



**UNITED STATES DEPARTMENT OF COMMERCE**  
**National Oceanic and Atmospheric Administration**

*National Marine Fisheries Service*

*P.O. Box 21668*

*Juneau, Alaska 99802-1668*

November 1, 2004

Nancy Sonafrank  
Water Quality Standards Section Manager  
Alaska Department of Environmental Conservation  
610 University Drive  
Fairbanks, Alaska 99709

Dear Ms. Sonafrank:

The National Marine Fisheries Service (NMFS) has reviewed proposed revisions to the Mixing Zone sections of the Alaska Water Quality Standards. The Alaska Department of Environmental Conservation (DEC) is proposing several changes to the Mixing Zones sections of the Water Quality Standards in 18 AAC 70.240 through 70.270 to:

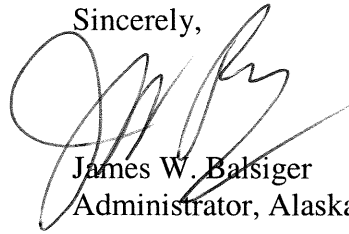
- Improve clarity and reduce redundancy;
- Allow mixing zones in certain fish spawning areas where there will be no adverse effect on the capability of an area to support fish spawning, rearing, or incubation (the proposed regulations provide for consideration of measures that would offset the potential adverse effects of mixing zones on aquatic resource); and
- Simplify some technical provisions including those dealing with risk assessment, flow calculations, and mixing zone models.

NMFS supports efforts by DEC to improve clarity and reduce redundancy in regulation, and many of the proposed amendments to the Alaska Water Quality Standards appear to yield no substantive change. However, NMFS opposes the proposed regulation to allow for mixing zones in certain fish spawning areas. This proposed revision is a significant change from the existing prohibition on mixing zones in spawning and rearing habitat of resident and anadromous fish. Water quality standards are premised on levels that do not adversely affect fish and/or their habitat, and any mixing zone (i.e., an allowable variance from water quality standards) could adversely affect fish and their habitat. The proposed change allows for the interpretation of data that are weak (based on acute toxicity information) and the interpretation or extrapolation of that data may be flawed. Applying acute toxicity data to a situation where chronic impacts occur (as in a mixing zone) and fish embryos and larvae are present may adversely affect living marine resources including Essential Fish Habitat (EFH). Our specific comments on the amendment to the Water Quality Standards at 18 AAC 70 are enclosed and include comments from our research scientists at the Auke Bay Laboratory.



Should you have any questions regarding these comments please contact Ms. Jeanne Hanson at (907) 271-3029.

Sincerely,



James W. Balsiger  
Administrator, Alaska Region

Enclosure

cc: NMFS HCD – Susan Walker  
NMFS Auke Bay Lab – Jeep Rice  
Records – Lori Durall

USFWS, ADNR-OHMP, ADEC – Anchorage

Lisa McGuire  
U.S. EPA, Region 10  
Water Quality Standards and Planning Unit  
1200 Sixth Avenue  
Seattle, Washington 98101

**National Marine Fisheries Service (NMFS)**

**Comments on the Draft Revision of Mixing Zone Regulations, 18 AAC 70.240-270**

NMFS opposes the proposal to allow mixing zones in areas of spawning and rearing habitat. Scientific research, including research conducted by NMFS, increasingly indicates that these habitats are critical to the survival of numerous fish species, including prey species, and that the early life stages that utilize these habitats are extremely sensitive to chronic pollution. Chronic pollution over time in spawning and rearing habitats will place those populations at risk over the long term, leading to a diminishing of the stock and possibly to localized extinction.

Allowing mixing zones for pollutants in water generally is based on two assumptions: (1) exposure to fish will be transient, and (2) short exposures to toxicant levels will be below acute toxicity levels. These assumptions might be met with adult transient fish moving through an area, but are not likely to be met if there are resident fish, a rearing area, and most importantly, a spawning area. Developing salmon eggs and larvae will be in spawning gravels for months, and any exposure they receive from elevated levels in a mixing zone would be chronic. Predicting impacts of chronic toxicity using acute toxicity information is fundamentally flawed because the mechanisms of toxicity are intrinsically different between acute toxicity (narcosis) and chronic toxicity. In other words, simply applying a dilution safety factor of 100 or some other value does not have relevance when calculating the potential chronic impacts to embryos and developing larvae.

