Reviewer's report

**Title:** Affinity for risky behaviors following prenatal and early childhood exposure to tetrachloroethylene (PCE)-contaminated drinking water: a retrospective cohort study

**Version:** 1 **Date:** 26 May 2011

**Reviewer:** Sylvaine Cordier

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**Introduction**

1. Most references refer to occupational exposures which occur mostly through inhalation or dermal contact. How is the general population exposed to PCE through water contamination: ingestion only? or showering and bathing as this is the case for disinfection by-products?
2. Bockelman 2002 is not in the reference list.
3. A recent review (Brown Dzubow R, 2010) deserves to be cited.

**Methods**

4. What is the justification of one mile of VL/AC pipe as the threshold for exposure?
5. Comparison between participants and non participants need to include prevalence of smoking and of alcohol drinking among parents, and whether these differences are similar among exposed and non exposed
6. How is “binge drinking” assessed from the questions asked since quantity is asked per day?
7. Maternal water uses were known for only a portion of the study population, but the influence of taking into account these habits should be evaluated on this sub sample.
8. What is the influence of water source on PCE concentration? The assumption that water sources have not changed between 1970 and 1988 (the end of the 5-year “childhood exposure” if I understand right) is strong, and from what the authors write, not likely to be true after 1980. How would deviance from this assumption influence estimates of early childhood exposure?
9. Seasonal variations of exposure (likely to occur because of high volumes of water use during the touristic season for instance) are important to take into account to estimate exposure during the prenatal period. Has this been checked?
10. The decision to group together “prenatal and childhood exposure” on one side and “childhood only” on the other side is debatable. Why not build separate indices, one for prenatal exposure, one for childhood exposure and let them compete for prediction of outcome?
11. The procedure for the choice of confounders is not clearly described. In particular, I do not understand the following sentence: “Categorical variables were retained for further consideration if there was more than a 5% difference (of what??) between compared groups”. Please explain.

Results
12. present separate distributions for cumulative PCE exposures for prenatal and childhood
13. in most of the associations with PCE exposure tertiles, the increased risk, if any, is apparent only in the highest tertile. Do the corresponding communities have something in common (socioeconomic environment for example) besides PCE exposure
14. tables 4-9: the eligible population in each table (definition, size) should be clearly defined for each table, perhaps in the title. It appears to be 1364 in Table 4, 1339 in Table 5, 1139 in Table 6 etc…
15. tables 5, 6, 7, 9: delete “continued” in the title of the first page of each table
16. table 5, first page: the highest tertile is missing for the variable “Drank >=5/4 drinks/drinking day as teen”

Discussion
17. please discuss possible confounding by contextual municipality environment
18. add discussion about possible differential participation of families with smoking or alcohol drinking mothers during pregnancy
19. use of alcohol consumption studies as an example of solvent-induced developmental effects is debatable since intergenerational transmission of addictive behavior is also likely to be involved (see Melchior, 2010; 2011)
20. how does the estimated level of exposure to PCE compares to LOAEL in experimental studies or other human effects?

Level of interest: An article of importance in its field

Quality of written English: Acceptable

Statistical review: Yes, but I do not feel adequately qualified to assess the statistics.

Declaration of competing interests:
I declare that I have no competing interests