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Water quality proposal favors industry

Once it's dirty it costs billions to clean up, if at all. I'm talking about water—a universal requirement for life and one of Alaska's greatest assets. From an Eskimo's gift to the soul spirit to holy baptism and everything in between, we rely on clean water.

As residents we know Alaska is the last best place and many of us shoulder an obligation to see that it stays that way. Gov. Hickel's confused, on again, off again stripping of the water quality standards is clearly an immediate threat to Alaska. If we ever needed an example of political expediency paving the way for industrial pollution, we wouldn't look further than the Department of Environmental Conservation's recommendations on the National Toxics Rule.

Its net effect is to ease the way for accelerated resource extraction the state has proposed to lower the limits of arsenic, dioxin, and hydrocarbons in our water. These carcinogens are residues from mining operations, pulp mills, and the oil companies. They are released into rivers and lands when industry says it would cost too much to contain them.

After severe criticism from ordinary citizens, conservationists, and the fishing and tourism industries, DEC has backed away from, but not repudiated, its initial language. As proposed, revisions of the water quality standards would permit pulp mills at Sitka and Ketchikan to dump 85 times more cancer-causing dioxin than federal regulations recommend. In Juneau, the gold mines would get the green light to discharge 250

GUEST OPINION

Douglas Vaden

times more arsenic than the federal government is likely to suggest. The Alyeska pipeline terminal in Valdez would be allowed to continue releasing hydrocarbons, oil, and grease into Prince William Sound. If regulations were adopted as originally proposed, Alaska's reputation for clean water would become a memory.

John Sandor, DEC commissioner, rationalizes this strategy as a balancing act between economics and health. Tipping the scales in industry's favor, Sandor has quietly asked the U.S. Environmental Protection Agency to loosen the limit on dioxin from one cancer in every 1 million people to one case in 100,000. Without comment, without notice, Sandor is assuming that Alaskans agree this is an acceptable risk level. DEC bases this standard on estimates that Alaskans eat five pounds of fish a year. Data gathered by the Department of Fish and Game suggests Alaskans generally consume between 20 to 300 pounds of fish a year. In aligning itself with industry DEC is putting Alaskans' future health for short-term economic gain.

Alaska's fishing industry is prized for both abundance and purity. Each value is threatened when Alaska's waters are degraded. Whether the silver market perceptions bench the fishermen the result is the same. Millions in lost wages and another black eye for Alaska. Tourism brings additional millions of dollars into the state each year. Visitors come north because they think the land and water haven't been spoiled yet. Imagine the word passing through the RV parks: "We're too late, we should have come last year. Except for the glaciers it's as polluted as New Jersey."

Last month when this newspaper approved DEC's proposed changes a companion editorial acknowledged the Interior Alaska Ecumenical Peace with Justice Council's positive efforts to raise awareness of world issues. A litany of social ills, from violence to poverty to HIV, were cited as topics for thoughtful public discussion. Glaring in its omission was any mention of environmental justice. Though it does indicate a certain editorial consistency, the failure to acknowledge Alaskans' trust in unbiased scientific research cannot go unchallenged.

If we in Alaska fail to plan for our long-term residency we will join just as surely millions of environmental refugees who are now pressing in on their not-so-well-off neighbors. In Africa and Asia an estimated 10 million people have lost their homelands through the collapse of natural systems. Human-induced deserts, droughts, floods, landslides, destruction of forests, croplands and coral reefs have impoverished whole cultures.

While much of the rest of the world is coping with a biosphere under seige, Alaska still possess large unfragmented watersheds and coastlines that serve intact ecosystems. We serve the future when we consider genetic diversity. If prophets of infinite growth like Sandor are allowed to dictate water policy, images of "pristine Alaska" will exist only in photographs. Instead of giving industry a license to pollute we should demand that industrial cleanup be figured into the cost of doing business. Anything less should be considered a threat to our national security.

Indeed, some defense analysts contend that the deterioration of the environment must be considered as one of the gravest threats facing all people. In Alaska this means that weakening the water quality standards holds the potential to erode the biological foundations of human life. Rather than spur economic development it will create economic instability, injustice and violence.

Stung by criticism, DEC now wants to start again with a new set of standards that will address scientific data and appropriate risk levels for human health. This time the agency will consult with a panel of business people, fishermen, conservationists and others. DEC is taking nominations for this committee until the end of the month. You may submit your name by writing to DEC, 410 Willoughby Ave., Suite 105, Juneau, AK 99801-1795.

Douglas Vaden is an Eagle resident who followed mining and water quality issues.

Critics attack state water-quality standards

Juneau Empire

By JAY STANGE Tues, Aug 4, 92

THE JUNEAU EMPIRE

In front of 80 people at a crowded and hot public hearing Monday night, Haines resident Tim June unceremoniously yanked his long-sleeve shirt up to his armpits.

His turn to testify had come.

From his right nipple, all the way across his chest and around his back was a quarter-inch-wide reddish scar. Another large cut on his abdomen showed another cancer autograph, a souvenir from a bout with the deadly disease 20 years ago.

June's dramatic testimony before the crowd and Department of Environmental Conservation officials came at a hearing on proposed changes to the state's water-quality standards. The proposal for regulating carcinogens allowed in Alaska waters includes an assumption that an acceptable risk of cancer is one in 100,000 people.

The U.S. Environmental Protection Agency allows states to select an appropriate cancer risk ranging from one in 100,000 to one in 10 million. Alaska has proposed the least stringent factor of one in 100,000 for regulation of dioxin, arsenic and chloroform – all suspected caremogens.

June said he was outraged. The cancer victim has spent the past three weeks reading technical manuals, speaking to EPA and DEC officials, and taking is message to the streets.

"Where did I get it (cancer) from?" June asked as he pulled his shirt back down over his scars. "The best guess I can come up with after thinking about this for 20 years, probably every day of my life, was I made the mistake of living close to Kaiser Steel in Southern California.

"I was one of the one in 100,000 from that plant ... I mean where are we going with these regulations?"

At Monday's hearing at Centennial Hall, an overwhelming number of those in the audience seemed to share June's sentiment. Representatives from the Sierra Club Legal Defense Fund, the Southeast Alaska Conservation Council, Alaskans for Juneau and others spoke up against the plan to change the state's water-quality regulations.

DEC is mandated by amendments to the federal Clean Water Act of 1990 to rewrite its standards for how much pollution is acceptable in Alaska's waters. If the state does not, the EPA is ready and willing to do so from Seattle.

The state standard will be used by the EPA to issue new permits for pulp mills in Ketchikan and Sitka and for proposed mining activity in Juneau, among other activities statewide.

Critics of DEC's changes, including many of the environmental groups speaking at the meeting Monday, believe Gov. Walter J. Hickel's administration, through DEC com-

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Water...

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missioner John Sandor, has been politically motivated to create lax water-pollution standards.

Sandor was a few rows back of June's scar tactics Monday.

After the hearing, Sandor said the proposal did not involve a cabinet level action but was developed by DEC water-quality staff on their own initiative.

"I'm confident that the professionals (DEC staff) will come up with their professional recommendations and we who are involved with policy will be able to deal with those," Sandor said. "And within the limits of that, I'm absolutely positive that the EPA is not going to simply blindly accept what any state proposes."

Sandor said the proposed regulations would protect the public interest.

Some in the audience agreed.

Chuck Achberger, executive director of the pro-mining Alliance for Juneau's Future, said there was room for protecting the environment and allowing industry to operate. Achberger supported the regulations as proposed.

Donald Burford said the Ketchikan pulp mill has operated for 40 years with people harvesting oysters in front of the mill without, to his knowledge, a single death due to cancer.

Burford said the thought that mills will operate for a short time, lay waste to the land, and cause cancer deaths to be "improbable and frightening and I refuse to accept it."

Anthony Williams said, "If pulp mills were to shut down, what is the risk factor involved in that?" If the risk factor would put jobs in jeopardy, perhaps the water standard should be lowered even further, Williams said.

But the tide of the public hearing was overwhelmingly against the proposed standards, despite any risk to jobs.

Caryl Boehnert of Alaskans for Juneau, a mine watchdog organization, said "we should call the it the Department of Environmental Economics, not the Department of Environmental Conservation, then at least we would be saying it up front."

Boehnert, a private research scientist, said that in terms of economic impacts the state's proposed water-pollution policy discriminated against smaller industries such as fishing and tourism by lowering water standards in favor of larger industries such as hard rock mining, pulp and paper mills, and oil and gas operations.

"It's clearly an economic war when fishing is taking a third or fourth place," Boehnert said.

Kate Troll, executive director of the Southeast Alaska Seiners Association and a member of the habitat committee for United Fishermen of Alaska, said she has seen three family members succumb to cancer and though the proposed standards would cripple the reputation of Alaska's wild fish.

Troll said inferior standards would belie the message told with millions of dollars of state funds spent on marketing salmon.

"Stringent water-quality standards are in themselves an excellent marketing tool," she said.

Sandor said after the hearing, "I think you should know that the governor has assured the United Fishermen association and he has certainly given us direction. He said he did not want to see aquatic life changed or modified. And they are not being modified even by this proposal."

Sandor said DEC has a mandate to protect fisheries. "We devote a lot of time to that."

He said public concerns would be addressed and he downplayed the importance of emotional public testimony in DEC's adoption of its final standards.

"The purpose of the public comment is not to take a vote based on the number of people who are for or against the proposal," Sandor said. "The purpose of the public comment is to make absolutely certain that the comments and the suggestions are addressed and incorporated into the development of the regulations."

Other concerns addressed included the timing of the public comment process, which – though it has been extended six weeks to Sept. 30 – coincides with the commercial fishing season, making it difficult for fishermen to participate. Troll and others asked for the comment period to be extended another month to Nov. 1.

Their request has not been acted on.

A public hearing has been set for Sept. 24 in Haines and a teleconference for Sept. 25 for Juneau additional comments.

Misguided satellite

The European Retrievable Carrier (EURECA) satellite released by the shuttle Atlantis has been inadvertently

Shuttle...

Continued from Page 1

up to 5,000 volts of electricity as it cuts across Earth's magnetic field at 17,500 mph.

A Cover-Up on Agent Orange?

Critics charge that the Centers for Disease Control sabotaged an investigation of the defoliant's effects on Vietnam veterans

The medical detectives at the Atlanta-based Centers for Disease Control have a well-earned reputation for relentlessly tracking down the causes of such mysterious ailments as Legionnaires' disease. But the agency's record is in danger of being blemished by a bitter controversy over Agent Orange, a defoliant containing dioxin, a suspected carcinogen.

Critics charge that the agency and one of its senior officials, Dr. Vernon Houk, helped scuttle a $63 million study that might have determined once and for all whether U.S. troops exposed to Agent Orange suffered serious damage to their health. Houk maintains he recommended that the study be canceled on strictly scientific grounds. Yet there is evidence that the CDC suppressed reports from the National Academy of Sciences that directly challenged its position, and spurned extensive help from the Pentagon, leading the White House to kill the study.

Agent Orange was widely used in Vietnam to strip the thick jungle canopy that helped conceal enemy forces; only later did scientists become aware of the potentially dangerous long-term effects of dioxin, which has produced cancers in animals. The defoliant has been suspect ever since unknown numbers of Vietnam veterans developed various cancers or fathered seriously handicapped children. Based on the inability to prove a conclusive link between those ailments and Agent Orange, the Reagan and Bush administrations refused to compensate veterans for all but a few of these health problems. But critics charge that no clear connections have been established because no serious large-scale study of exposed veterans has been done.

The most forceful complaints about the CDC have been leveled by former Chief of Naval Operations Elmo R. Zumwalt Jr. As the Navy's top commander in Vietnam, he ordered that Agent Orange be sprayed in the Mekong Delta region to destroy vegetation from which the Vietcong regularly launched ambushes against U.S. patrol boats. In 1988 Zumwalt's son Elmo III, a former lieutenant who had served in the "brown-water Navy," died from a rare lymphoma. Zumwalt believes his son's exposure to Agent Orange was responsible.

Last month Zumwalt told a House subcommittee that the CDC's work on Agent Orange had been "a fraud." He singled out Houk for having "made it his mission to manipulate and prevent the true facts from being determined." New York Congressman Ted Weiss, chairman of the panel, charged in an interview that the CDC appeared to have "rigged" its investigation to support its view that a large study of exposed veterans was not feasible.

Congress authorized the CDC study in 1982 after receiving thousands of complaints from Vietnam vets about Agent Orange. Houk, director of the agency's Center for Environmental Health and Injury Control, was placed in charge. At the White House, a science panel of the Agent Orange Working Group supervised the CDC's investigation. The Pentagon assigned its Environmental Support Group to provide the CDC with Agent Orange spraying records and those of the deployment of soldiers who may have been exposed.

But the study soon bogged down in a complex dispute over identifying which soldiers were likely to have been exposed to Agent Orange. The CDC considered a company of 200 men potentially exposed if it passed within 1.3 miles of a recently sprayed area. The Army had fairly detailed records on the daily positions of its companies during the fighting. There were gaps, but the Pentagon group repeatedly told the CDC that other documents, such as daily journals and situation reports, could be used to pinpoint which units had ventured into areas sprayed with the defoliant. Houk's team complained that the Pentagon data were too spotty to determine whether companies had been deployed in normal formations spread over 200 to 300 yards or dispersed over distances of up to 12 miles. It stubbornly refused to make use of the other records.

By late January 1986, Dr. Carl Keller, chairman of the White House science panel, and several other of its members concluded that Houk had already decided that the CDC study was not feasible and was trying to pin the blame on the Pentagon. To break the impasse, retired Army Major General John Murray was asked by Defense Secretary Caspar Weinberger to review the Pentagon records. After a four-month study, Murray thought the records were useful. But as a nonscientist he did not feel competent to rebut the objections raised by Houk and the White House scientists. He gave up, agreed that the information was inadequate and suggested cancellation of the project.

Unknown to Murray and the White House, the Institute of Medicine, an arm of the National Academy of Sciences, then turned in a contracted consultants' report to the CDC on the Agent Orange study. It concluded that the Pentagon group was fully capable of "determining locations and filling gaps" in the troop movements and criticized the CDC's study for excluding many of the veterans most likely to have been exposed. The CDC never

Veterans march in Washington to protest the poison used to protect them

Shortly before a tragedy, Admiral Zumwalt ordered spraying; his son died

The father claimed there had been a "mission" to conceal "the true facts."

turned the institute's report over to the White House.

Murray presented his conclusions at a White House meeting on May 27, 1986. The White House moved to kill the study unless other ways could be found to identify exposed soldiers. Much later, Murray learned of the institute's report and began to doubt his recommendation. "I may have been a babe in the woods," he said in an interview. "My feeling now is that this whole thing deserves another look."

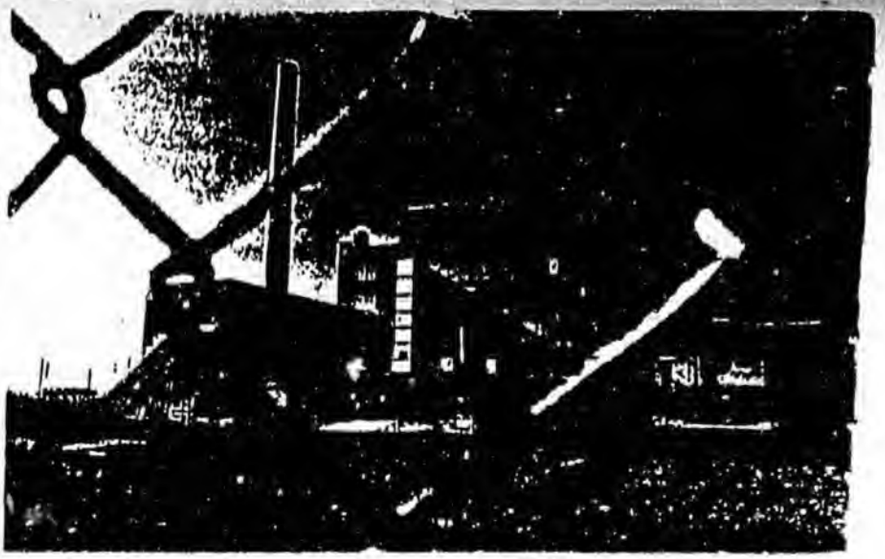
Instead of killing the project outright, the White House panel accepted a proposal by Houk to take blood tests of 646 Vietnam veterans, selected on the basis of their probable exposure, to see if they had elevated blood levels of dioxin. The tests showed that none had abnormal blood levels—not surprising, given that the exposure would have taken place 20 years earlier and that none of those tested had handled Agent Orange directly.

Though many scientists ridiculed the blood tests, Houk used them to contend again that the Pentagon records could not be used to pinpoint exposure to Agent Orange. He recommended canceling the study; the White House Science Panel agreed, and the Domestic Policy Council did so in September 1987. This was after $43 million had been spent.

Once again the White House had acted without having all the facts. The Institute of Medicine only weeks earlier had written a blistering review of the CDC's work. It urged that each of the agency's major conclusions be deleted because the evidence presented by the CDC did not support them. The White House never received this devastating report.

The CDC's Dr. Vernon Houk

Houk insists that his opposition to continuing the project was based solely on rigorous scientific principles. "If we could find a population of people who were exposed in sufficient numbers, we would have proceeded with our study," he says. "We just simply could not find them." Skeptics like Congressman Weiss suspect that the CDC did not want to antagonize the Reagan Administration, which was worried about the huge liability costs if Agent Orange was shown to cause the veterans' ailments. Whatever the reasons for its failure, the decision not to complete the study leaves open a vexing problem: whether Agent Orange will exact a toll on Vietnam vets and their descendants for generations to come. —*By Ed Magnuson. Reported by Jay Peterzell/Washington*



A reactor at the suspected plant, shut down since the 1960s

There Was Death in the Milk

Maybe in fish too, according to a new study of long-ago radiat releases from the nuclear reservation in Hanford, Wash.

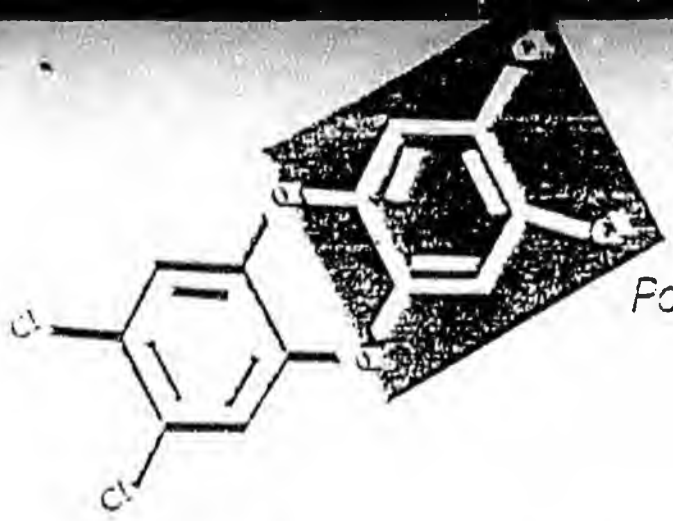
Growing up in Moses Lake, Wash., Vicki Skipper suffered stomach cramps that continued even after her family moved to Connecticut in 1962, when she was eight. Later, she reports, "I got swollen glands unde my arms, and I had my thyroid removed, and they never figured out what it was. I always thought it was from the plant, but I could never prove it." A federally sponsored panel of scientists and medical experts last week, however, indicated that her suspicions—and those of thousands of others who, from the late 1940s until well into the 1960s, lived in eight Washington and two northern Oregon counties near the Hanford, Wash., nuclear reservation—are far from groundless.

The panel found that between 1944, when it opened, and 1947, the Hanford weapons plant poured so much radioactive iodine into the air that 1,200 children living nearby were exposed to cumulative doses ranging from 15 to 650 rads (one rad is roughly equal to the radiation from a dozen chest X rays). About 13,500 people, or 5% of the area's total population, may have taken in doses of 33 rads or more—about twice the three-year dosage the Nuclear Regulatory Commission considers safe for workers exposed to radiation as an occupational hazard.

The iodine was released as a gas when fuel rods were chemically dissolved in acid as the last step in producing plutonium, the explosive material in some nuclear weapons. It got into humans mostly because they drank milk from cows that had grazed on grass contaminated by airborne iodine. In human bodies the iodine tended to concentrate in the thyroid in amounts that would have been enough to cause at least some cases of

Though the releases were heavi tween 1944 and 1947—one reason th el picked that period for study—th not stop then. "Regulatory standard not developed until the 1950s," th noted, and not until 1973 did the of radiation in the atmosphere decr the point that it could no longer be measured. The panel, funded by t partment of Energy, also studied of radioactive substances from nu actors into the Columbia River 1964 and 1966, when some of the discharges occurred. River wat pumped through the reactors to co Radioactivity—in lower doses than borne iodine—entered the bodies ple who swam in the river or lived near it or ate fish caught in it.

The Hanford plant and react shut down in the late 1960s; milk, vegetables from the area by now ou radiation-free. But that is no cons those exposed to dangerous radiat er. A study of health effects of the by the Centers for Disease Contr be complete until 1993. If the gove unwilling to offer compensation who lived near the plant and fell ill time of heavy discharges, or to the if they have died, Washington Sen. Adams promises to introduce leg compel it to do so. Meanwhile, Vi has perhaps the last word: "Whe byl hit, I remember thinking th sure had a lot of nerve talking ab when we've been doing the same people." —*By George J. Church. Ellis E. Conklin/Seattle and Rosan Washington*



Dioxin's Other Face

Portrait of an "environmental hormone"

By KAREN F. SCHMIDT

When a villain starts looking like a friend, it's time to look again. Take TCDD, the most notorious and potent member of the dioxin family. Once demonized as "the most toxic synthetic chemical known to man" because of its exquisitely lethal effect on guinea pigs, TCDD now appears "no more risky than spending a week sunbathing," as a recent New York Times article put it.

In 1982, scares over TCDD forced several thousand residents of Times Beach, Mo., to permanently flee their tainted community. But "given what we now know about this chemical's toxicity and its effects on human health, it looks as though the Times Beach evacuation was unnecessary," Vernon N. Houk — the scientist at the Centers for Disease Control who originally spearheaded the evacuation — acknowledged, according to the Times article last August.

