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Proposed Amendment to California's Recycled Water Policy

Comments

The California State Water Resource Control Board (SWRCB) is requesting comments on a Draft Amendment to the Recycled Water Policy that was developed in response to a June 2010 report titled, "Monitoring Strategies for Chemicals of Emerging Concern (CECs) in Recycled Water - Recommendations of a Scientific Advisory Panel."

The Panel report on p 58 provided recommendations for monitoring specific CECs in recycled water used for groundwater recharge reuse. SWRCB incorporated the Scientific Advisory Panel's recommendations into the proposed Draft Amendment, which includes two parts: some editing of the original Recycled Water Policy and a new Attachment A, which describes the monitoring requirements.

This comment below contains an extract from the Orange County Groundwater Replenishment (GWR) Independent Advisory Panel report of the March 30-31, 2010 meeting of that group. It is self-explanatory. It addresses the matter of appropriate rationales for monitoring water reuse product water. In general, the GWR panel disagreed with the rationales in the SWRCB report that identified triclosan and caffeine to be monitored as chemicals of **health concern** in water reuse applications. The GWR panel felt that those chemicals are of little toxicological relevance. In addition, their exposures from other sources are several orders of magnitude greater than from treated reuse water, and so listing them as of toxicological concern was not supported. It is important that there be a clear distinction between CECs used to evaluate operations versus those which pose health risks.

The GWR Panel recommended that Orange County Water (OCWD) develop a monitoring strategy and rationale for the inclusion of analytes and frequencies and provide them for review by the Panel before making a full commitment to this endeavor. Case-by-case flexibility should be provided so that monitoring can be tailored to the specific source/technology relationship, and wasteful monitoring for the sake of monitoring can be avoided. The data from several reuse projects demonstrate that there are very limited occurrences of any chemicals of interest in today's well designed reuse projects. NDMA and 1,4 dioxane are probably the best candidates, by far, because of the difficulty of removing them. On the other hand, evidence shows that exposures to NDMA and nitrosamines from drinking water, say at 10 ng/L, are considerably less

than 0.1% of body burden from total dietary and endogenous sources. For dioxane, the USEPA HA is 0.035 mg/L @ nominal 1/10,000 hypothetical lifetime risk. Although 17 β -estradiol would be a concern, if present in processed water for drinking water, the likelihood of its presence is quite small in a process that included ozone or granular carbon or reverse osmosis.

From the report:

“The Panel had considerable difficulty understanding the rationale for the recommendations in the SAP report on chemicals in regard to toxicological relevance. The Panel concluded that the most reasonable basis for monitoring in a GWR-type treatment system was to utilize the analytes as treatment performance indicators and to select surrogates for numerous other chemicals that would therefore not be monitored. Among the chemicals considered by the SAP, those of greatest interest for that purpose were NDMA and 17 β -Estradiol (although it is unlikely to survive the process) which combine potential presence and potential health concern if they occurred at higher than expected levels. 1,4-Dioxane is also a good choice due to its known inefficient removal by RO. UCMR3 chemicals would best be addressed selectively, unless there is a regulatory requirement to analyze them. Chemicals like triclosan, caffeine, and sucralose and other artificial sweeteners are of no toxicological interest. Hydrazine and quinoline are of little interest and unlikely to survive an RO/AOP process. Nicotine and cotinine could be considered for inclusion since they are cigarette-related and likely to be in sewage as well as being of toxicity interest if at high enough levels in the finished water. They are also relatively lower molecular weight molecules that could challenge RO, but not likely ozone or AOP.”

7.4 “Two of the four chemicals that were identified by the SWRCB-sponsored SAP for monitoring due to toxicological relevance were caffeine and triclosan. The Panel could not understand a rationale for including those two chemicals as being of toxicological concern. The triggering values likely were adopted from the Australian Guidelines for Water Recycling without apparent understanding of the process that was used to derive the levels of health concern. Essentially, the Australian Guidelines developed these figures utilizing the Thresholds of Toxicological Concern (TTC), and it appears that they did not appreciate the inappropriate use of this approach for chemicals of known toxicity. The intent of the TTC was to identify compounds whose exposure was of sufficient concern that compound-specific data were needed to make a risk assessment (Kroes et al., 2004)¹. The process was initially based on an analysis of the distributions of no observed adverse effect levels (NOAELs) in toxicological studies in the U.S. Food and Drug Administration databases, but has been expanded to include many chemicals with diverse uses. The exposure thresholds are intentionally conservative and are used as a means of triage among chemicals without the toxicological data needed for proper risk assessment. Chemicals with exposures below the threshold are considered to pose little hazard, while those above the threshold are identified for toxicological characterization to develop a dataset that is appropriate for risk assessment. In no case are these thresholds looked upon as risk assessments. They are only used for compounds for which there are

¹ Kroes, R., A.G. Renwick, M. Cheeseman, J. Kleiner, I. Mangelsdorf, A. Piersma, B. Schilter, J. Schlatter, F. van Schothorst, J.G. Vos, and G. Würtzen. 2004. Structure-based thresholds of toxicological concern (TTC): guidance for application to substances present. *Food Chem. Toxicol.* 42(1):65-83.

no toxicological data. This latter assumption is not true of either caffeine or triclosan. A large body of literature exists on the adverse and beneficial effects of caffeine – more than enough to assess risks and derive figures appropriate as goals. There also is a substantial database for triclosan as presented in the Registration Eligibility Document (RED) published by the Office of Prevention, Pesticides & Toxic Substances (OPPTS) (U.S. EPA, 2008)². The Panel does not object to the use of these chemicals for monitoring the effectiveness of treatment, but in neither case do the concentrations of these two contaminants approach levels of health concern in wastewater, much less water treated for potable reuse. Both NDMA and 17 β -estradiol would represent health concerns at appropriate low levels. To place the same emphasis on caffeine and triclosan greatly distorts their importance as health concerns.”

