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November 29, 2007

Ms. Jeanine Townsend, Acting Clerk to the Board
Executive Office, State Water Resources Control Board
P. O. Box 100
Sacramento, California, 95812-0100

Re: Comments on sediment quality objectives proposed in the SWRCB draft policy.

Dear Chair Doduc and Members of the Board:

By introduction, I am a toxicologist who has taught and practiced toxicology in California for the past 33 years. Since 1984, I have worked on sediment quality issues for stakeholders in the Newport Bay Watershed. My curriculum vitae is attached.

I am writing to provide comments on the draft report: Water Quality Control Plan for Enclosed Bays and Estuaries. Part I. Sediment Quality. The report was issued on September 27, 2007 by the staff of the State Water Resources Control Board (SWRCB). The comments will deal specifically with the proposed sediment quality objectives (SQOs) for DDT and chlordane.

My concern with the draft report is the lack of application of basic toxicological principles in deriving SQOs. Toxicology relies on establishing dose-response by studying the effects of different doses of a chemical on a biological system. These are controlled studies that provide information on the dose-response relationship and toxicity thresholds. Modeling and statistics are then applied to address uncertainty. The methods are well established and can be found in numerous texts and in U. S. EPA reports on human and ecological risk assessment. These well established risk assessment methodologies appear to have been overlooked in the derivation of sediment quality objectives.

The draft report proposes the use of multiple lines of evidence to determine which chemicals are causing toxicity in sediments. The triad of chemical analysis, sediment toxicity and benthic health are cited as the basis for the multiple lines of evidence. However, the most important line of evidence in determining causation is the result of spiked-sediment bioassays. In the absence of spiked-sediment bioassay results, sediment thresholds for toxicity can be estimated from water column bioassays by equilibrium partitioning. Toxicity thresholds and dose-response from these two types of bioassays, since they directly measure toxicity due to a

chemical, should have the greatest weight in determining whether a chemical is toxic at a given level in sediments.

Unfortunately, neither of the two proposed SQOs are based on dose-response or toxicity thresholds. The California LRMs and CSIs are statistical metrics of paired chemistry and sediment toxicity/benthic health measurements. The presence of a chemical in a toxic sediment does not prove causation and should not be the basis for any SQO. Hundreds of chemicals are often present in toxic sediments, some known to be at clearly toxic levels, although most are not (for example, see Chapman et al., 1987).

To assert that 1.52 ppb DDT in sediment is the threshold for moderate toxicity to the benthos (Table 6, page 14 of Appendix A), stated on page 14 of Appendix A to mean that moderate toxicity is: "Clear evidence of sediment pollutant exposure that is likely to result in biological effects;...", is incorrect and illogical in light of the findings of sediment bioassays. Schuytema et al. (1989) observed no mortality in *Hyalella azteca* at 1,300 ppb DDT. At levels of 573 ppb and 1,230 ppb, DDT in sediments did not significantly increase mortality in *Rhepoxynius abronius* (EVS Environmental Consultants, 1994). Donald D. MacDonald concluded that the benthic toxicity threshold for DDT in sediments in the Southern California Bight was 7,210 ppb (MacDonald, 1994).

The California LRMs and CSIs are validated in the draft report by comparison to published sediment quality guidelines (SQGs). Unfortunately, the comparison SQGs also rely largely on association rather than on dose-response and toxicological thresholds. In addition, the ERM for chlordane (Long and Morgan, 1990) and the PEL for DDT (MacDonald et al., 1996) contain flawed data sets (see Flow Science et al., 2006). The 1995 publication cited as the source of the ERM for DDT (Long et al., 1995) does not contain the data set from which the ERM is derived. It is not possible to know whether an ERM is valid without knowing the underlying data.

In assessing chemical causation of sediment toxicity, <u>all</u> lines of scientific evidence, particularly spiked-sediment bioassays and equilibrium partitioning from water column bioassays, should be considered. The proposed California LRM and CSI sediment quality objectives overestimate the toxicity potential of DDT and chlordane by several orders of magnitude. The use of these proposed objectives for DDT and chlordane would waste valuable resources that could be used to address the real causes of sediment toxicity.

I would be happy to respond to any questions concerning the above comments.

