test, EPA stated that it was only suited for pure compounds, and often gave false positive or negative results.[67]

C. Federal Program

Considerable controversy revolved around two sections of the FWCPA passed by Congress in 1972. Section 307 required very strict standards for toxic and carcinogenic pollutants to be met within a year of their promulgation, and section 301 required best available control technology economically achievable (BAT) for all pollutants (including persistent organics) to be installed by 1983. Only very limited standards for a few pollutants have been set under the stringent provisions of section 307. This resulted in a major law suit by environmental groups against EPA. The outcome of the suit was a settlement which recognized that more implementation time and reliance on technology based standards was needed to control toxics. EPA was thus ordered to add 129 toxic pollutants, including many carcinogenic organics, to the list of parameters for which BAT effluent standards would be set for various industrial categories, including all chemical plants, and enforced under section 301. The limits must now be met no later than 1984.

A concerted effort was launched by chemical companies and others to convince Congress to delay or eliminate the general 1983 BAT

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standards. A majority of the industry-oriented National Commission on Water Quality voted that the costs of BAT were not worth the benefits, and supported a 5 to 10 year delay of the BAT deadline.[73] A minority of that Commission supported BAT and the 1983 deadline, stressing that there will be benefits not measured in terms of the traditional water pollution parameters considered by the majority. Among the reasons cited as justification for this view were:

- BAT will for the first time require control of chlorinated and other harmful organics which affect water supplies but not oxygen demand.
- BAT will help reduce the potential hazards of complex pollutant mixtures for which it is not possible to set water quality standards.
- BAT will help mitigate the effects of population and industrial growth.
- BAT will encourage innovative technology.
- Installation of BAT controls would ~~not~~ adversely affect the economy.[73]

EPA urged Congress to retain the 1983 BAT deadline because it is vital to the control of toxic pollutants.[64]

Unfortunately, Congress went ahead in 1977 to pass a major revision of the 1983 BAT requirement. The new federal amendment basically sets out three categories of pollutants:

- "Conventional pollutants," to be defined by EPA, which will essentially only have to continue to meet "best practicable control technology" effluent limits.
- "Toxic pollutants," as defined by the 129 compounds listed in Table IV, which will require best available technology by 1984, without waivers.

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- "Nonconventional pollutants," which includes everything not specified in the above two categories. These will be subject to best available technology by 1984, but with waivers allowed on economic grounds if sufficient environmental protection can be shown.

Regarding organics, EPA has proposed to implement the new amendment by requiring additional controls only for those on its list of 129. They have done this by proposing to classify COD as a conventional pollutant. They are considering having COD used as an "indicator" for designated toxics in some cases, but this is only in cases of a simple effluent containing only a few organics, not a complex chemical plant. Since the decision is not yet finalized, there is a possibility that EPA could redesignate COD a "nonconventional pollutant" which would satisfy the concern that persistent organics be subjected to the best available control technology.

EPA will probably not reclassify COD, thus chemical plants will be able to comply with the next round of NPDES permits by simply installing process changes or filtration in those relatively few areas of the plant complexes which handle the designated toxics (from the list of 129). Thus the estimated 50,000 lb./day wasteload of persistent organics now discharged by Kanawha Valley chemical plants will be reduced very slightly if at all.

Spill prevention and enforcement is regulated under section 311 of the Federal Act. Unfortunately, the way this was written by congress and interpreted by EPA and the Courts, only a relatively small portion of industrial chemicals subject to spills are actually regulated. All other non-designated chemicals are not subject to

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reporting and fines if a spill occurs and, more importantly, are not subject to approved spill prevention plans (i.e., dikes around tanks and precautions on barges, etc.). The shortcomings of the EPA list of hazardous chemicals designated for spill enforcement is highlighted by the fact it does not include some of the toxics from the list of 65 EPA is setting effluent standards for and some chemicals addressed in Safe Drinking Water Act standards.[76]

