August 2, 2010

Information Quality Guidelines Staff (2811R)
U.S. Environmental Protection Agency
1200 Pennsylvania Avenue, N.W.
Washington, D.C. 20460

Re: Request for Correction of EPA's Action Plan for Bisphenol A Pursuant to EPA's Data Quality Guidelines

Dear Sir or Madam:

The American Chemistry Council (ACC) submits this Request for Correction to the U.S. Environmental Protection Agency under the Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by the Environmental Protection Agency (the Guidelines). Because EPA relied on a limited number of studies and did not evaluate the scientific data in a weight-of-the-evidence analysis, ACC believes that recent EPA statements regarding Bisphenol A (BPA) are not supported by accurate, reliable and unbiased data and therefore are not supportable under EPA's Guidelines. Accordingly, this Request seeks the correction of the underlying assumptions, and the resulting preliminary conclusions, of EPA's Bisphenol A Action Plan issued on March 29, 2010.

Please feel free to contact me directly regarding any questions you may have about this request at 703-741-5588.

Very truly yours,

Steven G. Hentges, Ph.D.
Polycarbonate/BPA Global Group

cc: David Rostker, Office of Management and Budget
Dominic Mancini, Office of Management and Budget
Nancy Beck, Office of Management and Budget

Request for Correction of EPA's Action Plan for Bisphenol A

The American Chemistry Council (ACC) submits this Request for Correction to the U.S. Environmental Protection Agency under the Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by the Environmental Protection Agency (the Guidelines)[1]. Because EPA relied on a limited number of studies and did not evaluate the scientific data in a weight-of-the-evidence analysis, ACC believes that recent EPA statements regarding Bisphenol A (BPA) are not supported by accurate, reliable and unbiased data and therefore are not supportable under EPA's Guidelines. Accordingly, this Request seeks the correction of the underlying assumptions, and the resulting preliminary conclusions, of EPA's Bisphenol A Action Plan issued on March 29, 2010[2].

As detailed below, EPA's BPA Action Plan does not meet the Guidelines' quality standards for influential scientific information. EPA issued the Guidelines to ensure and maximize the quality of disseminated information, particularly with respect to the objectivity, utility, and integrity of scientific information. Contrary to these requirements, information disseminated by EPA regarding BPA has not been "presented in an accurate, clear, complete, and unbiased manner" with substance that "is accurate, reliable, and unbiased."[3] Instead, EPA's conclusions in the BPA Action Plan rest upon flawed scientific data and analyses that do not meet the heightened quality standard applied to information that substantially impacts key public policies and use decisions. Because the BPA Action Plan fails to satisfy the Guidelines' influential data standards for objectivity and reliability, and instead proposes precautionary action based on flawed data, it must be amended immediately or withdrawn from EPA's website. More importantly, ACC believes existing relevant data is sufficient to assess BPA in a weight-of-the-evidence analysis and EPA should complete such an analysis before pursuing any further actions.

I. BPA Background

BPA is one of the most thoroughly studied chemicals in commerce with over fifty years of research and data. Many government regulatory agencies across the globe have assessed the extensive body of scientific research and data and concluded that BPA does not pose a risk to human health or the environment. According to the BPA Action Plan, EPA intends to initiate immediate actions addressing BPA in the environment based on concerns for potential effects in aquatic species.[4] EPA's Action Plan is not supported by quality science, appears to simply dismiss findings supporting the safety of BPA, and proposes to act on precaution not risk.

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[1] United States Environmental Protection Agency, *Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by the Environmental Protection Agency*, EPA/260R-02-008 (Oct. 2002) *available at* http://www.epa.gov/quality/informationguidelines/documents/EPA_InfoQualityGuidelines.pdf (EPA Guidelines).

[2] United States Environmental Protection Agency, *Bisphenol A Action Plan*, CASRN 80-05-7 (Mar. 2010) *available at* http://www.epa.gov/oppt/existingchemicals/pubs/actionplans/bpa_action_plan.pdf (BPA Action Plan).

[3] EPA Guidelines at 15.

[4] Bisphenol A Action Plan at 1-2.

BPA does not pose a risk to the environment at the levels at which it is found. Monitoring data from extensive sampling across North America show that BPA concentrations in the environment are well below concentrations that might cause harm.[5] The results from multiple, valid toxicity studies across a range of taxonomic groups support this conclusion.[6] Research has also shown that BPA rapidly biodegrades and does not bioaccumulate.[7] Comprehensive environmental risk assessments recently conducted in Europe and Japan have affirmed that BPA is not a risk to aquatic or terrestrial species at the low levels found in the environment.[8]

Multiple international regulatory bodies have repeatedly determined that BPA does not pose a threat to human health at currently measured exposure. In January 2010, the U.S. Food and Drug Administration reiterated that standard toxicology tests support the safety of BPA at typical human exposure levels. For many years, the European Food Safety Authority has supported the safety of BPA, including in applications causing exposure to infants and children.[9] Other international regulatory bodies that have recently assessed the science and determined BPA is not a risk to human health, including the Swiss Federal Office of Public Health (February 2009), the French Food Safety Authority (November 2008), the German Federal Institute for Risk Assessment (September 2008, January 2010), the Japanese National Institute of Advanced Industrial Science and Technology (November 2005), and Health Canada (October 2008, July 2009).

