UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION 2
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OCT 2 1 1999

Dr. George Putman
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Dear Dr. Putman:

This is in response to your July 1, 1999 letter commenting on the U.S. Environmental Protection Agency's (EPA's) April 1999 Responsiveness Summaries for the Phase 2 Ecological Risk Assessment Scope of Work and the Phase 2 Human Health Risk Assessment Scope of Work.

As you know, the public comment period on the Human Health Risk Assessment Scope of Work ended on August 31, 1998 and the public comment period on the Ecological Risk Assessment Scope of Work ended on November 2, 1998. EPA responded to all significant comments in the Responsiveness Summaries, but did not solicit public comment on the Responsiveness Summaries. Nevertheless, EPA's responses to comments made in your July 1, 1999 letter are enclosed.

If you have any questions regarding this matter, please contact me at (212) 637-3959.

Sincerely yours,

Alison A. Hess, C.P.G.
Project Manager
Hudson River PCBs Site

Enclosure

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Enclosure
EPA's Response to Dr. George Putman's 7/1/99 Letter Commenting on the Responsiveness Summaries for Phase 2 Human Health Risk Assessment Scope of Work and Ecological Risk Assessment Scope of Work

Human Health Risk Assessment

In your July 1, 1999 letter, you state that, upon review of the Responsiveness Summary for the Human Health Risk Assessment Scope of Work, you believe that the Scope of Work is biased by EPA's 1) refusal to consider epidemiological (human) data from localities and sites other than the Hudson River area in the Human Health Risk Assessment for the Hudson River PCBs site; 2) reliance on toxicity data from studies of Aroclors or congener mixtures different than those found in the Hudson River; 3) a failure to consider the results of occupational mortality studies and current opinion/debate among members of the medical profession; and 4) refusal to develop or consider medical history of workers and residents in Fort Edward and Hudson Falls who were exposed to PCBs.

You did not submit comments on the Human Health Risk Assessment Scope of Work during the public comment period. However, many of the concerns you raise in your July 1 letter were expressed by others in comments on the Scope of Work and, consequently, were addressed by EPA in the Responsiveness Summary. Each of your specific concerns is addressed below.

Epidemiological Data

Consistent with EPA's "Risk Assessment Guidance for Superfund - Part A" (EPA, 1989), the Agency prepares site-specific, baseline risk assessments by comparing exposures specific to the site (e.g., fish ingestion rates, concentrations of PCBs in fish, exposure durations) to toxicity values for the contaminants of concern (i.e., cancer slope factors (CSFs) and non-cancer reference doses (RfDs)). The toxicity values for PCBs are obtained from the Integrated Risk Information System, known as IRIS, which is EPA's database of consensus toxicity values. This general approach is the basis for the July 1998 Human Health Risk Assessment Scope of Work.

EPA has classified PCBs as probable human carcinogens based on a number of studies in animals. Human carcinogenicity data for PCB mixtures is currently "inadequate but suggestive" (see, Human Health Risk Assessment for the Upper Hudson River at p. C-2, citing EPA, 1996c). In the Responsiveness Summary, EPA discussed a number of epidemiological studies of PCB exposure, including occupational exposure at capacitor plants in upstate New York and elsewhere, that were used to develop the Weight of Evidence for PCBs in IRIS (see, Responsiveness Summary for the Human Health Risk Assessment Scope of Work at pp. 25-26).

Use of Toxicity Values Based on Data from Exposure to Different PCB Mixtures

For Superfund risk assessments, EPA uses the toxicity values for chemicals of concern that are in its IRIS database, which are the Agency's consensus values based on the published scientific

literature. The CSFs for total PCBs are based on studies showing liver tumors in animals exposed to a number of different PCB mixtures, which are believed to span the range of congeners found in environmental mixtures (see, Human Health Risk Assessment for the Upper Hudson River at p. C-2, citing EPA, 1996c). Non-cancer toxicity is represented by separate RfDs for Aroclor 1016 and Aroclor 1254. In the Scope of Work, EPA stated that it would use the CSF for total PCBs and would evaluate the results of site-specific sampling and modeling to determine the appropriate RfD for the given medium being evaluated, such as sediment, water, or fish (see, Human Health Risk Assessment Scope of Work at pp. 14 and 21 and Responsiveness Summary for Human Health Risk Assessment Scope of Work at p. 24). This approach is consistent with Superfund risk assessment guidance.

Occupational Mortality Studies and Current Opinion/Debate Among Members of the Medical Profession

As noted above, human carcinogenicity data, including occupational mortality studies, are currently inadequate but suggestive, and EPA's classification of PCBs as probable human carcinogens is based on animal studies. A recent study of mortality of workers at two PCB capacitor plants in upstate New York, Kimbrough et al. (1999), had not been published by the close of the public comment period on the July 1998 Human Health Risk Assessment Scope of Work, so EPA did not address it in the Responsiveness Summary for the Human Health Risk Assessment Scope of Work. However, EPA was aware of this study when it was published and included it in the Human Health Risk Assessment for the Upper Hudson River (see, pp. C-2 to C-3).