Sincerely,

James L. Byard

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references and curriculum vitae attached

REFERENCES

Chapman, P. M., Dexter, R. N. and E. R. Long, Synoptic measures of sediment contamination, toxicity and infaunal community composition (the sediment quality triad) in San Francisco Bay. Marine Ecology Progress Series 37: 75-96, 1987.

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Flow Science Inc., Byard, J. L. Tjeerdema, R. S. and QEA Consultants Inc., DDT analysis for the Newport Bay Watershed, stakeholder technical report submitted to the SARWQCB for the OC TMDL, November 20, 2006.

Long E. R. and L. G. Morgan, The potential for biological effects of sediment-sorbed contaminants tested in the national status and trends program. NOAA technical memorandum NOS OMA 52, Seattle, Washington, 1990.

Long, E. R., MacDonald, D. D., Smith, S. L. and F. D. Calder, Incidence of adverse biological effects within ranges of chemical concentrations in marine and estuarine sediments. Environmental Management 19: 81-97, 1995.

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Schuytema, G. S., Nebecker, A. V., Griffis, W. L. and C. E. Miller, Effects of freezing on toxicity of sediments contaminated with DDT and endrin. Environmental Toxicology and Chemistry 8: 883-891, 1989.

CURRICULUM VITAE

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EDUCATION

B.S., Biochemistry, Cornell University, 1960-1964

Ph.D., Biochemistry, University of Wisconsin, 1964-1968

Postdoctorate, Biological Chemistry, Harvard Medical School, 1968-1970

HONORS

Babcock Fellow, University of Wisconsin, 1967-1968

Arthritis Fellow, Harvard Medical School, 1968-1970

CERTIFICATIONS

Diplomate of the American Board of Toxicology, 1980-present

PROFESSIONAL SOCIETIES

Society of Environmental Toxicology and Chemistry

Society of Toxicology

EMPLOYMENT

Sole Proprietor of James L. Byard, Toxicology Consultant, 1984 - present. Consulting in basic and applied research in toxicology, risk assessment, auditing toxicity studies, environmental fate of chemicals, and testimony as an expert witness.

Lecturer, Department of Environmental Toxicology, University of California, Davis, California 95616, 2006 - present. Teaching University courses in toxicology.

Adjunct Associate Professor, Distinguished Visiting Scholar, and Lecturer, Department of Environmental Toxicology, Univeristy of California, Davis, California 95616 (1984-1995). Teaching University courses in toxicology.

Assistant and Associate Professor of Environmental Toxicology, Department of Environmental Toxicology, University of California, Davis (1974-1984). Teaching, research, and public service in toxicology. Research in chemical carcinogenesis, metabolism, mechanism-of-action, and primary liver cell cultures.

Research Assistant Professor of Toxicology, Center of Experimental Pathology and Toxicology, Albany Medical College of Union University, Albany, New York 12208 (1970-1974). Teaching in toxicology and biochemistry. Research in metabolism and mechanism-of-action of saccharin, carrageenan, dieldrin, mirex, PCBs, hexachlorobenzene, methyl mercury, and freons.

CONSULTING EXPERIENCE

Reviewed NIOSH criteria document for benzylchloride.

Reviewed EPA drinking water criteria document for dibromochloropropane.

Participated in the laetrile hearings in the California Governor's Office.

Gave written and oral testimony to Proposition 65 Scientific Advisory Panels, State and Regional Water Boards, and District Air Pollution Boards.

Consulted with the California Department of Pesticide Regulation, Office of Environmental Health Hazard Assessment, U. S. Environmental Protection Agency, and the U. S. Food and Drug Administration

Toxicology consultant to the Health Effects Study of the Replenishment of Ground water with Treated Waste Water, County Sanitation Districts of Los Angeles County.

Member of the California Department of Health Service's Water Reuse Health Effects Panel.

Developed a surface and ground water monitoring program for Alpine County, California.

Chaired a two-day conference on chemical carcinogenesis and teratology for the California Air Resources Board.

Toxicology consultant to several engineering firms dealing with cleanup of hazardous wastes (e.g., Rocky Mountain Arsenal, Brio Refining, THAN- Fresno, BKK Landfill, Concord Naval Weapons Station; Operating Industries Landfill, Kopper's Oroville site, Silicon Valley groundwater contamination, Lincoln Village, etc.).