Most dioxin researchers now suspect that only very high doses of TCDD — as occur accidentally or in certain occupational settings — may increase the risk of cancer in humans. But that redefinition does not necessarily imply that the chemical is harmless at lower doses.

Indeed, this near-ubiquitous contaminant — a by-product of the paper, wood and herbicide industries and of the incineration of organic solvents — is gaining a new and nasty reputation among toxicologists: as an "environmental hormone" that subtly disrupts normal physiology in ways not completely understood. More potent than some of the body's natural chemical messengers, TCDD suppresses the immune system of mice at least 100 times more effectively than corticosterone, a hormone known for that effect, dioxin researchers say. In fact, increasing evidence suggests that TCDD's ability to mess with the immune system — not its carcinogenicity — may represent its greatest threat to public health.

All this flip-flopping on the chemical's toxicity may puzzle the public, but it has proved no less confusing to dioxin researchers. TCDD's toxic effects result from a perplexing web of interactions. Unlike most toxicants, dioxin causes an array of biological responses that vary widely according to tissue. For example, TCDD may goad one cell type to reproduce wildly and cause another to deviate from its normal path toward specialization.

Different animal species also vary in their responsiveness to dioxin. It takes several thousand times more TCDD to kill a hamster than it does to kill a guinea pig. Yet the hardy hamster is quite susceptible to TCDD's triggering of increased cellular levels of a P450 enzyme — a protein catalyst that plays a role in detoxifying certain chemicals within the body and rendering others more toxic.

Unfortunately, epidemiologic studies have done little to resolve toxicologists' muddy understanding of dioxin's human hazards. For instance, such studies rarely turn up consistent adverse effects among humans exposed to dioxin — with the exception of chloracne, the disfiguring skin eruptions associated with acute TCDD exposures.

Consider studies of U.S. troops potentially exposed to Agent Orange, a TCDD-tainted herbicide, while serving in Vietnam. An Air Force study of veterans who had participated in the Ranch Hand defoliation program found indications that these men faced an increased — though statistically insignificant — risk of skin, genito-urinary and otopharyngeal cancers and a tendency to develop underactive thyroids and diabetes (SN: 3/3/84, p.132). Another study found an increased incidence of high blood pressure, benign fatty tumors, sensitivity to light, and depression among these veterans and miscarriages among their wives (SN: 11/19/88, p.325). A third study found that Vietnam veterans suffer higher-than-normal rates of non-Hodgkins lymphoma, a deadly cancer of the lymph nodes, but it failed to tie the disease to Agent Orange exposure (SN: 4/14/90, p.236).

"If you think of TCDD as a hormone, it makes it easier to understand these very big differences," asserts Linda S. Birnbaum, director of environmental toxicology at the Environmental Protection Agency's Health Effects Research Laboratory in Research Triangle Park, N.C. A single hormone can induce an array of effects in different tissues and species, she explains.

The environmental hormone theory also helps explain why dioxin appears to induce a variety of cancers rather than a single hallmark type — such as the rare form of cancer, called mesothelioma, that signals asbestos exposure. Unlike most carcinogens, TCDD does not directly damage DNA in a target organ, notes George W. Lucier of the National Institute of Environmental Health Sciences in Research Triangle Park. However, he explains, dioxin clearly enhances abnormal cell growth and appears to cause cancer by amplifying the diverse activities of other carcinogens.

Two recent epidemiologic studies support the human carcinogenicity of TCDD, at least at fairly high doses. In one, researchers at the National Institute for Occupational Safety and Health examined health records for workers exposed to TCDD at a dozen chemical plants. Overall, the 5,172 workers appeared 15 percent more likely to die from cancer than the general population, Marilyn A. Fingerhut and her co-workers reported in the Jan. 24, 1991 NEW ENGLAND JOURNAL OF MEDICINE. However, records on the 1,520 workers whose exposures began at least 20 years ago — when plant dioxin levels were typically much higher than today — showed nine times the normal rate for one particular cancer, soft-tissue sarcoma.

A similar study of 1,583 pesticide-plant workers in Germany showed that, compared with the general population, TCDD-exposed workers experienced a 24 percent higher rate of death from all cancers. Among workers with more than 20 years' exposure, the cancer death rate increased to 87 percent above normal, according to Alfred Manz and his co-workers at the Center for Chemical Workers' Health in Hamburg. However, they reported in the Oct. 19, 1991 LANCET, the increases were not linked to any one

particular type of cancer.

On the basis of these and other studies, Birnbaum says, "I really feel that high-dose exposure to dioxin has the potential to cause cancer." However, she adds, "I'm very concerned that much lower exposure to dioxin may result in adverse health effects that are very subtle and difficult to detect."

In an effort to update federal regulatory guidelines for human exposures to dioxin – now considered a "probable human carcinogen" – EPA has begun reassessing the scientific data on dioxin. In its draft version of this document, due in June, EPA will focus much greater attention on toxicological data revealing TCDD's reproductive, developmental and immunotoxic effects. This document will also establish TCDD as the first pollutant to be regulated on the basis of toxicity observed at the cellular level.

Now that most dioxin researchers believe a single fundamental mechanism underlies all of TCDD's effects (see box, p. 26), toxicologists such as Lucier can construct a unifying mathematical model to describe how dioxin triggers biological effects in cells and organisms. Others, including Birnbaum and Nancy I. Kerkvliet of Oregon State University in Corvallis, will help flesh out the model by collecting specific data on the dose-response relationships between TCDD and its array of biological effects.

"Dioxin is no more and no less potent than it ever was," Kerkvliet says. "But understanding the mechanism can now help us better estimate the human risk."

So far, studies in mice suggest that dioxin's immunotoxic punch occurs in extremely low doses and may well be more important than cancer in determining dioxin's primary health risk, adds Birnbaum. At least in animals, some suppression of immunity consistently occurs at TCDD doses lower than or equal to those required for triggering increased production of a P450 enzyme – previously considered a liver cell's most sensitive response. In fact, Birnbaum's preliminary unpolished data suggest that immunotoxicity in mice could be occurring at TCDD doses one of that needed to boost levels of this enzyme, she says.

Even though scientists continue to debate whether an excess of this P450 enzyme causes any adverse health effects, "few people will contend that suppression of the immune system is not an adverse health effect," she observes.

To study TCDD's immunotoxicity, researchers generally use mice, whose immune systems model those of humans. In one typical test, EPA toxicologists exposed mice to TCDD, then injected them with a harmless, antibody-stimulating agent – red blood cells from sheep. An animal's ability to produce antibodies serves as one useful measure of its immunological health. Compared with normal mice, the TCDD-treated animals produced fewer antibodies to the sheep blood cells, Birnbaum says.

EPA researchers have also measured how well TCDD-treated mice respond to viral injections, such as influenza. Mice pretreated with dioxin readily die after exposure to a quantity of virus that rarely kills healthy mice. Gary R. Burleson of EPA's Research Triangle Park facility and his co-workers reported in the November 1990 JOURNAL OF TOXICOLOGY AND ENVIRONMENTAL HEALTH. Birnbaum's team is now trying to determine the dose-response relationships of these immunosuppressive effects.

Because "there are so many ways to cause immune suppression," Birnbaum explains, scientists can only speculate as to how TCDD weakens immunity. Indeed, she notes, "there could be multiple mechanisms."

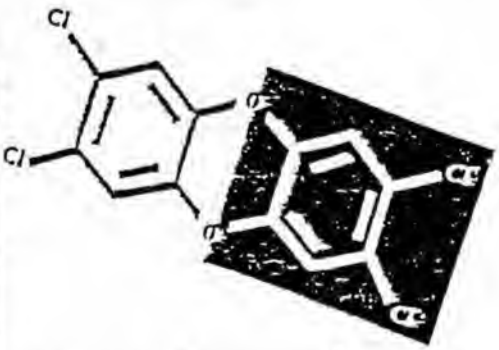
At a minimum, TCDD probably interferes with the normal influences of hormones on the immune system, Kerkvliet posits. She says that it appears TCDD can combine with a particular type of receptor protein inside a cell's fluid interior, and then inappropriately turn on specific genes. Some of the victimized cells may reside in glandular tissues, such as the thymus, where hormones influencing immunity are produced.

Dioxin also appears to act directly on the immune system, says Kerkvliet, who studies TCDD's effects on a group of white blood cells called T-lymphocytes. She and her co-workers were initially confounded when they observed that although TCDD boosts production of T-lymphocytes – which referee the total immune response – it still causes an overall decline in the mouse immune system's ability to fight foreign substances, be they viruses or pollutants.

"We think TCDD is turning on certain T-helper cells inappropriately, which then makes the overall immune response suppressed," Kerkvliet now says. This idea fits with a new hypothesis that not all of the specialized T-lymphocytes called T-helper cells "help" strengthen the immune response; some may actually inhibit it, she notes.

For the most part, Kerkvliet believes that dioxin initiates its direct immunotoxic effects by binding to the dioxin protein receptor – perhaps in the bone marrow, where white blood cells are produced – and by toying with the normal functioning of genes. Recently, her research group studied how TCDD affects a mouse's production of cytotoxic T-lymphocytes, which destroy cells infected with viral invaders.

The team compared responses in TCDD-treated mice with normal and defective dioxin receptors, and found significantly greater immune suppression in the mice with normal receptors. They also compared the responses of these mice to a variety of polychlorinated biphenyls (PCBs), chemical relatives of dioxin. Immunity suppression indeed correlated with each chemical's ability to bind to the protein receptor. Kerkvliet's group reported in the April 1990 FUNDAMENTAL AND APPLIED TOXICOLOGY. These findings suggest that dioxin's protein receptor plays an important role in its immunotoxicity, they say.



During the Vietnam war, the U.S. military dumped millions of gallons of TCDD-tainted Agent Orange over South Vietnam. Veterans who participated in this defoliation program, called Operation Ranch Hand, have experienced a variety of health problems that might be related to dioxin exposure.

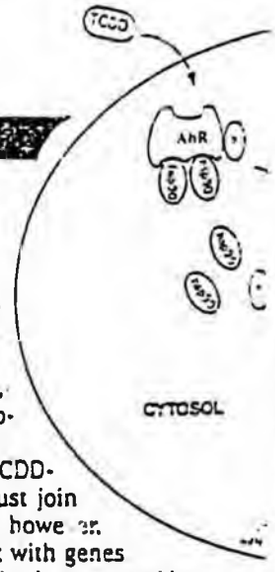
Given the complexity of the immune system, however, not all dioxin researchers are ready to settle on a single receptor-based mechanism to describe all of TCDD's immunosuppressive effects.

Michael P. Holsapple of the Medical College of Virginia/Virginia Commonwealth University in Richmond has also observed that "when we give dioxin to animals or white blood cells, we see problems with their immune function." However, he adds, "the immune system is probably just a microcosm of the whole complex story for dioxin." He suspects that TCDD may employ different routes of attack depending on the conditions of exposure, he says.

For instance, his team compared the effects of acute versus chronic TCDD exposures on the ability of mice to produce antibodies to sheep red blood cells. After a single acute dose, mice with normal dioxin receptors suffered greater immune suppression than mice who had defective receptors. However, when mice received this same amount of TCDD over a two-week period, both mouse strains showed similar immunosuppressive responses, he and his colleagues report in the January 1992 TOXICOLOGY AND PHARMACOLOGY. Holsapple now theorizes that TCDD's mechanisms may not always involve the receptors and may differ at high and low doses.

Throughout the developed world, humans already experience chronic low-dose exposures to dioxins, primarily through their diet (SN: 7/13/85, p.26). Holsapple and his co-workers suspect that people "exposed to low doses over an extended period of time (i.e. months to years) may be at increased risk to immunotoxic effects by these chemicals through additional and presently unidentified mechanisms."

One such mechanism can be inferred from developing research in the field of endocrinology, Holsapple says. Scientists had assumed that, much like dioxin, all steroid hormones act exclusively through an intracellular protein receptor that helps it target a particular gene (SN: 8/10/91, p.85). But Holsapple points to new evidence suggesting that some steroid hormones — including progesterone, estrogen and testosterone — can also bind to other receptors on the outside of a cell membrane, where they can regulate the flow of salts into and out of a cell. TCDD might also tinker with a cell's physiology through such a mechanism, he suggests.



Dioxin's Cellular Siege

Dioxin may cause everything from immune suppression and liver tumors to cleft palate in mice, but all of these adverse effects begin with the same initial cellular changes, most dioxin toxicologists now believe.

This "new" view — the impetus behind the Environmental Protection Agency's (EPA) current reassessment of dioxin's risks — actually traces back to 1976. That year scientists reported discovering that TCDD — the most toxic and best studied of the 75 dioxin species — binds with a receptor protein residing in the cells it invades. Only recently, however, did a group of 38 international dioxin experts unanimously conclude that *every one* of TCDD's myriad effects appears to begin with the compound's binding to this receptor — a mechanism resembling that of the body's own steroid hormones.

"Those biological responses [to TCDD] that have been examined in great detail have all been shown to involve this receptor," says EPA toxicologist Linda S. Birnbaum, one of the scientists who reached agreement at the dioxin conference held at Cold Spring Harbor Laboratory (N.Y.) in late 1990. She says EPA hopes to base a new assessment of human health risks from dioxin — and new regulations — on the recently recognized universality of this receptor in TCDD's effects.

In the 15 years since scientists first realized that dioxin binds to a receptor, called aryl hydrocarbon (Ah), they have developed a detailed picture of how TCDD acts on individual cells. For example, the Ah receptor actually comprises several proteins that cluster together in the liquid interior of most cells in the body. Once dioxin seeps into a cell and links up with these proteins, the TCDD-protein complex can enter the cell's nucleus and cause trouble by meddling with the on-off switches of genes.

Cells of some tissues, such as the liver, teem with Ah receptor proteins, while others may contain only a few. Why our cells should produce such receptors for dioxin remains a mystery.

Perhaps the body produces a hormone that normally operates through the Ah receptor, speculates Thomas A. Gasiewicz of the University of Rochester (N.Y.) School of Medicine. As scientists come to understand the similar and overlapping actions of our natural chemical messengers — hormones and neurotransmitters — with toxicants and drugs, traditional definitions are blurring, he says.

"Just because a compound binds to a receptor doesn't mean it's necessarily going to be toxic," Gasiewicz observes. Any natural hormone that binds to the Ah receptor probably plays a healthy role in regulating cell growth, he says. Even steroids — vitally important hormones that act through protein receptors — can turn "toxic" when their levels get out of whack, he adds. For instance, excess estrogen can lead to cancer.

Scientists have no clues as to the identity of the hormone that normally binds to the Ah receptor, but they assume it physically resembles TCDD, for which there's a perfect docking site on one protein subunit of the Ah receptor. Once TCDD enters the cell, it binds with the receptor and evicts other subunits, called heat shock protein 90.

The remaining TCDD-receptor complex must join yet another protein, however, before it can interact with genes in the cell's DNA, Gasiewicz reported in the March 19, 1991 BIOCHEMISTRY. This additional protein, called the Ah receptor transforming protein (Art), does not directly bind to TCDD, he found, but instead seems to enable the whole complex to hook up with DNA. Gasiewicz now theorizes that Art, which may vary slightly in structure according to the tissue, might steer the complex to act on certain genes.

To get at those genes, the TCDD-receptor complex must first enter the cell's nucleus. Although it's not clear just which events occur in the liquidy cytosol surrounding the nucleus, Oliver Hankinson of the University of California, Los Angeles, has found a protein that must join the complex before the ensemble can gain passage into the cell's center. This protein bears a basic helix-loop-helix structural motif common to DNA-binding proteins. Hankinson reported in the May 17, 1991 SCIENCE. In fact, he told SCIENCE NEWS, it may be the same Art protein that Gasiewicz discovered.

Although they are still identifying the receptor's protein players, Gasiewicz and Hankinson know that it takes at least two proteins and TCDD to create a

In mice, it takes far smaller quantities of TCDD to suppress immunity than it does to unleash most of TCDD's other toxic effects. And white blood cells in both mice and humans respond similarly to TCDD. But to date, there's little evidence to suggest that low-dose exposures to TCDD suppress immunity in humans. Birnbaum, Kerkvliet and Holsapple contend that studies of dioxin-exposed humans have asked the wrong questions.

"If I were to take mice and ask the same [research] questions that are routinely asked of the populations at Times Beach, or in the Ranch Hand study, I would come up with a very nebulous picture [of TCDD's immunotoxicity]," says Holsapple. "But when we ask different questions [in mice], we can certainly show very strong effects on the immune response."

Birnbaum is now calling for a study that will determine how well TCDD-exposed people mount an antibody response to a novel antigen. Perhaps a new flu vaccine – one that uses an influenza strain that hasn't previously infected humans – can serve the function of the sheep red blood cells given to laboratory mice, she says.

But Kerkvliet says EPA shouldn't hold its breath waiting for the definitive epidemiologic study. It would be next to impossible to prove beyond a doubt that dioxin causes immune suppression in humans, she asserts. Unlike sheltered laboratory mice, people come in contact with many immunity-altering forces – such as stress, drugs and disease. Regulators should therefore base their limits for safe exposures to dioxin on animal models and on our developing scientific understanding of TCDD's mechanisms of action, she says.

Kerkvliet suspects that most Americans – who harbor about 30 parts per trillion (ppt) of dioxins in their blood, including about 7 ppt of TCDD – fall below the range of dioxin exposures that can jeopardize immunity. However, she adds, populations that commonly receive higher doses, such as nursing infants (SN: 4/20/96, p.254), chemical workers and people who consume large quantities of fish, could conceivably experience compromised immunity.

"The fact that you can't clearly show the effects in humans in no way lessens the fact that dioxin is an extremely potent chemical in animals – potent in terms of immunotoxicity, potent in terms of promoting cancer," says Kerkvliet. "I simply don't believe that humans represent some unique species." □

Once TCDD (T) seeps into a cell, it binds to an aryl hydrocarbon receptor (AhR) and kicks off the heat-shock protein (hsp90) subunits. The complex then joins an Ah-receptor-transforming protein (Arnt) and passes into the nucleus, where the ensemble binds to DNA and switches a gene on or off. Unidentified "mystery proteins" (?) may also participate throughout this process. An activated gene triggers production of messenger RNA (mRNA), the instructions that a cell then uses to build a specific protein, such as a P450 enzyme.

complex that can turn on a gene. And once that complex binds to DNA, it can activate a gene and thereby cause the cell to produce excessive quantities of a certain protein. Theoretically, dioxin could also turn some genes off, which can also cause ill effects.

Unlike the steroid hormones, which degrade in a few hours, TCDD molecules require seven years to reduce their concentration by half. Because of TCDD's long half-life, it appears that the body cannot regulate this process and the gene's "switch can be turned on for inappropriately long periods of time," Gasiewicz points out. Thus, one TCDD molecule can continuously disrupt normal cell physiology.

In developing a model to explain dioxin's cellular actions, scientists have primarily studied how TCDD turns on a gene for a P450 enzyme. While this specific enzyme normally helps the body excrete toxic substances, it sometimes renders them more potent instead. Though scientists don't know if increased levels of P450 enzymes contribute to any of dioxin's toxic effects, they do know that the TCDD-receptor complex probably flips the P450 gene switch by a mechanism that applies to many other genes as well.

"We're beginning to know the beginning of the story, which is how the receptor activates genes," says Hankinson. "And to some degree we understand the end product [why animals get cancer and why they die]. The real black box is *which* genes are turned on and how they relate to the biological effects of dioxin."

Recently, William F. Greenlee and his colleagues at Purdue University in West Lafayette, Ind., found several new genes targeted by the TCDD-receptor complex. In the Oct. 18, 1991 SCIENCE, they describe identifying two dioxin-responsive genes in human skin cells. The first directs the production of plasminogen activator inhibitor-2, a protein that functions in embryonic development, wound healing, inflammation and cancer. The second gene contains the code enabling a cell to produce cytokine interleukin 1-beta, a protein involved in inflammation and immune responses.

These are the first genetic targets of dioxin to be discovered since the P450 gene, and Greenlee says "these [new] genes are likely to play an important role in the toxicity of TCDD." He says they could plausibly be involved in chloracne – the hallmark skin reaction that usually signals acute human exposure to dioxin. These findings lend credence to a unifying mechanism for all of dioxin's diverse effects, Greenlee says.

"If you look at the broad range of events, it all comes back to a very generic process," he says.

Making the leap from a generic cellular mechanism to guidelines for human exposure – as EPA proposes to do – could prove tricky, however. Some toxicologists argue that receptor involvement implies a certain rate-limiting event – perhaps a minimum number of TCDD molecules needed to bind – before a cell or animal responds with a measurable change in its physiology. This in turn suggests that a "threshold" concentration may exist, below which dioxin causes little or no harm (SN: 5/18/91, p.308).

However, scientists should not assume a safe threshold exists, argues George W. Lucier of the National Institute of Environmental Health Sciences in Research Triangle Park, N.C. To date, his research team has found no predictable, consistent pattern in the dose-response relationships for a number of dioxin's toxic effects – nor evidence of any thresholds.

"My data might not prove that a threshold doesn't exist," Lucier concludes, "but there's also no evidence to support that one does exist."

Still, which ever way the chips fall, Lucier says he's pleased that EPA is finally attempting to incorporate recent research findings into an updated view of dioxin's human toxicity.

"A lot of dollars are spent doing mechanistic research," he comments. "There are thousands of papers on dioxin. We ought to be able to use some of that information in the risk assessment process." – *K. Schmidt*

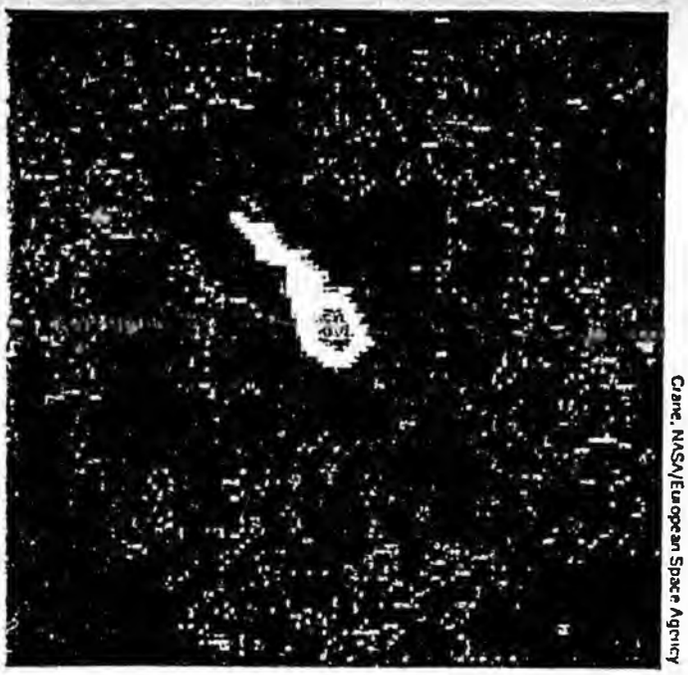
Hubble images reveal unusual galactic jet

Viewed from Earth in visible light, the elliptical galaxy NGC 3862 doesn't look like much. Indeed, its flat emission pattern, apparently devoid of sharp peaks and dips in intensity, once prompted researchers to describe this galaxy as "optically dull."