Regarding Kanawha River chemical spills, an EPA official stated that the 1977 carbon tetrachloride slug was unique only in that for the first time it was analyzed and followed as it went down the Ohio River. He said that many toxic slugs have gone down the Ohio undetected in the past thirty years.[70] EPA has been unable to take enforcement actions against several other recent Kanawha chemical plant spills because its regulations under section 311 of the FWPCA have not been finalized. Meanwhile, the interstate organization ORSANCO, is requesting EPA funding to continue a network of 10 gas chromatograph monitoring stations in the Ohio River Basin to help detect and track future spills.[71]

Problems with enforcing section 311 are apparently due to the documentation required for each chemical placed on the official "hazardous pollutant" list. The original law passed in 1972, but did not propose a list of "hazardous pollutants" until 1974, then this took an incredible four years to finalize. Then it was overthrown by Federal Court around September 1978. Another list of approximately 300 chemicals is expected to be proposed in 1979. This does not include dozens of chemicals handled by Kanawha plants known to be dangerous and subject to spills, including dimethyl sulfate, butanol, ethylene oxide, toluene diisocyanate, crotonaldehyde, and propylene glycol. These are simply not regulated by EPA because

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they are either not a national problem, or they did not have the resources to put together adequate documentation. More significantly, it should be understood that even relatively "safe" industrial chemicals can cause problems in spill situations. Thus there is justification for regulating at least to some degree, the discharge of all industrial chemicals.

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CHAPTER EIGHT: STATEMENTS IN SUPPORT OF BEST AVAILABLE TECHNOLOGY TO TO CONTROL PERSISTENT ORGANICS

"Only 10% of the total organic contamination of our drinking water has been identified and of those 309 identified compounds, 65% have never been tested for chronic toxicity of cancer. Said another way, greater than 95% of the organic chemicals currently in Cincinnati's drinking water have never been tested for their chronic toxicity nor for their possible carcinogenic potential. We do know, however, that in collaborative studies, done by EPA, the University of Cincinnati and the Stanford Research Institute, mixtures of organic compounds extracted from Cincinnati's drinking water produced mutations in bacterial systems used as screening tools for chemicals causing cancer - an ominous sign indeed. To complicate the situation further, even less is known about the interaction of these small amounts and the health effects thereof.

It is therefore quite clear that we will have the technology available (we almost do already) to stop the pollution of the Ohio River long before we will have all the information needed to decide the degree of these organic contaminants. Therefore, it is imperative that the federal government require that polluters use all available technology and skills to decrease the introduction of these chemical contaminants into the river and help local governments remove any pollutants that arrive by other means." Dr. Arnold Leff, Cincinnatti Health Commissioner[76e].

"The fact that most of the toxic organic chemicals listed are relatively nonbiodegradable indicates that general limitations on BOD are insufficient in themselves to limit the quantity of toxic chemicals which are discharged. . . The best available control technology requirements of 1983 had as their major benefits the control of toxic pollutants. BAT technologies, such as activated carbon filtration, are specially designed for removal of recalcitrant chemical pollutants." Dr. Robert Harris, Environmental Defense Fund.[76f]

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"Two principal strategies exist in current laws. The first is the pollutant-by-pollutant assessment of effects, which is the basic strategy of Section 307(a) of the FWPCA and of the SDWA, but which has had only limited success, e.g., it has resulted in standards for only 6 toxic chemicals under Section 307(a). The second strategy involves the installation of best-available-control-technology on an industry-by-industry basis, as in the permits system of the FWPCA, so far used mainly to control traditional pollutants. Most witnesses agreed that the task of identifying every possible toxic contaminant and establishing a standard for each would be a monumental if not impossible task given the infinite number of chemical combinations and data and resources limitations." Staff summary of House of Representatives—1977 Hearing on Toxic Water Pollutants. 76i

"It is quite reasonable to assume that there are halogenated organics that may be equally or more toxic than those appearing on the list in the NRDC-EPA consent decree...