“To indicate the breadth of the literature base for these two compounds, we have selected a few of the publications that are available and appropriate to the purposes of performing risk assessments for both. These references are provided in **Appendix D below**”.

APPENDIX D – References for Caffeine and Triclosan

Caffeine

Browne, M.L. 2006. Maternal exposure to caffeine and risk of congenital anomalies: a systematic review. *Epidemiology* 17(3):324-331.

Carrillo, J.A. and J. Benitez. 1996. CYP1A2 activity, gender and smoking, as variables influencing the toxicity of caffeine. *Br. J. Clin. Pharmacol.* 41:605-608.

Christian, M.S. and R.L. Brent. 2001. Teratogen update: evaluation of the reproductive and developmental effects of caffeine. *Teratology* 64(1):51-78.

Geleijnse, J. M. 2008. Habitual coffee consumption and blood pressure: An epidemiological perspective. *Vascular Health and Risk Management* 4(5):963-970.

Kaida, A., L. Yu, E. Oztas, and J.F. Chen. 2006. Novel neuroprotection by caffeine and adenosine A(2A) receptor antagonists in animal models of Parkinson’s disease. *J. Neurol. Sci.* 248(1-2):9-15,

Lopez-Garcia, E., F. Rodriguez-Artalejo, K. M. Rexrode, G. Logroscino, F. B. Hu, and R. M. van Dam. 2009. Coffee consumption and the risk of stroke in women. *Circulation* 119(8):1116-1123.

² U.S. EPA. 2008. Reregistration Eligibility Decision for Triclosan. List B, Case No. 2340. EPA 739-RO-8009. Office of Prevention, Pesticides and Toxic Substances (7510P), U.S. Environmental Protection Agency, Washington, DC

Nehlig, A. and G. Debry. 1994. Potential teratogenic and neurodevelopment consequences of coffee and caffeine: a review on human and animal data. *Neurotoxicol. Teratol.* 16(6):531-543.

Nyska, A., E. Murphy, J. F. Foley, B. J. Collins, J. Petranka, R. Howden, P. Hanlon, and J.K. Dunnick. 2004. Acute hemorrhage myocardial necrosis and sudden death of rats exposed to a combination of ephedrine and caffeine. *Toxicol. Sci.* 83:388-396.

Rashid, A., M. Hines, B.J. Scherlag, W.S. Yamanashi, and W. Lovallo. 2006. The effects of caffeine on the inducibility of atrial fibrillation. *J. Electrocardio.* 39(4):42-435.

Sata, F., H. Yamada, K. Suzuki, Y. Saijo, E. H. Kato, M. Morikawa, H. Minakami, and R. Kishi. 2005. Caffeine intake, CYP1A2 polymorphism and risk of recurrent pregnancy loss. *Mol. Human Reprod.* 11(5):357-360.

Zhang, W. L., E. Lopez-Garcia, T.Y. Li, F.B. Hu, and R.M. van Dam. 2009. Coffee consumption and risk of cardiovascular events and all-cause mortality among women with type 2 diabetes. *Diabetologia* 52(5):810-817.

Zhang, W., F.B. Hu, E. Lopez-Garcia, R.M. van Dam, and T. Y. Li. 2009. Coffee consumption and risk of cardiovascular diseases and all-cause mortality among men with type-2 diabetes. *Diabetes Care* 32(6):1043-1045.

Triclosan

Kumar, V., A. Chakraborty, M.R. Kural, and P. Roy. 2009. Alteration of testicular spermatogenesis and histopathology of reproductive system in male rats treated with triclosan. *Reprod. Toxicol.* 27(2):177-185.

Paul, K.B., J.M. Hedge, M.J. DeVito, and K.M. Crofton. 2010. Short-term exposure to triclosan decreases thyroxine in vivo via upregulation of hepatic catabolism in young Long-Evans rats. *Toxicol. Sci.* 113(2):367-379.

Rodricks, J.V., J.A. Swenberg, J. F. Borzelleca, R.R. Maronpot, and A. M. Shipp. Triclosan: a critical review of the experimental data and development of margins of safety for consumer products. *Crit. Rev. Toxicol.* 40(5):422-484.

Stoker, T.E., E.K. Gibson, and L.M. Zorrilla. 2010. Triclosan exposure modulates estrogen-dependent responses in the female Wistar rat. *Toxicol. Sci.* 117(1):45-53.

U.S. EPA. 2008. Reregistration Eligibility Decision for Triclosan. List B, Case No. 2340. EPA 739-RO-8009. Office of Prevention, Pesticides and Toxic Substances (7510P), U.S. Environmental Protection Agency, Washington, DC