Health Canada's determination to ban the use of BPA in baby bottles, but no other application, was based on the application of the precautionary principle, which is embodied in Canadian law. In the following language, Health Canada made clear that infants were not exposed to a health risk from polycarbonate baby bottles:

> *"Our focus now is on the health of newborns and infants under 18 months. Science tells us that exposure levels are below those that could cause health effects; however, due to the uncertainty raised in some studies relating to the*

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[5] Klecka, G. M., Staples, C. A., Clark, K. E., van der Hoeven, N., Thomas, D. E., and Hentges, S. G. 2009. Exposure analysis of bisphenol A in surface water systems in North America and Europe. Environmental Science and Technology. 43(16):6145-6150.

[6] Staples, C. A., Woodburn, K. B., Klecka, G. M., Mihaich, E. M., Hall, A. T., Ortego, L., Caspers, N., and Hentges, S. G. 2008. Comparison of four species sensitivity distribution methods to calculate predicted no effect concentrations for bisphenol A. Human and Ecological Risk Assessment. 14(3):455-478.

[7] West, R.J., Goodwin, P.A., and Klecka, G.M. 2001. Assessment of the ready biodegradability of bisphenol A. Bulletin of Environmental Contamination and Toxicology. 67:106-112.

[8] European Commission, *European Union Risk Assessment Report –4,4'Isopropylidenediphenol (Bisphenol-A)*, 3rd Priority List, Volume 37, European Commission Joint Research Centre, EUR 20843 EN, Brussels, Belgium *(*2008); Japanese National Institute of Advanced Industrial Science and Technology (AIST), *AIST Risk Assessment Document Series 4: Bisphenol A* (2007).

[9] European Food and Safety Authority, *Summary Opinion of the Scientific Panel on food additives, flavourings, processing aids and materials in contact with food related to 2,2-BIS(4-HYDROXYPHENYL) PROPANE*, Question number EFSA-Q-2005-100 November (2006); European Food and Safety Authority, *Scientific Opinion of the Panel on food additives, flavourings, processing aids and materials in contact with food on a request from the Commission on the toxicokinetics of Bisphenol A*, THE EFSA JOURNAL 759, 1-10 (2008); European Food and Safety Authority, *Statement of EFSA prepared by the Unit on food contact materials, enzymes, flavourings and processing aids and the Unit on Assessment Methodology on a study associating bisphenol A with medical disorders*, THE EFSA JOURNAL 838, 1-3 (2008).

potential effects of low levels of bisphenol A, the Government of Canada is taking action to enhance the protection of infants and young children."[10]

Similarly, Canada took a precautionary approach in its analysis of environmental risk when it failed to apply a species sensitivity distribution approach, described in Canada's own CCME protocol as the best available scientific method,[11] and instead relied on the most sensitive endpoint in a flawed study – Lahnsteiner et al. (2005)[12] In addition, rather than base its predicted environmental concentration for surface water on readily available representative concentrations, Canada again used a precautionary approach when it chose an unrepresentative, single maximum concentration of BPA in sewage treatment plant effluent as the predicted environmental concentration.[13]

Statements made by EPA in the Action Plan regarding the safety and health impacts of BPA are highly influential with respect to actions proposed by EPA under TSCA §§ 4 and 5(b)(4). For that reason, EPA must ensure that the science underlying its statements regarding BPA – including references to other regulatory analyses – reflects the weight of the evidence and "adhere to a rigorous standard of quality."[14] The accuracy of the information presented in the BPA Action Plan will directly affect the scientific integrity of both the EPA's potential actions and the regulatory message conveyed to policymakers, the market place, and the general public. This request for correction is, therefore, of considerable significance to the member companies represented by ACC.

II. The American Chemistry Council: An Affected Stakeholder

The ACC Polycarbonate/BPA Global Group consists of a majority of the manufacturers of polycarbonate plastic and BPA worldwide, and promotes the business interests and general welfare of this industry through relevant technical, communications, and public policy activities.

BPA is widely used in all areas of commerce as it is an integral monomer in the production of epoxy resins and polycarbonate plastic. Examples of products made with polycarbonate plastic include compact discs (CDs) and digital video disks (DVDs), eyeglasses/safety glasses, safety helmets, bullet and blast resistant glazing, cell phones and smart phones, solar panel covers, life-saving medical devices such as kidney dialyzers, and automotive headlamps, to name a few. Restricting or banning BPA poses risks to both the manufacturing supply chain and potentially to human health and the environment, as alternatives to BPA do not have the same high performance properties and do not have the decades-long safety track record of BPA.

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[10] Health Canada, Bisphenol A Fact Sheet, *available at* http://www.chemicalsubstanceschimiques.gc.ca/fact-fait/bisphenol-a-eng.php.

[11] Canadian Council of Ministers of the Environment, *A protocol for the derivation of water quality guidelines for the protection of aquatic life* (2007), *in* Canadian Water Quality Guidelines 1999, Canadian Counsel of Ministers of the Environment, Winnipeg.

[12] Lahnsteiner, F., Berger, B., Kletzl, M., and Weismann, T. 2005. Effect of bisphenol A on maturation and quality of semen and eggs in the brown trout, Salmo trutta f. fario. Aquatic Toxicology. 75(3):213-224.

[13] Environment Canada, Health Canada, *Screening Assessment for the Challenge Phenol, 4,4'(1-methylethylidene)bis-(Bisphenol A)CAS 80-05-7,* (October 2008), *available at* http://www.ec.gc.ca/substances/ese/eng/challenge/batch2/batch2_80-05-7_en.pdf

[14] EPA Guidelines at 20.