With respect to the issue you raise of "contemporary opinion/debate in the medical profession about PCB type and toxicity effects in humans," EPA stated that it would summarize information on the toxicity of PCBs for both cancer and non-cancer health effects from the IRIS files for PCBs and Aroclors and the most recent reported scientific studies, including currently available information on the potential effects of PCBs on the endocrine system (see, Human Health Risk Assessment Scope of Work at pp. 14 and 21 and Responsiveness Summary for Human Health Risk Assessment Scope of Work at pp. 27-28). This information is found in Appendix C of the August 1999 Human Health Risk Assessment for the Upper Hudson River.

Medical History of Workers and Residents of Fort Edward and Hudson Falls Exposed to PCBs

EPA has evaluated the epidemiological studies of Brown and Jones (1981), Brown (1987), and Kimbrough et al. (1999), which include workers at two PCB capacitor plants in upstate New York, presumably General Electric Company's Fort Edward and Hudson Falls plants. The Brown and Jones (1981) and Brown (1987) studies were evaluated as part of EPA's 1996 reassessment of its CSFs and are included in the IRIS file for PCBs. EPA has also evaluated Kimbrough et al. (1999) with respect to its CSFs, and expects that the study will not lead to any change in its CSFs for PCBs (see, Human Health Risk Assessment for Upper Hudson River at pp. C-2 to C-3.)

EPA is not conducting human epidemiological studies of workers and residents in Fort Edward and Hudson Falls. Such studies are not necessary to prepare baseline risk assessments under the Superfund program and were not part of the Human Health Risk Assessment for the Upper Hudson River released in August 1999. Human epidemiological studies are outside EPA's responsibilities and are generally conducted by researchers in other federal or state agencies, such as the Agency for Toxic Substances and Disease Registry or the New York State Department of Health (NYSDOH). Such studies can be submitted to EPA for evaluation, and possible inclusion in the IRIS database, through the Agency's established toxicity assessment procedures. However, due to the limitations of epidemiological studies (see, Responsiveness Summary for the Human Health Risk Assessment Scope of Work at p. 25), such studies have not been recommended as the basis for deriving CSFs and are generally inappropriate for use in the exposure assessment portion of a Superfund baseline risk assessment.

EPA is aware of the human epidemiological studies of the Akwasasne on the St. Lawrence River conducted by Dr. Fitzgerald of NYSDOH and the planned studies of residents in the Glens Falls/Hudson Falls area who were not occupationally exposed to PCBs. Due to the fish consumption advisories for the St. Lawrence and Upper Hudson Rivers, among other reasons, these studies are not appropriate for use in developing the site-specific exposure parameters for the Hudson River. However, when the planned epidemiological studies of residents living near the Hudson River are completed, this information can be submitted to EPA for review through the above-mentioned toxicity assessment procedures. The exposure to PCBs emanating from unlined municipal dumps, which you mention, highlights one of the limitations in using epidemiological studies of local residents in Superfund risk assessments; namely, that such studies may include exposures beyond those being evaluated in the baseline risk assessment.

Ecological Risk Assessment

In your July 1, 1999 letter, you state that, upon review of the Responsiveness Summary for the Ecological Risk Assessment Scope of Work, you believe that the Scope of Work is biased by EPA's reliance on toxicity data from studies of Aroclors or congener mixtures different than those found in the Hudson River.

For Superfund ecological risk assessments, EPA calculates Toxicity Quotients by comparing site-specific exposures to toxicity values for a contaminant of concern. This approach, which is outlined in the September 1998 Ecological Risk Assessment Scope of Work, is consistent with EPA's guidance on conducting ecological risk assessments (see, EPA's 1997 *Ecological Risk Assessment Guidance for Superfund: Process for Designing and Conducting Ecological Risk Assessments* at p. 7-3, "...the quotient method of comparing an estimated exposure concentration to a threshold for a response can be used...").

The commercial Aroclor mixtures used in laboratory studies to derive toxocity values for PCBs may not represent the environmental exposure to that Aroclor, which, in turn, may be different than the original Aroclor mixture used. Because the toxicity of PCB mixture varies depending on its composition of PCB congeners, EPA determined that it would use two approaches to assessing toxicity: the total PCBs and Aroclor mixture toxicities approach, and the PCB congener-specific toxicities and Toxic Equivalency Factors (TEF) approach for dioxin-like PCBs (see, Ecological Risk Assessment Scope of Work at pp. 38-39 and Responsiveness Summary for Ecological Risk Assessment Scope of Work at pp. 27-28). In this way, EPA would reduce uncertainties associated with the difference between the PCB congeners used in studies to derive toxicity values and the PCB congeners that are bioavailable to ecological receptors of concern living in and near the Hudson River.