Our view [is] that the only sure way of dealing with toxic pollutants is to achieve (eventually) complete abatement...The Senate language [1977 Amendments to FWPCA] may not allow enough flexibility...Since BAT regulations often will cover toxic pollutants not on the lists mentioned above, the Administrator may want more flexibility...Conventional pollutants often serve as surrogates for control of toxic pollutants. It would be important to be able to include this consideration in any determination regarding the waiver from BAT for conventional pollutants." EPA Response to questions from Congressman Bo Ginn. 76

"I think your suggestion is an excellent one and entirely consistent with the spirit and intent of the section of the bill to which you referred. I will bring it to the attention of the conference committee now meeting on the legislation and try to work out some sort of agreement which will implement the concept you have in mind." Senator Jennings Randolph (letter to Ed Light 11/3/77 in response to a request that the 1977 Amendments to the FWPCA not allow a waiver from BAT standards for persistent organics).

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"Thanks for letting me know of your concern over proposed listing of chemical oxygen demand (COD) as a "conventional pollutant".

I share your reservations about this proposal and your view that, if adopted, it could weaken industrial water pollution control efforts to a significant degree and make it much more difficult to regulate chemical toxic pollutants.

To the degree that it occurred, such a result would run directly counter to the intent and spirit of the Clean Water Act of 1977, and would represent a major setback in the effort to improve the Nation's water quality." Senator Jennings Randolph (letter to Ed Light 4/20/78) in response to request that he comment on EPA's decision to classify COD as a conventional pollutant.)

"The following water quality objectives must be achieved by 1983 [for Kanawha Valley chemical plants]:

Best Available Control Technology Economically Achievable for persistent organics (as included in a general parameter such as total organic carbon)..."

- Resolution endorsed by 5 members of Charleston area 208 study industrial waste advisory committee.[78]

"BAT controls on [chemical] oxygen demanding wastes are an alternative means of controlling the discharge of toxic materials contained in those wastes and of controlling nontoxic organic pollutants in our waters that combine with chlorine in the drinking water treatment process to produce deadly carcinogenic substances... Implementation of BAT could reduce oxygen demanding loads by...over half the industry contribution...Reducing industrial oxygen demanding loads to this extent would result in significant reductions of toxic compounds." Jim Banks, Attorney for Natural Resources Defense Council (NRDC).[76g]

"The Subcommittee found that EPA has not vigorously exercised its authority to issue regulations to protect the public from organic chemical contaminants in drinking water. EPA failed to set maximum contaminant levels or to impose treatment

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technique requirements for total concentrations of organic chemicals thereby prolonging public exposure to potentially dangerous substances. And, EPA did not utilize proper procedure in reacting to the carbon tetrachloride contamination incident... We recommend that EPA be given authority to require pollution discharge testing by companies to prove safety of discharged materials, require monitoring without first having set maximum contaminant levels, and require reporting and public notice of all contaminants including unregulated contaminants.

The report also recommends that EPA take rapid steps to promulgate total organic chemical contaminant levels, create a national early warning system for major surface water streams, require industrial reporting of known and suspected toxins and carcinogens and promulgate regulations immediately for hazardous substances spills as required by the Federal Water Pollution Control Act." Congressman John Moss in 9/27/77 letter to Harley Staggers.

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CHAPTER NINE: STATEMENTS IN SUPPORT OF SPILL CONTROLS FOR ALL CHEMICALS

Congressmen Miller: "When section 311 is fully implemented, can we expect incidents such as the carbon tet and the HCP incidents to cease?"

EPA: You will continue to have spills because you will have substances, out of the thousands of chemical substances, that you have not gotten around to putting on the list yet." Jeff Miller, Enforcement Director, EPA.[76]

"The 306 Compounds designated as hazardous substances...do not necessarily cover all the hazardous substances handled by the industrial plants in the Kanawha Valley." Charleston Area 208 Study.[78]

"Concerning the problem of spills, while some 300 chemicals are on the proposed Section 311 list, most of the severe contamination events have involved unlisted chemicals. This does not necessarily mean the list was poorly prepared: what it means is that of the thousands of chemicals produced, used, and disposed, it is very difficult indeed to predict which will in fact be mishandled or spilled. This suggests the need for some authority requiring reporting and cleanup even though a spilled substance is not listed." House Subcommittee Report on Toxic Water Pollutants 9/77/ [76g].

