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Re: Proposed Permits for Application of Herbicides by the Alaska Railroad Corporation

Comments Prepared by:
Pamela Miller, M.En., Executive Director and Biologist; Alaska Community Action on Toxics
Heather McCausland, Development and Communications coordinator

Dear Ms. Colvin:

These comments are presented on behalf of Alaska Community Action on Toxics.

Alaska Community Action on Toxics ("ACAT") is a statewide non-profit public interest environmental health research and advocacy organization dedicated to protecting environmental health and achieving environmental justice. Alaska Community Action on Toxics *mission: to assure justice by advocating for environmental and community health. We believe that everyone has a right to clean air, clean water and toxic-free food*. We work to stop the production, proliferation, and release of toxic chemicals that may harm human health or the environment.

Alaska Community Action on Toxics firmly opposes the use of herbicides and associated chemicals for vegetation management purposes by the Point MacKenzie Correctional Farm. Please enter these comments into the public record and confirm receipt. We appreciate your careful consideration. We maintain that there are viable, economical alternatives that preclude the need for chemical treatments. Our comments provide justification for our opposition on the basis that herbicide use poses an unacceptable threat to water quality, fish, wildlife, habitat, workers, and public health. We assert that the Alaska Department of Environmental Conservation should deny the permit application of the Point MacKenzie Correctional Farm in order to meet the Department's obligation to protect human health and the environment.

Over the past three decades, citizens of Alaska have consistently voiced strong opposition to the use of herbicides in Alaska. Prompted by the concerns of people about the harmful effects of herbicides on human health and the environment, Governor Jay Hammond banned the use of herbicides by state agencies in 1978. Community members along the railway initiated a lawsuit to stop herbicide use by the Alaska Railroad. A federal judge determined in 1983 that herbicides could not be used without preparation of an Environmental Impact Statement as mandated by the National Environmental Policy Act. In 1985, the state assumed control of the railroad and the state ban on the use of herbicides was

applied and continues to apply to the present.[i] Alaskans have particular concern about the use of herbicides because many people in this state "participate in the harvest and consumption of various wild plants, game, and fish." Also, our economy depends on "several of Alaska's major industries such as commercial fishing and tourism…, on the image, as well as the reality, of a pristine, non-toxic environment," and the "unique environmental conditions inherent in the sub-Arctic and Arctic environment." In a study published in 1991, the University of Alaska Fairbanks "found a greater persistence of the parent herbicide compounds and far more extensive downward migration of the herbicides than had been anticipated based on the available scientific literature."[ii]

The Department must respect and heed the substantive on-going public opposition to past and current permit applications for the proposed use of pesticides. Resolutions and letters have been formally adopted by local tribes, borough governments, municipalities, and community councils—these resolutions and letters still stand as formal and currently relevant opposition to the Railroad's herbicide use and apply to the proposed uses at Point MacKenzie Correctional Farm. To ignore the long-standing opposition from local governments, tribes, and citizens is a violation of public trust. Standing resolutions and letters expressing opposition to the use of herbicides by the Alaska Railroad include: Native Village of Eklutna (resolution and letter from the tribal government), Montana Creek Native Association, Inc. (resolution), Municipality of Anchorage (letter), City of Seward (resolution), Kenai Peninsula Borough (resolution), Matanuska-Susitna Borough (resolution), Denali Borough (resolution), Birchwood Community Council, and Talkeetna Community Council. The federally recognized tribal government of the Native Village of Eklutna states particular concerns in their resolution and letter about threat of proposed herbicide use to the safe harvest of berries, medicinal plants, fish and wildlife that are vital to their spiritual, cultural, and physical sustenance. In addition, the Chickaloon Village Traditional Council (CVTC) opposes the herbicide applications by the Alaska Railroad. CVTC is a federally and internationally recognized as a Traditional sovereign government with a nine member traditional council that is the governing body for the Tribe, Chickaloon Native Village. CVTC's purpose is to perpetuate their ancestors' beliefs, customs, traditions, and values and steward the environment to help their citizens thrive. The proposed herbicide applications by the Alaska Railroad would infringe on the Tribe's right to a safe and healthy environment for their tribal members.

Environmental and Human Health Effects of the Proposed Chemical Mixtures

The following sections provide a review of some of the peer-reviewed literature concerning the environmental and health effects associated with the active ingredient and herbicide mixture. Although not intended as an exhaustive review of the literature, the summary presented here clearly demonstrate that the active ingredients and herbicide mixtures proposed for use by the Point MacKenzie Correctional Farm are unacceptable for use because it will likely harm the health and well-being of fish, wildlife and communities in close proximity to the Point MacKenzie Correctional Farm, workers, prisoners, and individuals who use adjacent lands and waters for berry-picking, fishing, and hunting. These comments include a literature review included in our comments from 2009 for the ARRC's permit applications to ADEC and are updated with current research. A 1991 study by the University of Alaska Fairbanks states: "Should herbicides contact groundwater in significant concentrations, considerable liability could result from cleanup efforts."[iii] The Point MacKenzie Correctional Farm has failed to

consider this potential liability and other externalities and monitoring requirements in their permit application.

On August 1, 2006 the Attorney General of Alaska announced that Alaska "joined with 13 other states and the U.S. Virgin Islands to petition the Environmental Protection Agency (EPA) to require pesticide manufacturers to disclose on the label of their product all hazardous ingredients…The EPA currently requires that pesticide labels disclose only the product's "active" ingredients that contain toxic materials intended to kill insects, weeds, or other target organisms. Pesticide products also contain many other "inert" ingredients, which are intended to preserve or improve the effectiveness of the pesticides' active ingredients. These "inert" ingredients may be toxic themselves…" The news release further states that "people who use or who are impacted by the use of a pesticide should have notice of all that product's potential health risks." Thus, it would be wrong for the State to issue a permit to the Point MacKenzie Correctional Farm to apply pesticides for which the manufacturers do not disclose ingredients that may harm human health.

Dr. Warren Porter, Professor of Environmental Toxicology at the University of Wisconsin, Madison, completed a review of the literature concerning the environmental health effects of low-dose chemical mixtures of pesticides.[iv] He concluded:

> Pesticides have interactive effects and ultra low-level effects that are below EPA allowable levels. These effects include adverse neurological, endocrine, immune, reproductive and developmental health outcomes.

EPA assessments of biological risk can be off by a factor of 10,000 at ultra low doses. Scientists call for a new type of risk assessment in the open literature because of the inadequacies of the current EPA pesticide registration system.

Pesticides have broad biological effects that are unintended and often unpredictable because of physicochemical properties engineered into their molecules.

Pesticides of different classes can have similar impacts on endocrine disruption and sexual development. Chemicals affect development at levels in the tenths of a part per billion range.