But orbiting 380 miles above Earth, the Hubble Space Telescope now reveals that the nucleus of NGC 3862 spews out a short jet of radiation, too short to have been detected with ground-based telescopes, in both visible light and the near ultraviolet. Moreover, this jet shines more brightly in the ultraviolet — at the shorter end of the electromagnetic spectrum — than at longer wavelengths, a feature diametrically opposite to the energy output of any other galactic jet yet observed.

"It appears that we are seeing a new type of phenomenon," says Philippe Crane of the European Southern Observatory (ESO) in Garching, Germany.

Researchers believe that a typical jet, possibly powered by a massive black hole or other potent energy source at the center of a galaxy, radiates because of the acceleration of electrons that circle strong galactic magnetic fields. This radiation, known as synchrotron radiation, has a higher intensity at redder, or longer, wavelengths. The well-studied jet in the galaxy M87, for example, fits this model perfectly, says Crane (SN: 1/25/92, p.52).



Ultraviolet image of the galaxy NGC 3862, viewed with Hubble's Faint Object Camera, shows jet (elongated white area) extending from the galaxy's core.

Crane, NASA/European Space Agency

Crane speculates that the unique radiation pattern from NGC 3862, a resident of the Abell cluster of galaxies, could represent a combination of two types of emissions: standard synchrotron radiation as well as radiation, primarily in the ultraviolet, from atoms that surround the jet and are heated by it. If this interpretation proves correct, it would mark the first time that astronomers have observed both types of radiation from a jet.

Crane notes, however, that recent observations with the ESO's New Technology Telescope in La Serena, Chile, found no evidence of atomic emissions. Alternatively, he adds, the jet's output may stem from galactic mechanisms not yet understood. — *R. Cowen*

Perinatal dioxin feminizes male rats

When delivered to pregnant rats, a very low dose of dioxin can not only demasculinize but also feminize the sexual development of male offspring, a trio of new studies shows. The lasting reproductive effects — both behavioral and physiological — occur at doses well below those causing visible toxicity.

Scientists at the University of Wisconsin-Madison had shown that overtly toxic doses of TCDD, the most potent dioxin, can reduce concentrations of androgens — the male sex hormones, such as testosterone — in the blood of adult animals. Because TCDD crosses the placenta, these researchers wondered if dioxin exposures before and immediately after birth might also alter androgen levels and the role of these hormones in a male animal's sexual development.

Thomas A. Mably and his co-workers provided such perinatal exposures with a single oral dose of TCDD to female rats on day 15 of their pregnancy — a time when organ formation in the fetal pups was nearly complete and the males were ready to produce androgens. Though the pups' TCDD exposure undoubtedly began *in utero*, notes Dick Peterson, who led the three Wisconsin studies, earlier data indicate a pregnant animal will eliminate most of the fat-seeking toxicant through breast milk. Peterson therefore believes nursing provided the bulk of the pups' dioxin.

In the May TOXICOLOGY AND APPLIED PHARMACOLOGY, his team reports finding that perinatal exposures to TCDD produced dose-dependent changes in androgens and their reproductive effects "into adulthood."

Compared to male pups whose mothers received no dioxin, TCDD-exposed pups developed smaller accessory sex organs (such as the ventral prostate), appeared to mature sexually more slowly, exhibited distinctly feminine-style regulation of one hormone related to testosterone production and expressed a greater willingness to assume a receptive-female posture when approached by a sexually stimulated male. Even the lowest dose of TCDD delivered — 0.064 microgram per kilogram of the mother's body weight, a level well below what the researchers had expected would produce any quantifiable effects — yielded consistent reductions in a male offspring's daily sperm production and sperm reserves.

Other recent studies suggest that TCDD may act as an "environmental hormone" (SN: 1/11/92, p.24). It now appears that the developing male reproductive system is more sensitive to the effects of this hormone-like toxicant than any other organ or organ system studied, the Wisconsin scientists write.

Though these changes did not affect the rats' fertility, Peterson notes, "that does not mean these findings do not have human health implications."

Male rats normally inseminate a female with up to 10 times as many sperm as are typically needed to ensure impregnation. Humans, by contrast, typically release only about as many sperm as would be required for fertilization. "As a result," Peterson and his coworkers write, human reductions in sperm production "similar in magnitude to that in rats would be expected to reduce fertility in man."

"Highly significant" is how Linda S. Birnbaum characterizes the findings. Director of environmental toxicology at EPA's health effects lab in Research Triangle Park, N.C., she was impressed by the subtle, permanent reproductive-system changes from very low-level TCDD exposures and by the "failure [of the team] to find a no-effects level."

"The real question is how general these effects are," Birnbaum says.

Her lab will repeat the studies with another strain of rats — and, eventually, other species. Unlike Peterson's group, EPA's experiments will also look at females, she said, "because there may be effects on them as well."

And if this effect holds in another species? "I would get very concerned [about the potential human-health implications]," Birnbaum says. — *J. Raloff*

World Watch
July/Aug '92

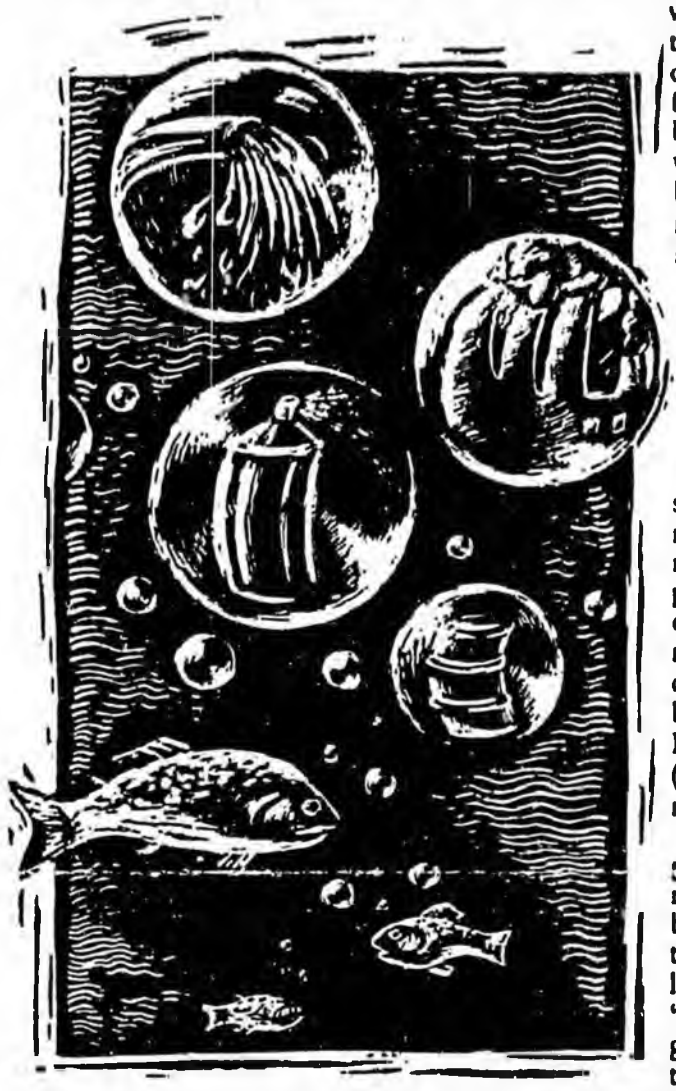
Chemical Reaction in the Animal Kingdom

BY ANN MISCH

Something strange is going on in the animal kingdom, especially among vertebrates living close to water. Scientists in the field have noticed immune suppression, abnormal behavior, wasting (a gradual shriveling and dying), and reproductive failure in beluga whales inhabiting Canada's St. Lawrence River, fish living in polluted urban bays, and numerous species of birds, fish, and mammals in and around the Great Lakes.

Viral epidemics have broken out four times in the past five years among striped and bottlenose dolphins—twice in the Mediterranean Sea, once off the Atlantic Coast of the United States, another time in the Gulf of Mexico—and twice in seals. Fish pathologists have also detected unusually high rates of liver tumors and other abnormalities, such as eroded fins, among fish drawn from the Chesapeake Bay, the Puget Sound, the Hudson and Buffalo rivers in New York, the Black River in Ohio, and coastal waters surrounding Los Angeles.

Just what is going on? Across this broad range of geography and malady



there is a common denominator: the presence of three families of industrial chemicals, both in the animals' habitats and in their tissues. What the field studies have found does not constitute a phenomenon quite like the DDT catastrophe of years past, when a single highly toxic chemical was found to be the cause of massive die-offs of birds. Here, the pattern is subtler and more complex—a spectrum of chemicals linked to a pattern of biological abnormalities. The discovery has come about as a result of growing sophistication in the methodologies available to scientists monitoring the effects of toxins on biological systems.

The new evidence suggests that low-level chemical exposure resulting in long-term accumulation in the body may lead to effects quite different from those dramatic collapses that have previously driven the formation of public policy on chemical effluents. In fact, some scientists now believe these newly revealed effects—such as breakdowns in the immune or reproductive systems and normal development running amok—may replace cancer and mortality as the most significant health threats posed by these chemicals. The implicated substances are organochlorines (dioxin, PCBs, the now-banned DDT, and pesticides such as dieldrin and mirex), aromatic hydrocarbons (including the carcinogen benzo(a)-pyrene, or BaP), and heavy metals (cadmium, lead, and mercury).

In the past, toxicologists often assessed the risk posed by chemicals by the incidence of mortality or cancer caused in laboratory animals. "These are relatively gross health indicators," though, says Katherine Davies, one of many scientists who have contributed to discussions on pollution and wildlife in the Great Lakes held by the International Joint Commission. Laboratory researchers are now uncovering an array of subtler, non-lethal "endpoints"—or measurable effects—that earlier studies had overlooked.

Many of these effects are unleashed at lower levels than those at which cancer appears. This has led some scientists to call for changes in state and federal standards of safe exposure, which are traditionally based on doses set low enough to avoid cancer. Theo Colburn, a toxicologist and senior fellow at the World Wildlife Fund in Washington, D.C., said in recent testimony before the Senate Committee on Governmental Affairs, "you can't find the science behind the regulatory decisions that are being made."

What researchers are especially alert for now are hidden effects of exposure to toxic compounds that persist in the environment for many years and accumulate in the fat of animals high up in the food chain. These effects may be found either in animals directly exposed or in their young, who in some cases inherit the burden of their parents' exposure.

Some synthetic chemicals and heavy metals now appear to disrupt vital physiological processes, such as the regulation of hormones, the functioning of the immune and nervous systems, growth, and reproduction. They might also be responsible for altered behavior, such as the indifference Forster's terns around Lake Michigan show toward their young. Normally, biologists expect to see adult terns lavish attention on their helpless, nest-bound offspring.

Dioxin, for instance, is known to toxicologists as an "environmental hormone" because it seems to offer itself as a hormone substitute. Depending on the situation, dioxin can either block or enhance the effects of the animal's own hormones.

Dioxin's effects can show up in offspring even when the parents exhibit no outward signs of exposure. For instance, exposing pregnant rats to dioxin lowers testosterone levels in the blood of their newborn rat pups, finds Professor Dick Peterson and coworkers at the University of Wisconsin's School of Pharmacy. As the male rats mature, their sexual development, which is normally propelled by testosterone surges, lags behind. ①

TRENDS • Contd.

②

Exposure to toxics may also impair an animal's ability to produce offspring. Pierre Beland, director of the St. Lawrence National Institute of Ecotoxicology, and Daniel Martineau, a pathologist at Cornell University's School of Veterinary Medicine, have observed reproductive complications and other health problems among an isolated population of beluga whales that inhabit a polluted stretch of Quebec's St. Lawrence River. They have found fewer juvenile belugas among this group than among beluga populations in the Arctic. Their analyses of tissue samples from dead whales have revealed high levels of mercury, lead, cadmium, PCBs, DDT, mirex, dioxin, and furans, among other toxics.

Beland and Martineau conclude that a "direct cause and effect relationship is likely to exist between the various toxic compounds present in tissues and the health and reproductive status of this population."

While absolute proof that chemicals *cause* disease will always elude field biologists, researchers nonetheless have two important tools to assist them in tracing connections between pollutants and disease. First are epidemiological studies drawing correlations between measured levels of chemicals and observed symptoms in wildlife, such as those carried out by Colburn, Beland, and Martineau. Second are more precise lab tests, such as those performed by Dick Peterson, that draw a clearer picture of the multiple effects of toxins within cells and among different organs and systems.

When associations between chemicals and diseases are established in field studies, and the toxicity of those chemicals can be recreated in the laboratory, further "proof" becomes moot. Daniel Martineau dryly comments that to actually *prove* that particular chemicals caused the problems he and others have observed in the beluga whales, "you'd have to have a perfectly clean St. Lawrence on one side and a polluted St. Lawrence on the other." The inherent uncertainty involved in tracing a single chemical to a particular symptom may itself be the best argument for taking correlations—which scientists now have in abundance—more seriously.

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IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF NEW YORK

Agent Orange
Vernon Houk
+
Dioxin

SHIRLEY IVY, Individually and as Representative of the Estate of DONALD IVY, *et al.*

Plaintiffs,

v.

DIAMOND SHAMROCK CHEMICALS COMPANY, *et al.*

Defendants.

CV-89-03361 (E.D.N.Y.) (JBW)

[B-89-00559-CA (E.D.TEX.)]

AFFIDAVIT OF CATE JENKINS, PH.D.

RECENT SCIENTIFIC EVIDENCE DEVELOPED AFTER 1984 SUPPORTING A CAUSAL RELATIONSHIP BETWEEN DIOXIN AND HUMAN HEALTH EFFECTS

V. CONCLUSION

420. A large volume of new research published since 1984 demonstrates a wide range of adverse health effects associated with dioxin. The populations where these effects are found include Vietnam veterans, residents living in Missouri where contaminated oil was used on roads, chlorophenol and phenoxyacetic acid chemical production workers in the U.S. and other countries, farmers using phenoxyacetic acid herbicides, and residents exposed to dioxin contamination in Seveso, Italy from an industrial explosion.

421. Many Vietnam veterans and other populations exposed to dioxin have experienced more than one of the adverse health effects associated with dioxin. Such a coincidence of injuries increases the probability that the common causal factor for the multiple injuries was dioxin rather than two or more coincidental factors. The range of human populations exposed to dioxin experiencing these heath effects (Vietnam veterans, farmers, forestry workers, residential populations in Missouri and Italy, and chemical production workers in the U.S. and other countries) establishes a firm basis for concluding that dioxin, and not some other unique factor related to service in Vietnam, was responsible for these health effects. Furthermore, many Vietnam veterans as well as other populations exposed to dioxin have experienced dose-related increased rates of these adverse health effects, providing strong epidemiologic evidence that the effects were caused by, and not merely associated with, dioxin. In all cases, animals have experienced these same health effects when dioxin is administered in a controlled laboratory setting, thus providing a plausible biological basis for the health effects observed in humans.

422. The effects demonstrated by these new studies to be significantly associated with dioxin exposures include elevated cancers of all sites combined (representing a general carcinogenic effect of dioxin), as well as cancers of specific sites, namely: soft tissue sarcomas; non-Hodgkin's lymphoma; Hodgkin's disease; leukemias, lymphomas, and other hematologic cancers; respiratory system cancer; skin cancer; testicular cancer; and cancers of the brain, stomach, colon, rectum, prostate, hepatobiliary tract, pancreas, and kidney. One adverse effect in addition to cancer significantly associated with dioxin is organic nerve damage, including peripheral as well as central nervous system damage, and the severe consequences of central nervous system damage, such as suicide and fatal accidents, depression, anxiety, and other neuropsychological problems. Other adverse effects significantly associated with dioxin include reproductive abnormalities; immunological abnormalities; dermatologic abnormalities; hepatotoxic effects; gastrointestinal ulcer; cardiovascular disorders; metabolic disorders such as porphyria cutanea tarda, thyroid dysfunction, diabetes, and altered lipid metabolism; and lung and thorax abnormalities.

423. On the basis of the evidence analyzed and summarized above, including epidemiologic evidence concerning the consequences of exposure to dioxin and phenoxyacetic acid herbicides, it is my opinion to a reasonable scientific certainty that the above mentioned adverse health effects and reproductive outcomes, and physical, social, and neuropsychiatric and neuropsychological consequences of these adverse health effects, were probably caused by the plaintiffs' exposures to Agent Orange while in service to our country in the Vietnam War.

The foregoing affidavit and appendices are a true and accurate statement of my scientific assessment in the above-captioned case to the best of my knowledge and belief.

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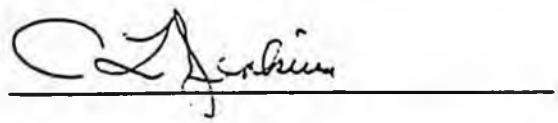

Cate Jenkins, Ph.D.

Signed and sworn before me by CATHERINE L. JENKINS this 3RD day of SEPTEMBER, 1991

Elizabeth H Smith
Notary Public, District of Columbia
My Commission Expires Sept. 14, 1995

My commission expires the _____ day of _____.

Elizabeth H. Smith
Notary Public, District of Columbia
My Commission Expires Sept. 14, 1995

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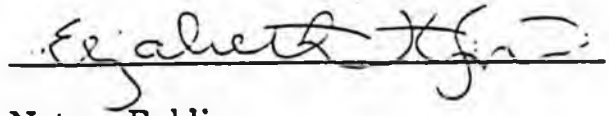

Notary Public

EXHIBIT I

IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF NEW YORK

SHIRLEY IVY, Individually and as Representative of the Estate of DONALD IVY, et al.	[]	
Plaintiffs,	[]	CV-89-03361 (E.D.N.Y.) (JBW)
V.	[]	[B-89-00559-CA (E.D.TEX.)]
DIAMOND SHAMROCK CHEMICALS COMPANY, et al.	[]	
Defendants.	[]	

AFFIDAVIT OF ADMIRAL ELMO R. ZUMWALT, JR.

BEFORE ME, the undersigned authority, appeared Admiral Elmo R. Zumwalt, Jr., USN (Ret.), 1500 Wilson Blvd., Arlington, VA 22209, and after being duly sworn does testify and declare as follows:

1. From 1968 to 1970 I served as the Commander of U.S. Naval Forces, Vietham. From 1970 to 1974 I served as the Chief of Naval Operations and a member of the Joint Chiefs of Staff.

2. On October 6, 1989 I was appointed Special Assistant to Secretary Derwinski of the Department of Veterans Affairs ("VA") to assist the Secretary in determining whether there is a significant ("as likely as not") statistical association between exposure to Agent Orange and any specific adverse health effect.

3. As Special Assistant, it was my duty, with the assistance of independent scientific experts, to do the following: 1) evaluate scientific studies regarding the health effects of Vietnam veterans exposed to Agent Orange, as well as numerous studies concerning the health hazards of civilian exposure to dioxin contaminants; 2) review and evaluate the protocol and standards employed in the major government sponsored studies, in order to assess their credibility, fairness and consistency with generally accepted scientific practices; and 3) review and evaluate the work of the Scientific Council of the Veterans' Advisory Committee on Environmental Hazards.

to harmful doses of Agent Orange.

30. As the Committee concluded, the blood serum analysis, used as proof that an exposure study could not be conducted, was based on erroneous assumptions and a flawed analysis. For example, CDC's conclusion that the half-life of dioxin in the human body is 7.1 years was reached in disregard of warnings from CDC's own scientists and the National Academy of Sciences Institute of Medicine ("IOM") peer review committee that there was not sufficient evidence to support the longer half-life. IOM informed CDC that, because of the incorrect assumptions about the half-life of dioxin, the conclusions of the blood study were not supportable. IOM also rejected CDC's conclusion about the inadequacy of military records as a basis for exposure estimates, independent of any blood serum analysis.

Vernon Houk

31. In his testimony, under questioning from the Subcommittee Chairman, Rep. Weiss, Dr. Vernon Houk admitted that the senior statistician on the Agent Orange project believed that the dioxin blood analysis was so flawed that it had "a substantial likelihood that there would be essentially no correlation" between the exposure scores and the blood levels. Dr. Houk disagreed with this officially expressed opinion of the project's senior statistician and supported the validity of CDC's blood serum analysis. 1989 Agent Orange Hearing at 67 (statement of Vernon N. Houk, M.D., Director, Center for Environmental Health and Injury Control, Centers for Disease Control, U.S. Department of Health and Human Services).

32. In the course of my duties as Special Assistant to the Secretary of Veterans Affairs I have specifically reviewed the work of Dr. Vernon Houk in connection with Agent Orange studies, both in his capacity as a member of the AOWG and as a CDC official. It is my conclusion that Dr. Vernon Houk has made it his mission to manipulate scientific data and procedures so as to prevent the true facts about dioxin from being determined. Continuing to pursue this effort, Dr. Houk has been recently quoted in Time magazine, Aug. 26, 1991, The New York Times, Aug. 15, 1991, St. Louis, Washington, and other newspapers and media outlets, in an apparent public relations campaign, falsely claiming that previous assessments of the harmful effects of dioxin have been overestimated. These articles cite no credible basis for Dr. Houk's opinion. I consider this media campaign further evidence of Dr. Houk's attempts to cover up emerging evidence strongly confirming the harmful effects of dioxin.