"An acceptable spill prevention and control plan shall be submitted to EPA for all compounds handled by Kanawha Valley chemical plants. Any subsequent industrial spill of these compounds in the B-C-K-P Region shall be considered a violation of section 311 of PL 92-500." Resolution endorsed by 5 members of Charleston area 208 Study Industrial Waste Advisory Committee.

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TABLE IV. EPA DESIGNATED TOXIC POLLUTANTS

RECOMMENDED LIST OF 129 PRIORITY POLLUTANTS COMPOUND NAME, APRIL 29, 1977

(1) *acenaphthene; (2) *acrolein; (3) *acrylonitrile; (4) *benzene; (5) *benzidine; (6) *carbon tetrachloride (tetrachloromethane).

*CHLORINATED BENZENES (OTHER THAN DICHLOROBENZENES)

(7) chlorobenzene; (8) 1,2,4 trichlorobenzene; (9) hexachlorobenzene.

CHLORINATED ETHANES (INCLUDING 1,2 DICHLOROETHANE, 1,1,1-TRICHLOROETHANE AND HEXACHLOROETHANE)

(10) 1,2 dichloroethane; (11) 1,1,1-trichloroethane; (12) hexachloroethane; (13) 1,1-dichloroethane; (14) 1,1,2 trichloroethane; (15) 1,1,2,2-tetrachloroethane; (16) chloroethane.

*CHLOROALKYL ETHERS (CHLOROMETHYL, CHLOROETHYL AND MIXED ETHERS)

(17) bis(chloromethyl) ether; (18) bis(2-chloroethyl) ether; (19) 2-chloroethyl vinyl ether (mixed).

*CHLORINATED NAPHTHALENE

(20) 2-chloronaphthalene.

*CHLORINATED PHENOLS (OTHER THAN THOSE LISTED ELSEWHERE; INCLUDES TRICHLOROPHENOLS AND CHLORINATED CRESOLS)

(21) 2,4,6 trichlorophenol; (22) parachloromeda cresol; (23) *chloroform (trichloromethane); (24) *2-chlorophenol.

*DICHLOROBENZENES

(25) 1,2-dichlorobenzene; (26) 1,3-dichlorobenzene; (27) 1,4-dichlorobenzene.

*DICHLOROBENZIDINE

(28) 3,3'-dichlorobenzidine.

*DICHLOROETHYLENES (1,1-DICHLOROETHYLENE AND 1,2-DICHLOROETHYLENE)

(29) 1,1-dichloroethylene; (30) 1,2-trans-dichloroethylene; (31) *2,4-dichlorophenol.

*DICHLOROPROPANE AND DICHLOROPROPENE

(32) 1,2 dichloropropane; (33) 1,2-dichloropropylene (1,3-dichloropropene); (34) *2,4-dimethylphenol.

*DINITROTOLUENE

(35) 2,4-dinitrotoluene; (36) 2,6-dinitrotoluene; (37) *1,2-diphenylhydrazine; (38) *ethylbenzene; (39) *fluoranthene

*HALOETHERS (OTHER THAN THOSE LISTED ELSEWHERE)

(40) 4-chlorophenyl phenyl ether; (41) 4-bromophenyl phenyl ether; (42) bis(2-chloroisopropyl) ether; (43) bis(2-chloroethoxy) methane.

*HALOMETHANES (OTHER THAN THOSE LISTED ELSEWHERE)

(44) methylene chloride (dichloromethane); (45) methyl chloride (chloromethane); (46) methyl bromide (bromomethane); (47) bromoform (tribromomethane); (48) dichlorobromomethane; (49) trichlorofluoromethane; (50) dichlorodifluoromethane; (51) chlorodibromomethane; (52) *hexachlorobutadiene; (53) *hexachlorocyclopentadiene; (54) *isophorene; (55) *naphthalene; (56) *nitrobenzene.

*NITROPHENOLS (INCLUDING 2,4-DINITROPHENOL AND DINITROCRESOL)

(57) 2-nitrophenol; (58) 4-nitrophenol; (59) *2,4-dinitrophenol; (60) 4,6-dinitro-o-cresol.

*NITROSAMINES

(61) N-nitrosodimethylamine; (62) N-nitrosodiphenylamine; (63) N-nitrosodi-n-propylamine; (64) *pentachlorophenol; (65) *phenol.