The preponderance of recent scientific evidence to emerge from toxicological studies suggests that concentrations as low as one part per billion of PAH (polynuclear hydrocarbons) can adversely impact developing fish embryos. As our understanding of toxic effects of contaminants increases, the evidence suggests that the goal of most fisheries pollution mitigation should be to avoid exposing spawning areas to environmentally persistent contaminants, especially in situations such as mixing zones where fish eggs and larvae are exposed chronically.

Streams and estuaries sustain the vulnerable early developmental life stages of many fish species. Salmon eggs, larvae, and juveniles use both stream and estuary for much of the first year of life, and the juveniles of many marine species use the estuaries for nursery grounds. The principal threat to these species is not from acutely toxic concentrations that can result in mortality, but in the more subtle effects of low-level contaminant exposures to these sensitive life stages. Incubating eggs are very sensitive to long-term exposures to those pollutants such as hydrocarbons, pesticides, and PCBs that sequester onto lipid molecules in the developing embryos. These environmentally persistent organic pollutants remain in the eggs as they develop. The result is that fewer juveniles survive, so that recruitment from the early life stages is reduced and adult populations may not be replaced at sustainable levels.

Most of the toxicity models currently in use for fish assume a disruption of the nervous system leading to narcosis and eventual death. Water quality standards for mixing zones are predicated on this model of how toxic chemicals work. Recent research indicates that pollutants such as polycyclic aromatic hydrocarbons (PAHs) are unlikely to act as narcotic agents in early life stages of fish that have been chronically exposed as embryos. Instead embryonic exposures result in edema of the yolk sac,

hemorrhaging, disruption of cardiac function, binding of aryl hydrocarbon receptors, enzyme induction, mutation and heritable changes in progeny, craniofacial and spinal deformities, neuronal cell death, anemia, reduced growth, and impaired swimming (White et al. 1999, Barron et al. 2003, Billiard et al. 1999, 2002, Brinkworth et al. 2003, Marty et al. 1997, Incardona et al. in press). Exposure to sunlight results in a 48 fold increase in toxicity of PAH to fish larvae (Barron et al. 2002) and resulted in 2 ppb becoming toxic to the calanoid copepods that form much of the diet of developing juvenile fishes (Duesterloh et al. 2002).

Early life stages are more vulnerable to both the chemical effects of toxicants, and to the ecological consequences. In a developing embryo, cellular damage may be replicated many times as tissues develop and enlarge, resulting in a tissue that is severely compromised in function at later life stages. The embryo may develop normally for some time, as it may not need this tissue until a later life stage, thus the consequences of damage may not be apparent immediately. This concept was demonstrated eloquently when salmon embryos were exposed in controlled laboratory conditions to low levels of crude oil, and then tagged and released to the marine environment. A variety of symptoms were evident in many of the resulting larvae, from mortalities to deformities, to even the more subtle effects on growth from very normal appearing animals. Delayed impacts on growth were evident at 19 parts per billion total PAH, and the ecological consequence (suffering increased levels of predation) was evident when fewer adults returned from the oil exposed groups (Heintz 2000). Exposure of developing embryos to 5 and 19 ppb total PAH resulted in a subsequent 20% and 40% reduction in marine survival among the returning adults. Exposure of incubating herring eggs to weathered oil caused significant morphological defects at 9 ppb, and effects of more weathered oil were significant at concentrations as low as 0.2 ppb PAH; chromosomal aberrations were observed at 0.7 parts per billion (ppb) (Carls et al. 1999). Over time, reductions in adult recruitment will slowly and subtly result in lower populations, eventually leading to extinction of the stock. This situation has probably been the primary mechanism leading to missing populations in urban estuaries.

Allowing persistent organic pollutants into the environment also results in creating long-term reservoirs of pollution that are available to organisms. Hydrocarbons, pesticides, and PCBs tend to adsorb to organic tissue, and can be transferred up the food chain, or can be adsorbed to inorganic matter in sediments, where they can remain bioavailable for years. There is a substantial uptake of these compounds, especially in many invertebrate species that do not have the biochemical mechanisms to purge their systems of the pollutants (Varanasi et al. 1989, 1992; Meador et al. 1995). There is a growing body of evidence of accumulations of persistent organic pollutants in the cold sink of the arctic, and Alaskan waters with many lipid rich compartments (copepods, forage fish, target species, marine mammals) is becoming more contaminated each year from pollutants originating in other major geographic areas. We would not want to accelerate this problem with local releases with more direct routes to Alaskan fish resources.

