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California Farm Bureau Federation
California Rice Commission
East San Joaquin Water Quality Coalition
San Joaquin County-Delta Water Quality Coalition
Western Growers Association
Western Plant Health Association
Westside San Joaquin River Watershed Coalition

August 21, 2012

Via Electronic Mail Only

Charles R. Hoppin, Chairman and Members
c/o Jeanine Townsend, Clerk to the Board
State Water Resources Control Board
1001 I Street, 24th Floor
Sacramento, CA 95814
commentletters@waterboards.ca.gov

Re: Comments on Draft Policy for Toxicity Assessment and Control (Public Review Draft, June 2012)

Dear Chair Hoppin:

The above-named agricultural organizations appreciate the opportunity to review the State Water Resources Control Board's (State Water Board) draft *Policy for Toxicity Assessment and Control* (Draft Toxicity Policy). While we appreciate the efforts that the State Water Board has made to address concerns expressed previously, we continue to have significant concerns with the Draft Toxicity Policy and its application to agricultural dischargers.

20.1 → As a preliminary matter, we continue to believe that it is inappropriate for the Draft Toxicity Policy to apply to agricultural dischargers. The primary purpose of this policy is to supersede Toxicity control provisions in the state's *Policy for Implementation of Toxics Standards for Inland Surface Waters, Enclosed Bays, and Estuaries of California* (SIP), which is a Toxicity policy that applies only to point source dischargers subject to federal National Pollutant Discharge Elimination System (NPDES) permit requirements. Moreover, ← 20.15 the June 2012 Draft Staff Report and Environmental Checklist to the Draft Toxicity Policy (Staff Report) states that the "State Water Board's goals for this project are to have the Regional Water Boards convert the Policy's toxicity objectives into effluent limitations in order to: protect aquatic life beneficial uses; provide regulatory consistency; provide a basis for equitable enforcement; and fulfill the requirements of State Water Board Resolution No. 2005-0019." (Staff Report, p. 12.) The aforementioned resolution pertains specifically to the State Water Board's intent to adopt amendments to the SIP, and it directed staff to "introduce amendments to the SIP to address narrative toxicity control provisions . . . ."

20.1

20.15 (Resolution No. 2005-0019, p. 2.) None of the reasons or purposes for development of the Draft Toxicity Policy applies to nonpoint source dischargers such as agriculture. Agricultural dischargers are exempt from federal NPDES requirements, and not subject to the SIP. Accordingly, all provisions in the Draft Toxicity Policy with respect to agriculture, or "channelized dischargers" as defined in the Draft Toxicity Policy, need to be removed.

Our more specific concerns with respect to the provisions that are applicable to agriculture are provided here. Attached are additional technical comments with respect to the proposed null hypothesis as a numeric water quality objective.

20.2 **Definition of Channelized Dischargers**

The Draft Toxicity Policy proposes to create a new definition that would apply almost exclusively to agricultural dischargers. We find this proposed new definition inappropriate for several reasons. Most importantly, the definition is inconsistent with applicable state law and could arguably expand application of the Draft Toxicity Policy to agricultural conveyance facilities that are not waters of the United States or surface waters of the state. Specifically, the proposed definition would define agricultural dischargers as those that discharge through a directed channel that are not regulated under the NPDES permit program. All channels are not necessarily surface waters of the state. In fact, we contend that channels that are man-made agricultural conveyance facilities are not surface waters of the state and therefore discharges to such channels are not subject to the Clean Water Act (CWA) or the Porter-Cologne Water Quality Control Act (Porter-Cologne). Further, unless specifically identified in a water quality control plan (Basin Plan), constructed agricultural drains do not have designated beneficial uses and therefore the toxicity objectives that are designed to protect aquatic life beneficial uses would not apply. (See State Water Board Order WQO 2002-0016, at p. 5.) By including all nonpoint source discharges to channels as part of the definition of channelized discharges in this policy, the Draft Toxicity Policy implies that all "channels" are surface waters of the state subject to this policy. That is factually and legally incorrect.