In a recent issue of the preeminent peer-reviewed environmental health journal published by the National Institute for Environmental Health Sciences, *Environmental Health Perspectives*,[v] the authors warn:

> "Inert ingredients may be biologically or chemically active and are labeled inert only because of their function in the formulated product…Inert ingredients can increase the ability of pesticide formulations to affect significant toxicological endpoints, including developmental neurotoxicity, genotoxicity, and disruption of hormone function. They can also increase exposure by increasing dermal absorption, decreasing the efficacy of protective clothing, and increasing environmental mobility and persistence. Inert ingredients can increase the phytotoxicity of pesticide formulations, as well as toxicity to fish, amphibians, and microorganisms."

In this case, the active ingredient, glyphosate, cannot be used without an adjuvant and/or surfactant. The scientific literature supports the fact that the use of surfactants/adjuvants increases the bioavailability, toxicity, persistence, and bioaccumulation of the active ingredient.

Further, the ADEC cannot provide assurance of compliance and monitoring. We assert that the applicant should not be granted the permit to apply herbicides; however, at a minimum the Point MacKenzie Correctional Farm would be required to obtain a Clean Water Act discharge permit because of the potential damage to water quality and health. The applicant does not fulfill the condition set forth in 18 AAC 90.515(8)(D) which states that the permit must adequately provide a description of the affected area including, "each potentially affected surface water or marine water body within 200 feet of the treatment area."[vi] It is the responsibility of ADEC to closely examine the list provided by the Point MacKenzie Correctional Farm to confirm that every water body is, in fact, catalogued in compliance with 18 AAC 90.515(8)(D). Numerous surface waters (and catalogued anadromous waters)may intersect with the proposed treatment areas. The Point MacKenzie Correctional Farm application does not include maps with sufficient detail to identify all surface water, wetlands, or close marine waters. Again, this information is easily available in online databases (for example, cataloged anadromous streams are at http://www.adfg.alaska.gov/sf/SARR/AWC/index.cfm?ADFG=maps.interactive), and the Point MacKenzie Correctional Farm is negligent in failing to provide detailed information about surface water as required.

Environmental and Human Health Concerns of Listed Active Ingredients

Chlorothalonil
Cymoxanil
Rimsulfuron
Metribuzin
2,4-D, (2,4-dichlorophenol)
Mecoprop-p
Dicamba
Metiram
Azoxystrobin
Glyphosate
diquat dibromide
maleic hydrazide
LI-700

Environmental and Human Health Concerns of Chlorothalonil

Bravo Weather Stick with EPA registration number 50534-188-100, with active ingredient chlorothalonil.

Chlorothalonil is irritating to eyes and skin. People exposed to chlorothalonil can become sensitized to the fungicide and develop severe or persistent reactions.

In laboratory tests, chlorothalonil causes kidney damage, mild anemia, liver damage, embryo loss during pregnancy, oxidative DNA damage (damage to the cell's genetic material), and cancers of the kidney and forestomach. Most of these effects have been observed in several test species. It is classified as a "probable human carcinogen" by the U.S. Environmental Protection Agency.

Chlorothalonil residues are found regularly on celery and green beans. It has been found in groundwater in four states, in the air approximately a mile from chlorothalonil-treated fields, and in Bering Sea fog and seawater samples.

Chlorothalonil is very highly toxic to fish, and concentrations as low as 2 parts per billion can cause gill damage and anemia. It is also toxic to shrimp, frogs, beneficial microorganisms, and earthworms. In plants it causes a variety of effects, including reductions in yield.

Chlorothalonil is contaminated with the carcinogen hexachlorobenzene. Its major breakdown product is about thirty times more acutely toxic than chlorothalonil itself and is more persistent in soil.

Chlorothalonil's mode of action involves its combination with a molecule called glutathione inside fungus cells. As these glutathione-chlorothalonil derivatives form, they tie up all of the cells' available glutathione, leaving enzymes glutathione-dependent unable to function. Several enzymes that are important in cellular respiration, the process by which large molecules are broken down and provide the cell with energy, are glutathione dependent. Their inhibition leads to chlorothalonil's toxic effects. [vii]

Chlorothalonil's toxicity is much greater when exposure occurs through inhalation. The median lethal concentrations (LC50s) of chlorothalonil or chlorothalonil-containing products are between 0.09 milligrams per liter (mg/l) of air and 0.54 mg/l. This places its inhalation toxicity in the highest two toxicity categories defined by the U.S. Environmental Protection Agency (EPA).[viii]

When people are exposed repeatedly, their skin can become sensitized so that they develop allergic reactions to the fungicide. Greenhouse workers,[ix] [x] nursery workers,[xi] [xii] field workers on banana plantations,[xiii] workers in chlorothalonil manufacturing plants,[xiv] painters, [xv] and home gardeners have all developed skin rashes and sensitivities. Probably the most notorious case of chlorothalonil sensitivity involves Lieutenant George Prior, who died a month after playing golf in Arlington, Virginia in 1982. During that month he suffered from a mysterious illness that began with headache and fever, and ended with large blisters on his arms and back, kidney failure, aspiration pneumonia, and extreme pain. Navy pathologists concluded that the disease was caused by the chlorothalonil used on the golf course twice during the week prior to his game.[xvi]

Chlorothalonil Subchronic Toxicity:

Tests with laboratory animals have shown that consumption of a chlorothalonil - contaminated diet over a period of weeks or months has caused a variety of adverse health effects, mostly involving the kidney.[xvii][xviii]

Oxidative damage was studied in one experiment "oxidative damage causes misreplication of DNA that may lead to mutations or cancer." Chlorothalonil caused DNA damage when administered alone. The

authors conclude that excluding the use of Chlorothalonil on food for human consumption would reduce the risk of DNA damage from ingestion of food. [xix]

Two studies of cell cultures have shown that chlorothalonil induces genetic damage: DNA damage occurred in cultures of human white blood cells when chlorothalonil exposures were at low concentrations and of short duration, [xx] and mutations occurred in cultures of mouse lymphoma cells.[xxi]

Although forestomach cancers in mice may not be relevant to humans, certainly the kidney cancers occur in an important human organ. Kidney cancers occur in mice, whose ability to form thiol derivatives of chlorothalonil is comparable to that in humans. [xxii]

Chlorothalonil in water:

Chlorothalonil has been found in groundwater in four states: California, Florida, Massachusetts, and Maine. In addition, it binds strongly to organic acids in water which can result in elevated concentrations in water, like cranberry bogs, that contains these acids.[xxiii] Chlorothalonil's ability to contaminate water long distances from where it is used was startlingly demonstrated in a U.S. Dept. of Agriculture study of the Bering Sea. Chlorothalonil was found in every fog sample collected, and in several of the sea water samples collected. [xxiv]

Chlorothalonil drift in air:

Chlorothalonil is able to travel in the air a significant distance from an application site. A study in North Dakota found chlorothalonil in air sampled almost a mile from farmland where the fungicide was used.[xxv]

Chlorothalonil persistence in soil:

Chlorothalonil's half-life in soil (the length of time required for half of the quantity of chlorothalonil applied to break down or move away from the application site) is identified by both EPA and the World Health Organization as approximately 1 to 2 months. [xxvi], [xxvii]

Chlorothalonil Effects on Fish:

Chlorothalonil is acutely toxic to fish: concentrations of less than 100 parts per billion (ppb) are typically fatal. For example, the median lethal concentration (LC_{50}) for rainbow trout varies between 10 and 76 ppb; [xxviii] for channel catfish LC_{50}s between 52 and 90 ppb have been reported; [xxix] and an LC_{50} of 27 ppb has been measured for the stickleback. Chlorothalonil is "very highly toxic" to all of these species of fish by EPA criteria.

