Richard Kapuscinski

The subject (draft) Memo (page 8) claims that "The damage [i.e., serious heart defects in a developing fetus due to a brief exposure] can be caused by relatively low TCE concentrations that are similar to concentrations associated with other critical cancer and non-cancer effects that result from long-term (many years) exposure". The location of that sentence, relative to the preceding information, in the Memo (e.g., "According to the EPA,") could leave a casual reader with the impression that the U.S. Environmental Protection Agency (EPA) (e.g., in its toxicity assessment in its Integrated Risk Information System) is the source of that claim. To the contrary, the TCE-related memo issued by the federal Superfund program (Compilation of Information Relating to Early/Interim Actions at Superfund Sites and the TCE IRIS Assessment. Memorandum from Robin Richardson. Office of Superfund Remediation and Technology Innovation, Washington, D.C. August 27, 2014. Currently available on-line at: https://semspub.epa.gov/work/HQ/174044.pdf) observes that: "the RfC for a single exposure hasn't been determined yet by EPA." I am unaware of any subsequent derivation by any EPA office or program of a single-exposure or short-term RfC considering potential cardiac effects in fetuses.

To the extent that the Washington Department of Ecology (WADoE) intends to retain the draft claim (highlighted above) in its final Memo, I would ask and encourage that you make clear that the claim is yours (or, alternatively, cite supporting, peer-reviewed evidence published by other parties). On the other hand, perhaps WADoE's proposed guidance and policy can instead be rationalized on the basis of "an abundance of caution", rather than that fetal damage is necessarily "caused by low TCE concentrations" (e.g., by any exceedance of the chronic RfC for TCE in IRIS).