*PHTHALATE ESTERS

(66) bis(2-ethylhexyl) phthalate; (67) butyl benzyl phthalate; (68) di-n-butyl phthalate; (69) di-n-octyl phthalate; (70) diethyl phthalate; (71) dimethyl phthalate.

*POLYNUCLEAR AROMATIC HYDROCARBONS

(72) benzo(a)anthracene (1,2-benanthracene); (73) benzo (a)pyrene (3,4 benzopyrene); (74) 3,4-benzofluoranthene; (75) benzo(k) fluoranthene (11,12-benzofluoranthene (76) chrysene; (77) acenaphthylene; (78) anthracene; (79) benzo(ghi)perylene (1,12-benzoperylene); (80) fluorene; (81) phenanthrene; (82) dibenzo (a,h)anthracene (1,2,5,6-dibenzanthracene); (83) indeno (1,2,3-cd)pyrene (2,3-o-phenylenepyrene); (84) pyrene; (85) *tetrachloroethylene; (86) *toluene; (87) *trichloroethylene; (88) *vinyl chloride (chloroethylene).

PESTICIDES AND METABOLITES

(89) *aldrin; (90) *dieldrin; (91) *chlordane (technical mixture and metabolites).

*DDT AND METABOLITES

(92) 4,4'-DDT; (93) 4,4'-DDE (p,p'-DDX); (94) 4,4'-DDD (p,p'-TDE).

*ENDOSULFAN AND METABOLITES

(95) a-endosulfan-Alpha; (96) b-endosulfan-Beta; (97) endosulfan sulfate.

*ENDRIN AND METABOLITES

(98) endrin; (99) endrin aldehyde.

*HEPTACHLOR AND METABOLITES

(100) heptachlor; (101) heptachlor epoxide.

*HEXACHLOROCYCLOHEXANE (ALL ISOMERS)

(102) a-BHC-Alpha; (103) b-BHC-Beta; (104) r-BHC (lindane)-Gamma; (105) g-BHC-Delta.

*POLYCHLORINATED BIPHENYLS (PCB's)

(106) PCB-1242 (Arochlor 1242); (107) PCB-1254 (Arochlor 1254); (108) PCB-1221 (Arochlor 1221); (109) PCB-1232 (Arochlor 1232); (110) PCB-1248 (Arochlor 1248); (111) PCB-1260 (Arochlor 1260); (112) PCB-1016 (Arochlor 1016); (113) *Toxaphene; (114) *Antimony (Total); (115) *Arsenic (Total); (116) *Asbestos (Fibrous); (117) *Beryllium (Total); (118) *Cadmium (Total); (119) *Chromium (Total); (120) *Copper (Total); (121) *Cyanide (Total); (122) *Lead (Total); (123) *Mercury (Total); (124) *Nickel (Total); (125) *Selenium (Total); (126) *Silver (Total); (127) *Thallium (Total); (128) *Zinc (Total); (129) **2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD)).

*Specific compounds and chemical classes as listed in the consent degree.

**This compound was specifically listed in the consent degree. Because of the extreme toxicity (TCDD) We are recommending that laboratories not acquire analytical standard for this compound.

UCC 006830

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WV-0085 Title #1

Ethylene oxide
Effect of insulation on the auto ignition temperature

The Effect of Insulation on the Auto ignition Temperature of Ethylene Oxide.

Test Procedure.

Operations Involved

i) Loading test section with sample

ii) Placing test section in salt bath

iii) Heating Salt Bath

iv) Adjusting gas flow rates.

v) Removal of sample.

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Apparatus

The apparatus comprises a molten salt bath, gas delivery and mixing system and a sample test section. These are shown on the accompanying drawings and schematic diagrams. In view of the hazardous nature of the materials involved (molten salt, ethylene oxide) the test equipment is located in Cell 2 of building 776. No one is permitted to enter the cell whilst ethylene oxide is flowing through the apparatus.

All tubing carrying ethylene oxide to the test section and ethylene oxide/air

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mixture away from the apparatus is 1/4" stainless steel. This diameter has been found adequate in preventing flashback.

