Title of project
Evaluating the sublethal impacts of current use pesticides on the environmental health of salmonids in the Columbia River Basin.

BPA project number 35024

Business name of agency, institution or organization requesting funding
National Marine Fisheries Service/Northwest Fisheries Science Center

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The ISRP gave our proposal a very careful and thorough review, and we appreciate the opportunity to respond to the their comments. Our responses and clarifications are given below (the ISRP comments are italicized).

Do pesticides in question exist in sufficient quantities in the environment such that those concentrations are likely to have measurable effects and can be partitioned and separated from other large and known sources of salmon mortality. Although a variety of pesticides are used and detectible in the environment, what are the concentrations and identities that are expected at levels in which salmon would be affected. If there is evidence, it should be cited.

First, water quality criteria to protect salmon and other aquatic organisms in North America have only been developed for a few current use pesticides. These criteria have been promulgated by the Environmental Protection Agency, the National Academies of Sciences and Engineering, the Canada and United States International Joint Commission, and the Canadian Council of Resource and Environment Ministers. Although pesticides are used throughout the Systemwide Province, the USGS NAWQA program has only monitored the surface water concentrations of these chemicals in two of the major tributaries to the Columbia River (the Yakima and Willamette basins). Using the Willamette River system as an example, intensive environmental monitoring has shown that aquatic life criteria are exceeded for atrazine, azinophos-methyl, carbaryl, carbofuran, chlorpyrifos, diazinon, diuron, lindane, and malathion (Wentz et al., 1998). Therefore, these chemicals have been measured in salmon habitat at levels that are toxic to aquatic organisms. The ISRP is referred to Table 1 of our proposal and the USGS NAWQA pesticide monitoring reports for the Yakima basin (Ebbert and Embrey, 2002) and the Willamette basin (Wentz et al., 1998) for more detailed information.

Second, pesticides representing several of the major use classes have been shown to significantly impair the physiology or behavior of salmonids at concentrations that have been detected in the natural environment. Fate and transport models (*e.g.,* GENEEC) and direct environmental monitoring (Wentz et al., 1998) have shown that most pesticides occur in river systems at concentrations of 10 ppb or less. Pesticides within a class generally share a common mode of action, and the classes that have been evaluated for direct toxicity to salmonids (at concentrations of 10 ppb or less) include the organophosphates (*e.g.*, azinphos-methyl, chlorpyrifos, diazinon, fonofos, malathion), the carbamates (*e.g*., carbaryl, carbofuran), the pyrethroids (*e.g.*, permethrin, cypermethrin, esfenvalerate), the triazines (*e.g*., atrazine, cyanazine, simazine), and the antisapstains (TCMTB). These pesticides have been shown (again, at concentrations below 10 ppb, or levels that have otherwise shown to be environmentally-realistic) to interfere with migratory behavior (chinook salmon; Scholz et al., 2000), foraging behavior (Atlantic salmon; Morgan and Kiceniuk, 1990), predator detection (chinook salmon; Scholz et al., 2000), predator avoidance (chinook salmon; Kruzynski and Birtwell, 1994); olfactory function (coho salmon; N.L. Scholz, unpublished results – see Figs. 8 & 10 in the proposal), reproductive priming (Atlantic salmon; Moore and Waring, 1996; 1998; Waring and Moore, 1997), and fertilization success (Atlantic salmon; Moore and Waring, 2001).

Is the model realistic? The use of a copper based model and surrogate for organic pesticides seems convenient but not necessarily realistic. No evidence was provided to show copper concentrations exist in the Columbia River or most tributaries of the levels likely to be toxic or have sub-lethal impacts.