In rainbow trout, concentrations of 2 ppb reduced the diffusive capacity of the gills to about 40 percent of that of unexposed fish. The same concentration reduced hematocrit levels in the trout's blood to 65 percent of normal, resulting in "severe anemia."[xxx]

A test of the effects of exposure to sheepshead minnows during the entire life cycle showed that chlorothalonil concentrations of 6.5 ppb caused decreases in the number of eggs, the hatchability of the eggs, and the survival of fry.[xxxi]

Chlorothalonil tends to concentrate in fish tissues (bioconcentrate) above the levels found in the water in which the fish are living. Measured bioconcentration factors vary from 16 in catfish to 940 in rainbow trout. [xxxii]

Fish kills and respiratory distress in fish at trout farms have been reported after spraying of potato fields, 47 and fish kills have been reported after spraying of cranberry bogs [xxxiii] with chlorothalonil.

Chlorothalonil Effects on Other Aquatic Animals:

By EPA criteria, chlorothalonil is "highly toxic" (with an LC_{50} between 100 and 1000 ppb) to Dungeness crab larvae, pink shrimp, frogs, and water fleas. As observed with fish, sublethal effects occur at much lower concentrations. For example, 7 ppb causes reduced shell growth in oysters, less than 100 ppb causes immobilization of dungeness crab larvae, and 2 ppb causes immobilization of water fleas. [xxxiv] Less than 40 ppb reduced activity of the neurotransmitter acetylcholinesterase in three species of freshwater crustaceans. [xxxv] Frog kills have been reported after chlorothalonil treatment of cranberry bogs. [xxxvi]

Chlorothalonil reduced the viability of spores of Bacillus popillae, the causal agent of milky disease which is used as a biological control for the Japanese beetle. Chlorothalonil also inhibited vegetative cell growth of the milky disease microorganism at concentrations less than those that recommended for use on turf. [xxxvii] Use of chlorothalonil disrupts biological control. [xxxviii]

Eating a chlorothalonil-contaminated diet caused reproductive impairment in bobwhite quail at the middle and high dose used in a feeding study. Effects included reduced survival of quail offspring. A study with mallard ducks measured a reduction in egg production and hatching success at the high dose. [xxxix]

Chlorothalonil is contaminated with a known carcinogen

Chlorothalonil is contaminated during its manufacture with hexachlorobenzene.[xl] Hexachlorobenzene (which was itself used as a pesticide until all uses were cancelled in 1984) is classified by EPA as a probable human carcinogen, like chlorothalonil, because it causes liver, kidney, and thyroid tumors in rats, mice, and hamsters. [xli] It also causes a wide spectrum of other adverse health effects: impaired immune system function, porphyria, kidney damage, effects on the thyroid, tremors, and reduced fertility. Hexachlorobenzene bioaccumulates in both animals and plants and is persistent, with a half-life in soils of between 3 and 6 years. [xlii]

Chlorothalonil Metabolites

The primary metabolite (breakdown product) of chlorothalonil is 4-hydroxy-2,5,6 trichloroisophthalonitrile. It is found in soil, plants, and animals during the breakdown of

chlorothalonil.[xliii] It about 30 times is more acutely toxic than chlorothalonil itself and is more persistent 68 and mobile in soil. [xliv]

Environmental and Human Health Concerns of Cymoxanil

Dupont Curzate 60 DF Fungicide with EPA registration number 352-592, with active ingredient cymoxanil.

European Chemicals Agency is meeting in June to consider classifying cymoxanil for reproductive toxicity and specific organ toxicity[xlv]. Effects have been seen on male reproductive organs in repeated dose studies providing evidence for adverse effects of cymoxanil on sexual function and fertility. Haematology[xlvi] and thymus atrophy effects observed in 90-day dog studies, along with the effects on the eye, may constitute significant toxic effects of cymoxanil after repeated exposure.

Effects have been seen on male reproductive organs in repeated dose studies summarized in the CLH report provide evidence for adverse effects of cymoxanil on sexual function and fertility.

Haematology[xlvii] and thymus atrophy [xlviii] effects have been observed in 90-day dog studies, along with the effects on the eye (retina and lenticular degeneration seen in a two-year rat and one-year dog study [xlix,l]), these effects may constitute significant toxic effects of cymoxanil after repeated exposure.

Classification with the European Chemicals Agency is under consideration for Acute toxicity; Skin sensitization; Specific target organ toxicity – reported exposure; Reproductive toxicity; Aquatic acute; Aquatic chronic. [li]

Environmental and Human Health Concerns of Rimsulfuron

Dupont Matrix Herbicide with EPA registration number 352-556, with active ingredient rimsulfuron.

Rimsulfuron is a Urea compound. Symptoms of Poisoning with Urea Compounds

Many ureas are irritating to eyes, skin and mucous membranes. Symptoms include coughing and shortness of breath, nausea, vomiting, diarrhea, headache, confusion and electrolyte depletion. Protein metabolism disturbances, moderate emphysema, and weight loss with chronic exposure.