33. Dr. Houk's politically motivated efforts to cover up the true effects of dioxin, and manipulate public perception, coincide with the similar, economically motivated, efforts of chemical companies that produce dioxin. They are, in my judgment, responsible for letters and articles that have been published in the media discounting the effects of dioxin on human

health. These chemical companies that place profits above other concerns, were very cunning in working out the 1984 Agent Orange settlement deal before all the scientific information -- such as that now presented in Dr. Jenkins' affidavit in this case or the disclosures recounted here about the way in which the supposedly objective government studies were manipulated -- became available. It is apparent that these same companies are now attempting to support the validity of that settlement, at the very time it is under review in this case, by means of a public relations campaign centered around the statements of Dr. Vernon Houk, whose work on the CDC Agent Orange study has been thoroughly discredited.

34. Upon discovering the irregularities in CDC procedures, Dr. Philip Landrigan, who was the former Director of the Environmental Hazards Branch at the CDC, stated: "Suspicion abounds that CDC did not look deeply enough into the existing records and did not exercise sufficient ingenuity in seeking to identify a potentially heavily exposed subset of veterans. . . . Further, I would argue that CDC should itself be raising the question." 1989 Agent Orange Hearing at 229 (statement of Philip J. Landrigan, M.D.).

35. In 1986, the Subcommittee on Oversight and Investigations of the Committee on Energy and Commerce documented how political officials of the Office of Management and Budget ("OMB"), who were untutored in science, interfered with and second-guessed the professional judgments of agency scientists and multidisciplinary panels of outside peer review experts to effectively alter or forestall CDC research on the effects of Agent Orange, primarily on the grounds that "enough" dioxin research had already been done. OMB Review of CDC Research: Impact of the Paperwork Reduction Act: A report Prepared for the Subcommittee on Oversight and Investigations of the Committee on Energy and Commerce, 99th Cong., 2nd Sess. (1986).

36. The Committee on Government Operations' Agent Orange hearings revealed additional examples of political interference in the CDC's Agent Orange projects by members of the White House Agent Orange Working Group. Evidence of political interference in the design and implementation of the CDC study and drafting of the results of the CDC study by Administration officials rather than CDC scientists, further destroys the credibility of the CDC exposure study efforts. The Committee concluded from this evidence, as do I, that the CDC study of the effects of dioxin on Vietnam veterans was controlled and obstructed by the White House, primarily through its AOWG and the OMB so as to prevent any useful findings by the CDC concerning the effects of Agent Orange. This obstruction was done pursuant to a strategy for denying liability in cases of toxic contamination.

I. NEW EVIDENCE SHOWING A SIGNIFICANT ASSOCIATION BETWEEN AGENT ORANGE EXPOSURE AND CERTAIN HEALTH EFFECTS

4. There is mounting evidence of a causal connection between certain illnesses and exposure to dioxins. After reviewing the scientific literature, I have concluded that the following illnesses are significantly associated with exposure to Agent Orange: non-Hodgkin's lymphoma, chloracne and other skin disorders, lip cancer, bone cancer, soft tissue sarcoma, birth defects, skin cancer, lung cancer, porphyria cutanea tarda and other liver disorders, Hodgkin's disease, hematopoietic diseases, multiple myeloma, neurological defects, auto-immune diseases and disorders, liver cancer, nasal/pharyngeal/esophageal cancers, leukemia, malignant melanoma, kidney cancer, testicular cancer, pancreatic cancer, stomach cancer, prostate cancer, colon cancer, brain cancer, psychosocial effects, gastrointestinal diseases, diabetes, and cardiovascular abnormalities.

5. In addition to my report on the scientific studies showing an association between health effects and Agent Orange exposure, I have reviewed the affidavit of Dr. Cate Jenkins submitted in this action. Dr. Jenkins' affidavit compiles a growing number of studies showing a link between dioxin exposure and serious health effects. This extensive list of positive studies, all published in 1984 or later, further supports my conclusions stated above.

II. FAULTY CONCLUSIONS, FLAWED METHODOLOGY AND NOTICEABLE BIAS OF THE VA ADVISORY COMMITTEE ON ENVIRONMENTAL HAZARDS

6. In 1984, Congress passed the Veterans' Dioxin and Radiation Exposure Compensation Standards Act, Pub. L. No. 98-542, 98 Stat. 2727 (1984) (the "Dioxin Standards Act") to provide disability compensation to Vietnam veterans exposed to herbicides containing dioxin. Congress authorized the VA to conduct rulemaking to determine which diseases were entitled to compensation as a result of a service-related exposure to Agent Orange. The Dioxin Standards Act required the VA to appoint a Veterans' Advisory Committee on Environmental Hazards (the "Advisory Committee") to review the scientific literature on dioxin and submit periodic recommendations and evaluations to the Administrator of the VA. The Advisory Committee is composed of experts in dioxin and epidemiology, as well as interested members of the public. The responsibility of the experts is to evaluate the scientific evidence pursuant to regulations promulgated by the VA and thereafter to submit recommendations and evaluations to the Administrator of the VA (subsequently the Secretary of Veterans Affairs) on whether "sound scientific or medical evidence" indicated a connection between exposure to [por-fu-rea] the manifestation of various diseases.

Water Quality Regulations Hearing

10-03-92

file 2

Alaska State Legislature

Please enter into the record my testimony to the House Resources Committee
committee name

committee on water quality standards, dated Oct 3, 1992.
bill/subject

I would first like to comment that this hearing should have been organized differently since all members of the public who wish to have testified were penalized by your lack of time control on the first part of your agenda.
I am against change to the water quality standards. I believe they are a short-sighted attempt to protect some industries at great cost to other renewable industries and the future of Alaska. I feel that costs to ecosystems should be considered more, as well as human health, particularly with regard to dioxin, where costs to non-humans are well-proven. I believe we have inadequate oceanographic knowledge to safely use mixing zones, and they should be forbidden. I will submit further written comments when I have time to prepare them. Thank you.

Signed: Page Else
Testifier

Sitka Conservation Society
Representing (Optional)

2219 Sawmill Creek, Sitka
Address

747-7448
Phone No.

MICHAEL J. FRANK
2224 TURNAGAIN PARKWAY
ANCHORAGE ALASKA 99517

PH: 248-5078

Representative Cliff Davidson
Alaska Legislature
House Resources Committee
3111 C. Street Suite 415
Anchorage, Alaska 99503

Dear Rep. Davidson:

On October 3 I attended the House Resources Committee's oversight hearings on DEC's proposed amendments to the water quality regulations, but had to leave before public testimony was taken. Enclosed are comments I submitted to DEC on September 30 which may be of interest. I have a few additional observations to make.

Both Commissioner Sandor and Mr. Sturdevant asserted during the oversight hearing that DEC was not proposing to weaken the water quality standards ("WQS") for aquatic life. This claim must be facetious, since in practice DEC has ignored the WQS for aquatic life by routinely granting generous mixing zones.

The current regulation for mixing zones permits "(t)he water quality standards set out in this chapter ...(to) be exceeded within a mixing zone prescribed by the department." 18 AAC 70.032(a)(1). DEC's current regulatory proposal would add a new subsection (e) to the mixing zone regulation that would further allow the WQS for aquatic life to be exceeded except when some undefined measure of permanent damage occurred. See proposed 18 AAC 70.032(e)(3)(i) and (iii).

DEC has already given generous mixing zones to virtually every major point source of pollution in the state, including Alyeska's Valdez Marine Terminal, the southeastern pulp mills, most fish processors, Cominco's Red Dog Mine facility, publicly owned treatment works --- you name it and odds are that you'll find a mixing zone in which the WQS for aquatic life are, at least potentially, exceeded. Thus, DEC's claim that its current proposals do not reduce the WQS for aquatic life is essentially meaningless since DEC already allows the WQS for aquatic life to be ignored by granting generous mixing zones.

I argue in the enclosed comments that mixing zones are of very questionable legality. I think they originally were granted in order to give industries in place when the Federal Water Pollution Control Act Amendments of 1972 were passed time to install advanced pollution control technology. While they were allowed to use mixing zones, in the meantime point sources were supposed to be making reasonable further progress towards the Act's goal of no pollution discharges by 1985. Unfortunately, in the Reagan era under EPA-administered NPDES permits, mixing zones became the rule, not the exception, and therefore DEC could hardly resist the temptation to allow mixing zones as part of NPDES certifications and state wastewater discharge permits.

I believe it would be very revealing if the House Resources Committee could have DEC inventory the existing mixing zones, giving the date they were created and their size, describing any expansion that has occurred, detailing the bioassay or other testing that has occurred within and without the mixing zones, describing what public comment was solicited, and so on.

On a different subject, arsenic, the House Resources Committee may also want to speak with State Epidemiologist Dr. John Middaugh. I spoke with him recently about DEC's use of a study he coauthored concerning arsenic in Fairbanks area water wells. He thought DEC had completely misinterpreted the results of his study, and was somewhat taken aback that he was not consulted before DEC issued its arsenic issue paper.

I appreciate the time that you have devoted to this subject, and hope you continue to keep an active role in DEC oversight.

Sincerely,

Michael J. Frank

Michael J. Frank

encl.

MICHAEL J. FRANK
2224 TURNAGAIN PARKWAY
ANCHORAGE, ALASKA 99517

Ph: 248-5078

September 28, 1992

Dave Sturdevant
Water Quality Management
Department of Environmental Conservation
410 Willoughby Avenue, Suite 105
Juneau, Alaska 99801-1795

Re: Water Quality Regulations - Proposed Amendments

Dear Mr. Sturdevant:

Please accept the following comments on the Alaska Department of Environmental Conservation's ("DEC's") proposed amendments to certain of DEC's water quality regulations which proposals were noticed for public review this past summer.

Before addressing specific regulatory changes, I would like to make some general comments on DEC's proposals.

First, some of the suggested changes appear designed to update the regulations to reflect current scientific thinking --- or at least one side of current scientific thinking --- and DEC's motivations in that regard are good. The general tenor of the proposals, however, seems to ignore the fundamental motivation behind the pollution laws enacted over the last twenty years: to pass on a clean world to the next generation. Certain of the proposals (e.g., those dealing with dioxin, arsenic and chloroform, and the 96 hour LC 50) seem bottomed on an evaluation of the comparative risks of pollutants to this generation of water users or on presumed "temporary" effects of a pollutant on aquatic life. But as a doctrine comparative risk is blind to inter-generational values. Its use

as a rationale for allowing certain levels of pollutants to enter otherwise clean water systems virtually assures that the next or following generations of Alaskans will not have as clean an environment as this generation.

Even when comparing and taking risks is necessary in making regulatory judgments, DEC must base it on reasonably complete scientific data. When the data is incomplete or highly disputed (e.g., in the case of dioxin), the choice should be easy: prevention of the introduction of any level of pollution until more is known about the risks. This choice is consistent with DEC's statutory purposes. Instead, DEC's discussion of the purposes behind some of the regulatory changes (in particular those for dioxin, arsenic and chloroform) seems to shift burden of proof: DEC won't regulate any potential pollutant very strictly absent proof of the certainty of the pollutant's harmful effects. This is simply inconsistent with DEC's "responsibility as trustee of the environment for present and future generations." AS 46.03.010(b).

Second, some of DEC's proposals also do not insist on economic efficiency (that each economic transaction should reflect its true cost) or on fairness (that those who cause costs should pay for them). These principles are consistent with mainstream principles of environmental protection *and* free-market economics. As an example, DEC proposes a new human health sta dard for dioxin which purportedly would have the effect of freeing the Ketchikan and Sitka pulp mills of stricter NPDES permit requirements otherwise derivative of EPA's proposed National Toxics Rule. The effect, however, may be to convert the mills' cost of controlling dioxin pollution into a long term social cost borne by the public. The adoption of a more lenient standard for dioxin may also be unfair to competing firms in the same industry which are incurring pollution control costs that reflect either more stringent pollutant discharge standards or a stronger long term commitment to install advanced pollution control technology. In that regard DEC's regulatory issue papers are myopic in their failure to even discuss what the industry is capable of or is doing elsewhere to meet pollution control requirements

Third, in many respects the Public Review Packet makes a sincere effort to lay out the rationale for DEC's proposals in separate issue papers and "fact" sheets. DEC should be applauded for developing separate issue papers, as they ordinarily make it easier for a member of the public to understand an issue and determine DEC's position on it.

Nonetheless, many of the DEC informational documents fall short of good quality and provide an inadequate basis for understanding DEC's rationale in making particular proposals. Among the defects are (1) a redundancy in the information supplied in the fact sheets and issue papers, which makes it appear that there is more information given on a certain topic than is actually there; (2) a failure to have a bibliography in some of the issue papers or accompanying fact sheets (see e.g., the proposed change with respect to mixing zones) so that a member of the public can review informational documents beyond the documents DEC has provided in the Public Review Packet; (3) a failure to cite to scientific information sources *within* an issue paper or a fact sheet even when DEC has otherwise given a bibliography, and instead citing to inappropriate secondary and tertiary sources (see e.g., citations to newspapers and non-scientific magazine articles in the arsenic and dioxin documents); (4) an incomplete citation to sources [see e.g., citation to "(Dickason, 1991)" in the issue paper for the Total Hydrocarbon standard, at page 10, and citation to NCASI, 1990 in the chloroform regulatory issue paper, at page 1, without further identifying information]; and (5) a citation to an industry source as the sole, apparently unchecked, evidence for a proposition (see e.g., citation to the National Council of the Paper Industries for Air and Stream Improvement for the proposition that "the body weight method is more appropriate, etc." in the chloroform issue paper, at page 1).

While DEC is not obligated to create a formal rule making record in support of a regulatory proposal, the Alaska Administrative Procedures Act, AS 44.62, requires that public notice and agency documentation of the proposed changes be sufficient to give a member of the public enough data for the further pursuit of data gathering, informed comment and decision-making. In this regard DEC's supporting documentation often fails the test.

Fourth, and an equally crucial defect that relates to public participation, the Public Review Packet fails to address in any comprehensive fashion considerations highly relevant to certain of the proposals DEC makes. Among these omissions are (1) a failure to include at least a summary of Triennial Review comments received in 1990, although the Triennial Review is DEC's expressed basis for proceeding ahead with the instant proposals; (2) a failure to identify water bodies on which permitted operations exist or are likely to be established (e.g., an existing mining operation with arsenic discharges to the proposed AJ Mine reservoir which may benefit from the proposed re-definition of "water") and which will be impacted in the near term by DEC's proposals, thus making

it impossible for the public to relate a particular regulatory proposal to water bodies that may deserve special protection or for which there is other information relevant to the proposal; (3) a failure to identify existing and major state and federal wastewater discharge permits that will be altered if the proposals become effective, and in that same context (4) a failure to explore in any detail the potential interplay of the proposed amendments, existing wastewater discharge permits and the current and more stringent water quality standards ("WQS") with the anti-backsliding rule of the Water Quality Act of 1987 ("WQA"), Pub. L. 100-4, February 4, 1987, § 404(a), 33 USC § 1342 [amending ¶ 402 of the Federal Water Pollution Control Act of 1972 ("FWPCA"), as amended by the Clean Water Act Amendments of 1977].

With respect to the anti-backsliding rule in particular, the public cannot make an informed decision as to the potential of DEC's proposals to impact existing permittees without knowing whether the rule will or will not prevent changes in the permits if the regulation amendments are adopted. This issue should have been more fully explored in the Public Review Packet.

Fifth, while providing the public a wholly inadequate data set for making the decision, DEC improperly proposes to adopt a 1 in 100,000 "acceptable" risk level for the establishment of WQS for toxic pollutants. This highly significant decision may cross DEC regulatory lines at many interstices. For the choice of an acceptable risk level has effects not simply at the pollution prevention stage but also in how DEC might deal with the existing threats caused by pollution and any cleanups that will be required or are now ongoing. Each of these stages may require different risk choices, and within each stage risk levels might vary depending upon the pollutant, its environs, the local biotic population at risk, etc. If 1 in 100,000 is an acceptable risk level for establishing a human health WQS for arsenic, will it be for BETX air pollutants from Alyeska Pipeline Service Company's Valdez Marine Terminal? Will it become the acceptable risk level determinant for cleaning soil contaminated with fuel from a leaking underground storage tank in a remote area with no groundwater?

Despite the risk-decision's importance, however, DEC seems nearly flippant in its choice, saying: "Establishing a risk level is a social and economic public policy decision. There is no scientific basis for favoring one risk level over another." *Regulatory Issue Paper: Human Health Criteria For Dioxin*, at page 3. Even were this statement completely true, it argues for a highly focused rule making that allows for an

informed public policy discussion of the issue rather than DEC's near inadvertent, ad hoc adoption of a risk level that seems prearranged only to help the operations of certain industries. In fact, in DEC's *Summary of Changes to Alaska's Water Quality Standards Proposed for 1991* (January 1991), at page 6, DEC indicated that a "human health risk level for carcinogens will be identified", intimating that a separate regulation would address it.

Moreover, DEC's statement is misleading. It erroneously asserts that there is no science used in establishing a risk level. Although defining the magnitude and probability of harm often present problems fraught with difficulties and pure value judgments, this is not always so. Scientific knowledge varies in certainty and completeness, and therefore a decision to accept a 1 in 100,000 or 1,000,000 risk level may well be dependent on one's scientific evaluation of data which support an estimation of the risk. It is not simply a matter of deciding whether one is more or less risk adverse as a matter of general policy. The public may be willing to undertake a 1 in 100,000 risk for establishing a dioxin WQS knowing they will never visit Sitka or Ketchikan, but opposed to use of the same risk level in establishing an arsenic WQS because they often drink from placer-mined streams. These personal calculations are not choices devoid of science, and the public is entitled to demand the best evidence and discussion available before they are made. This is so because the risk of error will fall on them, not on the regulated industry.

The risk-decision's importance further suggests that a focused rule making address the context in which risk decision-making occurs and allocate the burden of proof where there is uncertainty. The burden should be on those who wish to pollute to show that there will be no adverse effects, as opposed to allowing the pollution absent proof of harm. This is only as it should be, in recognition of the fact that of 70,000 chemicals used in commerce, fewer than 10,000 have health effects data and but a 100 have some direct human health data. DEC *#3 Water Quality Standard Questionnaire/January 1992*, at page 1 (quoting a Dr. Kim from the New York Department of Health). A focused rule making should address the risks associated not only with cancer, but with effects on future generations in terms of genetic changes, birth defects, etc. It should address the question of comparative risks: whether it is reasonable, for example, to compare the risk of cancer to someone seventy years of age with the risk of genetic defects to an infant.

That DEC's documentation in the *Public Review Packet* is entirely deficient to form the basis for selection of a 1 in 100,000 risk level with respect to arsenic, chloroform and dioxin is most clearly evident in DEC's *Summary Response to Public Comments on Revision of Alaska's Water Quality Standards* (January 1991), § 12, at page 6. Therein DEC said it was considering a 1 in 1,000,000 as the risk level for human carcinogens. It now proposes 1 in 100,000, but does not given a reasoned basis for lowering the target. This illustrates that the entire topic deserves a thorough public airing divorced from any particular WQS or pollutant before a risk "standard" is adopted.

Sixth, underlying DEC's entire approach to the establishment of WQSs and regulation of wastewater discharges may be a fundamental misperception of the obligations imposed on states by the Federal Water Pollution Control Act ("FWPCA"), as amended, and the limitations and obligations imposed by Alaska Statutes. DEC seems to elevate to primacy WQSs in assuming that only from them does one derive operator discharge limits for NPDES and state wastewater discharge permits. See, e.g., DEC's *The Dioxin Issue: EPA, Alaska and the National Controversy*, (May 1992), at page 18 ("these criteria will be used on NPDES permits instead of EPA's limits where appropriate and as applicable to the specified designated uses"). While it is true that both DEC and EPA have relied on WQSs as the bottomline for establishing permit effluent limits, such water quality based permit limitations, to the extent they add to the pollutant load of a water body, are suspect under the FWPCA.

In fact, in enacting the FWPCA Congress expressly intended to focus attention *away* from WQS and *to* so-called "end of the pipe" limitations as the primary method of preventing pollution and maintaining the quality of unpolluted waters. The legislative history of the FWPCA shows that this is true.

The FWPCA of 1948, ch. 758, 62 Stat 1155 (1948) was the earliest comprehensive federal statute dealing with water pollution. It meekly authorized federal research and cumbersome measures to deal with interstate water pollution. 1965 amendments to the Act required states to classify all waters within the state by their intended uses (e.g., swimming, fishing, water supply, etc.). Thereafter, states were required to adopt ambient water quality standards appropriate to the use for which a water body was "zoned", and adopt implementation plans to control discharges sufficiently to achieve the various standards; all of this was subject to federal approval.

Time consuming enforcement procedures in the FWPCA, a perception that the nation's waters were growing more polluted, and dissatisfaction with the WQSs approach to pollution control kept the federal law in the environmental and Congressional limelight. In particular, WQSs proved difficult to establish when there were multiple points of discharge and pollutants, and proved even more difficult to enforce, since one had to trace back the violation of a WQS to the discharge source. Serious consideration of other amendments to the FWPCA began in 1970, and eventually the Nixon Administration and Senator Edmund Muskie became the driving force behind competing proposals.

The Nixon Administration proposals (made in four separate bills as part of a comprehensive package of amendments to the FWPCA) would have continued the water quality standards approach (per a proposed S. 1014), but the states would have also been required to establish effluent limitations for both municipal and industrial points of discharge as part of WQSs submitted for federal approval. In November, 1971 the Senate passed S. 2270, sponsored by Senator Muskie. S. 2270 rejected the Nixon Administration's proposal for continued reliance on WQSs, instead shifting emphasis to a mechanism of permitted effluent limitations designed to control pollutants at the source in order to prevent their discharge *before* they entered any "navigable" waters of the United States. Application of "best practicable technology" and later "best available technology" at the end of the pipe was required in pursuit of this national policy.

The House of Representatives began deliberations over companion legislation to S. 2270 in November 1971. In the House the Nixon Administration again opposed a shift of emphasis from WQSs to effluent limitations. EPA Administrator William Ruckleshaus favored House-introduced legislation which would have required that effluent limitations only be used as a last resort and then only if socially and economically achievable. U.S. House of Representatives, *Committee on Public Works, Water Pollution Control Legislation - 1971, (H.R. 11896, H.R. 11895).*, Hearings, 92nd Cong., 1st Sess., December 7 - 10, 1971, at page 286. A later House-passed bill in essence continued emphasis on WQSs, and forced a Conference Committee to resolve differences with S. 2270.