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Loading Test Section

Hazards Involved.

A.) Inhalation of insulation dust, contact with skin. (any commercially available insulation)

— MSDS —

Precautions.

Volume 18" x 3/4" ID pipe

a.) Supervisor will ascertain if insulation material has any dangerous properties.

b.) operator will wear a mask and gloves.

Procedure.

1.) Insulation material is crushed so that the largest particle size is less than 1/8".

2.) Crushed insulation is loaded into test section

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3.) Thermocouples and sampling tube are placed radially in position.

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Placing Test section in Salt Bath.

Unit is fastened to the floor & wall

Hazards Involved

A) Splashing of molten salt ~~the cover on salt bath will be covered~~

B.) Contact with hot metal (burns)

Precautions.

dry air for agitator

~~a.) The molten salt agitator will be turned off reduced removed during loading.~~

also retainers

b.) The operator will wear asbestos gloves, face shield and appropriate protective clothing.

Procedure

The test section is lowered into the outer tube, which stands in the

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salt bath, and screwed into position

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Heating Salt Bath

Hazards Involved

A.) Premature melting of the bottom of the salt bath before the top could lead to a violent eruption of the bath contents.

B.) Over-vigorous use of molten salt agitator (air-blower) could lead to a spray of molten salt.

Precautions.

a.) Heat-up sequence will be from top to bottom of salt bath, the lower heaters will not be used until upper layer is molten.

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This will be detected by thermocouple.

b.) The air blower agitator will be turned off before anyone enters cell.

c.) Face shield, asbestos gloves and protective clothing will be worn by anyone needing to enter cell.

Procedure.

manual
variac

1.) Turn on upper heater, monitor temperature on upper bath thermocouple

2.) When upper temperature is 230°C turn on middle heater, monitor middle bath temperature

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3.) When middle temperature is 230°C turn on lower heaters, monitor lower bath temperature.

4.) When all bath thermocouples show that temperature is above 230°C salt agitator may be turned on. Air flow rate is adjusted so that the bubbles break the surface without splashing. This can be checked with a mirror.

5.) Adjust heaters to give required bath temperature.

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Gas Mixture Flow Rate

Variable ratio of air & oxide

Hazards Involved.

A.) Ethylene oxide is a dangerous gas having a 4.4.4,W hazard rating in the Reactive and Hazardous Chemicals Manual.

3 sec. Contact time

B.) Preignition or flash back of ethylene oxide/air mixture could result in a violent explosion.

C.) A spill of ethylene oxide could cause a toxic, explosive cloud.

Precautions.

a.) The operator will study the section

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on ethylene oxide in the Reactive and Hazardous chemicals Manual before starting any experimentation and become familiar with the appropriate safety precautions.

b.) The ethylene oxide and air flows are separately monitored and mixed at the point of entry to the test section.

c.) Tubing is 1/4" stainless steel which will prevent flash back in the pure ethylene oxide stream.

d.) Ethylene oxide flow rate will be less than 0.7 cu. ft/hour

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e) The gases are exhausted to the atmosphere after passing through the test section.

f.) The cell is well-ventilated.

g) All metering is carried out remotely

Procedure.

1) When the test section is in position the apparatus is flushed through with ~~nitrogen~~ ~~or~~ air for 15 minutes.

2.) Ethylene oxide is vapourised in the steam heated water bath prior to metering.

3.) Ethylene oxide flow rate is adjusted

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to the required level, the gas pressure is held at 4 psig.

4.) The air flow rate is adjusted to give the desired stoichiometry.

5.) If the test section thermocouples indicate an exotherm, the ethylene oxide flow is shut off and the air is left to purge the apparatus.

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Removal of Sample

Hazards Involved.

A.) Splashing with molten salt, causing burns, eye damage.

B.) Release of ethylene oxide vapour.

C.) contact with hot surfaces.

Precautions.

a.) Salt agitator is turned off before operator enters cell.

b.) Operator is to wear face shield, asbestos gloves and protective clothing.

c.) N_2 purge is left on for 15 minutes prior to removal

1st step

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d.) Test section is allowed to cool before insulation is removed.