The overall economic impact of any proposal to restrict or ban BPA would be substantial, affecting the manufacture of BPA, epoxy resins and polycarbonate as well as the thousands of companies engaged in the manufacture and sale of the myriad applications dependent on these materials. Accordingly, the members of the ACC Polycarbonate/BPA Global Group will be directly impacted by any actions taken as a result of the BPA Action Plan.

III. EPA's Guidelines Require That Scientific Information in the BPA Action Plan Meet Higher Standards of "Objectivity" and "Utility"

EPA issued its Guidelines to ensure and maximize the quality of all disseminated information, particularly with respect to the information's objectivity, utility, and integrity. A review of these Guidelines makes clear that EPA's failure to rely on high quality data and to apply a weight-of-the-evidence approach to analyzing the science underlying the BPA Action Plan violates its Data Quality Guidelines, particularly as the Action Plan is "influential" information subjected to an even more rigorous standard of quality.

The EPA Guidelines "contain EPA's policy and procedural guidance for ensuring and maximizing the quality of information we disseminate" as well as specifically describing "new mechanisms to enable affected persons to seek and obtain corrections from EPA regarding disseminated information that they believe does not comply with EPA or OMB guidelines."[15] The Guidelines provide a pathway for the correction of any information disseminated by EPA that falls short of the "basic standard of quality, including objectivity, utility, and integrity."[16] These Guidelines stem from and adhere to the objectives set out by the Office of Management and Budget (OMB) in its own Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by Federal Agencies issued in response to a Congressional mandate.[17]

Like OMB, EPA defines "objective" information as information that is "presented in an accurate, clear, complete, and unbiased manner, and as a matter of substance, is accurate, reliable, and unbiased."[18] The "utility" criterion relates to "the usefulness of the information to the intended users."[19] Notably, unlike certain regulatory agencies such as Environment Canada or Health Canada, EPA does not subscribe to a precautionary approach, but requires a fair weighing of all the evidence before it.

A. The BPA Action Plan qualifies as "influential" information

"Influential" information, which is information that will have a clear and substantial impact on important public policies or private sector decisions, must "adhere to a rigorous standard of quality" and "should be subject to a higher degree of quality."[20] As noted in the Guidelines, information that can "adversely affect in a material way the economy, productivity,

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[15] *Id*. at 3.
[16] *Id.*
[17] United States Office of Management and Budget, *Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by Federal Agencies*, 67 FED. REG. 8452 (Feb. 22, 2002), *available at* http://www.whitehouse.gov/omb/fedreg_reproducible/ (OMB Guidelines).
[18] *Id.* at 15; OMB Guidelines § V.3, 67 FED. REG. at 8459.
[19] EPA Guidelines at 15; OMB Guidelines § V.2, 67 FED. REG. at 8459.
[20] EPA Guidelines at 20.

competition, jobs" or that addresses "precedent-setting or controversial scientific or economic issues" is considered influential. Further, certain "disseminated information that may have a clear and substantial impact on important public policies or private sector decisions" is also influential and subject to the higher degree of quality standard.

The BPA Action Plan is, thus, "influential" information. The Action Plan is EPA's roadmap on BPA, and includes EPA's articulated plans for its public policy position on BPA, specifically, EPA's determination to undertake rulemaking under TSCA §4 and §5(b)(4). Moreover, the Action Plan is "influential" because the TSCA §5(b)(4) rulemaking will be "precedent-setting" as EPA has never before employed that section of TSCA. The Action Plan also has the potential to significantly impact the U.S. economy by affecting more than $14.2 billion in sales and over 39,000 jobs in 1,400 plants that manufacture BPA, polycarbonate plastic, epoxy resins and end products made from these materials. Additional unquantified impacts will be felt by companies that use these materials to formulate, manufacture and use industrial and consumer goods, as well as the many consumers of those goods. Costs incurred by manufacturers to identify replacement materials through application development, performance testing, and regulatory compliance will be substantial. There can be no question that the statements made by EPA in the Action Plan and in any subsequent position papers or planned pending regulatory action are highly influential and thus, the underlying information must be of higher quality.

B. "Influential" BPA information must pass a two-step quality test

For "influential" information, such as the BPA Action Plan, EPA adopted a two-pronged approach to ensure that influential information will meet rigorous quality standards. First, EPA determined that when evaluating environmental problems it would apply a:

> *"weight-of-the-evidence" approach that considers all relevant information and its quality, consistent with the level of effort and complexity of detail appropriate to a particular risk assessment."*[21]

Second, EPA adapted the quality principles in the Safe Drinking Water Act Amendments (SDWA) of 1996 to ensure the objectivity of influential scientific information, as follows: [22]

(A) The substance of the information is accurate, reliable and unbiased. This involves the use of: (i) the best available science and supporting studies conducted in accordance with sound and objective scientific practices, including, when available, peer reviewed science and supporting studies; and (ii) data collected by accepted methods or best available methods (if the reliability of the method and the nature of the decision justifies the use of the data). (emphasis added).

(B) The presentation of information on human health, safety, or environmental risks, consistent with the purpose of the information, is comprehensive, informative, and understandable.

[21] *Id.* at 21.
[22] EPA Guidelines at 22.

The Guidelines clarify that with respect to "peer reviewed studies," EPA recognizes that there is not a requirement to have data peer-reviewed and that data are also reliable when "data are developed using test guidelines and Good Laboratory Practices (GLPs) in accordance with EPA regulations."[23]

EPA has not followed its own approach for assuring that the Action Plan, as "influential" information, is based on "accurate, reliable and unbiased" information. EPA did not conduct a weight-of-the-evidence analysis and failed to take into account extensive environmental monitoring data, which demonstrate BPA is present only at low levels in the environment, and the results of multiple guideline toxicity studies, which demonstrate BPA is not harmful to aquatic life at the low levels found in the environment.