The resulting bill approved in conference and in 1972 signed into law was, with minor changes, S. 2270. While the new law amending the FWPCA continued the use of WQSs, it abandoned reliance on them, and

instead switched to a set of technology based requirements specifically constructed for quick and simple implementation. Progressively tighter levels of effluent reduction were established so that the nation would make rapid progress to achieve the new national policy: "It is hereby declared to be national policy that the discharge of pollutants into navigable waters be eliminated by 1985." § 101(a)(1) of the 1972 FWPCA amendments. While the "policy' was not intended to be enforceable in and of itself, it was intended to establish the decisive guideline for implementation of the FWPCA. U.S. Senate, Committee on Public Works, *A Legislative History of the Water Pollution Control Act Amendments of 1972*, 93rd Cong., 1st Sess., 1973, Vol. II, at page 1262.

The new FWPCA amendments detailed factors for EPA to consider in setting effluent reduction levels in each case, including cost, technical feasibility and "non water quality environmental impacts". The impact on water quality, however, was expressly excluded, consistent with the desire to focus on end of the pipe discharge controls. *Weyerhauser Co. V. Costle*, 590 F.2d 1011, 1041-42 (D.C. Cir. 1978); *accord, Assoc. of Pacific Fisheries v. EPA*, 615 F.2d 794, 805 (9th Cir. 1980).

The federal policy of continued advancement toward the no discharge goal was reinforced with the Water Quality Act of 1987's adoption of an anti-backsliding rule, and more recently with passage of the Pollution Prevention Act of 1990. The latter Act indicates that it remains national policy that "pollution should be prevented or reduced at the source wherever feasible...." Pub. L. 101-508 (Nov. 5, 1990), 104 Stat. 1388, 42 USC § 13101 note).

Alaska law is written consistently with this national policy. AS 46.03.010(a) indicates: "It is the policy of the state to conserve, improve, and protect its natural resources and environment and control water...pollution, in order to enhance the health, safety and welfare of the people of the state and their overall economic and social well-being." See also id. at (b). Water pollution is flatly prohibited. AS 46.03.710 ("A person may not pollute or *add to* the pollution of the...water of the state." Emphasis added.) See also, AS 46.03.800(a). The "terms and conditions (of wastewater discharge permits) shall be directed to *avoiding* pollution and to otherwise carry out the policies of this chapter." AS 46.03.110(d)(emphasis added). Anti-degradation strictures are established by regulation. 18 AAC 70.101(b)-(c).

Under the FWPCA and state law, therefore, WQSs establish the fallback protection for Alaskan waters. The central focus, however, is on end of

the pipe prevention. In contrast, DEC seems to presume it acceptable to establish WQSs as a first, and the critical, step in determining end of the pipe discharge limits. It further presumes that mixing zones are acceptable as long as designated uses can be protected, when dilution was clearly abandoned as a solution to pollution with the FWPCA's 1972 amendments. None of this is consistent with either national or state goals as expressed in federal and state law. Existing law requires that no risk of error be discharged from the end of the pipe.

I will now turn to the more specific comments I have about individual changes DEC proposes in the regulations.

INDIVIDUAL REGULATION CHANGES.

Arsenic. The issue paper's heavy reliance (see issue paper, at page 6) on "two comprehensive epidemiology studies in Fairbanks" (id. at page 5) to justify its proposed human health criteria for arsenic is entirely misplaced. Neither study were "comprehensive." For example, the study co-authored by current state epidemiologist Dr. John Middaugh focused on natural levels of arsenic in Fairbanks groundwater and determined the rate at which the body would accumulate natural arsenic. The study was not a long term evaluation of the human health impact of ingestion of arsenic, and did not look at the ingestion of arsenic introduced into drinking water from placer mining activities. I urge you to consult with Dr. Middaugh and correct the issue paper's characterization of the results of his study.

Moreover, DEC's dismissal of the Taiwanese study's relevance to Alaskan conditions is unwarranted. DEC assumes that because the Taiwanese exposed to arsenic were of a different race than most Alaskans and have a different diet that most Alaskans the study's showing of adverse health effects in Taiwanese is not translatable to Alaska. In establishing a WQS, DEC cannot assume those exposed to arsenic ingestion from Alaskan waters will always be of a different race, have different genetics or have a different diet than those humans adversely affected in Taiwan. Such assumptions would be an absurd, if not constitutionally suspect and socially reprehensible, basis for establishing any environmental rule.

Given that DEC conceded in its *Summary Response* that it has no ability to comprehensively evaluate the natural or, for that matter, the unnatural characteristics of Alaskan waters, it cannot claim as it did in

the arsenic issue paper that trivalent, inorganic arsenic is not being discharged into Alaskan waters.

Dioxin. Aside from its expressed intent to insulate the southeastern pulp mills from a more stringent National Toxics Rule, what apparently drives DEC's motivation in setting a relatively lenient dioxin WQS is its incorrect assumption that "Current theory indicates that, in fact, there is a safe threshold level for dioxin in the body below which cancer would not occur." DEC's *Regulatory Issue Paper: Human Health Criteria For Dioxin*, at pages 4-5. This assumption is false. It perhaps derives from an inaccurate press release issued by the Chlorine Institute after an Institute sponsored scientific meeting at which dioxin was the topic. *Scientific American* (April, 1991), at page 24. Most current scientific thinking --- and recent scientific findings --- continue to support the generally accepted view (outside of the view shared by trade associations and industry sponsored studies) that there is no threshold for dioxin. *Science*, October 18, 1991, at page 377. Accordingly DEC should revisit the literature on this subject and reconsider its proposal.

Chronic Toxicity of Whole Effluent. DEC should not permit chronic toxicity within a mixing zone. To do otherwise means the designated uses within a mixing zone are not protected. Indeed, DEC agrees. See *Regulatory Issue Paper: Mixing Zones*, at page 3 ("The mixing zone language should assure that the water body has a continuous zone of passage for migratory species that meets water quality criteria.") Therefore, it is inappropriate for this regulatory change to require chronic toxicity to be measured at the *boundary* of the mixing zone. That DEC proposes otherwise strongly suggests that mixing zones are really pollution giveaway zones, a device to circumvent timely implementation of end of the pipe discharge goals.

Also, the language of the definition for a "chronic toxicity unit" seems limited to single generation impacts. What if the second or third generation of an exposed species displays genetic defects? This possibility does not seem to be encompassed within the language of the definition.

Mixing zones. Extensive use of mixing zones --- and any use of mixing zones where end of the pipe technology is readily available to prevent pollution discharges --- is inconsistent with existing law. Moreover, contrary to DEC's statements in the mixing zone issue paper, the proposed mixing zone regulation introduces more vagueness into the

existing regulation; it does not clarify it (although the existing regulation *does* need clarification).

If one looks at other states' regulations on this subject, virtually all set clear, easily enforceable numerical limits on the size of mixing zones. DEC's proposal, nonetheless, leaves the issue up in the air, giving the public and regulated industry no clear guidelines on what to expect. Also, allowing the entire width of a stream to be dedicated to a mixing zone would DEC leave no margin for safety, particularly at low water flow times of the year.

The current proposal also does not clarify that the burden of proof is on the applicant to justify a mixing zone. In fact, mention of "burden of proof" in amending language may suggest that DEC has the burden of proof on any thing not clearly laid at the feet of the permittee/applicant. The regulatory language should be clarified to say that the applicant has the burden of proof on all relevant elements, including those in subsection (a) of 18 AAC 70.032.

The introduction of the phraseology "adverse effects" and "significant" in subsection (a)(1), as DEC proposes, opens broad loopholes in the existing language. Moreover, the proposed limiting language for adverse effects on "human health at the location" ignores impacts on aquatic life, and leaves open the question of responsibility if human health effects are caused outside the boundary of the mixing zone "location" but clearly caused by pollutants in the waters of the mixing zone.

Further, the suggested phraseology "using methods found by the department to be most effective *and feasible*" in proposed subsection (a)(3) is inconsistent with the anti-degradation language and requirements of 18 AAC 70.010(c)(3)("all wastes and other substances to be discharged using the methods found by the department to be most *effective*").

Proposed language for subsection (e)(3)(iii) adds even more ambiguity, suggesting that "mixing zones may not result in permanent displacement of indigenous organisms or long term reduction in fish population levels" without defining "permanent" or "long term."

Adoption of a new category of protection for "resident game fish" is too limiting and inconsistent with the goal to protect all aquatic life. I do not think DEC intends to allow the destruction of fish species which are neither anadromous nor "resident game fish" but which are part of the

food chain on which anadromous fish and resident game fish depend for survival.

Establishment of a mixing zone, or its expansion, should be an event that is subject to public review and comment. The existing regulation is not clear in that regard. DEC should adopt an amendment which makes it clear that mixing zones will not be established or expanded without public notice and a comment period.

Natural characteristics. I do not understand what DEC's motivation is here. In DEC's *Summary Response, supra*, § 5, at page 2, DEC indicated that the existing regulations already provided an adequate basis for "accomodat(ing)" situations where the "natural pollutant levels exceed the criteria of the WQS". Moreover, DEC has already conceded its administrative inability to "comprehensively establish background levels of toxic compounds in waters of the state, except where special studies are done at specific sites." Id., § 11, at 5. Yet it proposes language that would allow it to do precisely that, without public notice or comment.

DEC's proposal suggests that it can ignore effluent standards and limitations on water bodies with high natural levels of pollutants, although this would be inconsistent with the FWPCA. It also would be inconsistent with AS 46.03.710 (may not "add to" the pollution of a water body).

DEC suggests no method for defining "natural." What are the parameters? How would DEC go about "administratively approv(ing)" natural levels? Permit by permit? Pollutant by pollutant? At what level of DEC'S administration would this occur, and with what public involvement?

96 Hour LC 50. The issue paper on this topic concedes it is a "cursory treatment of a complex subject." What is most troubling is that the paper does not more fully discuss the need to bridge between regulatory standards and in-the-field enforcement.

Since DEC indicates that "There is no current intent for the Department to get seriously into the business of toxicity testing" and the DEC Laboratory is not equipped to do so, what types of tests are left to DEC field officers to do that would test for chronic pollution problems from a permittee's facility? To quote from page 2 of the *#8 Water Quality Standard Questionnaire/March 1992 (Topic: Sediment Standard)* "[I]t is

necessary to have methods of measurement that are cost-effective and can be performed by the operator...." This should also be true for DEC, or else the monitoring will be totally within the control of the operator.

Yet DEC proposes to drop the 96 Hour LC 50 default test for chronic toxicity and substitute a very expensive, time consuming bioassay testing regime that it does not have the capacity to perform. The testing burden will fall on the regulated permittee. In effect DEC will allow the regulated permittee to test itself for pollution problems. This abdicates DEC's roles of surveillance and enforcement.

Moreover, the financial burden on smaller operators of chronic toxicity testing will be substantial, perhaps crushing at times, and likely will have the effect of DEC foregoing the requirement of such testing as a permit obligation.

While in an ideal world, with an adequate government laboratory and unlimited government funds for research and testing, DEC's proposal would make scientific sense, it is not a world that DEC lives in. DEC must, therefore, set default limits that are easy and inexpensive to enforce.

Total hydrocarbons. The proposal to drop this standard is troubling insofar as it leaves only the "no sheen" rule in place to prevent non-TAH hydrocarbon pollution. While it may make good science to better categorize hydrocarbon families for standards-setting, the no sheen rule's usefulness in all contexts is dubious. In the turbid waters of Cook Inlet or in the Alyeska mixing zone in Valdez (where there may be turbidity due to the Lowe River), one might see no sheen although a water quality test would show hydrocarbon presence of 100 mg/l, approximately 700 times greater than the current TII standard. It does not seem to make sense to ignore the heavy hydrocarbons and worry about the more volatile TAHs which may present a less acute (albeit, more chronic) threat.

Sediment. Lots of different types of and fines in sediment are not "settleable" and will not settle out in the hour provided for in the Imhoff Cone test method. One can have a very muddy stream and still meet the settleable solids standard. Accordingly, DEC's proposal threatens a huge change which can only degrade state waters. This change, when coupled with elimination of the "color" criteria, will turn aesthetically pleasing, clear water streams into cloudy flows...Ironically, it was DEC itself which indicated in its *Summary Response, supra*, § 14, at page 7, that the

existing sediment and turbidity criteria, for both fresh and marine waters, "*are necessary* to protect designated uses. ADEC believes there is sufficient flexibility in the WQS to accommodate regional or site-specific variations through short-term variances, site-specific standards, mixing zones, and reclassification."

Definition of water. I do not understand what the second "or" clause of the language proposed to be added to the definition adds to the definition. What does it include that the first part of the sentence doesn't? Only last year DEC stated that the existing definition is "essentially the same as the statutory definition." DEC's *Summary Response*, *supra*, §, 49, at 14. DEC now proposes a change to the definition which varies with the statutory definition, without explaining why.

What is troubling about the proposed definition is that it would exempt from the WQSs clean water flowing into a polluted treatment pond, simply because the operator has constructed the treatment system to capture the clean water. Waters in treatment facilities should be exempt, but not if the treatment facilities are interjected into the pathway of the natural flow of a clean water body. Operators should be obligated to divert clean waters around their treatment facilities. To allow otherwise in effect allows the creation of "mixing zones" upstream of the end of the pipe.

CONCLUSION

I hope my comments are helpful and are not seen as overly negative. (It is an unfortunate by-product of the regulation review/comment process that negatives are highlighted.) I sincerely appreciate the time you spent with me on the phone recently in discussing DEC's proposals.

Enclosed are completed questionnaires with respect to certain of the individual proposals.

Sincerely,

Michael J. Frank

encl.

CORDOVA DISTRICT FISHERMEN UNITED

P.O. Box 939

Cordova, Alaska 99574

Phone (907) 424-3447 Fax (907) 424-3430

September 30, 1992

Mr. Dave Sturdevant
Water Quality Management
Department of Environmental Conservation
410 Willoughby Avenue Suite 105
Juneau, Alaska 99801-1795

Dear Mr. Sturdevant:

This past February, the United Fishermen of Alaska held its annual board meeting in Juneau. During one of the sessions, Governor Hickel met with the UFA board and addressed the water quality issue. Enclosed is a copy of an Anchorage Times article, "Hickel Backs Fishermen on Water-quality Representation," dated February 13, 1992. The article summarizes Governor Hickel's comments regarding revisions to state water quality standards:

> "Hickel told the United Fishermen of Alaska board that he supports the group's request to have a fishing industry representative work closely with the state when it changes water-quality standards and regulations."

We interpreted this comment as a commitment on the part of the governor to work with the fishing industry on revising and updating the state's water quality regulations. Unfortunately, this has not been the case and commercial fishing interests have been disregarded throughout this process.

Cordova District Fishermen United is concerned that the state is moving too rapidly in its efforts to revise Alaska's water quality standards and is making critical decisions based on erroneous and incomplete information. The most glaring example of this is basing the human health criteria for dioxin on a fish consumption rate of five pounds per year. This rate of consumption is clearly inappropriate for setting dioxin standards for Alaska. The ADEC issue paper on dioxin cites data collected by the Subsistence Division of the Alaska Department of Fish and Game which indicates that fish consumption in Alaska generally ranges from 30 to 300 pounds per year. If ADEC had chosen to base the rate of fish consumption at the lower end of this range, it would only be twice the national average of fifteen pounds, as calculated by the National Marine Fisheries Service.

CDFU recognizes that the development and utilization of Alaska's natural resources is the key to our state's economic well-being. Alaska needs to make a concerted effort to balance our need to develop our natural resources while maintaining a healthy, productive environment. As far as water quality is concerned, that balance can best be determined by establishing a conflict resolution task force.

Sincerely,
CORDOVA DISTRICT FISHERMEN UNITED

Mary L. McBurney
Executive Director

cc: Governor Walter J. Hickel
Attorney General Charles Cole
John Sandor, Commissioner ADEC
Carl Rosier, Commissioner ADF&G
Glenn Olds, Commissioner DNR
Senator Curt Menard
Representative Gene Kubina
Representative Cliff Davidson

enclosure

UNITED FISHERMEN OF ALASKA

211 Fourth Street, Suite 112
Juneau, Alaska 99801
907/586-2820
Fax: 907/463-2545

September 30, 1992

Dave Sturdevant
Water Quality Management
Dept. Environmental Conservation
410 Willoughby Ave., Suite 105
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United Fishermen of Alaska is very concerned that the revisions proposed by the Department of Environmental Conservation to Alaska's water quality standards will lead to degradation of fish habitat, reductions in fish populations, and erosion of consumer confidence in Alaska's seafood quality. UFA believes that these proposed revisions will have tremendous negative economic impacts, both short- and long-term, which will be felt statewide throughout the entire seafood industry. UFA believes that these potential economic impacts to the seafood industry have not been addressed - AT ALL - by the DEC in its haste to implement its own standards before substantially higher standards are set by EPA through the National Toxics Rule. UFA finds that DEC's approach lacks both balance and foresight.

Seafood is Alaska's No. 1 export. Alaska's seafood production ranks first among states and fifth among nations. The seafood industry is Alaska's largest private sector employer, providing jobs for one in twenty Alaskans (i.e., 70,000 seasonal or 33,000 year-round jobs). The seafood industry is Alaska's second-largest revenue generator ($31.1 million in taxes in 1991).

Besides exporting this abundant resource, Alaskans eat it - lots of it. Alaskans may consume more fish and shellfish than residents from any other state: studies from the Subsistence Division estimate that average consumption of fish and shellfish among some subpopulations of residents exceeds 200 pounds per person per year.

CORRECTION

THIS DOCUMENT
HAS BEEN REPHOTOGRAPHED
TO ASSURE LEGIBILITY

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We interpreted this comment as a commitment on the part of the governor to work with the fishing industry on revising and updating the state's water quality regulations. Unfortunately, this has not been the case and commercial fishing interests have been disregarded throughout this process.

Cordova District Fishermen United is concerned that the state is moving too rapidly in its efforts to revise Alaska's water quality standards and is making critical decisions based on erroneous and incomplete information. The most glaring example of this is basing the human health criteria for dioxin on a fish consumption rate of five pounds per year. This rate of consumption is clearly inappropriate for setting dioxin standards for Alaska. The ADEC issue paper on dioxin cites data collected by the Subsistence Division of the Alaska Department of Fish and Game which indicates that fish consumption in Alaska generally ranges from 30 to 300 pounds per year. If ADEC had chosen to base the rate of fish consumption at the lower end of this range, it would only be twice the national average of fifteen pounds, as calculated by the National Marine Fisheries Service.

We realize that there is a difference of interpretation regarding ADEC's value for the consumption rate of fish. During the KCHU radio call-in show, "Coffee Break" (9/21/92) you were asked about the five pound fish consumption value. You responded that the value did not refer to a total consumption rate of five pounds of fish per person per year, but rather referred to an annual consumption rate of five pounds of contaminated fish. Indeed, if this is ADEC's position, then the public has been commenting on incorrect information. The dioxin issue paper does not mention that the five pound value represents contaminated fish ingested over the course of a year.

CDFU maintains that the proposed changes to the state's water quality standards are too much, too fast. While the Hickel Administration and ADEC have acknowledged that the new regulations will benefit the Southeast Alaska pulp and mining industries, these changes amount to throwing a regulatory blanket over the entire state in order to cover a few localized permitting problems. Since these new regulations will change the rules governing water quality throughout the state, it's unreasonable that the fishing industry and other interested parties weren't consulted or invited to help in their formulation.

CDFU is not satisfied that the data cited by ADEC illustrate or justify a clear need to change the existing water quality standards. Rather, CDFU urges ADEC to take a more conservative approach to the water quality issue and keep the administration's promise to work closely with the fishing industry in reviewing and revising the water quality standards. To meet this end, we support the establishment of a dispute resolution water quality task force to identify problem areas in the existing regulations and negotiate acceptable solutions. The task force would be composed of people representing all affected parties including various public and private interests and impacted industries. A dispute resolution process will help to avoid the time, energy and expense of legal action through the courts and encourage balance in formulating public resource policy.

CDFU does not support the water quality revisions proposed by ADEC. However, we do endorse the recommendations and technical comments submitted by United Fishermen of Alaska.

The seafood industry is Alaska's largest private sector employer and second-largest income producer. The Consumer Reports article, "Is Our Fish Safe to Eat?" was a wake-up call to the seafood industry that consumers are becoming more concerned about the wholesomeness of what they eat and where it comes from. Unlike many other seafood producers, Alaska has an enviable marketing edge for promoting its fisheries products: pure, uncontaminated water. We can't afford to lose this marketing advantage - clean water helps to sell fish.

CDFU recognizes that the development and utilization of Alaska's natural resources is the key to our state's economic well-being. Alaska needs to make a concerted effort to balance our need to develop our natural resources while maintaining a healthy, productive environment. As far as water quality is concerned, that balance can best be determined by establishing a conflict resolution task force.

Sincerely,
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Mary L. McBurney
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enclosure

2/13/90 Times

Hickel backs fishermen on water-quality representation

By ROGER F. NYHUS
ASSOCIATED PRESS

JUNEAU — Gov. Walter J. Hickel reassured a group of fisherman Wednesday that he sympathizes with many of their concerns, including maintaining strict water-quality regulations.

Hickel told the United Fishermen of Alaska board that he supports the group's request to have a fishing industry representative work closely with the state when it changes water-quality standards and regulations.

"You not only have a right, I think it's a protection of one of the greatest industries Alaska has," Hickel said.

Board member Riki Ott told the governor that the recent Consumer Reports article about salmon contaminated with cancer-causing PCBs highlights the need for clean water.

"High water-quality standards relate directly to high-quality fisheries resources," Ott said. "If industry wants to do a project, you follow the laws that are out there, and you don't get exemptions. You do it right."

Hickel responded: "I don't have any problem with that."

Ott said fishermen are concerned about changes in the state's water-quality regulations, including clean-water standards that relate to forest-practices legislation passed by the Legislature in 1990.

"The streams serve as our economic nursery," said board member Kate Troll, who represents the Southeast Seiners Association. "We feel that is somewhat threatened. We'd like to see our water-quality standards maintained. We don't want regulations that are full of loopholes."

The state Department of Environmental Conservation is reviewing changes to Alaska's water-quality standards as well as the forest-practices legislation, which requires [illegible] near streams.