Further, we do not believe that it is necessary or appropriate to include a definition for "channelized dischargers." To the extent that the State Water Board determines it is appropriate to apply the numeric toxicity objectives to agriculture and include agriculture in the implementation provisions of the Draft Toxicity Policy, it is not necessary to include this definition. The same result could be reached by deleting all references to channelized dischargers, and "Irrigated Agriculture." For example, on page 14 of the Draft Toxicity Policy, the section applies to irrigated agriculture subject to any conditional waiver, conditional discharge prohibition, or waste discharge requirement. It is not necessary to artificially create a new term that has no practical or legal application.

20.3 **Toxicity Objectives**

We are opposed to the adoption of numeric toxicity objectives for general application to inland surface waters, enclosed bays, and estuaries of the state. In particular, we are

20.3 opposed to the use of the proposed null hypothesis where all water quality is presumed toxic until sufficiently demonstrated that the water is non-toxic. Further, the proposed numeric 20.4 objective functionally indicates that a single Test of Significant Toxicity (TST) failure in a receiving water bioassay test represents an exceedance of the numeric objective. This, combined with the state's 303(d) Listing Policy, will result in the listing of a number of water bodies on the state's 303(d) List that are in fact non-toxic.

20.5 Next, the State Water Board has failed to comply with Porter-Cologne in its proposed attempt to adopt new water quality objectives for toxicity. Specifically, Water Code section 13241 requires the State Water Board to consider a number of factors when it adopts water quality objectives, including the water quality conditions that could reasonably be achieved, and economic considerations. (See Wat. Code, § 13241.) With respect to economic considerations, water boards are "under an affirmative duty to consider economics when adopting water quality objectives." (Memorandum to Regional Water Board Executive Officers from William R. Attwater, Chief Counsel (Jan. 4, 1994) at p. 1.) When considering economics, the economic assessment requires a determination of the following factors: (1) whether the objective is currently being attained; (2) what methods are available to achieve compliance with the objective, if it is not currently being attained; and, (3) the costs of those methods. (*Ibid*.) With respect to agriculture (and others), the analysis is superficial to non-existent. First, the economics analysis does not evaluate costs of compliance with the proposed numeric toxicity objective, but looks only to costs of toxicity testing. By looking only at monitoring costs, the Economic Considerations report claims that in the Central Valley, Central Coast, and Los Angeles regions the incremental costs would be minimal. However, its limited review of monitoring costs alone is inadequate. The Economic Considerations report fails to properly account for the added expense of the TST approach, and that the recommended monitoring for chronic toxicity would greatly increase toxicity monitoring costs for agriculture.

20.6 Taking the Central Valley region as an example, currently, chronic toxicity testing is not required by the Conditional Waiver and associated monitoring and reporting programs for the various coalition areas. Chronic toxicity testing is significantly more expensive than acute toxicity testing. Specifically, implementing chronic water column toxicity testing for *Ceriodaphnia dubia* and *Pimephales promelas* will result in an increase in toxicity testing costs of two times the current acute toxicity testing costs. All costs will double including the initial toxicity test of the sample, the reference toxicity tests, and any subsequent toxicity identification evaluations (TIEs). For example, based on current East San Joaquin Water Quality Coalition (ESJWQC) toxicity testing costs for three species (algae, fat head minnow, and water flea), the increase in costs for a single sample will be $3,250. If a TIE is required, the cost increase is estimated to be $2,780 for each TIE. These increases are in addition to the current costs associated with toxicity testing. Assuming that the ESJWQC would conduct toxicity tests for 12 months at 6 sites, the increase in costs is estimated to be a minimum of $234,000 annually when compared to current toxicity testing costs. The addition of TIEs and additional sampling to meet management plan requirements increases this amount further. The ESJWQC estimates that if chronic toxicity testing were required, the toxicity analytical

20.6 cost would increase from $381,000 to $690,000 annually. This is a substantial increase in cost that is not discussed in the Economic Considerations report.