First, we are not proposing to use copper as a surrogate for organic pesticides. Copper is one of the most widely used pesticides in the Columbia River Basin and is therefore a legitimate and stand-alone water quality concern. Second, there can be no doubt that the proposed copper exposures are environmentally realistic. Copper has been shown to impair the salmonid olfactory system by the lab of the Principal Investigator (PI) and other groups (e.g. Hansen et al., 1999; Hara et al., 1976). Relevant data are shown in Fig. 10 of the proposal. Importantly, the threshold for sublethal copper toxicity as measured in the lab of the PI is less than 6 ppb (data available to the ISRP upon request). This threshold concentration is well within the range of copper concentrations measured in the surface waters of the Willamette River Basin (Anderson et al., 1996) and other salmon habitats. In the Willamette, for example, copper was detected in 19 of 23 filtered water samples, with a 75^{th} percentile detected concentration of 7.4 ppb. Notably, copper was the most frequently detected trace element at agricultural and mixed-use sites. In urban areas, copper concentrations in storm water runoff are considerably higher (10 to 100 ppb or more; Bellevue, 1995).

Copper does exist in the Clarkfork River in high concentrations. There the homing instincts of rainbow trout and bull trout, two potradromous species, seem extremely well developed (Schmetterling, personal communication, MDFWP) in this extremely copper

contaminated system. Milltown Dam is one of the most studied and evaluated cleanup sites on EPA's superfund list.

The ISRP is assuming that anecdotal reports of homing efficiency in potadromous salmonids is evidence that anadromous Pacific salmon can navigate despite sublethal damage to their olfactory system. It has been firmly established that anadromous Pacific salmon fail to home when olfaction is blocked (Wisby and Hasler, 1954; A. Dittman, unpublished results). We cannot ascertain from the ISRP's comments whether: 1) the homing instincts of potadromous species are similarly reliant on olfactory cues, 2) the olfactory capacity of potadromous species in the Clarkfork system has been evaluated (and is functional), or 3) the susceptibility of potadromous and anadromous species to short-term, sublethal copper exposures is equivalent. It is entirely possible that local movements of bull trout within the Clarkfork system are guided by other sensory modalities, and these are less sensitive to the chronic effects of copper exposure than the olfactory system. Without additional information, we cannot respond to the Milltown Dam example in the larger context of the migratory behavior of anadromous stocks.

Is the work directly applicable to salmon? The proposed work is physiological research (driven by good ecological issues). The question arises is what is the actual relevance of the studies to the real world of salmon. For example, how applicable is the zebrafish model to salmon and what evidence is there that the rapid development rate of zebrafish embryos (a tropical species) represents much slower development of coldwater salmon embryos?

The issue of heterochrony has very little relevance to this particular problem. The genes that are responsible for morphogenesis, pattern formation, and organogenesis during embryonic development are highly conserved across all vertebrates (zebrafish, salmon, humans, or otherwise). This is precisely why large-scale genetic screens in zebrafish have identified hundreds of mutant phenotypes, many of which resemble human clinical disorders (Dodd et al., 2000; Dooley and Zon, 2000). This is also why the National Institute of Health is directing the zebrafish genome initiative. Put simply, the zebrafish model has extraordinary potential for the study of vertebrate biology, physiology and human disease (Fishman, 2001; Grunwald, 1996). From a phylogenetic perspective, zebrafish should be a better model for salmon than they are for humans.

In a related research effort, the PI has recently validated zebrafish as model for studying the effects of polycyclic aromatic hydrocarbons (PAHs) on salmon and other fish species. The effects of PAHs exposures on pink salmon were studied extensively following the 1989 *Exxon Valdez* oil spill (*e.g.*, Heintz et al., 1999). These effects include impaired cardiovascular function as well as spinal and craniofacial deformities, and they have also been documented in herring (Vines et al., 2000). Notably, these same effects occur in developing zebrafish embryos (J. Incardona and N.L. Scholz, unpublished results, available to the ISRP upon request). Consequently, we are now using molecular and physiological techniques that have been worked out for zebrafish to identify specific mechanisms of sublethal PAH toxicity in salmon.

Are natural concentrations and mortalities significantly detectable to warrant the research? The discovery of microscopic anatomical or physiological anomalies after exposure to high concentrations of a toxin in a rapid developing model (zebrafish) in vitro may present unrealistic results and lead to unwarranted conclusions (Type 2 statistical error- i.e. find problems that don't actually exist for example, that pesticides from lab data extrapolated to the wild indicate a mortality component for wild salmon when there may be none). Response?