"We have no intention to weaken our water-quality standards, although there are certain things we need to modify, and the standards may turn out higher or lower," Dave Sturdevant, DEC's water quality standards coordinator, said in an interview.

The state must update its water-quality standards every three years to comply with the federal Clean Water Act, he said. The state began reviewing its standards in 1990. It expects to hold a second round of public hearings on the proposed changes in early fall, Sturdevant said.

The state Department of Natural Resources, DEC and the U.S. Environmental Protection Agency are working on the proposed forest-practices regulations, he said. Fish and Game Commissioner Carl Rosier said the proposal should be completed within 30 days.

"To the extent that any of the departments in the state are even considering things like abandoning, tough water-quality standards — that's a total disaster," said Deming Cowles, United Fishermen's lobbyist in Washington, D.C.

"The state has some of the strongest water-quality standards in the nation. That's one thing that helps us sell our fish."

Hickel said he supports increased funding for domestic seafood marketing, primarily for salmon, and more regulation of [illegible] factory trawlers.

Gov. Walter J. Hickel

UNITED FISHERMEN OF ALASKA

211 Fourth Street, Suite 112
Juneau, Alaska 99801
907/586-2820
Fax: 907/463-2545

September 30, 1992

Dave Sturdevant
Water Quality Management
Dept. Environmental Conservation
410 Willoughby Ave., Suite 105
Juneau, AK 99801-1795

United Fishermen of Alaska is very concerned that the revisions proposed by the Department of Environmental Conservation to Alaska's water quality standards will lead to degradation of fish habitat, reductions in fish populations, and erosion of consumer confidence in Alaska's seafood quality. UFA believes that these proposed revisions will have tremendous negative economic impacts, both short- and long-term, which will be felt statewide throughout the entire seafood industry. UFA believes that these potential economic impacts to the seafood industry have not been addressed - AT ALL - by the DEC in its haste to implement its own standards before substantially higher standards are set by EPA through the National Toxics Rule. UFA finds that DEC's approach lacks both balance and foresight.

Seafood is Alaska's No. 1 export. Alaska's seafood production ranks first among states and fifth among nations. The seafood industry is Alaska's largest private sector employer, providing jobs for one in twenty Alaskans (i.e., 70,000 seasonal or 33,000 year-round jobs). The seafood industry is Alaska's second-largest revenue generator ($31.1 million in taxes in 1991).

Besides exporting this abundant resource, Alaskans eat it - lots of it. Alaskans may consume more fish and shellfish than residents from any other state: studies from the Subsistence Division estimate that average consumption of fish and shellfish among some subpopulations of residents exceeds 200 pounds per person per year.

Consumer confidence in Alaska's seafood is driven by the perception that Alaska's fish are pure because Alaska's water is pure. The state invests in and markets this image through the Alaska Seafood Marketing Institute (ASMI). For example, in February this year, the state went to bat for its seafood industry when Consumer Reports published an article titled "Is Our Fish Fit to Eat?" The report advised against eating salmon, among other fish, because of contamination with PCBs and heavy metals. Clean water, protected by strong water quality standards, played a key role in returning consumer confidence in Alaska seafood.

The state spent $13.5 million alone last year to market "pure" Alaska salmon from Alaska's pristine waters. Like many industries dealing with informed and concerned consumers, ASMI recognizes the economic advantage of perception and consumer confidence. As such, ASMI includes this statement in its press packets:

> "According to the National Oceanographic and Atmospheric Administration, Alaska has the world's most pristine waters. Analysis of strategic sample sites conducted by NOAA, such as the 1984-1985 "National Benthic Surveillance Project: West Coast" annual report shows Alaska's fishing grounds to be located in waters free from heavy pollutants."

DEC's proposed revisions would clearly undermine this economic advantage.

Gov. Hickel recognized the critical link between quality fish and clean water when he told the board of UFA this February that maintaining strong water quality standards was "a protection of one of the greatest industries Alaska has" (Anc. Times 2/13/92). Lack of adequate protection of water and habitat has led to deterioration of fisheries resources in virtually every other state in the nation. And many states are investing hundreds of millions of dollars to restore their water quality. Gov. Hickel recognized that Alaska must avoid repeating the mistakes which cost other states their fisheries.

Gov. Hickel supported UFA's request to have a fishing industry representative work closely with the state while it revised the water quality standards. DEC failed to carry through with the governor's promise, and made it difficult for commercial fishing industry to participate. DEC planned to hold its public comment period during the height of the summer fishing season when most fishermen were out of town.

Further, DEC denied access to the statewide public teleconference held on September 25, 1992, to at least one commercial fishing group (Petersburg Vessel Owners Association) on the grounds of a minimum notice "requirement" of 60 days. According to Legislative Affairs, sites can be scheduled momentarily, however,

the ruling on additions is up to the sponsor, in this case DEC. No one contacted by UFA at Legislative Affairs or the Ombudsman's Office had ever heard of a 60-day minimum notice requirement.

UFA is extremely concerned with DEC's apparent attitude that public hearings are a nuisance and public input is to be endured rather than used to influence public policy. This attitude gives the distinct impression that the department has an agenda with pre-determined goals. For example, Dave Sturdevant stated that DEC "has no intention to weaken our water quality standards, although there are certain things we need to modify, and the standards may turn out higher or lower" (Anc. Times, 2/13/92). However, DEC's revisions make the water quality standards less stringent in nearly every instance.

DEC is proposing a fundamental shift in public policy with major technical changes in the state's management of its water resources. Basically, the DEC is advocating for state control of discharge of its pollutants into its waterbodies. While UFA supports this fundamental shift in policy, it cannot support the state's current approach which favors short-term over long-term economic benefits, risks statewide public health, and sacrifices one resource-based industry - commercial fishing - for the benefit of the timber, mining, and oil industries.

The state's efforts to decrease protections for Alaska's waters diametrically oppose national and international efforts to increase protection of waterbodies. One of the principles adopted at the Earth Summit in Rio, and supported by the United States, seeks to control land-based sources of pollution that ultimately degrade the marine environment. In his speech at the Earth Summit, Governor Hickel called on the "nations of the world ... to protect our fisheries from the waste that comes from greed." The DEC must realize that the place to start is in its own state. Alaska's industries must internalize environmental costs. We firmly believe that this can be done without imposing economic hardships. Several of our technical comments address this achievable balance.

Instead of adopting its revisions on its own time line, UFA requests the department to establish a Clean Water Task Force with representatives from all interest (timber, mining, oil, commercial and charter fishing, tourism, Natives, and environmentalists) to determine acceptable risk levels for pollutants. The task force should adopt a dispute resolution format t set pollutant criteria, and it should be chaired by an objective moderator. Information on this format can be obtained through the Harvard Center for Dispute Resolution (617) 495-1684.

Although UFA strongly believes that this is the only fair approach for changes in public policy of this magnitude, UFA has taken the time to prepare specific comments on the water quality standards. These technical comments were written by Dr. Riki

Ott, the Chair of UFA's Habitat Committee, and approved by the board. As I am sure you are aware, Riki gillnets in Prince William Sound, and has a Masters in oil pollution/marine biology from the University of South Carolina, and a doctorate in heavy metal pollution of marine sediments/fisheries from the University of Washington.

The technical comments are enclosed.

Sincerely,

Jerry McCune
UFA President

JM::phl

cc: Governor Walter J. Hickel
Charles E. Cole, Attorney General
Carl L. Rosier, Commissioner, ADF&G
John A. Sandor, Commissioner, DEC
Glenn A. Olds, Commiesioner, DNR
Kim Elton, Executive Director, ASMI
Duncan Fowler, Ombudsman
Senator Curt Menard
Representative Cliff Davidson
Prince William Sound Aquaculture Corp.

9/30/92

SPECIFIC COMMENTS OF

UNITED FISHERMEN OF ALASKA

ON THE ALASKA WATER QUALITY STANDARDS

GENERAL 18 AAC 70.010.

UFA recommends first and most importantly that DEC adopt the following narrative statement relating to toxic discharges in the general section of the water quality standards 18 AAC 70.010:

> "There shall be no discharge of toxic materials in toxic amounts. For point source discharges, this shall be interpreted as no discharges in excess of the numeric criteria for acute toxicity testing of whole effluent as measured at the end of the discharge pipe, and no discharges in excess of the numeric criteria for chronic toxicity testing of whole effluent outside the boundaries of the mixing zone."

Justification: DEC is proposing a significant shift in public policy: proposed revisions to the mixing zone regulations will allow the state to exercise full regulatory control for discharge of all types of pollutants into all state waters through mixing zones.

In the past, mixing zone discharges of "nonconventional"[1/] and "toxic" or priority pollutants were regulated jointly by DEC and EPA through federal (NPDES) permits. The state primarily regulated mixing zone discharges of "conventional" pollutants. The state has technically had the authority to regulate discharge of nonconventional and toxic pollutants since 1985. However, in practice, the state chose not to regulate these pollutants independently of EPA because the state feared lawsuits: the standards regulating discharge of pollutants into mixing zones could be broadly interpreted.

1/"Conventional" pollutants include TSS (total suspended solids), pH, BOD (biological oxygen demand), fecal coliform bacteria, and oil and grease. "Toxic" pollutants are defined by 40 CFR 401.16 and include 126 compounds considered of "priority" concern to human health. Many toxic pollutants are known or suspected carcinogens, mutagens, or teratogens. "Nonconventional" pollutants are compounds considered a health risk, but not included in the other two lists, i.e., chlorine, ammonia.

Now the state is proposing significant revisions to the mixing zone language - revisions which will provide clear regulatory (and legal) guidelines, thereby allowing the state to exercise its authority to permit discharge of pollutants into state waters with minimal federal guidance. However, with this new state authority, comes the responsibility - and the challenge - of maintaining Alaska's high water quality as mandated by the Clean Water Act. UFA believes that it is important to clearly indicate the state's intent to maintain its high water quality standards in narrative language.

Secondly, UFA strongly recommends that the state develop a "Clean Water Task Force" with representatives from all interests, similar to the Forest Practice Act Task Force, to approach the challenge of maintaining high water quality while allowing discharge of nonconventional and toxic pollutants . Past experience shows that performance of the Clean Water Task Force would be improved with an objective moderator and a dispute resolution format.

Justification: UFA could support this fundamental shift in regulatory control of pollutant discharge *only if* the state takes a responsible approach by balancing concerns from all user groups, including industry and the public. However, UFA strongly disagrees with the state's current approach, i.e., that of basing its criteria on acceptable levels of risk versus acceptable levels of protection. Instead UFA believes that:

> "(e)stablishment of acceptable risk levels for chemicals is a task for the entire society and not only for the scientific community." In risk assessment, "models (are) developed for estimating effects of exposure ... with the goal that the risk will not be underestimated" (Klaassen 1986).

UFA believes that the "task force" concept - with an objective moderator and a dispute resolution format - may be the only fair approach to policy revisions which represent such a fundamental shift, and which have the potential to dramatically affect the integrity of entire ecosystems, fish and wildlife populations, and the health, lifestyles, and livelihoods of all Alaskans.

UFA recommends the following language for:

> "18 AAC 70.010(c): If the natural characteristics of a water exceed the numeric water quality criteria for the use classes 18 AAC 70.020 and 18 AAC 70.022, [THE DEPARTMENT WILL, IN ITS DISCRETION, ADMINISTRATIVELY APPROVE THE NATURAL LEVELS AS THE APPLICABLE CRITERIA FOR CORRESPONDING USE CLASSES] no additional discharges which would further degrade the water beyond the numeric water quality criteria will be allowed."

Justification: The approach proposed by the DEC could lead to permanent deterioration of water quality. Natural background water quality often exceeds the numeric criteria on a temporary or seasonal basis. For example, high total suspended solids are associated with spring breakup and heavy rainfalls; low dissolved oxygen is associated with water under ice at the end of winter. If DEC were to establish numeric criteria on such intermittent events, water quality of entire waterbodies could rapidly deteriorate and protection for fish stocks would erode. This is not the goal of the Clean Water Act.

Further, if natural background levels do exceed the state's water quality criteria, then discharge of additional pollutants in excess of the numeric criteria should be prohibited to avoid further degrading water quality.

UFA recommends that the following language be added to:

> "18 AAC 70.010(c)(1): reducing water quality is justified because of necessary economic or social development when considering all economic and social impacts that such a reduction in water quality may have;"

Justification: UFA finds that the DEC has not considered potential economic impacts on the seafood industry which could result from deterioration of water quality due to the extremely lenient criteria for several pollutants (dioxin, arsenic, chloroform). While this is discussed extensively elsewhere, the point here is that UFA does not want the state to ignore economic impacts on the seafood industry in the future, and feels that language stressing consideration of all economic and social impacts should be included in the general narrative.

MIXING ZONES 18 AAC 70.032
(Unless otherwise stated, citations are from regulatory issue paper on mixing zones.)

UFA recommends that DEC's proposed changes be revised as follows: (Note: DEC's proposed changes so fundamentally alter this provision, justification is given for both the recommended language and the deleted DEC language, just as if the latter were already in statute.)

> (a)(1) "pollutants discharged could result in acute toxicity, or could bioaccumulate, [IN FOOD CHAINS, OR] concentrate or persist in the environment [TO A LEVEL THAT CAUSES ADVERSE EFFECTS]; cause carcinogenic, mutagenic, or teratogenic effects on the biota or on human health [AT THE LOCATION]; or otherwise present a [SIGNIFICANT] risk to human health;"

Justification: It is UFA's intent to give the state the legal definitions it needs to exercise its full authority to regulate discharge of pollutants only if the state seeks a balance that will adequately protect the health of fish, wildlife, and humans. UFA believes that addition of the language "in acute toxicity" is a critical step towards achieving this balance: this addition is prohibits discharge of toxic pollutants in toxic quantities.

DEC's proposed language "in food chains" should be deleted for several reasons. It ignores potential instances in which pollutants are bioaccumulated by individual organisms, but not bioaccumulated "in the food chain." For example, mussels bioaccumulate many different pollutants to levels many orders of magnitude higher than levels in surrounding sea water, but organisms consuming mussels may or may not bioaccumulate the same pollutants. This language ignores effects on those individual species which tend to best bioaccumulate pollutants. Further, it is far more difficult to prove that compounds bioaccumulate "in the food chain" rather than in individual organisms.

DEC's proposed language "to a level that causes adverse effects" should be deleted because this is precisely the end result the Clean Water Act is trying to prevent, i.e., adverse effects on indigenous organisms through deterioration of waterways. By the time it is possible to prove that there have been "adverse effects" in the environment, it is too late. (It should be noted that the cost of restoring water quality is often multiple millions of dollars.)

DEC's proposed language "on human health" is too narrow in scope as it ignores carcinogenic, mutagenic and teratogenic effects on the biota - fish and wildlife. DEC's proposed language should either be modified or deleted. Left unchanged, it sets a standard which is virtually impossible to prove with current science: there are few known compounds for which there is an unequivocal cause-effect relationship established in which the

chemical in question is KNOWN to cause carcinogenic, mutagenic, or teratogenic effects on humans as opposed to other laboratory animals. Establishing an unrealistic or virtually unprovable standard will allow a proliferation of mixing zones - which will essentially defeat the purpose of the Clean Water Act.

DEC's proposed language "at the location" should be deleted because it is too restrictive. It ignores potential downstream effects of effluent on fish and shellfish caught "at the location" of a mixing zone, or downstream of a mixing zone, but consumed elsewhere. Downstream effects will be of increased importance as mixing zones are permitted in rivers and streams.

DEC's proposed language "significant" risk to humans should be deleted because it is too restrictive, it may be too difficult or unrealistic to prove, (depending on the definition of "significant" - the word is too subjective), and, again, this is one of the end results the Clean Water Act is trying to prevent.

UFA recommends the following change to DEC's proposed language for:

> (a)(3) "A mixing zone will be granted only after the applicant has shown to the department's satisfaction that the wastes or substances that may exceed the water quality criteria will be treated using [METHODS FOUND BY THE DEPARTMENT TO BE MOST EFFECTIVE AND FEASIBLE] all technological and managerial methods available for pollution reduction and removal, and[, AT THE DISCRETION OF THE DEPARTMENT,] will be discharged in a manner that maximizes initial dispersion and dilution."

Justification: The language proposed by DEC allows for too much discretion by the department. It needs to be more definitively worded to ensure that the goals of the Clean Water Act are met. The proposed language is adopted from Washington state (WAS 173-203-100) with modification.

UFA maintains that wastewater which exceeds the state's water quality criteria MUST be treated using best available technology. The cost does not matter: if industry cannot afford to treat it, industry should not discharge it. Alaska's industries should internalize the cost of maintaining high water quality. The seafood industry cannot afford potential impacts of pollution from ineffective treatment methods. UFA notes that other states that considered economic feasibility of treatment methods, rather than sticking with a set standard, notably Washington state, are now struggling to clean up their contaminated waterways. To prevent Alaska's waterbodies from reaching this level of pollution, UFA deleted all references to "economic achievability" from the Washington state language that UFA adopted, and from DEC's proposed revisions.

If the wastewater still exceeds the state standards after best available treatment, then a mixing zone will be granted, but wastewater MUST still be discharged into an area that maximizes initial dispersion and dilution. This is to ensure that the state's water quality criteria will be met at the boundaries of the mixing zone, and also that the effluent will be rapidly diluted. It has been found that some pollutants like metals and possibly other substances have an acute:chronic ratio of 2 for embryonic and larval life stages (EPA Gold Book).

UFA recommends that a new subsection be added and the other subsections be renumbered accordingly as follows:

> "(i) mixing zones are not authorized in anadromous and resident game fish waters, and other water with resident fish species of local cultural or social significance without the express concurrence of the Alaska Department of Fish and Game; and"

Justification: UFA maintains that if the state is going to exercise its authority to regulate discharge of pollutants, then it must be done in a responsible manner that balances environmental, economic and social concerns. ADF&G has the responsibility for protecting aquatic life and its habitat: the department must be able to exercise its authority. This includes authority to protect important commercial, sport, or subsistence fish stocks.

UFA recommends the following change to DEC's proposed language for:

(e)(3) mixing zones authorized in streams, rivers or other flowing freshwaters must maintain and fully protect existing uses in receiving and upstream waters.

Justification: If mixing zones are to be allowed in streams, some of which may include anadromous and/or game fish streams, then it is important to maintain and fully protect existing uses upstream as well as downstream. Upstream uses may include fish propagation and rearing. Reproductive and genetic impairment may result in fish swimming through a mixing zone that is downstream of a fish spawning area, thereby impairing upstream use.

UFA recommends the following language instead of DEC's proposed language for:

(e)(3)(i) "mixing zones are not authorized in anadromous and resident game fish waters where the effluent flow exceeds 10 percent of the cross-sectional width of the water body or three hundred (300) feet plus the horizontal length of the diffuser as measured perpendicularly to the stream flow, whichever is less; or is greater than 10 percent of the stream flow during periods of minimum flow as determined by accepted hydrologic gauging methodologies."

Justification: DEC's proposed language does not assure the goals of the Clean Water Act will be met. Under DEC's proposed language, it is possible that mixing zones could extend across the entire width of smaller (or larger) streams, thus forming a barrier to anadromous fish. This is not permitted under the Clean Water Act.

UFA's proposed language "exceeds 10 percent of the cross-sectional width of the water body or etc." adds a width restriction for mixing zones which would ensure a migratory corridor in all streams regardless of width. Ten percent was chosen to be consistent with existing (and proposed) regulatory language pertaining to mixing zones on lakes (e)(1), in addition to the rationale presented below. (It should be obvious that, under the UFA language, mixing zones would not be permitted in small anadromous streams in which this width restriction cannot be met. UFA believes this is necessary to protect existing uses in small streams which contain anadromous fish.)

UFA's proposed language is adopted from Washington (WAC 173-203-100) and Idaho (01.02060.01) with the following modifications. Washington's language was changed from 25 to 10 percent of the cross-sectional width, and from 25 to 10 percent of the flow. Further, "flow" was changed to "minimum flow." Idaho's language was changed from 300 meters to 300 feet. These modifications all reduce the maximum allowable size of mixing zones. UFA's justification for making these modifications is that, in both of these states, the original language did not prevent deterioration of waterbodies, nor did it protect existing uses as required

under the Clean Water Act. Experience has shown that damaged waterbodies are extremely expensive to repair. UFA maintains that Alaska does not have to repeat the mistakes of most lower 48 states. Further justification for the modification to Idaho's regulation is given under (e)(3)(ii).

UFA's proposed language "during periods of minimum flow" establish a base point from which to gauge stream flow similar to mean lower low water in navigational charts. Stream flow in most Alaskan streams is highly variable. Setting "periods of minimum flow" as the base to permit mixing zones not to exceed 10 percent of stream flow will ensure that mixing zones will never exceed 10 percent of stream flow during periods of low stream flow. "Minimum flow" should be statistically quantified and its definition relegated to the Clean Water Task Force where issues like the average of 3 or 7 days of low flow could be evaluated.

UFA's proposed language "accepted hydrologic gauging methodologies" recognizes that stream gauging is a site-specific measure, and that different methodologies may be more appropriate than others in different terrain.

UFA recommends the following language instead of the proposed wording for:

> (e)(3)(ii) "mixing zones shall not extend in a downstream direction for a distance from the discharge port(s) greater than three hundred feet plus the depth of water over the diffuser or point of discharge, or extend upstream for a distance of over one hundred feet."

Justification: The language proposed by DEC offers no definitive size restrictions for mixing zones in streams. It is the intent of the Clean Water Act that mixing zones must be as small as practicable. UFA's proposed language establishes a quantitative size for mixing zones and is adopted from Washington (WAC 173-203-100). To be relatively consistent with the length restrictions (maximum allowable length of 400 feet), UFA is proposing a width restriction of 300 feet - not meters - plus the horizontal length of the diffuser as measured perpendicularly to the stream flow in larger streams (see (e)(3)(i) above).

UFA recommends the following changes to DEC's proposed language for:

> (e)(3)(iii) mixing zones may not result in [PERMANENT] any displacement of indigenous organisms or [LONG TERM] any reduction in fish population levels; and

Justification: DEC's proposed language is extremely offensive to UFA. It shows a callous disregard for the short- and long-term interests of the commercial fishing industry. It also clearly

violates basic mandates of the Clean Water Act, i.e., to prevent degradation of water quality and to protect existing uses. Any reduction in fish population levels due to a mixing zone would constitute a failure by the state to protect existing uses under the Clean Water Act.