IV. The Scientific Foundation for the BPA Action Plan Violates the EPA's Guidelines

The scientific foundation of the BPA Action Plan fails to meet the rigorous standards of quality required for such influential information. Notably, the EPA apparently ignores the weight of an extensive body of scientific research and data that soundly supports a conclusion that BPA does not pose a risk to the environment. Data that apparently is ignored includes multiple, valid toxicity studies conducted "using test guidelines and Good Laboratory Practices (GLPs) in accordance with EPA regulations" and published in the peer-reviewed scientific literature. In addition, a substantial amount of environmental monitoring data is apparently ignored in favor of a single data point that is characterized in the Action Plan as an "outlier."

The data on which the Chemical Action Plan is based fall far short of embodying "the best available science and supporting studies conducted in accordance with sound and objective scientific practices" and the other components of "objectivity." The Action Plan should be amended immediately or withdrawn from EPA's website and, until a true weight-of the-evidence analysis is completed, EPA should not pursue any further actions.

A. More than a dozen specific errors undermine the scientific conclusions of the BPA Action Plan

In accordance with the Guidelines,[24] the following are thirteen specific statements contained in the BPA Action Plan that are incorrect and detract from the utility and reliability of the overall information that is presented in the Action Plan. Taken together these statements reveal a document that is so fundamentally in violation of the Guidelines that it should be amended immediately or withdrawn in its entirety.

The incorrect statements are presented in italics followed by an explanation regarding its inaccuracy and the recommendations for corrective action.

Statement 1:

> *"EPA intends to consider initiating rulemaking under section 5(b)(4) of the Toxic Substances Control Act (TSCA) to identify BPA on the Concern List as a substance that*

[23] *Id.* at 25.
[24] EPA Guidelines at 33.

may present an unreasonable risk of injury to the environment on the basis of its potential for long-term adverse effects on growth, reproduction and development in aquatic species at concentrations similar to those found in the environment. A notice of proposed rulemaking is intended to publish in autumn, 2010."[25]

Explanation of Inaccuracy: The basis for the conclusion that adverse effects in aquatic species could occur at concentrations similar to those found in the environment is not supported by the scientific evidence or statements in the BPA Action Plan. Using quality data and a weight-of-the-evidence approach, the concentrations found in the environment (predicted environmental concentration or PEC) do not exceed the concentrations of BPA where adverse effects on aquatic species occur (predicted no effect concentration or PNEC).

Specifically, the Action Plan states that the median concentration of BPA in U.S. waters is 0.14 µg/L, but appears to base its proposed TSCA § 5(b)(4) action on a maximum concentration of 12 µg/L, reported as a single data point by Kolpin et al. (2002)[26], which even the BPA Action Plan notes as an "outlier".[27] As reported in Klecka et al. (2009)[5], the author (Kolpin) indicated that the reported concentration of 12 µg/L was a sample taken from the Santa Cruz River, AZ, which was essentially 100% effluent dominated. A finding from a 100% effluent dominated water body is not an environmentally relevant concentration for use in risk assessment. Despite admitting that the 12 µg/L is an outlier, the Action Plan inexplicably uses this level as the basis for initiating rulemaking that will have significant impacts on BPA and the perceptions relating to this substance. In addition to relying on a single inappropriate data point, EPA failed to follow the Guidelines and perform a weight-of-the-evidence review which requires *"in the Agency's development of "influential" scientific assessments, . . . to use all the relevant information . . .and reach a position based on careful consideration of all such information (i.e. a process typically referred to as the "weight-of-evidence" approach).*[28] Indeed, EPA chose to ignore a highly relevant, peer reviewed, weight-of-the-evidence assessment performed by Klecka et al. (2009)[5] that was cited in the bibliography to the Action Plan. The assessment determined a median environmental concentration from 1068 samples of North American fresh surface water of 0.081µg/L, while the 95th percentile was 0.47µg/L.

Nor has EPA used quality data by simply citing the range of predicted no effect concentrations found in other assessments to conclude BPA is "*a substance that may present an unreasonable risk of injury to the environment on the basis of its potential for long-term adverse effects on growth, reproduction and development in aquatic species at concentrations similar to those found in the environment"*. There are three environmental risk assessments cited in the BPA Action Plan as providing information for the assessment of BPA. The risk assessments from Japan[8] and the EU[8] provide a comprehensive evaluation of the existing science, rate it based on data quality, identify Predicted No Effect Concentrations (PNEC) of 1.6 µg/l and 1.5 µg/l respectively based on accepted guideline studies, and conclude that BPA does not present a

[25] Bisphenol A Action Plan at 1.
[26] Kolpin, D.W., Furlong, E.T., Meyer, M.T., Thurman, E.M., Zaugg, S.D., Barber L.B., and Buxton, H.T. 2002. Pharmaceuticals, hormones and other organic wastewater contaminants in U.S. streams, 1999-2000: a national reconnaissance. Environmental Science and Technology. 36(6):1202-1211.
[27] Bisphenol A Action Plan at 10.
[28] EPA Guidelines at 26.

risk to the environment. However, the third risk assessment from Canada does not reach a conclusion based on the weight of the evidence, but instead relies on a single, novel low-dose study (Lahnsteiner et al., 2005[12]) with non-standard effect endpoints and methodology to calculate a PNEC for the pelagic compartment. Any reliance in the Canadian assessment to support EPA's conclusion that TSCA §§ 4 and 5(b)(4) action is warranted is not appropriate under the Guidelines; EPA cannot blindly rely on Canada's screening risk assessment (particularly as Canada's assessment incorporates the precautionary principle), but must perform its own weight-of-the-evidence assessment.