We reiterate the specific aim of our proposal. Referring to the final paragraph of Task 1 (pp. 11-12):

"Finally, it is important to note that we will evaluate the chemicals above for their toxicological effects on as many biological processes as is technically feasible given the current "state of the science" in the zebrafish model system. We have selected endpoints (see below) that are presumably fundamental to all fish species and will therefore have clear significance for salmonids. However, we are not proposing that zebrafish are surrogates for salmon in the strict sense that zebrafish toxicity data alone should be sufficient to guide salmon policy in watersheds with pesticide pollution. Rather, we view zebrafish as an experimental system that we can use to identify pesticides (or classes or pesticides) that may be harmful to salmon or other fish species in the Columbia River Basin. Results in zebrafish can then be validated, as appropriate, in the native species of concern. We have not included such groundtruthing experiments in this proposal because it will take our group a full three years to conduct the zebrafish phenotypic screens."

There is no possibility of unwarranted conclusions (or Type II errors) because we have no intention of extrapolating results in zebrafish to salmon without direct validation in the latter species. We did not include these additional experiments in this proposal because the zebrafish component alone is ambitious in terms of the number of chemicals in the screen and the diversity of biological endpoints that must be examined. When the phenotypic screens are finished, we will make the results and experimental tools available to the larger research community. Our group (or other groups) can then propose groundtruthing studies to establish toxicity thresholds in salmon, sturgeon, or other species of concern.

Are there potential affects on other species of fish and if so, what would be consequences to salmon? The authors hypothesize that predators may gain some advantage over smolts with impaired olfactory senses; however, should such a pesticide condition occur, why wouldn't the predators species have similar impairment?

Not all sensory systems are equivalent in terms of their vulnerability to toxic injury. For example, we have previously shown for juvenile chinook salmon that pesticide exposures impair olfactory-mediated predator avoidance behaviors and while leaving visually-guided behaviors (prey capture) intact (Scholz et al., 2000). Olfactory receptor neurons are directly exposed to the animal's surrounding environment and are therefore more

sensitive to injury than other areas of the nervous system. Natural predators will not have a similar impairment because fish and birds use vision, and not olfaction, to capture juvenile salmon and steelhead. Prey animals in substandard condition are often consumed in higher proportions by predators (Mesa et al., 1994; Temple, 1987).