UFA recommends the following change to DEC's proposed language for:

> (e)(3)(iv) mixing zones are prohibited in anadromous and resident game fish spawning areas [EXCEPT WHERE WATER QUALITY CRITERIA FOR THE AQUATIC LIFE DESIGNATED USE ARE NOT EXCEEDED IN SUCH AREAS. AQUATIC LIFE CRITERIA MUST BE MET AT END-OF-PIPE IN SUCH AREAS]; and

Justification: UFA does not believe that mixing zones should be permitted in fish spawning areas, period. This state has exhibited an appalling inability to enforce its environmental laws. UFA has absolutely no guarantee that water quality criteria, as DEC proposes, would be enforced. This would be to the clear detriment of fisheries resources. Protecting water quality in fish spawning areas is essential to the health of the resource.

NO MIXING ZONES IN FISH SPAWNING AREAS!

UFA wishes to point out an inherent problem with allowing mixing zones in streams as encapsulated in the proposed change for:

> (e)(3)(v) "mixing zones may be limited or prohibited in other identified special resource or critical freshwater areas, including identified anadromous fish escapement index streams and etc."

Problem: One way to determine potential reductions in fish populations is from escapement data. If mixing zones are not allowed in fish index streams, (and UFA agrees with DEC that they should not be), then index streams become also "control streams." That is, index streams are supposed to be representative of other streams in the area: however, if mixing zones are not allowed in index streams, but are allowed in other streams, then the underlying assumption for fish forecasting using index streams is invalidated. Further, any reductions in fish populations from streams with mixing zones may not be readily evident based on forecast data from streams without mixing zones.

A possible solution is to have ADF&G start a separate program of indexing representative streams in which mixing zones are allowed - and having industries/persons requesting mixing zones in streams required to fund this program. UFA believes further consideration on this matter is warranted and would like the opportunity to discuss this jointly with ADF&G and ADEC.

This problem - and its solution - are just one example of the issues that could be best dealt with through a "Clean Water Task Force."

REGULATING DIOXIN
(Unless otherwise stated, citations are from regulatory issue paper (IP) or fact sheet (FS) on dioxin.)

UFA recommends for marine water adopting a dioxin standard of **0.00006 ppq** for human health based on the risk level of one in a million (10^{-6}), a fish consumption rate of 65 g/day, a bioconcentration factor of 210,000, and a cancer potency factor of 86,750.

Justification: UFA has determined, based on available information, that neither DEC's proposed dioxin criteria of 1.2 ppq nor EPA's recommended criteria of 0.014 ppq are sufficient to protect the unique circumstances in Alaska. The individual factors in EPA's formula for human health criteria are discussed separately below.

HEALTH RISK LEVELS: UFA finds that the state's rationale for selecting a health risk of 10^{-5}, as opposed to 10^{-6}, is flawed and should not be accepted. DEC states:

> "Alaska has relatively little industry to generate toxic pollutants. In the case of dioxin, the only two known sources affecting water in the state are the two pulp mills in Ketchikan and Sitka. It is these sites where the dioxin criteria would be applied to waters receiving wastewater discharges from the two mills." (IP, pg. 6.)

It should be acknowledged that DEC is proposing a statewide health risk of 10^{-5}: it will not just be applied to "the two pulp mills in Ketchikan and Sitka." Health risk levels factor into an equation to establish numeric criteria for all carcinogenic compounds, not just dioxin and chloroform. How could DEC justify regulating some dischargers at a health risk of 10^{-5} and others at 10^{-6}? UFA finds that DEC has not been up front with the public in discussing this significant issue.

Further, Alaska may have relatively "little industry" generating dioxins, but both of the dioxin-generating pulp mills have been cited by EPA for fines totaling hundreds of thousands of dollars for a variety of violations of federal laws including toxic substance, hazardous waste and disclosure laws, and clean air and clean water legislation (Anc. Daily News 8/1/91, A1; Anc. Daily News, 9/11/92). These fines are some of one of the largest environmental fines ever proposed against Alaska-based industries.

DEC is largely justifying its choice of a health risk of 10^{-5} on what DEC claims are "economic" considerations: DEC is concerned that a more stringent standard would bankrupt at least one of the pulp mills (Anc. Daily News, 9/13/92, A1). This rationale is faulty for two main reasons.

First, DEC has failed to consider economic impacts on the commercial fishing industry in proposing dioxin criterion, according to Dave Sturdevant of DEC (KCHU "Coffee Break" 9/21/92). The seafood and charter industries combined outrank the two pulp mills in terms of employment and overall value in the southeast region. Further in Alaska, commercial fishing is the largest private sector employer and the second largest revenue generator in the state: a market scare from dioxin-contaminated fish could have ripple effects statewide, much like the botulism scare in 1981 which resulted in depressed fish markets and prices for several years.

Sturdevant stated that DEC has commissioned a study from the University of Alaska to consider economic impacts on other industries, but UFA finds this action is somewhat after the fact given that DEC has proposed standards *first*, and commissioned the study *second* (KCHU "Coffee Break" 9/21/92). Further, according to Sturdevant, the study will be completed after the public comment period ends on September 30. Because of DEC's clear intentions to adopt a dioxin criterion less stringent than the standard recommended by EPA, UFA is skeptical that DEC will give adequate - if any - consideration of economic risk to the commercial fishing industry of its actions.

Secondly, DEC has failed to consider that the cost for pollution control, $100 million per mill according to company officials, is artificially inflated because these mills have not been achieving the same level of pollution control as the rest of the country. This is extremely ironic given that both these mills are owned by Outside interests at least one of which - Louisiana Pacific - is not only meeting national standards at its other mills throughout the country, but is setting new levels of pollution control.

By not requiring stringent standards (health risk of 10^{-6}) for dioxin, DEC provides no incentive for these Outside interests to incorporate (or develop) new pollution control technologies in their Alaska-based operations. For example, Louisiana-Pacific Corp. recently announced plans to produce chlorine-free pulp for paper at its plant in Samoa, CA, after a $10 million modification project is completed in 1995 (Anc. Daily News, 9/12/92, Business, pg. 2). Producing pulp without chlorine eliminates dioxin from plant effluent. Presumably, Louisiana-Pacific could develop parallel technology to produce chlorine free pulp for rayon at its plant in Ketchikan given the right economic incentives. The Japanese-owned mill in Sitka, which also produces pulp for rayon, uses about 30 million pounds of chlorine a year.

It is time for these Outside interests to internalize environmental costs at their Alaska-based plants similar to other pulp mills throughout the country. To do less perpetuates this grievous assault by Outside interests on small Alaskan-owned businesses, Alaskan public health, and Alaskan fisheries resources. The longer DEC waits to require pollution control at these two mills, the more it will cost. This artificially high

cost for pollution control should not be used by DEC as a basis for lowering the statewide health risk.

DEC argues that EPA's dioxin criteria of 0.014 ppq is undetectable and therefore, too low, implying that the criteria must be raised to the detection point to be a valid standard. However, the detection limit for dioxin is currently about 8-10 ppq, and DEC's proposed standard of 1.2 ppq is still below the detection limit. A dioxin standard of 0.014 ppq does not create a problem for pulp mills in other areas of the country which meet this standard by taking measurements from the internal wastestream in the bleach plant before it goes through treatment: if dioxin is nondetectable in this internal wastestream, then there is adequate assurance that a standard of 0.014 ppq will be achieved in the receiving waters after plant effluent is treated and diluted 100:1.

DEC has decided to allow people to take more of a chance of developing cancer or other illnesses in order to help these two pulp mills, but DEC has failed to consider that all the people may not be willing to assume that risk. UFA finds that the arguments presented by DEC in its issue paper and "fact" sheet to minimize the health risk of dioxin are flawed at best and purposely misleading at worst.

For example, DEC cites extensively from the writings of Dr. Vernon Houk, supposedly the "government's leading official on environmental hazards" (FS, pg. 8). However through inquiries, UFA determined that Dr. Houk is not actively involved in the current research on dioxin. Specifically, Houk is not the primary author on any of the articles included in the most recent EPA draft report on the health effects of dioxin - a multi-chaptered report involving leading experts from the scientific community worldwide.

Further, the scientists involved in reassessing the health risk of dioxin for the EPA report have found that while dioxin may not be as strong a carcinogen as previously thought, the effects (reproductive and developmental toxicity, immunotoxicity, and acute, subchronic, and chronic toxicity) are much stronger than EPA first predicted. While initially, the cancer risk drove the formula to establish a dioxin criteria, EPA now suspects that other effects will drive the formula in the future. Some of these effects appear to have virtually no threshold concentration, i.e., even minute amounts of dioxin could result in an effect.

Although EPA has not yet committed on this issue, it is highly likely, based on this information, that EPA's new criterion for dioxin, due after completion of the ongoing dioxin reassessment in about 1-1.5 years, will be lower than 0.014 ppq. This may be especially true in light of evidence that the present criterion may not be low enough to protect human health: humans carry a

body burden of 5-10 parts per trillion of dioxin picked up from the environment (Sturdevant, KCHU "Coffee Break" 9/21/92).

A study on bioaccumulation and toxicity of dioxin in fish found that the no observable adverse effect level (NOAEL) for the most sensitive life stage of Lake Superior lake trout - the sac fry - was 34 picogram (10^{-12}) dioxin per gram fish: using a very liberal bioconcentration factor of 51,000 for fish with 7% fat, the water concentration corresponding to the NOAEL is 0.6 ppq (Cook et. al. 1991). A more realistic water concentration assuming 100% lipid in the sac fry would be 0.04 ppq. Recent research from the Great Lakes area shows that dioxin levels as low as 0.0085 ppq are necessary to protect sensitive wildlife (EPA 1992a, pg. 23).

These are significant findings because Alaska does not have a dioxin criterion for aquatic life: by default, the human health criterion will be applied to aquatic life. In the absence of aquatic life criteria, it is important that the human health criteria for dioxin, including health risk, be protective of fish and wildlife.

UFA finds it difficult to believe that DEC was not aware of any of this information on the range of effects from dioxin as is seemingly indicated by the absence of such information in DEC's "fact" sheet, and by DEC's advocation of a higher health risk (10^{-5}). UFA considers the above rationale amply justification for maintaining a health risk of 10^{-6} one in a million.

WEIGHT: UFA accepts EPA's value of 70 kilograms as an average value for adult weight in deriving the dioxin criteria.

WATER CONSUMPTION RATE: UFA accepts EPA's value of 2 liters per day as an average water consumption rate in deriving the dioxin criteria.

BIOCONCENTRATION FACTOR: UFA finds that the bioconcentration factor (BCF) recommended by EPA and DEC - 5,000 - is unrealistically low for Alaska. Instead UFA recommends a BCF of 210,000.

Dioxin is concentrated in the lipids of organisms, i.e., the greater an organism's lipid content, the greater that organism's ability to bioconcentrate dioxin. Justification for a dioxin BCF, therefore, should involve a discussion of lipid content. It should also be noted that "bioconcentration" does not take into account dioxin entering the fish (or consumers) through the food chain, only absorption through the skin. "Bioaccumulation" takes into account all factors, (ingestion, sediment adsorption, etc.), and is the more relevant fac- (Cook 1992). UFA is basing its BCF on bioaccumulation rates.

A 1991 joint study by EPA and the University of Wisconsin on bioaccumulation and toxicity of dioxin in Great Lake fish found that the BCF for fish with 7% lipid was 51,000 under laboratory exposure conditions (Cook, et. al. 1992, pg. 143). Michigan took this finding at face value and adopted a BCF of 51,000 to establish its dioxin criterion. However, Minnesota adjusted the BCF for fish and fish life stages with lipid content greater than 7% (i.e., lake trout with 18% lipid - Cook et. al. 1991, pg. 159) and adopted a BCF of 276,000.

Certain species of salmon, sablefish, and Pacific herring are all considered "fatty" fish, i.e., fish with more than 10% fat (Sikorski, pg. 29). The lipid range in these fatty fish may range from 1% to more than 25% wet weight, depending on environmental and biological factors (Sikorski, pg. 44). For example, adult salmon returning to spawn would have highest lipid content when at "peak" condition before the fish enter the rivers and stop feeding. Commercial fishermen are, therefore, harvesting, selling, and consuming salmon at peak lipid content.

Further, the lipids are stored in the muscles and subcutaneous fat of medium-fat and fat fish which includes most all species of salmon (Sikorski, pg. 44). Retention of this fat was found to be 100% for sockeye salmon cooked four different ways (bake, broil, canning, and microwave) (Sikorski, pg. 127). This means that once dioxin is taken up by salmon, at least, there is a very high potential for human exposure through consumption.

This can also be assumed to hold true for subpopulations of Alaskans who consume the entire fish, not just the muscle tissue. Several studies performed in the Great Lake Basin on fish-eating wildlife indicate that whole fish carcasses have an average fat content of 7.9% (EPA 1991a). Further, studies have shown close agreement of dioxin concentration with the lipid percent in all fish organs (Cook et. al. 1991, pg. 165).

The latest research has found that dioxin bioaccumulation increases by a factor of 10,000-21,000 for every 1% increase in percent fat content of fish residing in large-cold temperature waterbodies with low levels of dissolved or suspended organic matter (Cook et. al. 1991; Cook, 1992). UFA calculated a BCF for dioxin based on 10% average lipid content for fish and an accumulation factor of 21,000, i.e., 10 x 21,000 = 210,000.

UFA believes that this BCF is reasonable as an interim standard to use until the EPA dioxin reassessment is completed. This standard was calculated based on bioaccumulation rates in adult fish and does not consider that younger life stages (sac fry) may accumulate dioxin at much higher rates because of their high proportion of lipid (Cook et. al. 1991). UFA believes that a BCF of 210,000 is the <u>minimum</u> necessary to protect aquatic life, particularly "fatty" fish, and that with more data, this BCF may have to be increased.

FISH CONSUMPTION RATE: UFA finds that DEC, in its enthusiasm to make the dioxin criteria less stringent for two pulp mills, has chosen an unrealistically low fish consumption rate (FCR) for Alaska, and in doing so, may have grossly underestimated the health risk from consumption of potentially contaminated seafood. UFA recommends a fish consumption rate (FCR) of 65 g/d (about 50 pounds per person per year).

DEC initially maintained that the "most recent comprehensive data available" indicate a national average for consumption of fish and shellfish of 6.5 grams/day (g/d) which translates to approximately 5 (not 7) pounds per person per year (IP, pg. 9). After public outcry over the low FCR, DEC asserted that 6.5 g/d represented consumption of *contaminated* fish only (Sturdevant, KCHU "Coffee Break" 9/21/92, and public teleconference 9/25/92). Whatever the rationale, it - and the standard - are all incorrect for several reasons.

First, according to the National Marine Fisheries Service, which updates national FCR each year in its annual publication "Fisheries of the United States", the national FCR for 1991 was 15 pounds/year or about 19 g/d. DEC chose 6.5 g/d based (initially) on EPA's standard estimate of "average consumption of fish and shellfish from estuarine and freshwaters" by the entire U.S. population (EPA 1991b, pg. 37). However, in selecting this standard, DEC fails to account for consumption of fish and shellfish from *marine* water. EPA's standard estimate of fish and shellfish consumption from all waters is 20 g/d, virtually identical to NMFS's estimate of 15 pounds/year (EPA 1991b, pg. 37).

Secondly, according to EPA-Seattle risk assessment staff, its standard FCRs are based on average fish and shellfish consumption, not average *contaminated* fish and shellfish consumption. The reason for this is the underlying assumption that some consumers may eat predominantly locally-caught fish. For example, if there is a point source discharge, like the Sitka or Ketchikan pulp mills, then there is a risk that 100% of the locally-caught fish and shellfish could be contaminated. Thus, basing a FCR on average consumption would protect these consumers.

UFA firmly believes that Alaskans consume much more than 5 or even 15 pounds of seafood per person per year. DEC acknowledges that "Alaska clearly has subpopulations of sport and subsistence fishermen who consume much greater amounts of fish than the national average" (IP, pg. 6). DEC notes that a survey conducted by ADF&G during the 1980s indicate that consumption data are "variable, ranging generally from 30 to 300 pounds per year, with a few lower values and some values as high as 700 pounds per year" (IP, pg. 6). Despite this information, DEC has selected the lowest FCR in the nation. DEC has also neglected to account

for seafood consumption by commercial fishermen, another "subpopulation" which, UFA believes, consumes more seafood than the 15 pound/year national average.

Six other states have chosen a FCR higher than the national average to protect the high end consumer - subpopulations with a high fish diet such as, in the Great Lakes Basin, subsistence anglers (EPA 1992b). Deleware has the highest FCR of 37 g/d. (Minnesota has the second highest FCR of 30 g/d.)

Based on this information, UFA proposes that DEC adopt a FCR of 65 g/d or roughly a pound of seafood per person per week. UFA maintains that DEC should use this FCR when considering health risk from all toxic and nonconventional pollutants, including dioxin, to all Alaskan residents. This FCR is especially important when dealing with areas of the state, like Sitka, Ketchikan, and other southeast communities, which have large populations of commercial and sport fisherpeople, and subsistence users.

If anything, even the 65 g/d standard is too low to protect the high end consumer. The FCR is based on "cleaned" seafood or consumption of muscle tissue only ("guts" and carcass discarded). However, many subsistence users consume the entire animal and could be exposed to much higher levels of contaminants because pollutants tend to be stored in the "guts." Further, the 65 g/d standard may be too low to protect consumers, especially high end consumers, who eat predominantly locally-caught seafood.

It is interesting to note that research from the Great Lakes Basin found that piscivorous (fish-eating) species of birds and mammals were extremely sensitive to dioxin (EPA 1992c). The overall dioxin criterion recommended to protect these species (mink, otter, eagle, osprey, and kingfisher) is 0.0085 ppg, as mentioned earlier. It is interesting to note that this low criterion was driven by the extreme sensitivity of fish-eating mammals (EPA 1992d).

DEC's assumption that "(m)ost Alaskan fish taken by sport and subsistence users [and presumably commercial fishermen although DEC never once accounts for this group] are migratory, and so would not be expected to contain the projected bioconcentration levels, even if taken from a contaminated area" (IP, pg. 6) is not valid for several reasons.

All species taken by the commercial and sport fishing industries and by subsistence users as varying amounts of time feeding in nearshore waters. The highly migratory salmon and herring feed as juveniles in nearshore areas as do the less migratory halibut, cod, ling cod, and rockfish. Comparatively nonmigratory species like crab - and other shellfish (mussels) taken by subsistence users - can spend the majority of their lives in nearshore areas.

Sublethal effects have been demonstrated with fish exposed to pulp mill effluents containing dioxin (In Cook et. al. 1991, pg. 149). Massive fish kills in Sitka's Silver Bay are a reoccurring event - 1970, 1982, 1990 - and are thought to be related to disturbances of the sludge mat, a mixture of chemicals, waste and wood fibers that carpets the bottom of parts of Silver Bay (Anc. Times, 8/15/91, A9-10). Scientists say little if anything can survive in bottom areas covered with sludge mats: at least one bottom sample of Sitka sludge analyzed by the U.S. Fish and Wildlife Service "was extremely toxic and would be toxic to almost any organism encountering it" (U.S. Fish & Wildlife Service 1990).

Despite the seemingly obvious, DEC maintains that it does not know the cause of the fish kills. DEC also claims that it does not have the expertise to determine if Sitka seafood poses a public health risk (Anc. Times 8/15/91: A1). Yet this same agency is now trying to minimize the health risk which DEC admits it has no expertise to judge. UFA requests that the agency adopt a more conservative approach by selecting an FCR of 65 g/d.

CANCER POTENCY FACTOR: UFA finds that DEC has underestimated the cancer potency factor (CPF) and recommends that DEC adopt a standard of 86,750 rather than 17,500.

Reassessment by EPA of the dioxin health risk indicates, as mentioned earlier, that the cancer potency of dioxin may not be as great once thought. The ten fold difference between the FDA and the EPA CPFs (17,500 and 156,000, respectively) can be attributed to three variables: (1) EPA accounted for the high early mortality in the early dose group (FDA did not); (2) EPA looked at a wider variety of tumor types (FDA limited their analysis to certain tumors); and (3) EPA took a more conservative approach when extrapolating the data from animals to man.

The latter variable alone accounts for nearly half of the difference between the two CPFs (Devoli 1992). The three federal agencies that have published CPFs have since agreed to an alternate extrapolation method based on body weight (raised to the 3/4 power) which will probably work to lower EPA's CPF and raise FDA's CPF, absent consideration of the two other variables. The arithmetic mean of the FDA and the EPA standards is 86,750. This is the amount of the difference between the two methods that can be attributed to differences in the extrapolation methods.

DEC is apparently willing to ignore differences between the two methods based on the other two variables, i.e., early mortality and number of tumors, and so has proposed the lowest CPF available. UFA maintains that it may not be wise to ignore these variables absent other information from the dioxin reassessment.

So at this time UFA recommends adopting a CPF of 86,750 as an interim standard. It should be noted that this reflects the current thinking that the CPF initially recommended by EPA may be too high, while the FDA factor may be too low.

REGULATING ARSENIC
(Unless otherwise stated, citations are from regulatory issue paper (IP) or fact sheet (FS) on arsenic.)

UFA recommends that Alaska adopt human health arsenic criteria of 8.2 ppt and 14 ppt for fresh and saltwater, respectively, based on a risk level of 10^{-6}, a fish consumption rate of 65 g/d, a bioconcentration factor of 44, and a cancer potency factor of 1.75.

Justification: UFA finds that the state has substantially underestimated the health risk from arsenic in order to "help the A-J and Kensington gold mines" (Anc. Daily News, 9/12/92, A1). UFA finds that this approach lacks scientifically defensible arguments.

Arsenic is a carcinogen and should be regulated as such. UFA has adopted EPA's bioconcentration factor and cancer potency factor, but selected a health risk of 10^{-6}, and a fish consumption rate of 65 g/d for reasons consistent with the discussion presented for dioxin.