Moreover, EPA cannot rely on an assessment founded on the Lahnsteiner study because it is severely flawed and does not meet quality criteria for influential information, for the following reasons:

- In the Lahnsteiner study, no analytical determination of concentration was performed – not at the beginning nor at the end of the exposure period – so the actual concentrations to which the fish were exposed were unknown. The uncertainty of the exposure concentrations were cited in the European Union Risk Assessment Report[8] (Appendix 1, page 158) as the reason the Lahnsteiner study was considered of low relevance and was not used in the EU's comprehensive assessment of BPA toxicity and risk. EPA's standards for study quality should be no lower than the EU's, particularly with respect to "influential" data.

- The Lahnsteiner study used an excessive concentration of solvent and did not employ a non-solvent or clean water control critical to assuring the validity of fish reproduction studies. Globally accepted OECD guidance for fish short-term reproduction studies specifies a solvent concentration of no greater than 100 µg/L and use of a non-solvent or clean water control. In contrast to OECD guidance, the Lahnsteiner study employed a top solvent concentration of 541µg/L, which is well in excess of accepted solvent levels. In addition, a solvent control, but not a clean water control (without solvent), was used in the study. Thus, this study does not meet OECD criteria for validity and, on this basis alone, should not be considered reliable, objective data for EPA's Action Plan.

- As a complicating factor, the fish used in the study were wild-caught and of undetermined ages, although it was stated in the paper that they were within 3 years of each other. Prior exposure to other chemicals is also not known and only "unpublished data" for no prior chemical exposure is reported. No replication of test levels was employed and the estimated concentrations of exposure were very tightly spaced between 1.75 and 5 µg/L. The lack of replicate vessels and the tightly spaced treatment concentration does not allow careful statistical assessment of the intrinsic variability of the measured endpoints. Statistical analysis for intrinsic variability is especially crucial when the endpoint being evaluated is non-standard and non-validated, as is semen quality, which is the basis for the effect in the Lahnsteiner study.

- The protocol did not follow a "longitudinal" approach (addition of a "control" pre-incubation period without BPA exposure followed by exposure to BPA), which would allow each individual fish to be used as its own control. As spawning is not expected to be synchronous in wild-caught fish, such a procedure would allow for the high inter-fish

variability with respect to semen quality to be addressed. In the absence of such a procedure, it is very difficult to relate any of the claimed effects to the presence of BPA at the low nominal concentrations used (between 1.75 and 5.0 µg/L), especially given that the 1st stripping happened 5 days after BPA exposure started. More recent work by Bjerregaard et al. (2008)[29] with the same fish species at sensitive juvenile stages did not find any adverse effects on gonad development at 50 µg/L BPA.

- The extensive database of fish growth, development, and reproduction studies that have been reviewed and deemed valid in the European Union Risk Assessment[8] does not corroborate effects at the low estimated concentrations reported in the Lahnsteiner study. EPA's own guidelines would require a similar weight-of-the-evidence assessment, which would not support reliance on the Lahnsteiner study.

Rather than rely on a single study to develop a predicted no effect concentration (PNEC), the U.S. EPA directs[30] a species sensitivity distribution approach when the toxicity database includes, at a minimum, eight unique families of aquatic organisms from a diverse array of taxonomic groups. The database for BPA includes at least 19 valid chronic studies covering 14 different species from 10 unique families. Under EPA's own guidelines for evaluating a data rich compound such as BPA, the PNEC for aquatic organisms is 71 µg/L[6]. The evaluation and statistical analysis of the aquatic toxicology data in accordance with EPA's method, as well as three other species sensitivity distribution methods, was readily available to EPA in a peer-reviewed article[6], as was the EU's weight of evidence assessment that produced a PNEC of 1.5µg/L. Both Staples et al. (2008) and the EU assessment provide a higher quality, more objective assessment of the toxicity data that would be more consistent with EPA's stated preference for a species sensitivity distribution and weight-of-the-evidence approach than Canada's reliance on a single flawed study.

Taken together, neither the PEC nor the PNEC apparently used by EPA are supported by either the quality of data or a weight-of-the-evidence analysis that is needed to reach the conclusion in this statement from the Action Plan.

Recommendation for Corrective Action: Remove the statement as it is not supported by the existing quality data.

Statements 2 and 3:

"Because BPA is a reproductive, developmental, and systemic toxicant in animal studies and is weakly estrogenic, there are questions about its potential impact particularly on children's health and the environment." [31]

[29] Bjerregaard, L. B., Lindholst, C., Korsgaard, B., and Bjerregaard, P. 2008. Sex hormone concentrations and gonad histology in brown trout (Salmo trutta) exposed to 17beta-estradiol and bisphenol A . Ecotoxicology. 17(4):252-263.
[30] USEPA, *Guidelines for Deriving Numerical National Water Quality Criteria for the Protection of Aquatic Organisms and their Uses*, PB85-227049, National Technical Information Service (1985).
[31] Bisphenol A Action Plan at 2.