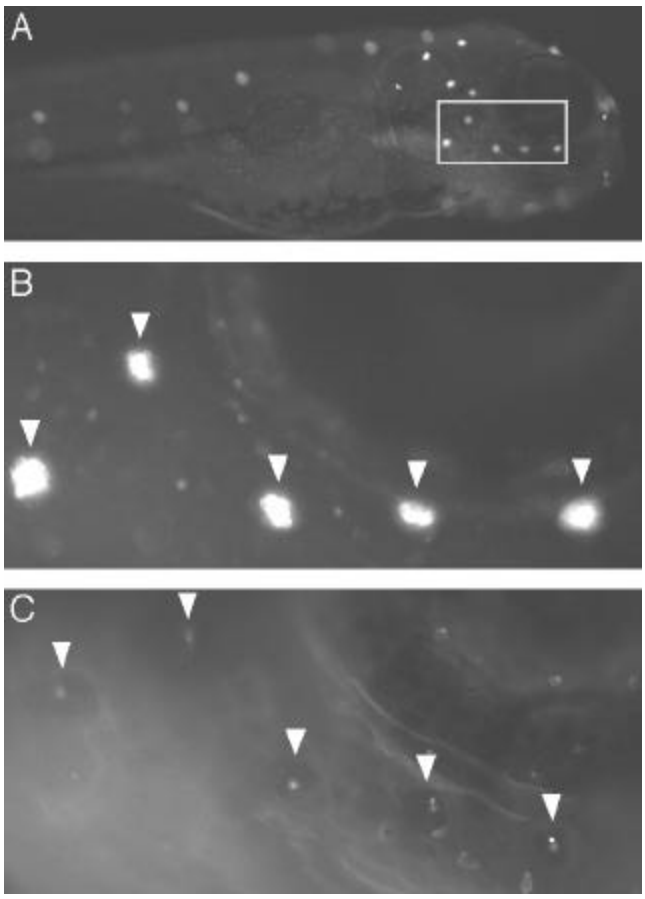

Figure 5. Epifluorescent micrographs showing lateral line neuromasts in a zebrafish larvae. The receptor neurons of the lateral line system have been labeled with a fluorescent vital dye (DASPEI). *A*, Low magnification image showing the distribution of neuromasts along the body of a control animal. The box indicates the region shown in *B*. *B*, Higher magnification image of the suborbital neuromasts from the same fish. Note that numerous sensory neurons are labeled within each neuromast. *C*, Suborbital neuromasts in an animal exposed to an environmental stressor (50 µg/L copper). Note the marked reduction in labeled cells within each neuromast. Kao and Scholz, unpublished results.

It may interest the ISRP to know that recent experiments in the PI's lab have shown that copper also targets the ciliated receptor neurons of the lateral line system. Lateral line neurons are similar to olfactory receptor neurons in that they are also in direct contact with dissolved pesticides in salmon habitat. An example of the effects of copper on lateral line neuromasts is shown in Fig. 1. Preliminary evidence indicates that the olfactory and lateral line systems are similar in terms of their sensitivity to copper. Significantly, the mechanosensory capacity of the lateral line system underlies schooling and predator avoidance behaviors, and sublethal injury to the lateral line has been shown to increase rates of predation in chinook salmon (Mesa and Warren, 1997). Thus, short term, sublethal copper exposures are likely to impair at least two sensory modalities that are critical for predator avoidance. At the same concentrations, copper is not likely to reduce the foraging efficiency of birds or predatory fish. Based on this new data, we plan to incorporate additional lateral line experiments into Objective #2, Studies 1&2.

Does evidence exist in nature that shows any unusual patterns of straying that might be connected with pesticides? The authors hypothesize olfactory impairment from pesticides

may be a source of straying. Is there even anecdotal evidence that specific basins, sub-basins or tributaries with higher levels of pesticides (even use, if not data) show higher rates of straying than other basins?

This is a good idea, and one that we've considered. There are two major difficulties: 1) the absence of pesticide exposure data for the majority of the tributaries to the mainstem of the Columbia River, and 2) the absence of within-population straying data for salmonids, and wild stocks in particular. Because the spatial data are so incomplete (and therefore difficult to reliably quantify), we chose instead to investigate the impacts of pesticides on straying at a more mechanistic level, validate the findings with a field study, and then extrapolate the results to the population scale using a metapopulation model. If our hypothesis is correct, and sublethal exposures to common pesticides at environmentally-realistic concentrations leads to an increase in straying, then it might be worth: 1) developing a GIS database for cropping patterns and other indicators of pesticide use in unmonitored basins, and 2) mining the available data for stray rates in different basins (*e.g.*, the coded wire tag inventory). We didn't include this in the proposal because of the labor involved and the poor resolution of the existing data.

Can we really get at the question of genetic consequences of pesticides? The authors hypothesize that genetic integrity of certain weak stock ESU's may be compromised by straying. The introduction of artificial selection to 95% of the salmon migrants via various artificial propagation (hatchery) techniques and selective harvest of the *past 50 years seems gargantuan compared to the potential problem of incremental straying from a pesticide source. A simpler hypothesis is that if pesticides are entering the salmon's life cycle, it is likely that those individuals and populations are carrying an additional genetic or environmental load. If this is true, a more direct approach to warrant the physiological studies proposed herein would be to find at least one watershed that has physiological detectable concentrations of copper (or other toxins) and then emulate that condition in the lab. At the same time, the problem of toxicity can be addressed as a preventative measure while research confirms the extent of the mechanism and the extent of the problem in the lab.*