Over 50 unpublished studies on the effects of rimsulfuron on dogs and rats have indicated some testicular and other effects.[lii] It has been shown to be toxic to rainbow trout and various ducks. [liii]

Environmental and Human Health Concerns of Metribuzin

Metribuzin 75 DF with EPA registration number 66222-106, with active ingredient metribuzin

Do not let this chemical enter the environment, it is combustible, metribuzin decomposes on burning producing toxic fumes including nitrogen oxides and sulfur oxides. [liv]

A known endocrine disruptor, metribuzin is Included in the class Triazines and triazoles with high exposure concern. [lv]

Environmental and Human Health Concerns of 2,4-D, (2,4-dichlorophenol)

Perfection Weed and Feed 21-7-14, with EPA registration number 2217-532-2935, with active ingredients 2,4-D, mecoprop-p; and dicamba;

2,4-dichlorophenol may represent a risk of potential endocrine disrupting effects in wildlife but further study may be needed. [lvi]

Persistence and Potential for Leaching into Wetlands, Streams, and Groundwaters

EPA includes 2,4-D among the list of compounds that are likely to leach from soil. In oxygenated waters, the half-life of 2,4-D is one week to several weeks. Despite its relatively short half-life in soil and aquatic environments, the chemical has been measured in groundwater supplies of at least 5 states and in Canada.[lvii]

2,4-D has a soil half life of one to two weeks. However, persistence and accumulation of 2,4-D residues from normal use is possible in cold northern environments according to a review of the chemical by the World Health Organization.[lviii] Also, when tracked indoors and not exposed to direct sunlight, 2,4-D can persist in carpets for up to one year after a single application at a concentration of approximately 0.5 µg/g.lix

Toxicity to Non-Target Fish and Wildlife Species and Human Health Effects

The Material Safety Data Sheet (MSDS) provided by the manufacturer of the chemical, Nufarm Americas Inc., states that this product is "toxic to aquatic invertebrates. Drift or runoff may adversely affect aquatic invertebrates and non-target plants." The MSDS warns: "**do not apply directly to water, or to areas where surface water is present**…" The MSDS also states: "Do not enter or allow workers entry into treated areas during the Restricted-Entry interval of 48-hours." The U.S. EPA requires manufacturers of 2,4-D to label the product with a warning about toxicity to fish. Toxic effects to fish occur at minute concentrations and adversely affect reproduction and development.[lx]

2,4-D has been linked to cancer, endocrine disruption, reproductive toxicity, neurotoxicity, kidney and liver damage, and toxicity to fish and wildlife. In a review of nearly 120 studies, the Lymphoma Foundation of America states that the pesticides "more frequently associated with increased lymphoma incidence and/or deaths" include the herbicides 2,4-D and the triazines.[lxi] Studies in Canada and Sweden of members of the general public found a 30-50% higher odds of 2,4-D exposure among people with non-Hodgkin's lymphoma.[lxii],[lxiii]

2,4-D causes significant suppression of thyroid function.[lxiv] Thyroid function plays a critical role in the development of the brain and metabolism. Slight thyroid suppression adversely affects neurological development in developing children, resulting in lasting effects on learning and behavior. 2,4-D is associated with reproductive disorders and abnormalities.[lxv] The chemical also interferes with the neurotransmitters dopamine and serotonin. In young organisms, esposure to 2,4-D results in delays in brain development and abnormal behavior patterns.[lxvi] This herbicide specifically impairs normal

deposition of myelin in the developing brain.[lxvii] The neurotoxic and anti-thyroid effects of 2,4-D make it highly likely that developing children will be more susceptible to long-term adverse health effects from exposure to this chemical. Recent research has revealed that 2,4-D accumulates and is transferred to the developing baby in breast milk.[lxviii]

Dioxin, a highly toxic compound known as carcinogenic and mutagenic at minute levels, contaminates 2,4-D as byproducts during the manufacturing process.[lxix]

Symptoms of 2,4-D poisoning in exposed people include irritation and inflammation of eyes and skin, hives, nausea, vomiting, throat irritation, headache, dizziness, coughing, and difficulty breathing.

In laboratory animals, human cells, and exposed people 2,4-D caused genetic damage. Scientists have also demonstrated that 2,4-D affects hormones in exposed people and laboratory animals. Three recent laboratory studies indicate that 2,4-D has the ability to reduce the effectiveness of the immune system.

2,4-D (and the entire family of phenoxy herbicides) is classified as possibly carcinogenic by the International Agency for Research on Cancer. Studies of exposed farmers support this classification.

New studies indicate that 2,4-D reduces fertility in several ways. 2,4-D exposure is associated with low sperm counts.

2,4-D also damaged sperm and male sex organs in laboratory studies. When low doses of a commercial 2,4-D herbicide were fed to pregnant laboratory animals, average litter size was reduced by about 20 percent.

According to the most recent data collected by the U.S. Environmental Protection Agency, some 2,4-D is contaminated with 2,3,7,8-TCDD, a potent dioxin.

Monitoring by the U.S. Geological Survey showed that 2,4-D is frequently found in rivers and streams. It is also often measured in air samples.

2,4-D use on lawns is linked with an increased risk of cancer in dogs.

2,4-D causes genetic damage in plants in amounts too small to cause visible damage to the plants.

Environmental and Human Health Concerns of Mecoprop-p

Perfection Weed and Feed 21-7-14, with EPA registration number 2217-532-2935, with active ingredients 2,4-D, mecoprop-p; and dicamba

Symptoms of exposure to mecoprop include burning skin and eyes, nausea, dizziness, and headaches. [lxx], [lxxi]

In laboratory tests, mecoprop has inhibited the synthesis of DNA (the molecules that contain genetic information), interfered with blood clotting, and inhibited the production of important components of the immune system. The National Institute for Occupational Safety and Health labels mecoprop as a "mutagen" [lxxii] because it inhibited the synthesis of DNA in a laboratory study of mice. DNA is the

"molecular basis of heredity," [lxxiii] the molecules that contain genetic information. A single dose of mecoprop reduced DNA production by 60 percent.[lxxiv]

In addition, tests conducted for a mecoprop manufacturer as part of the process of registering it as a pesticide, showed other types of genetic damage. These included chromosome damage in bone marrow cells in hamsters and human blood cells, as well as a kind of genetic damage called sister chromatid exchanges in hamster bone marrow cells.[lxxv] (Sister chromatid exchanges are exchanges of DNA within a chromosome as it duplicates. [lxxvi])

In 1994, EPA listed mecoprop as a toxic chemical under the Emergency Planning and Community Right-to Know Act because of its toxicity to the liver and kidneys.[lxxvii]

Laboratory tests using a commercial mecoprop-containing herbicide showed that the fertility of mice who drank water contaminated with low levels of the herbicide was less than that of mice who drank uncontaminated water. Even the lowest dose level tested in this experiment reduced litter size. [lxxviii] In a three month feeding study with rats, mecoprop doses of 9 milligrams per kilogram (mg/kg) of body weight per day caused a change in liver and kidney weights. [lxxix]

A regional study in Canada found that exposure to mecoprop was associated with an increased risk of the cancer non-Hodgkin's lymphoma. They found that "the risk of NHL was statistically significantly increased" [lxxx] by exposure to three phenoxy herbicides: mecoprop, 2,4-D, and dicamba. The odds ratio (a statistical measure of the increased cancer risk) was over 25 percent larger for mecoprop than for the other two phenoxy herbicides.