EPA did not suggest that the Agency would not examine or consider relevant site-specific data and findings, such as field studies of ecological receptors (see, for example, Sections 3.2: Observed Exposure Concentrations, and 5.0: Risk Characterization, of the Ecological Risk Assessment). However, because a baseline risk assessment is site-specific, it is not appropriate to compare ecological risks associated with PCBs in the Hudson River to risks at other PCB-contaminated sites in the Ecological Risk Assessment (see, Responsiveness Summary for Ecological Risk Assessment Scope of Work at p. 13).

rec'd 7/19/99 AL

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July 1, 1999

Mr. Douglas Tomchuk
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Dear Mr. Tomchuk:

I have reviewed the Response summaries for the Human Health and Ecological Risk Assessment SOWs issued in April, 1999. In my opinion there are many relevant, and a number of critical, issues raised by various responders to both assessments which are not adequately addressed in these response volumes. However, I cannot presume to speak for other respondents and my remarks here are specific to my letter of 10/28/98.

In my view these assessment scopes are biased by 1) refusal to consider comparative data (e.g. background, exposures, effects, medical history, and outcomes) from other localities or sites of PCB contamination; with human exposure risk; 2) a reliance on toxicity or carcinogenic reference values extrapolated from indirect, and in some cases arbitrary, sources of data; nearly all of which are derived from other localities, or laboratory work, involving arochlors or congener mixtures different from those now present in the Hudson; 3) a failure to consider the results of occupation mortality studies and contemporary opinion/debate in the medical profession about PCB type and toxicity effects on humans; and 4) a refusal to develop or consider the very relevant medical history data of the Ft. Edward/Hudson Falls workforce and general population, who were subject to PCB exposure by all of the exposure pathways identified.

Points 1, 3, and 4 are central to an assessment of human health risk, because the potentially available medical data is certainly much more relevant, detailed, and timely than the approach described in the SOW, namely (as in point 2, above) an extrapolation from limited studies based on laboratory animals fed PCB assemblages not comparable to those in the Hudson.

The bottom line in this RI/FS is to provide and project an accurate assessment of human health risk, and to this end any and all documented human PCB exposure and related medical data is certainly relevant.

The EPA "ERA "objective" to assess risk on a site-specific basis is, of course, logical to quantifying the parameters and conditions particular to an individual case, but it cannot mean that data and findings from other cases are not to be examined or considered, if relevant. If the latter is indeed EPA's ERA "guidance" (ERA, SOW p. 13), then it is tantamount to saying that

the principle of precedents and discovery in law and evidence progression in scientific investigation (also as applied in peer review) does not apply here; in other words the EPA is to be the sole judge of the basis of estimating risk; and of what risk data is relevant, and therefore can be presented (used in the work plan).

Considering the current debate about the toxicological effects of human exposure to PCBs, including alleged neurological impacts, an examination of the medical history data from documented studies of occupational and general public PCB exposure is certainly relevant, as is the exposure record and current status or recovery of the affected populations, especially where a fish ingestion pathway is present, as in the case of The Great Lakes study data. Moreover, the New York State Department of Health (NYSDOH) has investigated the PCB exposure of the Akwesasne (Mohawk Indian Reservation) on the St. Lawrence River, which includes ingestion of fish and animals. The NYSDOH investigation includes body burden data, and targeted infants and nursing mothers. Since the PCB involved was dominantly the more toxic arochlor 1260, the medical history of the Akwesasne should be very appropriate to consider in evaluating human health risk.

Finally, extensive medical history data is available for the Hudson Falls and Ft. Edward populations through a combination of occupational data from G.E.; local, County, and State medical statistics, and NYSDOH data. Furthermore, these populations have had long term exposure to the same Hudson PCBs now subject to risk assessment, and by every identified pathway including ingestion (local garden crops) and inhalation. The unlined municipal dumps of the Towns of Ft. Edward and Kingsbury contain more PCB than is buried in Hudson River sediments, and area residents have generally been exposed for years to much higher PCB vapor fluxes (discernable odor) than obtained over the River proper. *(Note comment of Dr. Brian Bush at the 6/16/99 STC meeting).

To ignore, or fail to develop, this medical background information under the guise of some EPA technical "objective", would in my view immediately invalidate the Human Health risk assessment and I request my remarks be submitted to the peer review panel. I cannot imagine that in the course of a "scientific" investigation of human risk, with a potential impact of hundreds of millions of dollars that every available and relevant source of risk information and data would not, or is not, to be consulted, weighed, tested for validity, and carefully considered in the outcome.

Very truly yours,

George W. Putman

George W. Putman, Ph.D.
Emeritus Faculty

cc: J. Haggard, GE
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