The concern regarding chronic pollution to embryos applies to non-organic pollutants as well, because the concepts are the same, although the literature is sparse. In this case, the contaminant is not necessarily trapped in the tissues for long periods of time because of the high affinity of large organic molecules in lipids, but remains in tissues because of the continuous replacement by the chronic mixing zone source. In other words, in both cases a tissue load is present. The second major principle is that chronic exposure to embryos and larvae are special biological situations. Because of the long

incubation time, the exposure to embryos and larvae is chronic, and any extrapolation from acute toxicity tests and mechanisms does not apply. Small and seemingly innocuous interruptions or interferences with the embryo development process can have lethal effects, whether they are immediately obvious or delayed, and are very subtle to detect. Chronic studies with embryos and subsequent wild releases are the only concrete way to detect sublethal effects that will affect returning adult numbers. NMFS is concerned that the proposed changes to the water quality standards would shift the burden of proof – meaning that non-harmful acute tests could be used inappropriately to judge the potential harm of chronic exposures to embryos and larvae, thereby underestimating actual effects.

Based on research conducted by NMFS and others, we recommend that DEC not approve mixing zones for areas of fish spawning and rearing. Using acute toxicity data and toxicity models is inappropriate for the protection of fish embryos and larvae in a chronic exposure situation. The populations exposed will be at considerable risk over time, and those exposed stocks can be expected to diminish over time to levels that may not support subsistence, sport, or commercial harvests.

### **Specific Comments**

18 AAC 70.240 (c)(4)(B) As written this section states that DEC will approve a mixing zone as proposed or with conditions, only if it finds that available evidence reasonably demonstrates that an effluent or substance will “... be treated to remove, reduce, and disperse pollutants, using methods found by the department to be the most effective and technologically and economically feasible, consistent with the highest statutory and regulatory treatment requirements.”

The regulations should be more specific. Statutory and regulatory treatment requirements differ around the country, and for certain discharges there are areas in the country with higher treatment requirements than Alaska. What standard will be used to assess whether a mixing zone is “consistent with the highest statutory and regulatory treatment requirements”?

18 AAC 70.240 (c)(4)(B) This section states that mixing zones will not “create a public health hazard through encroachment on existing uses of the waterbody for water supply or contact recreation.”

How is encroachment on existing uses defined? Should this be cross referenced to statutes that cover water rights?

18 AAC 70.240 (c)(4)(D) “the mixing zone will not in streams, rivers, or other flowing fresh waters, result in a reduction in fish population levels;”

Are individual fish kills acceptable? How will the population be tracked to insure that there will not be a reduction in fish populations?

18 AAC 70.240 (c)(4)(E) “the mixing zone will not in streams, rivers, or other flowing fresh waters, adversely affect the capability of an area to support spawning, incubation or rearing of anadromous or resident fish;”

Water quality standards are premised on levels that do not adversely affect fish and/or their habitat, and any mixing zone (i.e., an allowable variance above water quality standards) could adversely affect fish and their habitat.

18 AAC 70.240 (c)(4)(F) “the mixing zone will not in streams, rivers, or other flowing fresh waters, result in permanent or irreparable displacement of indigenous organisms;”

Again, if a mixing zone is a variance to water quality standards, indigenous organisms may find areas where the levels of pollutants exceed water quality standards to be inhospitable.

18 AAC 70.240 (d)(8) DEC will approve a mixing zone, as proposed or with conditions, only if the DEC finds that available evidence reasonably demonstrates that within the mixing zone the pollutants discharged will not “exceed acute aquatic life criteria at and beyond the boundaries of a smaller initial mixing zone surrounding the outfall, the size of which shall be determined using methods approved by the department.”

With no spatial limits on the size of the smaller initial mixing zone, a very large initial mixing zone could be permitted, allowing large areas to exceed acute aquatic life criteria. We recommend specifying that the initial mixing zone must not extend beyond the immediate vicinity of the outfall. Also, this section needs to clarify what is meant by “... methods approved by the department.” Some examples of the types of those methods would be helpful. In addition, NMFS suggests that the sentence read “... aquatic life criteria *to* and beyond.

18 AAC 70.240 (e)(1)(A) DEC will approve a mixing zone, as proposed or with conditions, only if it finds that the mixing zone is as small as practicable and will comply with the following size restrictions, unless DEC finds that evidence is sufficient to reasonably demonstrate that these size limitations can be safely increased: “(1) for estuarine and marine waters, measured at mean lower low water, (A) the cumulative linear length of all mixing zones intersected on any given cross section of an estuary, inlet, cove, channel, or other marine water may not exceed 10 percent of the total length of that cross section;”

Does the cumulative linear length refer to the perimeter of the waterbody?