Mecoprop is frequently found in urban streams. One study (done in King County, Washington) found mecoprop in every urban stream sample analyzed. [lxxxi]

Environmental and Human Health Concerns of Dicamba

Perfection Weed and Feed 21-7-14, with EPA registration number 2217-532-2935, with active ingredients 2,4-D, mecoprop-p; and dicamba;

A regional study in Canada found that exposure to dicamba was associated with an increased risk of the cancer non-Hodgkin's lymphoma. They found that "the risk of NHL was statistically significantly increased" [lxxxii] by exposure to three phenoxy herbicides: mecoprop, 2,4-D, and dicamba.

Acute exposure to dicamba causes skin irritation and some skin sensitization in laboratory tests, as well as severe eye irritation. The eye damage can be irreversible. [lxxxiii]

Dicamba also causes other acute effects. Congested lungs, hemorrhages, poor digestion, inflamed kidneys, and engorged livers occured in sheep fed doses of 500 mg/kg. [lxxxiv]

Acute effects can occur in exposed humans. Symptoms in worker poisonings reported to EPA included muscle cramps, shortness of breath, nausea, vomiting, skin rashes, loss of voice, and swollen glands. [lxxxv]

Reproductive Effects: Exposure of mallard eggs to Banvel caused reduced, stunted growth in the mallard embryos as well as eye malformations. [lxxxvi]

Concerns about reproductive effects are heightened by a manufacturing contaminant, 2,7-dichlorodibenzo-p-dioxin. In pregnant rats, this contaminant causes abnormalities, suppression of tissue growth, and lesions in fetal hearts. [lxxxvii]

Mutagenicity A 1990 study showed that injections of dicamba significantly increased the "unwinding rate" (single strand breaks) of the genetic material (DNA; deoxyribonucleic acid) in rat livers. The same study also looked at effects on human blood cell cultures and found that exposure to dicamba caused an increase in unscheduled DNA synthesis as well as a slight increase in sister chromatid exchanges (exchange of genetic material between chromosome pairs). [lxxxviii]

Two potentially carcinogenic contaminants of dicamba increase concerns about cancer.

The contaminant 2,7-dichlorodibenzo-p-dioxin 29 is not as potent a carcinogen as its notorious chemical cousin 2,3,7,8-TCDD, but it has caused leukemia and lymphoma, liver cancer, and cancer of the circulatory system in a 1979 study of male mice conducted by the National Toxicology Program. (No significant increases in cancer were found in female mice or rats of either sex.) [lxxxix]

Dicamba's dimethylamine salt can be contaminated with dimethylnitrosamine, small amounts of which cause cancer in laboratory animals. [xc]

Dicamba is "relatively water-soluble" and "mobile in soils." [xci] This means that it is likely to contaminate both ground and surface water. In a study that compared soil mobility of 40 pesticides, dicamba was one of three with the highest mobility;[xcii] one study found that the concentration of dicamba required to kill half of a test population (called the LC_{50}) of bluegill was 600 parts per million (ppm). In the same study, researchers determined that if the herbicide was absorbed onto vermicullite, it was 30 times more toxic. [xciii]

In another study, no effects on yearling coho salmon were observed at concentrations of dicamba up to 100 ppm. However, yearling coho were killed by much smaller doses (0.25 ppm) during a seawater challenge test which simulates their migration from rivers to the ocean. [xciv]

In addition, acute toxicity varies widely among fish species. For example, rainbow trout are killed by concentrations less than a tenth as great as those that kill mosquito fish. [xcv]

The toxicity to fish of dicamba-containing herbicides may be increased by the products used with them. For example, in 1992, forty fish were killed in Douglas County, Oregon, by the adjuvant added to Weedmaster, an herbicide containing dicamba and 2,4-D. [xcvi]

Environmental and Human Health Concerns of Metiram

Polyram 80 DF, with EPA registration number 7969-105-34704, with active ingredient metiram;

Metiram is listed as a substance with high evidence on endocrine disruptive effects from exposure. [xcvii]

Environmental and Human Health Concerns of Azoxystrobin

Quadris Flowable Fungicide, with EPA registration number 100-1098, with active ingredient azoxystrobin;

Azoxystrobin is highly toxic to aquatic fish. Azoxystrobin is of low acute and chronic toxicity to humans, birds, mammals, and bees but is highly toxic to freshwater fish, freshwater invertebrates, and estuarine/marine fish, and very highly toxic to estuarine/marine invertebrates. [xcviii]

Environmental and Human Health Concerns of Glyphosate

Round-up Pro with EPA Registration Number 524-475, with active ingredient glyphosate;

Glyphosate has been linked to certain cancers, endocrine, reproductive and developmental health effects in humans.

The U.S. Environmental Protection Agency (EPA) is currently conducting a Registration Review for glyphosate, a process to be completed in 2015. Prior to this, the EPA had not conducted a review of glyphosate since 1993. Since that time, many articles in the peer-reviewed literature have presented new evidence concerning the harm to health and environment associated with glyphosate and glyphosate-based formulations. The Glyphosate Final Work Plan (Registration Review Case No. 0178) identifies data gaps of concern that will be addressed in the ecological risk assessment including:[xcix]

- AMPA, the primary degradate of glyphosate, has been evaluated for ecological risk only to a few species. The EPA anticipates considering AMPA more thoroughly in the ecological risk assessment.
- Glyphosate needs to be reviewed for acute avian oral toxicity, acute toxicity for estuarine/marine mollusks, invertebrates, and fish species.
- Human health risks of glyphosate were most recently evaluated in 2006, and in accordance with new requirements under 40 CFR part 158, additional evaluation of the acute and subchronic neurotoxicity, and immunotoxicity to humans are needed.

These data gaps indicate potential for serious harm to environmental and human health. Widespread use of a chemical product years before the EPA risk assessment is complete is irresponsible and dangerously negligent of potential harm to the health of those living along the railroad corridor.