Consultant to several chemical companies (e. g., Monsanto, Syntex, IBM, U. S. Borax, Du Pont, TH Agriculture and Nutrition, etc.). Assignments include risk assessment, audits of toxicology studies, human exposure studies, and genetic toxicology studies.

Consultant to the California Rice Industry Association (risk assessment of rice pesticides and rice smoke).

Consultant to The Irvine Company (predevelopment hazard assessments, Proposition 65 compliance, pesticides and metals in aquatic environments).

Evaluation of the hazards of consumer products to meet regulations of the Consumer Product Safety Commission.

Consultant/expert witness for numerous legal cases involving human exposure to aldrin, ammonia, asbestos, benzene, brodifacoum, cadmium, carbon monoxide, chlordane, chlorine, chloroform, chlorpyrifos, chromium, creosote, 2,4-D, DBCP, DDT, diazinon, dieldrin, diesel fuel, dioxin, endrin, ethyl ether, formaldehyde, freon 113, gasoline, heptachlor, hexane. isopropyl alcohol, lead, marijuana, mercury, methyl bromide, methylene chloride, methyl ethyl ketone, methyl isobutyl ketone, mixed hydrocarbon solvents, paraquat, parathion, PAHs, PCBs, pentachlorophenol, perchlorate, perchloroethylene, phosdrin, selenium, silica, silvex, sulfur oxides, 2,4,5-T, toluene, toxaphene, trichloroethane, trichloroethylene, vinyl chloride, vinylidene chloride, xylene, etc.

EXAMPLES OF TECHNICAL REPORTS

1. **Assessment of legacy pesticide and PCB TMDL targets**
2. **Selenium concentrations in waterfowl eggs from the San Joaquin Wildlife Refuge.**

3. Risk assessment of the Denver Rail Yard, site of the Coors Baseball Field.

4. Risk assessment of vehicle emissions contaminating the Sweetwater Reservoir.

5. Comparison of hazardous materials in household wastes and industrial liquid wastes.

6. Annotated bibliography of industrial vitiligo.

7. Annotated bibliography of carbon monoxide poisoning.

8. Report on monitoring of rice pesticide residues in the United States and Japan.

9. Hazard assessment of amorphous silica in rice straw smoke.

10. Annotated bibliography of 2,4-D, 2,4,5-T, and 2,3,7,8-tetrachloro-dibenzo-*p*-dioxin.

11. Annotated bibliography of the acute dose-response of ammonia in humans.

12. Annotated bibliography of the acute dose-response of sulfur dioxide in humans.

13. Toxicant dynamics in an urban watershed.

PUBLICATIONS

1. Byard, J. L., The Impact of Rice Pesticides on the Aquatic Ecosystems of the Sacramento River and Delta (California). Reviews of Environmental Contamination and Toxicology 159: 95-110, 1999.

2. Byard, J. L., Hazard Assessment of 1,1,1-Trichloroethane in Ground Water. In The Risk Assessment of Environmental Hazards, D. Paustenbach, ed., pp 331-344, John Wiley & Sons, New York, 1989.

3. Byard, J. L., The Toxicological Significance of 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Related Compounds in Human Adipose Tissue. Journal of Toxicology and Environmental Health 22: 381-403, 1987.

4. Byard, J. L. and Dougherty, K. K., Comparative Metabolism and Toxicity of Chemical Carcinogens in Primary Cultures of Hepatocytes. In Vitro 21: 489-494, 1985.

5. Milam, K. M. and Byard, J. L., Acetaminophen Metabolism, Cytotoxicity, and Genotoxicity in Rat Primary Hepatocyte Cultures. Toxicol. Appl. Pharmacol. 79: 342-347, 1985.

6. Byard, J. L., editor, Biological Effects of Toxicants. A textbook in toxicology, 1983.

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8. Knadle, S. A., The Kinetics of Benzene Metabolism in Rhesus Monkey Hepatocytes Cultured in Glass T-flasks, Ph.D. Thesis, University of California, Davis, 1982 (Chairperson of thesis committee).