"There is general agreement that BPA is a reproductive and developmental toxicant at doses in animal studies of > 50 mg/kg-bw/day (delayed puberty in male and female rats and male mice); > 235 mg/kg-bw/day (reduced fetal or birth weight or growth early in life, effects on testis of male rats); and > 500 mg/kg-bw/day (possible decreased fertility in mice, altered estrous cycling in female rats, and reduced survival of fetuses)."[32]

Explanation of Inaccuracy: These statements are inaccurate and incorrect. BPA is not "a reproductive or developmental toxicant in animal studies." As recognized in the EPA Guidelines for Developmental Toxicity Risk Assessment (1991)[33], virtually all chemicals have the potential to cause developmental or reproductive toxicity when they are given at sufficiently high doses to kill or damage the health of the mother. What is critical is to determine whether a substance is a selective reproductive or developmental toxicant.

In BPA animal studies, the reproductive and developmental effects were secondary effects that were the result of maternal toxicity. For example, among the eight studies evaluated by the National Toxicology Program/Center for the Evaluation of Risks to Human Reproduction in 2008, with respect to reproductive and developmental toxicity:

- Two studies did not evaluate maternal toxicity; they do not permit a determination of whether the effects seen are related to maternal toxicity or to reproductive or developmental effects. These two studies are not of sufficient quality to support EPA's statement in the Action Plan that BPA is a developmental and reproductive toxicant.

- Six remaining studies that did evaluate maternal toxicity all showed that developmental effects were always associated with significant or even excessive maternal toxicity and all show types of developmental effects (e.g. reduced weight, not malformations) consistent with maternal toxicity.

EPA referenced the highly relevant assessment by the seven expert members of the California Developmental and Reproductive Toxicant Identification Committee (DARTIC). The Committee unanimously determined that BPA is not shown to cause developmental toxicity or to cause reproductive toxicity in either males or females.[34] The EU Risk Assessment also concluded that BPA is not a reproductive, developmental or systemic toxicant[8]. EPA apparently ignored both of these assessments' conclusions.

More recently, researchers in the EPA's Reproductive Toxicology Branch of the Office of Research and Development published a peer-reviewed study, Ryan et al. (2009)[35], examined whether maternal exposure to low doses of ethinyl estradiol (EE2) and BPA *in utero* and during

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[32] *Id.* at 5.

[33] Guidelines for Developmental Toxicity Risk Assessment, EPA/600/FR-91/001, 56 FED. REG. 234: 63798-63826 (Dec. 5, 1991).

[34] California Environmental Protection Agency's Office of Environmental Health Hazard Assessment, *Meeting Agenda and Audiocast Of The Science Advisory Board's Developmental And Reproductive Toxicant Identification Committee: Hearing Before The Developmental and Reproductive Toxicant Identification Committee of OEHHA's Science Advisory Board* (July 15, 2009), *available at* http://www.oehha.ca.gov/prop65/public_meetings/dart071509ag.html.

[35] Ryan, B. C., Hotchkiss, A. K., Crofton, K. M., and Gray Jr., L. E. 2009. In utero and lactational exposure to bisphenol A, in contrast to ethinyl estradiol, does not alter sexually dimorphic behavior, puberty, fertility and anatomy of female LE rats. Toxicological Sciences. 114(1):133-148.

lactation would alter the expression of well-characterized sexually dimorphic behaviors or alter the age of puberty or reproductive function in the female Long-Evans rat offspring. They concluded, as follows, that while EE2 produced permanent adverse effects, BPA did not affect either reproduction or sexually dimorphic behavioral endpoints.

> *"In conclusion, the current study demonstrates that maternal exposure to 5–50 µg EE2/kg/day during gestation and lactation produces permanent adverse effects on the developing female rat reproductive system. EE2 affected several reproductive measures at doses ranging from 5 to 50 µg/day, dosage levels within the dose range used by girls and women for therapeutic purposes. In contrast, exposure to BPA at dosage levels 40-, 400-, and 4000-fold above the estimated median human exposure (Calafat et al., 2008) did not alter any end point included in our studies in F1 male (Howdeshell et al., 2008) or in female LE rats. In the current study, we also found that doses of BPA ranging from 2 to 200 µg/kg/day did not affect maternal pregnancy or weight gain or F1 female birth weight, AGD, age at VO, reproductive morphology, fertility, fecundity, or sexual dimorphic behaviors (lordosis, Figure-8 maze activity or saccharin preference). The lack of effect of BPA on female and male rat offspring after oral exposure to low doses in our studies is consistent with the lack of adverse effects on growth, VO, fertility, and fecundity of low doses of BPA in several other robust, well-designed, properly analyzed multigenerational studies (Cagen et al., 1999; Ema et al., 2001; Tinwell* et al., 2002; Tyl et al., 2002)."[35]

No conclusion on the primary mode of action or effect can be drawn from the extensive dataset of valid ecotoxicity studies. Studies performed according to internationally recognized guidelines for the assessment of aquatic and terrestrial effects are not conducted to delineate maternal toxicity from reproductive and developmental effects.

Neither recent scientific studies nor recent assessments of all of the data support EPA's characterization of BPA as "a reproductive, developmental, and systemic toxicant."

Recommendation for Corrective Action: Remove from Statement 2 "*is a reproductive, developmental, and systemic toxicant in animal studies and"* and remove from Statement 3 the phrase "*there is general agreement that BPA is a reproductive and developmental toxicant"* as neither is supported by quality data.