Arsenic is a very complex chemical and exists in several forms, as recognized and discussed by DEC in its issue and fact papers. While UFA agrees with DEC that toxicity is associated with the inorganic form "arsenite" [arsenic(III)], DEC's implication that other forms can be ignored are without scientific basis. Arsenic cycling in marine and other ecosystems is dependent upon multiple variables, such as temperature, salinity, presence of particulates, and reduction/oxidation potential (Maher & Butler 1988, pg. 197). Many organisms are reported to influence arsenic speciation, such as bacteria, macroalgae and their epiphytes. Marine organisms in general are thought to accumulate more arsenic than fresh-water organisms (Maher & Butler 1992, pg. 197).

It is because of arsenic's complex nature - primarily, its ability to change from the nontoxic to the toxic form - that EPA recommends that health risk be determined based on total arsenic, not just concentration of arsenic(III). DEC's approach, i.e., that of assuming minimal presence of the toxic form of arsenic (III), is without scientific merit.

Further, DEC's assumption that "fish are not a significant source of inorganic, trivalent arsenic" (IP pg. 5), while true for fish from waters with low levels of arsenic, may not hold true for fish from waters with high arsenic levels. The toxic form arsenic(III) tends to be concentrated in certain organs such as the liver, gills, and heart. In many parts of Alaska, subpopulations of residents consume the entire fish. DEC's proposed criteria would not be protective of these subpopulations.

UFA recognizes that Alaska has high naturally occurring levels of arsenic. However, it is precisely because of this fact that UFA is advocating a conservative approach for regulating arsenic. Additions to the total level of arsenic already in environment would most likely result in increased levels of arsenic(III), assuming the arsenic species occur in a dynamic steady state. Allowing more arsenic to be discharged could potentially exacerbate the risk posed by the high natural background levels.

REGULATING CHLOROFORM

UFA recommends tnat Alaska adopt a human health criterion for chloroform of 5.11 ppb based on a risk level of 10^{-6}, a fish consumption rate of 65 g/d, a bioconcentration factor of 3.75, and a cancer potency factor of 0.0061.

Justification: UFA has adopted EPA's bioconcentration factor and cancer potency factor, but selected a health risk of 10^{-6}, and a fish consumption rate of 65 g/d for reasons consistent with the discussion presented for dioxin.

DEFINITION OF "WATER" 18 AAC 70.1109(46)
(Unless otherwise stated, citations are from regulatory issue paper on definition of water.)

UFA recommends that the definition of "water" NOT be amended to exclude unlined impoundments and other surface water bodies that are integral parts of wastewater treatment systems.

Justification: Unlined tailing ponds and other wastewater treatment facilities should not be allowed to contain concentrations of toxic wastes which exceed state water quality standards, because water in these facilities could contaminate ground and surface water resources, and find its way into drinking water supplies and fish spawning areas.

0.01 TIMES THE 96 HOUR LC_{50}
(Unless otherwise stated, citations are from regulatory issue paper on color, 0.01 LC_{50}, total hydrocarbons, sediment, and fecal coliform bacteria.)

UFA recommends that DEC's proposal to discontinue "0.01 times the lowest measured 96 hour LC_{50}" as a term to establish water quality criteria for toxic substances be approved. However, UFA recommends that the DEC adopt in its place a provision requiring mandatory application of chronic water quality criteria based on direct "No Observed Effects" toxicity testing for total hydrocarbons. UFA recommends the following changes be adopted for the narrative criteria for:

Toxic Substances

Substances shall not [INDIVIDUALLY OR] in combination exceed, at the end of the discharge pipe, [0.01 TIMES THE LOWEST MEASURED 96 HOUR LC_{50} (SEE NOTE 8)] acute criteria based on the "No Observed Effects Level" of chronic toxicity as determined through toxicity testing of the most sensitive and biologically important life stages of resident aquatic organisms using methods approved by EPA, or exceed criteria cited in EPA, Quality Criteria for Water (See Note 5) or Alaska Drinking Water Standards (18 AAC 80), whichever concentration is less. Individual substances shall not exceed, at the end of the discharge pipe, acute criteria based on the "No Observed Effects Level" of chronic toxicity as determined through toxicity testing of the most sensitive and biologically important life stages of resident aquatic organisms using methods approved by EPA. Substances shall not ... etc., Notes 5 and 8)."

Petroleum Hydrocarbons for Aquaculture

Shall not exceed [0.01 TIMES THE CONTINUOUS FLOW 96 HOUR LC_{50} OR, IF NOT AVAILABLE, THE STATIC TEST 96 HOUR LC_{50}] chronic toxicity criteria for whole effluent for the species involved. (See Notes 8 and 9). Total aromatic hydrocarbons in the water column shall not exceed 10 ug/l (See Notes 9 and 10), or exceed criteria [ESTABLISHED AT THE DEPARTMENT'S DISCRETION,] based on the "No Observed Effects Level" of chronic toxicity as determined through toxicity testing of the most sensitive and biologically important life stages of resident aquatic organisms using methods approved by EPA [OR OTHER METHODS APPROVED BY THE DEPARTMENT]. Concentrations ... etc., from floating oils."

Petroleum Hydrocarbons for Fresh and Marine Waters for "Growth and Propagation of Fish, Shellfish,, Other Aquatic Life, and Wildlife":

"Total hydrocarbons in the water column shall not exceed 15 ug/l, or [0.01 OF THE LOWEST MEASURED CONTINUOUS FLOW 96 HOUR

LC_{50}] exceed chronic toxicity criteria for whole effluent based on the "No Observed Effects Level" of chronic toxicity as determined through toxicity testing of the most sensitive and biologically important life stages of resident aquatic organisms using methods approved by EPA, whichever is less (See Notes 8 and 9). Total aromatic hydrocarbons in the water column shall not exceed 10 ug/l, or [0.01 OF THE LOWEST MEASURED CONTINUOUS FLOW 96 HOUR LC_{50} FOR LIFE STAGES OF SPECIES IDENTIFIED BY THE DEPARTMENT AS THE MOST SENSITIVE, BIOLOGICALLY IMPORTANT SPECIES IN A PARTICULAR LOCATION, WHICHEVER CONCENTRATION IS LESS] exceed criteria based on the "No Observed Effects Level" of chronic toxicity as determined through toxicity testing of the most sensitive and biologically important life stages of resident aquatic organisms using methods approved by EPA (See Notes 9 and 10)." (Note: remainder of narrative criteria for fresh and marine waters unchanged.)

Justification: Basically, UFA is recommending substitution of the outdated chronic toxicity test, based on 0.01 times the 96 hour LC_{50}, with the new chronic toxicity test based on "No Observable Effects Level" as is currently accepted by the scientific community. This is exactly what DEC is recommending for total aromatic hydrocarbons.

However, DEC is recommending discretionary application of the chronic toxicity test. UFA recommends mandatory testing as was previously required.

UFA proposed changes under "Toxic Substances" (i.e., "at the end of the discharge pipe") are consistent with our philosophy that discharge of toxic substances in toxic quantities should be prohibited. This discussion is expanded under justification for acute and chronic toxicity testing for whole effluent.

UFA believes that the state should assume responsibility for discharge of its pollutants into its waters *only if* the state clearly demonstrates its intent seek a balance among all user groups and the public health. This responsibility is not something to be taken lightly. There is more to it than simply dumping pollutants into state waters: there is a great deal of work in terms of monitoring, regulating, and enforcing state standards. The state should not be allowed to exercise its authority to control discharge of pollutants unless the state demonstrates it clearly intends to monitor, regulate, and enforce its standards. Further, monitoring itself must be conducted with rigorous scientific methodology, including standardized protocol and quality control/quality assurance: methodology should be approved by EPA.

To require any less of the state invites disaster.

UFA

The major difference between UFA's and DEC's language is that DEC recommends dropping the standard for total hydrocarbons, while UFA does not. This is discussed elsewhere.

TOTAL HYDROCARBONS
(Unless otherwise stated, citations are from regulatory issue paper on color, 0.01 LC_{50}, total hydrocarbons, sediment, and fecal coliform bacteria.)

UFA recommends that the standard for total hydrocarbons be changed as follows:

> "Total hydrocarbons in the water column shall not exceed 15 ug/l, or [0.01 OF THE LOWEST MEASURED CONTINUOUS FLOW 96 HOUR LC_{50} FOR LIFE STAGES OF SPECIES IDENTIFIED BY THE DEPARTMENT AS THE MOST SENSITIVE, BIOLOGICALLY IMPORTANT SPECIES IN A PARTICULAR LOCATION] exceed criteria based on the "No Observed Effects Level" of chronic toxicity as determined through toxicity testing of the most sensitive and biologically important life stages of resident aquatic organisms using methods approved by EPA, whichever concentration is less. Total aromatic hydrocarbons in the water column shall not exceed 10 ug/l, or [0.01 OF THE LOWEST MEASURED CONTINUOUS FLOW 96 HOUR LC_{50} FOR LIFE STAGES OF SPECIES IDENTIFIED BY THE DEPARTMENT AS THE MOST SENSITIVE, BIOLOGICALLY IMPORTANT SPECIES IN A PARTICULAR LOCATION] exceed criteria based on the "No Observed Effects Level" of chronic toxicity as determined through toxicity testing of the most sensitive and biologically important life stages of resident aquatic organisms using methods approved by EPA, whichever concentration is less. Concentrations of hydrocarbons, animal fats, or vegetable oils in the sediment shall not cause deleterious effects to aquatic life. Shall not cause a film, sheen, or discoloration on the surface or floor of the water body or adjoining shorelines. Surface waters shall be virtually free from floating oils. Substances shall not be present or exceed concentrations that individually or in combination impart undesirable odor or taste to fish or other aquatic organisms as determined by either bioassay or organoleptic tests (See Note 5)."

Further, UFA recommends changing the method specified in the definition of "total hydrocarbon" to methodologies specific to sewage wastewater treatment (total hydrocarbons, including animal and petroleum), and petroleum wastewater treatment.

Justification: The following discussion will focus solely on proposed deletion of the total hydrocarbon standard. Justification for substitution of chronic toxicity tests (chronic toxicity criteria based on NOEL for "0.01 of the lowest measured continuous flow 96 hour LC_{50}") is discussed elsewhere.

UFA finds that DEC's rationale for dropping the total hydrocarbon (TH) standard defies logic. Basically, the department is maintaining that the methodology for measuring total hydrocarbons is incorrect and, therefore, the TH standard needs to be dropped. DEC maintains that the required method is "erroneous" because (1)

it measures both dissolved and particulate hydrocarbons, and (2) the state's TH criterion of 15 ppb exceeds the detection limits of the methodology (200 ppb).

First, it is DEC's premise that TH should only measure soluble or dissolved TH, not all hydrocarbons, (i.e., dissolved, emulsified and particulate) that is erroneous. DEC maintains that the "dissolved biologically available hydrocarbons are the principal fraction affecting aquatic organisms" (pg. 10). This is simply not true: particulate hydrocarbons are biologically available to - and affect - filter feeders.

Particulate hydrocarbons are the principal component of a major source of oily waste generated by petroleum operations, i.e., oily sludge. Particulate hydrocarbons in oily waste are composed to a large extent of high molecular weight polynuclear aromatic hydrocarbons (HPAH). HPAH as a class tend to be persistent in the environment, readily transferable among organisms, and associated with sublethal effects such as reproductive impairment, liver dysfunction, hepatic tumors, etc. across a wide spectrum of organisms.

Under DEC's proposed revisions, unmonitored discharges of particulate hydrocarbons could be released into receiving waters. In some areas, this could have potentially enormous environmental impacts. For example, the Alyeska oil terminal in Port Valdez has a ratio of TH to total aromatic hydrocarbons (particulates to dissolved) in excess of 560 to 1 (IP, pg. 10). That is, the overwhelmingly dominant form of hydrocarbon in Alyeska's effluent is particulate hydrocarbon, released with about 1.5 tons per day of total suspended solids (TSS). According to data collected by Alyeska's contractors, the dominant taxon group in the Port Valdez benthos is a tiny filter feeder. Increasing the discharge of particulate hydrocarbons could affect both filter feeders and higher organisms, as filter feeders assimilate the particulate hydrocarbons and introduce HPAH into the food web (McCain et. al. 1990).

Based on its assumption that only total soluble hydrocarbons should be measured, DEC states that the method specified in the Water Quality Standards for measurement of TH is "erroneous" because it measure oil and grease, i.e., particulate as well as dissolved hydrocarbons (pg. 10). DEC claims that this "appears to be an inadvertent error not caught when the criterion and definition were adopted" (pg. 10). Nothing could be further from the truth: this method was selected because it does in fact measure TH, both dissolved and particulates. UFA supports continued use of this methodology.

Regarding DEC's second premise, DEC maintains that because the TH criterion is below the detection level of the required method, the standard itself should be dropped. DEC's rationale for dropping the standard is that most other states with numeric criteria for TH express them as ppm (parts per million) rather

than ppb (parts per billion) as Alaska, "making them less stringent than Alaska's criterion by roughly three orders of magnitude" (pg. 10).

The state has stringent standards for TH because Alaska led the nation in developing hydrocarbon standards based on Alyeska's promise in 1973 to use of Best Available Technology for the terminal pollution control equipment. But right from the beginning, Alyeska has lobbied regulators to reduce these very standards. In the late 1970's, Alyeska lobbied to increase the size of its mixing zone to accommodate its effluent when it was determined that water quality standards were not being met. Alyeska's wish was granted. In the late 1980's, Alyeska lobbied to increase the limit for flow rate and TSS (so it could legally increase its discharge of oily sludge - particulate hydrocarbons), despite evidence that removal of hydrocarbons would not be optimal at the higher flow rate. Alyeska's wish was granted again. Now Alyeska is lobbying to remove the state's standard for particulate hydrocarbons altogether so it can legally discharge unlimited quantities of oily sludge, despite evidence that HPAH are accumulating in the bottom sediment of Port Valdez (Feder & Shaw 1992). It is time to draw the line and require Alyeska to use Best Available Technology to control its particulate hydrocarbon emissions.

It is important to recognize that the total hydrocarbon standard applies to all facilities that discharge oily wastewater, not just Alyeska. This includes domestic sewage treatment facilities. In light of this, UFA would like to point out a substantive problem with method 503B: it may not be ideal to use the same analytical method to monitor TH from sewage wastewater treatment facilities and from petroleum-based wastewater treatment operations. Method 503B measures TH in terms of animal and petroleum hydrocarbons. Method 503E, for example, measures total petroleum hydrocarbons. While neither of these methods have detection limits as low as 15 ppb TH, according to Dr. Ihor Lysyj, an analytical chemist, "there are analytical procedures that can measure hydrocarbons, individually or collectively, at the 1 ppb level" (personal communication). However, it will take some research to determine the appropriate methodology: this is another example of an issue that could be dealt with by a Clean Water Task Force.

While DEC recommends deletion of the TH criterion, it recommends "retention of the narrative standard prohibiting hydrocarbons in sediments and prohibiting surface sheens" (pg. 11). UFA finds that this makes little sense. If the TH criterion is removed, the result will almost surely be an accumulation of hydrocarbons in the sediment as particulate hydrocarbons tend to settle to the bottom (as demonstrated by Alyeska's environmental monitoring data). Further, if industries are not required to monitor particulate hydrocarbons as a component of TH, then the state will not be able to identify the source of the particulate

hydrocarbons in the sediment, and will not be able to hold a party accountable for cleanup, if necessary.

Further, surface sheens become visible at approximately 10 ppm (parts per million), according to Dr. Lysyj, which is 667 times more oil than the current TH standard of 15 ppb. UFA does not recommend substituting the state's numeric criterion for TH for a narrative as nebulous - and as high - as "visible sheen."

The wording regarding organoleptic tests is currently in the state standards and appears to have been inadvertently left out of the revisions.

LIMITING ACUTE AND CHRONIC TOXICITY OF WHOLE EFFLUENT
18 AAC 70.022
(Unless otherwise stated, citations are from regulatory issue paper on chronic toxicity testing of whole effluent.)

UFA strongly recommends that the state adopt an acute toxicity criteria for whole effluent in conjunction with the proposed addition of chronic toxicity testing for whole effluent to read as follows:

> "18 AAC 70.022. ACUTE AND CHRONIC TOXICITY OF AN EFFLUENT. The acute toxicity of an effluent discharged to waters of the state, measured at the point of discharge, shall not exceed 0.3 acute toxic units (TU_a). The chronic toxicity of an effluent discharged to waters of the state, measured at the point of discharge or at the boundary of a mixing zone authorized by the department in a permit or certification, shall not exceed 1.0 chronic toxic units (TU_c).
>
> The department will require in its permits and certifications that acute and chronic toxicity testing of the whole effluent be conducted by the applicant to determine compliance with these criteria. Testing shall be based on the "No Observed Effects Concentration" according to methods and procedures approved by the U.S. Environmental Protection Agency. Testing must utilize sensitive standard bioassay organisms and the most sensitive and biologically important life stages of at least three resident species from ecological diverse taxa."

UFA further recommends that the state not adopt chronic toxicity testing without acute toxicity testing for whole effluent.

Justification: According to EPA's notice of March 9, 1984, (Federal Register Vol. 49, No. 48, pg. 9017), regarding development of water quality-based permit limitations for toxic pollutants, it is national policy that "all states have water quality standards which include narrative statements prohibiting the discharge of toxic materials in toxic amounts."

EPA's rationale in setting a general standard of "no toxic materials in toxic amounts" is that "for toxic and nonconventional pollutants it may be difficult in some situations to determine attainment or nonattainment of water quality standards and set appropriate limits because of complex chemical interactions which affect the fate and ultimate impact of toxic substances in the receiving water. In many cases, all potentially toxic pollutants cannot be identified by chemical methods."

By examining whole effluent toxicity and instream impacts using biological methods, it becomes possible to establish toxicity for toxic and nonconventional pollutants on a site specific basis.

Therefore, by requiring all states to adopt a generic standard of "no toxic materials in toxic amounts," it is possible to strive for and to maintain clean water in virtually all discharge scenarios.

According to EPA (1991b),

> "(a)t present, there are no national criteria developed under (Clean Water Act) Section 304(a) for whole effluent toxicity. Acute and chronic toxicity units (TUs) are a mechanism for quantifying instream toxicity using the whole effluent approach.
>
> "EPA's recommended magnitudes for whole effluent toxicity are as follows ...: a CMC (criteria maximum concentration) to protect against acute (short-term) effects and a CCC (criteria continuous concentration) to protect against chronic (long-term) effects (emphasis added). For acute protection, the CMC would be set at 0.3 acute toxic units (TU_a) to the most sensitive of at least three test species. For chronic protection, the CCC should be set at 1.0 chronic toxic units (TU_c) to the most sensitive of at least three test species" (pg. 35).

According to DEC, "(t)he State of Alaska does not apply acute aquatic life criteria at the boundary of the mixing zone, and does not propose to apply an acute limit on whole effluent toxicity at this time" (IP pg. 4). The state reasons that "(i)n most cases, the chronic toxicity limit is expected to be more stringent than the acute toxicity limit" (IP pg. 4).

UFA finds that these two statements by DEC indicate either a reprehensible lack of understanding of the nature and the effects of whole effluent toxicity, or are a calculated attempt further weaken the state's water quality standards. Of course acute aquatic life criteria are not applied at the boundary of a mixing zone: the whole purpose of a mixing zone is to dilute industrial wastestreams, usually by a hundredfold minimum, so that chronic criteria can be applied at the boundary of a mixing zone. Yes, in most cases, the chronic toxicity limit will be more stringent than the acute toxicity limit, but in most cases, that does not matter.

Under DEC's proposal, an industry could release a slug of extremely toxic material into the mixing zone. The toxic materials could then be diluted and successfully meet the chronic criteria for whole effluent. (EPA recommends an averaging period of 4 days for chronic criteria. That is, "the 4-day average exposure should not exceed the CCC" (EPA 1991b, pg. 35).)

This is a very significant issue in Alaska where many aquatic organisms are migratory. Migratory and other species swimming through a mixing zone during a release of extremely toxic materials could be exposed to lethal doses of pollutants. Also,

planktonic life stages of fish, shellfish and other organisms drifting through a mixing zone could also be exposed to lethal doses of pollutants.

It is also important to realize that some pollutants are extremely fast-acting and that it is possible for organisms to receive lethal doses of such chemicals during very short exposure times (e.g. ammonia, hydrogen sulfide). EPA recommends an averaging period of one hour for acute criteria. That is, "to protect against acute effects, the 1-hour average exposure should not exceed the CMC" (EPA 1991b, pg. 35).

DEC acknowledged that, by not adopting acute toxicity testing criteria for whole effluent, industries could discharge slugs of toxic materials into state waters, but rationalized that industry could not do this repeatedly without noticeable impacts on the environment (Sturdevant, KCHU "Coffee Break", 9/21/92). This rationale illustrates the extremes to which the state is willing to risk Alaska's high quality waters to benefit "dirty water" industries. The object of the state's water quality standards should be to prevent deterioration of its water bodies as mandated by the Clean Water Act, not to repair the water bodies after there has been "noticeable" harm.

EPA discusses four alternative ways to prevent lethality to organisms passing through the mixing zone (EPA 1991b, pg. 71). Of these, UFA recommends the first: "prohibit concentrations in excess of the CMC in the pipe itself, as measured directly at the end of the pipe." This is consistent with UFA's recommendation to include a narrative prohibiting discharge of "toxic materials in toxic amounts" as discussed earlier.

Prohibiting discharge of toxic materials in toxic amounts becomes more important now that the 100-to-1 safety margin for error, built into the chronic toxicity test of "0.01 times the 96 hour LC_{50}," has been eliminated. The replacement "NOEC" chronic toxicity test does not have such a safety margin. During periods of "upset" conditions, for example, it would be more likely under the new standards to exceed the chronic toxicity criteria.

<u>UFA maintains that chronic criteria for whole effluent testing cannot be applied independently of acute criteria for whole effluent testing without undermining the basic goals of the Clean Water Act</u>. To attempt to do so allows industry an opportunity to pollute.

UFA finds that DEC's proposed phrases regarding methodology ("to the extent feasible," and "alternative methods...specified by the department") leave too much to the discretion of DEC. If standardized protocols are not followed, and if tests are not conducted with the most sensitive and biological important life stages, as recommended by EPA (EPA 1991b, pg. 35), test results could be meaningless. Further, since the NOEL chronic toxicity testing is in its infancy, some applicants may wish to use

standardized test organisms as a check against the resident species.

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