We agree that harvest and hatcheries are important obstacles (past, present, and future) to the sustainability of wild salmon populations in the Columbia River basin. However, the ISRP is making a direct and somewhat questionable comparison between two major "H's" (harvest and hatcheries) and a single facet of another "H" – that is, habitat. Poor water quality conditions, when coupled with physical modifications of salmon habitat, are just as gargantuan as the ISRP's examples of harvest and hatcheries in terms of salmon and steelhead declines. Off-site habitat mitigation is a problem with many facets, and each deserves serious consideration.

We disagree with the ISRP's contention that straying is not a major concern for ESA-listed species in the Systemwide Province – see, for example, the discussion of straying and gene flow in natural populations by McElhany et al. (2000). Also, several RPAs in

the FCRPS 2000 Hydro Biological Opinion specifically address the importance of straying (*e.g.*, RPAs 107 and 184).

Finally, physiologically detectable concentrations of copper are found in most of the major river systems in the Systemwide Province. The laboratory and field experiments we propose in Objective 2 are specifically designed to emulate copper exposures under natural conditions in salmon and steelhead habitat, and they are therefore consistent with the ISRP's comments. In terms of preventative measures, the ISRP is advocating an approach consistent with the "precautionary principle" (European Environment Agency, 2001). While a precautionary approach to pesticides may be protective for salmonids in the face of scientific uncertainty, the implementation of the precautionary principal is beyond the scope of this technical research proposal.

If any results suggested that pesticides were an additive component of lifecycle mortality, how would such a finding be extrapolated over all populations and with all other causes of mortality? This becomes statistically daunting when in even a healthy system, 95-98% of the smolts do not return, mostly for unknown causes.

This is a rhetorical question. If we had sufficient data to make such an extrapolation, we would not have highlighted the importance of this issue in the problem statement for Objective 2. Is the ISRP suggesting that each individual modification of salmon habitat must have a statistical signature against a background of 98% smolt mortality to be considered a significant research priority? Among habitat variables, water temperature, dissolved oxygen, fine sediments, culverts, unscreened water diversions, large woody debris, and the availability of side channel habitat are all suspected to limit salmon and steelhead productivity. Restoring these physical processes is a major goal of the Fish and Wildlife Program's efforts to improve (or mitigate) off-site habitat conditions and enhance salmonid productivity throughout the Columbia River basin. Has the ISRP or any other entity required that the results of screening water diversions be measurable across all populations (where smolt mortality is 95-98%) before a research effort or restoration project goes forward? If not, why hold contaminants (*e.g.* pesticides) to a different standard?

Alternative Research Designs. Is there any possibility of an alternative experiment that would use salmon, perhaps in populations known to be exposed to certain toxins; e.g. are there known exposed and control populations in nature that could be used as subjects of this study? The current approach asks us to extrapolate from zebrafish to salmon and do theoretical population modeling using a large suite of hypothetical variables about olfactory impairment and gene flow. The ISRP would be more enthusiastic if the model were with salmon and in a location where pesticide is actually found in known concentrations of concern in the environment?

First, the ISRP's question is somewhat vague. There are, of course, natural and hatchery salmon populations that are exposed to more or less copper and other pesticides in the

Columbia River basin. These fish could (with appropriate ESA permitting, if necessary) be used for the proposed study. Importantly, the ISRP does not specify why, exactly, the quality of this proposal would be improved by working with "control populations in nature" vs. salmon reared at the NWFSC hatchery facility. We could evaluate the sensory capacity of juvenile salmonids in agricultural vs. pristine forested streams, but we would be unable to control the most important variable (pesticide exposure) and the observed differences between sampling groups, if any, would be subject to an array of different interpretations. By contrast, the approach we have defined will identify cause-and-effect relationships between real-world pesticide exposures and salmon health and performance.