Statement 4:

> *"Although there is disagreement about the interpretation of these low-dose studies, they do raise potential concerns for long-term effects at similar concentrations, and some authorities, including Canada and some U.S. state and county governments, have taken interim risk management action to protect certain sensitive populations, such as infants and toddlers."*[36]

Explanation of Inaccuracy: The phrase "and some authorities including Canada and some U.S. state and county governments" is misleading. While Canada has performed a screening risk assessment, the actions taken by U.S. states and county governments were the result of a political

[36] Bisphenol A Action Plan at 2.

legislative process and were not based on a scientific assessment. It is inaccurate and misleading to imply that these government actions support any conclusion about the interpretation of low-dose studies from a scientific perspective.

Recommendation for Corrective Action: Remove "*and some U.S. state and county governments"* from the statement.

Statement 5:

> *"There was a recent report in which a cross-sectional study design was used to suggest an association between BPA levels in humans and a higher risk of diabetes, heart disease, and elevation of certain liver enzyme activities (Lang et al., 2008). The authors examined the human data from the 2003-4 NHANES population. However, this report prompted an immediate review by the European Food Safety Authority (EFSA) (EFSA, 2008b) in late 2008 which concluded that the study did not provide sufficient proof for the stated associations. EPA notes that the same investigative group recently published an online research article repeating their original findings for heart disease but not diabetes on a second NHANES population from 2005-6 (Melzer et al., 2010)."*[37]

Explanation of Inaccuracy: The statement does not accurately reflect the fundamental issues with methodology and statistics that render the first study completely inaccurate and unreliable and which persisted in the second study by the same group. Neither of these studies comes remotely close to meeting EPA's criteria for quality data and should not be referenced or relied on in the Action Plan.

Specifically, with respect to the flawed methodology of the Lang et al. (2008)[38] study, EFSA stated that:

"[the study makes use of NHANES data], which comprises measurements of BPA in urine samples of individuals sampled once at the time the participants were asked about their health status, These data can be used as an estimate of the exposure to BPA within 24 hrs of sample collection. However, there is no information on exposure during the time period needed for development of diseases such as diabetes and cardiovascular conditions or changes in plasma liver-enzyme activities. Although the study authors attempted to rule out several commonly identified confounders of studies of this type, the observed association between urinary BPA elimination and the conditions mentioned above may have been a chance finding or may be due to non-identified confounders."[9] (Emphasis added.)

In a letter to the editor of JAMA, which published the Lang study, S. Stanley Young of the National Institute for Statistical Sciences, raised serious questions about the statistical methodology of the study:

> *[T]he potential for false positives, briefly mentioned but not analyzed is substantial when complete Centers for Disease Control and Prevention (CDC)*

[37] Bisphenol A Action Plan at 6.

[38] Lang, I. A., Galloway, T. S., Scarlett, A., Henley, W. E., Depledge, M., Wallace, R. B., and Melzer, D. 2008. Association of urinary bisphenol A concentration with medical disorders and laboratory abnormalities in adults. Journal of the American Medical Association. 300(11):1303-1310.

design is examined. . . . Focusing only on health outcomes selected by the authors, the analysis forms a 16x275 composite set of questions. However, there are more than 8 ways that the medical outcomes can be examined since 2 of the outcomes have subgroups, any 1 or combination of which could result in an association. Likewise there are more than 8 ways the clinical measurements can be examined because additional measurements and derived outcomes were reported. Overall, we counted 32 possible outcomes. . . . .

Given the number of questions at issue and possible modeling variations in the CDC design, the findings reported by the authors could well be the result of chance.[39] (Emphasis added.)

The Melzer et al. (2010) study by the same group of researchers used the same flawed study design and statistics with a new set of NHANES data[40]. Repetition of findings based on flawed methodology and flawed statistics does not make them more reliable nor does it indicate the presence of effects as wrongly indicated by the EPA statement.

Recommendation for Corrective Action: This statement should be removed from the BPA Action Plan because the information in the studies referenced is inaccurate and unreliable and therefore can support neither the statement nor the conclusions drawn from it.

Statement 6:

"Thirty-eight scientists (known as the "Chapel Hill Group"; vom Saal et al., 2007) concluded that: (1) there is relevance of in vitro data to in vivo effects; (2) ecological studies are consistent with lab animal studies; (3) the low doses in animal studies are relevant to BPA levels found in humans; and (4) life stage is important in pharmacokinetics, exposure, and effects in animals and humans."[41]

Explanation of Inaccuracy: The EPA Guidelines require that the substance of the information be "unbiased."[42] The Chapel Hill Group was substantially composed of scientists who conducted the very low dose studies that have been reviewed and rejected by government regulators, and scientists who received and continue to receive grant money from NIEHS for continued research on BPA. These scientists had a vested interest in concluding in the Chapel Hill Group report that their low dose studies and findings were relevant to humans. *See* Appendix A.

Recommendation for Corrective Action: Remove the reference to the Chapel Hill Group report, as the potential for bias is too high for the information to be relied on by EPA as "influential" information in such an important policy document.

[39] Young, S. S. and Yu, M. 2009. Association of bisphenol A with diabetes and other abnormalities. Journal of the American Medical Association. 301(7):720-721.
[40] Melzer, D., Rice, N. E., Lewis, C., Henley, W. E., and Galloway, T. S. 2010. Association of urinary bisphenol A concentration with heart disease: Evidence from NHANES 2003/06. PLoS One.5(1):e8673.
[41] Bisphenol A Action Plan at 6.
[42] EPA Guidelines at 22.