Procedure

1.) Air purge is turned off.

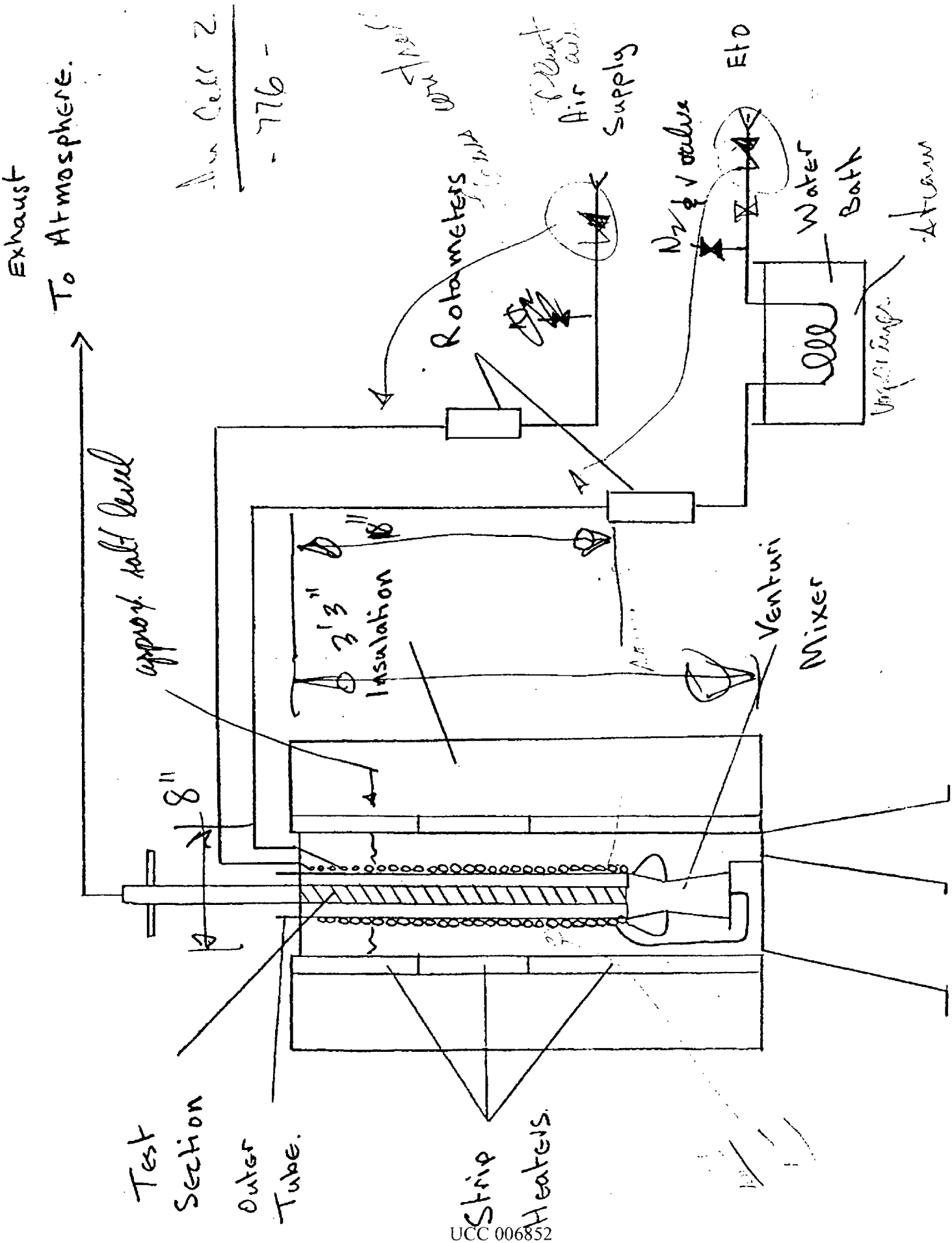
2.) connection between test section and exhaust pipe is broken

3.) Test section is unscrewed and removed from outer tube.

4.) Test section is allowed to cool

5.) The thermocouples and the sampling tubes are removed and the insulation sample is taken out and discarded.

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