Second, the ISRP is confusing Objectives 1&2. We are not asking the ISRP to extrapolate from zebrafish to salmon (see related comments above). Also, the theoretical population modeling in Objective 2 will not be based on "a large suite of hypothetical variables". Rather, the modeling will be based on the empirical results of Studies 3&4.

Finally, the model is specific to salmon (the entirety of Objective 2), uses exposure concentrations that are representative of actual conditions in salmon habitat (*e.g.*, the Willamette - see above), and focuses on biological endpoints that are a clear and significant concern for the recovery of threatened and endangered species in the Columbia River basin. This conceptual design should reinforce the ISRP's enthusiasm.

Summary.

1. Please address the basic evidence that pesticides in the environment are having a measurable and detectable impact on the return rates of salmon. Of the 100 pesticides identified, how many exist at concentrations are physiologically affecting sub-populations of salmon. Please identify evidence of specific impairment or straying or genetic or environmentally detectable load on any population. Address the concern that the model of zebrafish in the lab has several limitations to direct application to salmon in the wild. Address alternative approaches using salmon, potentially in nature.

Please see our answers the ISRP's earlier comments.

2. If a potential connection between copper and salmon is found, how significant is this source of mortality versus dozens of other sources not only of other pesticides, but also of dams, harvest, predation, ocean losses etc. Normal mortality rates are already above 95%. This speaks to the previously defined experiment, as the treatment and control will be exposed to all these cumulative impacts.

Please refer to our earlier response to this comment by the ISRP. Also, we emphasize that the impacts of pesticides relative to dams, harvest, and ocean conditions is not the focus of this research proposal.

3. The research seems extremely interesting to basic science, but please elaborate more directly on the likelihood that results will be directly applicable to the management of the FCRPS and salmon recovery. For example, hypothesize some expected and quantifiable impact, and what might be done about it. Wouldn't it be simpler to keep concentrations of harmful chemicals at sub-physiological impairment levels? This is EPA's mission and the toxicological research is usually in the realm of EPA chemical use and approval domains. Shouldn't this research wait for more details about natural concentrations of toxins? Some fascinating observations were cited by the authors about physiological and behavioral responses to predator alarm pheromones. What types of research can be done to develop more wild-like traits in hatchery reared smolts? Does this area of physiological research hold potential large benefits to salmon?

The proposed research is directly relevant to recovery planning throughout the Columbia River basin. The Fish and Wildlife Program is committed to restoring the quality of off-site salmon habitat, with the intention of increasing the productivity of threatened or endangered populations. Millions of dollars have recently been invested in environmental monitoring, and it is now well known that salmon are exposed to a large and complex array of chemicals that are specifically designed to kill or regulate the growth of biological organisms. What assurance does a recovery planner have that pesticides are not as important (or even more important) than conventional physical indicators of habitat degradation? In the absence of empirical data, recovery planners may very well commit a Type I error (to borrow the ISRP's analogy) – that is, assume that pesticides are not a significant limiting factor when, in reality, they are.

With respect EPA's mission, it's been nearly three decades since the Clean Water Act was passed by Congress. In the years since, aquatic life criteria have been developed for fewer than 10 of the 898 pesticide active ingredients that are currently registered for use. Also, pesticides are registered under the Federal Insecticide, Fungicide, and Rodenticide Act, and the national approval process does not specifically consider the biological requirements of salmonids. One could similarly argue that since the EPA is responsible for stream temperatures (again, under the Clean Water Act), the Fish and Wildlife Program should not be involved in temperature studies. This has not been the practice.

Additional monitoring data will only reveal that salmon and steelhead are exposed to different pesticides. It will not reduce the key biological uncertainty of the adverse effects of these pesticides on fish health and performance. This can only be accomplished by the kinds of research projects we are proposing here.

Finally, with respect to the alarm pheromone signaling system in juvenile salmonids, the experimental design for Study 2 (Objective 2) was adapted from a BPA-funded NATURES study (Berejikian et al., 1999). As suggested by the ISRP, this research has been used to develop more wild-like traits in hatchery fish.

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