In addition, the EPA has received a request (Federal Register Feb 22, 2012) from a manufacturer to cancel the registrations of two glyphosate products, Texcan Glyphosate Technical (EPA Reg. No. 0868068-00001) and Texcan 62% Glyphosate MUP (EPA Reg. No. 0868068-00002). Absent substantive comments before the deadline of March 23, 2012, the EPA will cancel the registrations for these pesticides, effective March 2013. Other glyphosate-based herbicides may still be available to the Alaska Railroad, but this cancellation indicates that even a large manufacturer doubts the product's future marketability.[c]

Glyphosate is the active ingredient in Aquamaster. Glyphosate is persistent in soils after application, especially in northern regions. In a Finnish study, the measured half-life of glyphosate was 249 days.[ci] In Ontario, Canada, glyphosate had a half-life in forest soils of 24 days with detectable residues persisting

for 335 days.[cii] On 3 British Columbia forestry sites, glyphosate persisted 360 days.[ciii] In a Swedish study, glyphosate persisted from one to three years on eleven forestry sites.[civ] Another peer-reviewed study reported that glyphosate has a half-life of 3 days to 25 weeks in soil and 1 day to 25 weeks in water with a pH of 7.[cv]

The herbicide has the potential of eliminating a wide variety of plants including desirable as well as "undesirable" vegetation, grasses and many broad leaf species. The main breakdown products of glyphosate are aminomethylphosphonic acid (AMPA).[cvi] Another reported degradation product of glyphosate is formaldehyde,[cvii] a known carcinogen. In a study of glyphosate degradation in Willapa Bay in Washington State, glyphosate concentrations in the estuarine mudflats took 119 days to decline to 72%, while AMPA did not degrade during that period.[cviii] Other studies show that the half-life for glyphosate in water ranges from 35-65 days. "In British Columbia, following application of glyphosate using a no-spray buffer and very low concentrations of glyphosate, the breakdown product AMPA was sometimes observed in water and sediment of streams after the first heavy rain following application.[cix] Another study of agricultural watersheds shows similar results, with the highest concentrations in runoff one to ten days, and detection up to 4 months after application.[cx] Higher peak concentrations were observed in water following heavy rain events up to three weeks after application and "sediment peaks were observed later and persisted in stream sediments for more than one year."[cxi] A fact sheet about glyphosate from the Oregon State University Environmental Toxicology and Chemistry Program does not recommend berry or mushroom consumption from newly-treated areas.

Herbicides cause "trophic cascades" including direct and indirect harmful effects on many species, including aquatic invertebrates that are food sources for salmonids and other fish.cxii In general, herbicides and other pesticides have long residence times in soils and waters at northern latitudes. Product formulations of active ingredients and proprietary, undisclosed additives such as solvents and surfactants can cause enhanced adverse effects to the environment and human health. Interactive and low-level effects at concentrations below EPA allowable levels have been found to cause profound impacts on neurological, endocrine, immune, and developmental processes including the development and function of the brain, as well as reproductive health.

Glyphosate and its primary degradation product aminomethylphosphonic acid (AMPA) were classified among the first contaminants in rivers.[cxiii] Glyphosate, the active ingredient in Aquamaster, is toxic to a variety of aquatic insects and tadpoles. Species richness of aquatic biota (copepods, *Daphnia*, snails, arthropods, amphibians) was reduced 22% in this 2005 study. Toad tadpole survival was reduced from 97 % to 0%.[cxiv] In a study published in 2000, Giesey and other researchers found toxicity of glyphosate can be quite high to some invertebrates.[cxv] Glyphosate also causes serious sub lethal effects in fish at low concentrations, including erratic swimming, gill damage and liver structure changes.[cxvicxvii] Fry and fingerlings are more vulnerable to the toxic effects of glyphosate than adult fish, with harmful toxicological effects on developing rainbow trout at 2-3 ppm.[cxviii]

In comments from the U.S. Fish and Wildlife Service on the proposed aerial application of glyphosate for forestry purposes in SE Alaska (letter to ADEC dated October 28, 2005), agency biologists stated: "We are concerned that these application restrictions for Accord [active ingredient is glyphosate] will not be

adequate to protect aquatic resources, based on recent glyphosate toxicity data. Accord [active ingredient glyphosate] does not appear to be an acceptable product due to its potential for aquatic toxicity." The Agridex surfactant has similar chemical properties in enhancing the toxicity of the herbicide glyphosate because the petroleum-based compound "carries" the herbicide more effectively into biological systems and across cell membranes.

Toxicity of glyphosate is affected by such environmental factors as water hardness, temperature, and pH. Toxicity increases at lower pH levels and higher temperatures.[cxix] Surfactants may exhibit increased toxicity in alkaline waters.[cxx] In addition, glyphosate has an antagonistic effect on the toxic action of a surfactant.[cxxi] High pH (7.5) "increased the toxic effects of the herbicide" [glyphosate] on survival, reproduction, and development time in zooplankton and amphibian species (frog species *Rana pipiens* and zooplankton species *Simocephalus vetulus*).[cxxii]

Glyphosate sub-lethal effects in fish and wildlife have not been well-studied, although available science indicates cause for concern. Chronic exposure to glyphosate for 14 days resulted in histopathological changes in gill and liver structure, as well as adverse effects to liver, heart, kidney and serum enzyme activity. Threshold gill and liver histopathological responses were observed at concentrations equal to 0.8% (5 ppm) and 1.6% (10 ppm), respectively, of the 96 LC50 for that species (620 ppm). Researchers surmised that the gill histopathological response was reparable if the fish were relocated to clean water, however the liver fibrosis was considered indicative of serious liver damage. Statistically significant changes in enzyme activity were observed at 0.4% of the 96 hr. LC50, the lowest exposure concentration, in liver (alkaline phosphatase, P less than 0.01; and glutamic-pyruvic transaminase, P less than 0.05) and kidneys (glutamic-oxaloacetic transaminase, P less than 0.05 and glutamic-pyruvic transaminase, P less than 0.05. Responses to chemical exposure vary by species, but equivalent exposure concentrations (0.4%, 0.8%, and 1.6% of the 96 hour LC50) for salmonids would be 4.4 ppm, 8.8 ppm, and 17.6 ppm.[cxxiii], [cxxiv] Rainbow trout fry were the most sensitive life stage followed by emergent fry.[cxxv] After treatment with Roundup, aquatic macro invertebrate density declined by 42% for a 1.5 year period.[cxxvi]

Studies show adverse effects on the following categories of beneficial insects: pollinators, soil aerators, predators, and soil producers. Glyphosate reduces the growth and survival of earthworms.[cxxvii] It is acutely toxic (at concentrations ranging from 2-55 ppm) and causes sub-lethal effects on fish.[cxxviii] Roundup (with associated proprietary surfactants and other additives) is 20-70 times more toxic than glyphosate alone.[cxxix] Toxicity of the surfactant increases with water temperature.[cxxx] Glyphosate causes an increase in water temperature for years following application through the destruction of shading vegetation—this increase is particularly dangerous to fish such as juvenile salmon, which depend on cooler water temperatures for survival.[cxxxi] In Nova Scotia, studies of treated forests revealed that songbird densities (white-throated sparrows and common yellowthroat) were reduced for two years after the glyphosate application.[cxxxii] A three-year study of treated forests in Maine demonstrated a decline in the abundance of songbirds.[cxxxiii] Declines in small mammal populations and adverse effects on moose, elk, and deer browse have also been documented.[cxxxiv] Glyphosate can adversely affect the health of soils and nutrient cycling by: 1) inhibiting nitrogen fixation[cxxxv], 2) causing a decline of beneficial mycorhizal fungi[cxxxvi], and 3) increasing the disease susceptibility of plants.[cxxxvii]