9. Salocks, C. B., Hsieh, D. P. H. and Byard, J. L., Effects of Butylated Hydroxy-toluene Pretreatment on the Metabolism and Genotoxicity of Aflatoxin B_1 in Primary Cultures of Adult Rat Hepatocytes: Selective Reduction of Nucleic Acid Binding. Toxicol. Appl. Pharmacol. 76: 498-509, 1984.

10. Steward, A. R. , Induction of Benzo(a)pyrene Metabolism by 2,3,7,8-Tetrachloro-dibenzo-p-dioxin in Primary Cultures of Adult Rat Hepatocytes. Regulation by Retinol Acetate and Serum, Ph.D. Thesis, University of California, Davis, 1982.

11. Loury, D. J. and Byard, J. L., Genotoxicity of the Cooked-Food Mutagens IQ and MeIQ in Primary Cultures of Rat, Hamster and Guinea Pig Hepatocytes. Environmental Mutagenesis 7: 245-254, 1985.

12. Loury, D.J., Kado, N.Y. and Byard, J.L., Enhancement of Hepatocellular Genotoxicity of Several Mutagens from Amino Acid Pyrolysates and Broiled Foods Following Ethanol Pretreatment. Food Chem. Toxicol. 23: 661-667, 1984.

13. DiRenzo, A. B., Gandolfi, A. J., Sipes, I.G., Brendel, K. and Byard, J. L., Effect of O_2 Tension on the Bioactivation and Metabolism of Aliphatic Halides by Primary Rat-Hepatocyte Cultures. Xenobiotica 14: 521-525, 1984.

14. Loury, D. J., Byard, J. L. and Shibamoto, T., Genotoxicity of N-Nitrosothiazolidine in Microbial and Hepatocellular Test Systems. Food Chem. Toxicol. 22: 1013-1014, 1984.

15. Byard, J. L., Metabolism of Food Toxicants: Saccharin and Aflatoxin B_1, A Contrast in Metabolism and Toxicity. In Nutritional and Toxicological Aspects of Food Safety, M. Friedman, ed., pp 147-151, Plenum Press, New York, 1984.

16. Loury, D. J., Hsieh, D. P. H. and Byard, J. L., The Effect of Phenobarbital Pretreatment on the Metabolism, Covalent Binding and Cytotoxicity of Aflatoxin B_1 in Primary Cultures of Rat Hepatocytes. J. Toxicol. Environ. Health 13: 145-159, 1984.

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20. Green, C. E., Segall, H. J. and Byard, J. L., Metabolism, Cytotoxicity and Genotoxicity of the Pyrrolizidine Alkaloid Senecionine in Primary Cultures of Rat Hepatocytes. Toxicol. Appl. Pharmacol. 60: 176-185, 1981.

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23. Steward, A. R. and Byard, J. L., Induction of Benzo(a)pyrene Metabolism by 2,3,7,8-Tetrachlorodibenzo- p-dioxin in Primary Cultures of Adult Rat Hepatocytes. Toxicol. Appl. Pharmacol. 59: 603-616, 1981.

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31. Byard, J. L., McChesney, E. W., Golberg, L. and Coulston, F., Excretion and Metabolism of Saccharin in Man. II. Studies With ^{14}C-Labelled and Unlabelled Saccharin. Food Cosmetics Toxicology 12: 175-184, 1974.

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PUBLISHED ABSTRACTS OF PRESENTATIONS GIVEN AT NATIONAL MEETINGS

1. Loury, D. J. and Byard, J. L., Subchronic Ethanol Administration Enhances the Hepatocellular Genotoxicity of Several Pyrolysate Mutagens. Toxicologist 4: 33, 1984.

2. DiRenzo, A.B., Gandolfi, A. J., Brendel, K., Sipes, I. G. and Byard, J. L., Effect of Hypoxia on the Bioactivation and Toxicity of CCl_4 and Halothane in Primary Rat Hepatocyte Cultures. Pharmacologist 25: 170, 1983.

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5. Knadle, S. A. and Byard, J. L., The Kinetics of Benzene Metabolism in Primary Hepatocyte Cultures of Guinea Pig and Rat Compared to Inhalation Uptake Kinetics. Toxicologist 3: 87, 1983.

6. Knadle, S. A., Salocks, C. B., Nakashima, J. and Byard, J. L., Comparative Rates of Benzene Metabolism in Primary Hepatocyte Cultures. Toxicologist 2: 22-23, 1982.