18 AAC 70.240 (f) “For streams, rivers, or other flowing fresh waters, in calculating the maximum pollutant discharge limitation, the volume of flow available for dilution must be determined using the actual flow data collected concurrent with the discharge or using other methods approved by the department.”

If the discharge can occur 24 hours a day seven days a week, year round, what part of the annual flow would be used?

## References

- Barron, Mace G., Mark G. Carls, Ron Heintz, and Stanley D. Rice. 2003. Evaluation of Fish Early Life-Stage Toxicity Models of Chronic Embryonic Exposures to Complex Polycyclic Aromatic Hydrocarbon Mixtures. *Toxicological Sciences* vol. 78 no. 1. p. 60
- Barron, M. G., M. G. Carls, J. W. Short, and S. D. Rice. 2002. Photoenhanced Toxicity of Aqueous Phase and Chemically Dispersed Weathered Alaska North Slope Crude Oil to Pacific Herring Eggs and Larvae. *Env. Tox. And Chem.* 22: 650-660
- Billiard, S.M., M.E. Hahn, D.G. Franks, R.E. Peterson, N.C. Bols, and P.V. Hodson. 2002. Binding of polycyclic aromatic hydrocarbons (PAHs) to teleost arylhydrocarbon receptors (AHRs). *Comp. Biochem. Physiol. B.* 133:55-68.
- Billiard SM, K Querbach and PV Hodson. 1999. Toxicity of retene to early life stages of two freshwater fish species. *Environ Toxicol Chem* 18: 2070-2077
- Brinkworth, L.C., P.V. Hodson, S. Tabash, and P. Lee. 2003. CYP1A induction and blue sac disease in early developmental stages of rainbow trout (*Oncorhynchus mykiss*) exposed to retene. *J. Toxicol. Environ. Health - Part A.* 66:47-66.
- Carls, M. G., S. D. Rice, and J. E. Hose. 1999. Sensitivity of fish embryos to weathered crude oil: Part I. Low-level exposure during incubation causes malformations, genetic damage, and mortality in larval Pacific herring (*Clupea pallasii*). *Environ. toxicol. chem.* 18:481-493.
- Duesterlo, Switgard, Jeffrey Short, and Mace G. Barron. 2002. Photoenhanced Toxicity of Weathered Alaska North Slope Crude Oil to the Calanoid Copepods *Calanus marchallae* and *Metridia okhotensis*. *Environ. Sci. Technol.* 36 : 3953-3959
- Heintz, R. A., S. D. Rice, A. C. Wertheimer, R. F. Bradshaw, F. P. Thrower, J. E. Joyce, and J. W. Short. 2000. Delayed effects on growth and marine survival of pink salmon *Oncorhynchus gorbuscha* after exposure to crude oil during embryonic development. *Mar. Ecol. Prog. Ser.* 208: 205-216.
- Incardona, J.P., T.K. Collier, and N.L. Scholz. In press. Defects in cardiac function precede morphological abnormalities in fish embryos exposed to polycyclic aromatic hydrocarbons. *Toxicological Applications in Pharmacology*.
- Marty, G. D., J. W. Short, D. M. Dambach, N. H. Willits, R. A. Heintz, S. D. Rice, J. J. Stegeman, And D. E. Hinton. 1997. Ascites, premature emergence, increased gonadal cell apoptosis, and cytochrome P4501A induction in pink salmon larvae continuously exposed to oil-contaminated gravel during development. *Can. J. Zool.* 75: 989-1007.
- Meador, J.P., J.E. Stein, W.L. Reichert, and U. Varanasi. (1995). A review of bioaccumulation of polycyclic aromatic hydrocarbons by marine organisms. *Reviews Environmental Contamination and*

Varanasi, U., J.E. Stein, W.L. Reichert, K.L. Tilbury and S-L. Chan. 1992. Chlorinated and Aromatic Hydrocarbons in Bottom Sediments, Fish and Marine Mammals in US Coastal Waters: Laboratory and Field Studies of Metabolism and Accumulation. In: *Persistent Pollutants in the Marine Environment*, Eds.: Colin Walker and D.R. Livingstone, Pergamon Press, New York, NY, p. 83.

Varanasi, U., Ed. 1989. *Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment*. CRC Press, Inc., Boca Raton, FL. 341p

White, P.A., S. Robitaille, and J.B. Rasmussen. 1999. Heritable reproductive effects of benzo[a]pyrene on the fathead minnow (*Pimephales promelas*). *Environmental Contamination and Toxicology* 18: 1843-1847.