Contrary to claims of safety by the applicant, recent research demonstrates serious harmful effects to human health associated with exposures to the herbicides. A 2002 study by Garry, et.al. found that glyphosate showed a significant correlation with excess adverse birth and neuro-developmental effects. The authors also note: "Regarding the herbicide glyphosate, our present study shows a tentative association between ADD/ADHD and use of this herbicide."[cxxxviii] A 2001 study concluded: "Preconception exposure to the pesticide active ingredients glyphosate, atrazine, carbaryl, and 2,4-D was associated with a 20-40% relative increase in risk…The herbicide glyphosate was associated with increased risks of late abortion, regardless of when exposure occurred."[cxxxix] "The genotoxicity of glyphosate has been positive in *in vitro* cultures of bovine and human lymphocytes and weakly mutagenic in a *Salmonella* assay."[cxl] One study observed the onset of parkinsonian syndrome following an accidental exposure to glyphosate. "A 54-year old man accidentally sprayed himself with the chemical agent glyphosate, an herbicide derived from the amino acid glycine. He developed disseminated skin lesions 6 hours after the accident. One month later, he developed symmetrical parkinsonian syndrome."[cxli]

Exposure to glyphosate is also associated with elevated risk of a rare form of non-Hodgkins's lymphoma (NHL), hairy cell leukemia: "The more recent study described two case-control studies, one on NHL alone and one specifically on hairy-cell leukemia, a rare form of NHL, with respect to pesticide exposure (with many different pesticides and exposure levels tested). A pooled analysis (done in order to increase numbers) revealed elevated ORs with statistical significance for herbicides in general, phenoxyacetic acids, glyphosate, and MCPA. Also, there were dose-response effects in these pesticide groups, most with statistical significance."[cxlii], [cxliii] A 2003 study confirmed the association of glyphosate exposure with increased incidence of non-Hodgkin's lymphoma.[cxliv]

Many currently used pesticides have the capacity to disrupt reproductive function in animals. Walsh and colleagues conducted a study concerning the underlying cause of reproductive endocrine disorders because "the possibility these compounds can affect the reproductive health of humans and wildlife in their natural habitats is of great concern. Little information is available regarding the effects of pesticides…on endocrine system function, despite their widespread use." The authors conclude: "Roundup [active ingredient glyphosate] disrupted steroidogenesis in Leydig cells through a post-transcriptional reduction in StAR (Steroidogenic Acute Regulatory) protein expression. Not only does StAR play an important role in steroid (hormone) production in gonads, but it is also indispensable for steroidogenesis in the adrenal glands. As a result, a disruption in StAR protein expression may impair more than just fertility. The adrenal glands synthesize glucocorticoids and mineralocorticoids, and a reduction in StAR expression in the adrenal glands may affect carbohydrate metabolism, immune system function, and balance. Because many toxicants that reduce StAR expression and steroidogenesis in the adrenal gland, a disruption in StAR protein expression may underlie many of the toxic effects of environmental pollutants."[cxlv]

Finnish researchers showed that Roundup's active ingredient, glyphosate, decreases the defenses of enzymes of the liver and intestines.[cxlvi] Roundup, as a mixture of all its ingredients, has been shown to shut down a powerful antioxidant in the liver that detoxifies harmful compounds so they can be excreted through bile. Glyphosate also alters gene expression and inhibits necessary steroid production

by disrupting a particular protein expression. In 2002, another study showed that glyphosate can also affect early cell division processes in embryos."[cxlvii]

In a systematic review of the peer-reviewed scientific literature concerning health effects of pesticides, a team of physicians from the Ontario College of Family Physicians concluded: "The literature does not support the concept that some pesticides are safer than others; it simply points to different health effects with different latency periods for the different classes…Some more surprising positive associations were found for pesticides that are considered less toxic in acute poisoning settings…The herbicides glyphosate and glufosinate had associations with congenital malformations [birth defects]. Parental preconception exposure to glyphosate was associated with late abortion."[cxlviii] A growing body of evidence implicates glyphosate and its formulations with health problems thus far ignored by the applicant and ADEC.

Recent papers published in peer-reviewed scientific journals provide additional evidence concerning the harmful environmental and health effects of glyphosate and its formulations:

- "Glyphosate-based herbicides belong to the first herbicides used worldwide, and are major pollutants of rivers and [other] surface waters….it s now well-documented that mixtures formulated with glyphosate and adjuvants are themselves not environmentally safe, in particular for aquatic life. They can even enhance heavy metal toxicity."[cxlix]
- Gasnier et al (2009) state: "In conclusion, according to these data and the literature, glyphosate-based herbicides provoke DNA damage, endocrine disruption *in vitro*, and CMR (carcinogen, mutagen, and reprotoxic) effects *in vivo*. The direct glyphosate action is most probably amplified by vesicles formed by adjuvants or detergents that allow cell penetration, stability, and probably change its bioavailability and thus metabolism." Results from the study also clearly show that the DNA of the human hepatoma cell line is damaged by a glyphosate-based herbicide.[cl]
- Glyphosate formulations affect human placental cell viability at levels significantly below prescribed application concentrations and sexual steroid biosynthesis at lower non-toxic doses. This effect was amplified with adjuvants, the so-called inert ingredients of herbicide formulations kept confidential by the chemical companies. Glyphosate is toxic to human placental cells at lower concentrations than those prescribed by label instructions. In a 2005 article published in *Environmental Health Perspective*[cli], the authors conclude that glyphosate acts as a disruptor of mammalian cytochrome P450 aromatase activity from concentrations 100 times lower than the recommended use; "this is noticeable on human placental cells after only 18 hours, and it can also affect aromatase gene expression. It also partially disrupts the ubiquitous reductase activity but at higher concentrations. Its effects are allowed and amplified by at least 0.02% of the adjuvants present in Roundup, known to facilitate cell penetration [Note: the surfactant in Agridex has similar chemical properties], and this should be carefully taken into account in pesticide evaluation. The dilution of glyphosate in Roundup formulation may multiply its endocrine effect. Roundup [and other formulations] may thus be considered as a potential endocrine disruptor. Moreover, at higher doses still below the classical agricultural dilutions, its toxicity on placental cells could induce some reproduction problems." Effects of the herbicide on aromatase are particularly significant because aromatase is the enzyme responsible for the irreversible androgen to estrogen conversion—if this system is disrupted, there are critical effects on sexual and several cell differentiations, reproduction, pregnancy, liver metabolism, bone development, behavior, and hormone-dependent cancers such as breast or

prostate cancer. “here we show that glyphosate is toxic to human placental JEG3 cells within 18 hours with concentrations lower than those found with agricultural use, and this effect increases with concentration and tie or in the presence of Roundup adjuvants.” The authors also conclude “endocrine and toxic effects of Roundup, not just glyphosate, can be observed in mammals.”