20.7 Further, the Staff Report does not discuss if the proposed numeric objectives can be reasonably achieved, considering the coordinated control of all factors that affect water quality. Considering the high bar that the numeric objectives proposed, and associated error rates with toxicity testing, it is not reasonable to expect that the numeric objectives will be achieved. 20.8 Accordingly, the purported Water Code section 13241 analysis is deficient and does not support the adoption of numeric toxicity objectives.

20.9 **Requirements for Channelized Dischargers Regulated Exclusively Under Porter-Cologne**

The Draft Toxicity Policy proposes to require use of the TST methodology to existing toxicity monitoring requirements, and recommends that all "channelized dischargers" implement a chronic toxicity testing monitoring program. We have several concerns with these requirements.

First, the introductory statement to this section states that, "[t]his section applies to monitoring of *discharges from channelized dischargers* regulated exclusively under the Porter-Cologne Water Quality Control Act (channelized dischargers) as defined in Part I(D)." (Draft Toxicity Policy, p. 14, emphasis added.) This statement implies that monitoring for toxicity should occur on the "discharge" – not the receiving water. Such a requirement is inappropriate. The irrigated agricultural programs primarily monitor surface waters to determine if the surface waters are meeting water quality standards. Water quality standards do not apply to end-of-pipe discharges from fields or to "channelized" discharges. Application of such standards at those points would constitute numeric effluent limitations, which are not practicable or feasible with respect to application to agriculture. (See, e.g., Staff Report, pp. 47-48.) If the State Water Board determines that the Draft Toxicity Policy should apply to agriculture, at the very least this language needs to be revised to clarify that the section applies to the monitoring of receiving waters by irrigated agriculture subject to conditional waivers, conditional discharge prohibitions, and/or waste discharge requirements.

20.10 Second, with respect to the use of the TST method, we echo many of the concerns raised by others with respect to the use of the TST method for acute and chronic toxicity testing. Specifically, the TST method as proposed will lead to a significant number of false positive test results (i.e., incorrectly identifying non-toxic samples as toxic). Such a result is significant considering the fact that toxicity test results trigger many different requirements on permittees. For irrigated agricultural entities in the Central Valley, successive toxicity results may trigger the need for accelerated monitoring, the need to conduct TIEs, and/or agricultural management plans. All of these actions take considerable time and resources and should only be required if toxicity is truly an issue. Thus, we encourage the State Water Board to not require the TST method for toxicity testing.

20.11 → Third, the Draft Toxicity Policy *recommends* that irrigated agricultural programs implement quarterly chronic toxicity testing. As indicated previously, chronic toxicity testing is significantly more expensive than acute toxicity testing. Further, for irrigated agriculture there is no scientific or technical reason that would justify the significant increase in cost for the change from acute toxicity testing to chronic toxicity testing. The proposed draft monitoring and reporting program (MRP) for the ESJWQC will require chemical-specific monitoring in ambient surface water. The cost of analysis for the multiple new chemicals (that are ultimately agreed upon as being appropriate) will result in a substantial increase in the cost of chemical analysis. Monitoring for specific chemicals in surface waters coupled with the establishment of trigger limits will be protective of aquatic life in waterways of the Eastern San Joaquin River watershed. Requiring chronic toxicity testing will not provide additional protection above that already provided by agreed upon chemical-specific monitoring and the establishment of trigger limits. Acute toxicity testing is sufficient to identify additional contaminants such as ammonium that could cause toxicity but are not discharged from irrigated agriculture. Thus, chronic toxicity testing requirements, or any recommendation for chronic toxicity testing requirements, must be removed.

In summary, we encourage the State Water Board to significantly revise the Draft Toxicity Policy. At a minimum, the proposed water quality objectives must be deleted and the policy's application to agricultural dischargers must also be deleted.

If you have any specific questions with respect to these comments, please contact Theresa "Tess" A. Dunham at (916) 446-7979. Thank you.