7. Steward, A. R. and Byard, J. L., Effect of Vitamin A on the Induction of Benzopyrene Metabolism by 2,3,7,8-Tetra- chlorodibenzodioxin in Primary Hepatocyte Cultures. Pharmacologist 23: 179, 1981.

8. Gill, S. S., Hammock, B. D. and Byard, J. L., Comparative Metabolism of Stilbene Oxides by Primary Hepatocyte Cultures. Toxicologist 1: 141-142, 1981.

9. Spilman, S. D. and Byard, J. L., Sulfate-Dependent Metabolic Activation of 2-Acetylaminofluorene by Primary Cultures of Adult Rat Hepatocytes. Toxicologist 1: 35, 1981.

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11. Salocks, C. B., Hsieh, D. P. H. and Byard, J. L., Butylated Hydroxytoluene Pretreatment Reduces Cytotoxicity and Covalent Binding of Aflatoxin B_1 in Primary Hepatocyte Cultures. Toxicologist 1: 108-109, 1981.

12. Green, C. E., Segall, H. J. and Byard, J. L., Metabolic Fate and Toxicity of Senecionine in Primary Hepatocyte Cultures. Abstracts of the Nineteenth Annual Meeting of the Society of Toxicology, A44, 1980.

13. Steward, A. R. and Byard, J. L., Induction of Benzopyrene Metabolism by 2,3,7,8-Tetrachlorodibenzo-p-dioxin in Primary Cultures of Adult Rat Hepatocytes. Abstracts of the Nineteenth Annual Meeting of the Society of Toxicology, A85, 1980.

14. Dougherty, K. K. and Byard, J. L., Induction of Mixed Function Oxidase by Phenobarbital, Hormones and Serum in Primary Cultures of Mouse Hepatocytes. Fed. Proc. 38: 846, 1979.

15. Spilman, S. D. and Byard, J. L., Metabolism of 2-Acetylaminofluorene in Primary Cultures of Rat Hepatocytes. Pharmacologist 20: 175, 1978.

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17. Wei, C. I., Decad, G. M., Wong, Z. A., Byard, J. L. and Hsieh, D. P. H., Characterization and Mutagenicity of Water-Soluble Conjugates of Aflatoxin B_1. Toxicol. Appl. Pharmacol. 45: 274, 1978.

18. Dougherty, K. K. and Byard, J. L., Induction of Mixed- Function Oxidase in Primary Cultures of Mouse Hepatocytes. Toxicol. Appl. Pharmacol. 45: 261, 1978.

19. Dougherty, K. K., Spilman, S. D., Green, C. E., Steward, A. R. and Byard, J. L., Primary Hepatocyte Cultures for the Investigation of the Fate and Mechanism of Action of Environmental Chemicals. Toxicol. Appl. Pharmacol. 41: 190, 1977.

20. Byard, J. L. and Pittman, K. A., Early Liver Changes Produced by Mirex and Their Reversibility. Toxicol. Appl. Pharmacol. 33: 130, 1975.

21. Griffin, T.B., Byard, J. L. and Coulston, F., Golberg, L. and Harris, E.S. Continuous Exposure of Rats to Hexafluoroethane. Toxicol. Appl. Pharmacol. 29: 82, 1974.

22. Byard, J. L., Koepke, U. Ch., Abraham, R., Golberg, L. and Coulston, F., Biochemical Changes Produced in the Liver by Mirex. Toxicol. Appl. Pharmacol. 29: 126-127, 1974.

23. Byard, J. L., McChesney, E., Golberg, L. and Coulston, F., Further Observations on the Metabolism of Saccharin in Man. Toxicol. Appl. Pharmacol. 29: 154-155, 1974.

24. Byard, J. L., Observations on the Metabolism of Saccharin. Toxicol. Appl. Pharmacol. 22: 291-292, 1972.

25. Byard, J. L. and Bauman, C. A., Protein-Bound Selenium in Rats Given Sodium Selenite. Fed. Proc. 27: 417, 1968.

26. Byard, J. L. and Bauman, C. A., Selenium Metabolites in the Urine of Rats Given a Subacute Dose of Selenite. Fed. Proc. 26: 476, 1967.

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