- Male offspring of female rats exposed to glyphosate-based Roundup during the perinatal period exhibited altered reproductive development, including excess androgen production and early puberty. The study’s authors concluded that maternal exposure to glyphosate reduced the aromatase activity in the offspring, causing detrimental changes in reproductive behavior, as well as permanent endocrine and histological problems[cliii]. The authors of this study note: “Even though glyphosate is ***considered*** a low-toxic herbicide, recent studies have revealed toxic effects ***resulting from even low-dose commercial formulations*** [emphasis added].” In their review of the literature, the scientists also note that “A reduction in aromatase activity was observed in placental and embryonic human cells treated with low concentrations of a commercial formulation of glyphosate, which disrupted the conversion of steroids to estradiol. Furthermore, a recent study showed a reduction in the expression of androgen and estrogen receptors, the expression and activity of aromatase, and genotoxicity in human liver HepG2 cells exposed to low concentrations of commercial glyphosate. Previous results from our laboratory demonstrated a significant reduction in serum testosterone concentrations and changes in the testicular morphology of male Wistar rats treated with Roundup (active ingredient glyphosate) during the prepubertal period, and these effects with e***ven the lowest dose used in the experiment*** (5 mg/kg). The authors concluded: “Thus the herbicide glyphosate may be characterized as a potential endocrine chemical disruptor. Endocrine disruptors were defined by Kavlock et al. (1996) as exogenous agents that interfere with the production, release, transport, metabolism, binding, action, or elimination of natural hormones responsible for the maintenance of homeostasis and the regulation of developmental processes.”
- In embryonic cells as well as in normal human placental and equine testis, there was glyphosate induced endocrine disruption through aromatase inhibition at low dose levels. Embryonic cells were even more sensitive. Adjuvants enhanced the cumulative effects with increased bioaccumulation or time-delayed effect. Adjuvants enhance absorption through plasmatic membranes.
- Glyphosate formulations cause total cell death within 24 hours through inhibition of mitochondrial succinate dehydrogenase activity and necrosis by release of cytosolic adenylate kinase measuring membrane damage. They also induce apoptosis via activation of enzymatic caspases 3/7 activity. The authors confirmed this by “characteristic DNA fragmentation, nuclear shrinkage (pyknosis), and nuclear fragmentation (karyorrhexis)”…These effects were observed at dilution levels far below label recommendations.[cliii]
- Three recent case-control studies suggest “an association between reported glyphosate use and the risk of non-Hodgkin lymphoma (NHL).”[cliv]
- “Similarly, use of the phosphonamino herbicides (glyphosate, Roundup) was overrepresented in the adverse birth and developmental effect group. Forty-three percent of the children (6 of 14) who had parent-reported ADD/ADHD used phosphonamino herbicides (OR = 3.6; CI, 1.35–9.65). No other commonly used pesticide compared by major organ and/or functional system was uniquely associated with specific adverse birth or developmental effects.”[clv]
- There is evidence that glyphosate disrupts development by disrupting DNA transcription. A study of the effects of glyphosate herbicides on sea urchins found that specific DNA transcription occurred later and to a lesser extent than transcription in control urchins. Further studies determined it was glyphosate and not other ingredient that causes this effect.[clvi]

These studies provide substantive evidence that glyphosate is likely an endocrine disruptor and supports several other studies that have shown toxic effects of both glyphosate and its formulations. Product formulations include added "inert" ingredients and adjuvant chemicals that make the product more toxic and help it to perform better (by causing it to stick, spread, reduce evaporation, or other desired function).

Environmental and Human Health Concerns of Diquat Dibromide

Reglone Dessicant with EPA registration number 100-1-61, with active ingredient diquat dibromide;

Diquat dibromide is very persistent in the environment, deadly and is used for suicide.[clvii]

Environmental and Human Health Concerns of Maleic Hydrazide

Royal MH-30 Xtra with EPA registration number 400-452, with active ingredient maleic hydrazide.

Recently, numerous experiments performed with various plant species have shown that maleic hydrazide (MH) acts as an inhibitor of the synthesis of nucleic acids and proteins. Similar results have been obtained with animal tumour cells. The chromosome-breaking effect of MH on plant chromosomes resembles very closely the chromosome-breaking properties of alkylating agents and other mutagenic compounds such as mitomycin C. MH-induced chromosomal aberrations have also been recorded in grasshoppers, fish and mice, although tests with some mammalian cell lines gave negative results. Among higher plants, selective sensitivity to the toxic effects of MH is well proved. This phenomenon seems to be due to the differential ability of various plant species to detoxicate the chemical. Plants can break down MH into several products, one of which, hydrazine, is a well-known mutagen and carcinogen. MH does not seem to be toxic to bacteria and fungi. The compound is degraded by soil microflora and hence can be utilized as a source of nitrogen nutrition. MH proved to be of low toxicity to mammals, but in some instances it decreased the fertility of rats. The reported carcinogenic effects of MH in mice and rats raise the question of its risks to man.[clviii]

Environmental and Human Health Concerns of LI-700

Some of these products require the use of a nonionic surfactant. The Point McKenzie Correctional Farm proposes to use the product **LI-700**, a surfactant.

LI-700 has been identified in a study of toxicity of surfactants to juvenile rainbow trout. [clix]

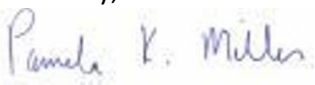
Critical Habitat for the Endangered Cook Inlet Beluga Whale

The Cook Inlet beluga whale has been listed as an endangered species under the Endangered Species Act (ESA) by the National Oceanic and Atmospheric Administration. Applications of pesticides pose direct and indirect threats to the development, survival, habitats, and food resources of the endangered beluga whale that must be considered by the ADEC, as these applications would occur within the Cook Inlet watershed. The listing of the Cook Inlet beluga whale means any federal agency that funds, authorizes, or carries out new projects or activities that may affect the whales in the area must first consult with NOAA's Fisheries Service to determine the potential effects on the whales. A federal action

must not jeopardize the continued existence of a listed species. The Point MacKenzie Correctional Farm receives federal funding and therefore must comply with consultation and other provisions of the ESA.

We assert that the Point MacKenzie Correctional Farm does not provide justification for use of the pesticides in the permit application. ADEC must deny all of these pesticides in this request in order to meet its legal obligation to protect vulnerable water bodies, aquatic habitat, subsistence resources, endangered beluga whales and other wildlife, and human health.

Sincerely,



Pamela Miller
Executive Director
Alaska Community Action on Toxics

cc Commissioner Larry Hartig, ADEC
Kristin Ryan, Director, Environmental Health, ADEC

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