Sincerely,

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Attachment
TAD:cr

ADDITIONAL TECHNICAL COMMENTS

The following technical comments address Part II (Toxicity Objectives) and Part III (Implementation Procedures) of the proposed Policy for Toxicity Assessment and Control. Comment 1 identifies a flaw in the basic toxicity testing methodology that compromises the ability of a toxicity test to identify true toxicity. Comment 2 points out the flaw in the rationale behind reversing the null and alternative hypotheses relative to classic statistical hypothesis testing.

20.12

1. Toxicity testing is based on pseudoreplication and therefore not a valid evaluation of the toxicity of environmental samples or effluent

The statistical testing of an environmental sample and the laboratory control are a classic case of "pseudoreplication" as originally defined by Hurlbert (1984). Pseudoreplication is defined as "the use of inferential statistics to test for treatment effects with data from experiments where either treatments are not replicated (though samples may be) or replicates are not statistically independent." In the case of toxicity testing, a single sample is collected from a water body in a single glass bottle. That bottle is returned to the laboratory and a small number of subsamples are poured from the same bottle into from 2 to 4 different containers. The test organisms are placed into the containers and the containers are treated as replicates. The null hypothesis is that the survival (as an example of one endpoint) of the organisms in the replicate containers from the environmental sample is the same as the survival of organisms in the replicate control containers. In the statistical world, performing this analysis involves performing the statistical test on subsamples from single samples from two treatments; one from the control water and one from the site (effluent or ambient) water. Hurlbert (1984) referred to this problem as "simple pseudoreplication." Suppose that two replicates are taken from a control sample and an environmental sample. Survival in the control containers are 8 and 9 organisms and in the environmental containers are 7 and 7 organisms. The means of these two groups are 8.5 and 7 and the t-test result is a failure to reject the null hypothesis of no differences between the means at $p = 0.1024$ (the control and the sample are not significantly different). Now suppose that there are four containers from each treatment and the control water survival in the four containers is 8, 9, 9, and 8 organisms and that survival in the environmental sample containers is 7, 6, 7, and 8 organisms. The means of these two groups are 8.5 and 7 and the t-test result is the rejection of the null hypothesis of no differences between the means at $p = 0.04$ (the control and the sample are significantly different). Statistical significance has been achieved simply by grabbing two additional subsamples of water from the same large container.

In the example above, the subsamples water are from the same bottle; the water has just been divided into four containers rather than two in order to gain statistical significance. Authors of the TST would argue that this is exactly the point of the new procedure; change the null hypothesis (but see below) and use more "replicates" to reduce the variance and make the statistical test more reasonable. However, this rationale demonstrates a fundamental misunderstanding of the term "replicates." Adding numerous small containers to the toxicity test only increases the number of subsamples from the single sample of environmental water or control water. It does not increase the number of replicates which by definition must be independent. Water from the same large bottle poured into several smaller containers does not constitute independent samples. There is still only one environmental sample and one control sample. The consequence of pseudoreplication is that it is not possible to assign a difference in the means of the two groups to any cause as there is only one sample for each of the two

20.12 treatments. In a laboratory setting, it may be claimed that the only variable that is different between the control and the treatment is a toxic compound, but there are other potential factors that could affect the outcome of the test including the location of the containers within environmental chambers, the way the water was replaced in one container relative to another, etc. In order for the toxicity test to be valid using the four small containers, the sample for each container must come from different bottles collected at slightly different times (for example a minute apart) from the field.

If the example used above is placed into the framework of the "null" and alternative hypotheses of the TST, the problem of pseudoreplication is the same. Using the survival of organisms in two containers, there is a failure to reject the null hypothesis of a difference between the two treatments, and with the results from the four containers in each treatment, one rejects the null hypothesis and accepts the alternative hypothesis of no differences. These tests are equally as invalid as the tests performed using the classical null and alternative hypotheses because there is still only a single sample of control water and a single environmental sample.

20.13 **2. Correct interpretation of statistical results of the TST does not allow an "acceptance" of the hypothesis of differences between control and treatment water**

Although there are several statistical arguments against using the TST, one of the basic aspects of the new procedure that has been overlooked is the interpretation of the results in the context of the null and alternative hypotheses. Traditional hypothesis testing in inferential statistics (like the t-test used in toxicity testing) is based on a null hypothesis of no differences among treatments and an alternative hypothesis that states that at least one of the treatments is different. A posteriori tests allow a determination of which treatments are different from other treatments, e.g. one of the treatments is different from the control group. But the important point is how the evidence from the statistical test is interpreted. Formally, the results of the statistical test allow one to either reject the null hypothesis of no differences, or fail to reject the hypothesis of no differences. "Accepting" the null hypothesis is not an option. Consequently, it is never possible to state unequivocally that the treatments are not different, or equivalently, that the treatments are "the same" (see example in point 2 below). All one can say is that the evidence presented using the sample drawn from the population does not allow the rejection of the null hypothesis at the confidence level (alpha value) established by the experimenter. The alpha value is typically 5% which means that the analyst is willing to fail to reject the null hypothesis 5 times out of 100 when the same statistical test is performed on 100 different samples drawn from a single population. Essentially, a failure to reject the null hypothesis means that there is a sample from the population which when used as the data in an inferential statistical test, results in a test statistic (e.g., t-value) for which the alpha value is greater than 0.05. The conclusion is that the null hypothesis is plausible. It is not possible to conclude that the null hypothesis is true. It is commonly misstated in numerous documents and on the internet that a statistical analysis leads to the conclusion that a hypothesis is true or has been proven. It is more correct to say that the evidence supports the hypothesis or is consistent with the hypothesis of no differences. Alternatively, one can definitively state that if the null hypothesis is rejected at $p < 0.05$, it can be concluded that the null hypothesis is false and the alternative hypothesis is true, i.e., there is a 95% probability that the differences between the control and environmental samples are due to the treatment rather than chance. Despite a plethora of published papers or sites on the internet that claim that if $p > 0.05$ in a statistical test, the null hypothesis is true, this interpretation is incorrect. The fact that this interpretation is commonly applied does not make it correct. **The correct interpretation of $p > 0.05$ as the failure to reject the null hypothesis is what should be used by the State and Regional Boards to use the results of toxicity**

20.13 **testing in a weight-of-evidence approach – not as a numeric water quality objective.** Using a weight-of-evidence approach, if other lines of evidence (e.g., water chemistry or benthic community analyses) suggest or indicate a negative impact of some stressor, the Regional Board could then conclude that beneficial uses are not being supported despite the failure of a toxicity test to indicate significant toxicity.

Interpreting the consequences of changing the null hypothesis to one that assumes a difference in some endpoint between the control and environmental samples can be placed into the context of the explanation provided above. Suppose, as proposed by the TST method, the "null" hypothesis being tested is that the survival of organisms in the ambient water (or effluent) is significantly lower than the survival of organisms in the control water. The alternative hypothesis is that there are no differences in survival between the control and any of the ambient samples. If the result of the test is a failure to reject the null hypothesis, i.e., the p value is greater than 0.05, the incorrect interpretation is that the null hypothesis is correct (or true) and there is a significant difference between the laboratory control and the environmental sample. It can never be concluded that those differences truly exist or if they are an artifact of the single sample collected or laboratory procedures. Failure to reject the null hypothesis only indicates that the single sample (and several subsamples poured from the single bottle) from the control and environmental water are different but the differences can't be attributed to any specific factor. The correct interpretation is that the results of the test are consistent with the hypothesis of a difference between the laboratory control and the environmental sample. Consequently, in a regulatory setting the TST is the opposite of what is needed to definitively state that a sample is toxic.

Literature cited

Hurlbert, S. H. 1984. Pseudoreplication and the design of ecological field experiments. *Ecological Monographs* 54:187 - 211.