Statement 7:

> *"In general, studies have shown that BPA can affect growth, reproduction and development in aquatic organisms. Among freshwater organisms, fish appear to be the most sensitive species. Evidence of endocrine-related effects in fish, aquatic invertebrates, amphibians and reptiles has been reported at environmentally relevant exposure levels lower than those required for acute toxicity. There is a widespread variation in reported values for endocrine-related effects, but many fall in the range of 1µg/L to 1 mg/L. (Canada, 2008)."* [43]

Explanation of Inaccuracy: The Action Plan does not provide any citation for the specific references used to support this statement. Notably, "[t]here is widespread variation in reported values for endocrine-related effects, but many fall in the range of 1 µg/L to 1 mg/L. (Canada, 2008)" is taken directly from the Canadian Screening Risk Assessment. The general reference to the Canadian Screening Risk Assessment fails to recognize that Environment Canada did not conduct a weight-of-the-evidence assessment, instead basing its conclusion on the precautionary principle. The precautionary principle is incorporated into Canadian law but not into U.S. law.

Before including such statements, EPA must independently analyze the underlying studies. Specifically, EPA must determine whether the studies and the hormonal effects they may potentially indicate are accurate and reliable under a weight-of-the-evidence analysis that:

> *"considers all relevant information in an integrative assessment that takes into account the kinds of evidence available, the quality and quantity of the evidence, the strengths and limitation associated with each type of evidence, and explains how the various types of evidence fit together*."[44]

Absent such an analysis, EPA cannot blindly rely on Environment Canada's conclusions.

A perfunctory review of Environment Canada's references reveals that only certain data were considered. For example, Table 7c of Environment Canada's screening assessment lists "Selected endpoint values relating to potential hormonal effects" for fish and includes the flawed Lahnsteiner study discussed above. However, this assessment failed to consider conflicting data provided in two multigenerational guideline studies conducted in accordance with Good Laboratory Practices[45] - Caunter et al., 2000[46] (noted in the Action Plan's references, but not analyzed) and Rhodes et al. 2008[47]. The latter study analyzed a range of population-relevant endpoints (e.g., survival, reproduction and development) alongside supplemental endpoints (e.g., vitellogenin and gonad histology) and showed that, overall, changes in gonad cell types and

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[43] Bisphenol A Action Plan at 8.
[44] EPA Guidelines at 26, fn. 29.
[45] "*Our test guidelines and Good Laboratory Practices (GLP) describe sound scientific practices for conducting studies needed to assess human and environmental hazards and exposures. Such studies are not required to be peer-reviewed." Id.* at 50.
[46] Caunter, J.E., Williams, T.D., Hetheridge, M.J., and Evans, M.R. 2000. Bisphenol A: Multigeneration study with the fathead minnow (*Pimephales promelas*). Brixham Environmental Laboratory, AstraZeneca UK Limited (unpublished).
[47] Rhodes, J.E., Wolf, J. C., van der Hoeven, N. 2008. Bisphenol A: Partial Life-Cycle Toxicity Test with the Fathead Minnow, *Pimephales promelas* (unpublished).

vitellogenin did not correspond to impacts on biologically meaningful population level endpoints. Thus, contrary to the suppositions in Environment Canada's Table 7c that vitellogenin induction indicates adverse effects, the robust, guideline, GLP study Rhodes et al. (2008) shows that vitellogenin induction does not correlate with adverse effects. Clearly, Environment Canada's conclusions are subject to question and cannot be relied on by EPA without an independent analysis of the quality of the data underlying Environment Canada's conclusions.

In stark contrast, the comprehensive risk assessments of from the EU (2008)[8] and Japan (2007)[8] provide a far more robust assessment of data quality. Only after reviewing and evaluating each study for validity and reviewing the endpoints for ecological relevance, the EU concluded that no risks to the fresh water or marine components are indicated. Similarly, the Japanese National Institute of Advanced Industrial Science and Technology (AIST) scrutinized the underlying data before concluding that "current levels of bisphenol A will not pose unacceptable risks to the local populations of aquatic life, particularly fish."[8]

Moreover, EPA has the means to assess whether there are endocrine-related effects from a substance such as BPA by applying the Tier 1 and Tier 2 tests of EPA's Endocrine Disrupter Screening Program. In Tier 1 tests, which show only the potential to interact with the endocrine system, BPA is shown to be weakly estrogenic (about 10,000 times less potent than estradiol.) Although the Tier 2 guidelines have yet to be validated, BPA has been the subject of extensive testing using protocols validated by EPA and OECD for assessing population level effects on endpoints such as reproduction and development. These Tier 2 type tests show that BPA does affect survival, growth or reproduction at environmentally relevant levels.

Recommendation for Corrective Action: Remove the reference to "endocrine-related," and "environmentally relevant exposure levels," and "there is a widespread variation in reported chronic toxicity values, but many fall in the range of 1 μg/L to 1 mg/L. (Canada, 2008)." The paragraph should be rewritten as follows:

> *In general, studies have shown that BPA can affect growth, reproduction and development in aquatic organisms at exposure levels lower than those required for acute toxicity.*

Statement 8:

> *"Canada concluded in its hazard characterization that "[c]onsidered together, the data provide strong evidence that bisphenol A is capable of eliciting adverse effects (1) following prolonged exposure at levels below those usually seen to elicit effects in standard toxicity tests (i.e., tests based on recognized methods which evaluate endpoints such as survival, reproduction and growth); (2) following brief low-dose exposure, particularly at sensitive developmental stages, with effects apparent later in the life cycle; (3) on filial generations following parental exposure; and (4) using more than one mode of action." (Canada, 2008)."*[48]

[48] Bisphenol A Action Plan at 8.

Explanation of Inaccuracy: While this is an accurate quote of the Canadian Screening Risk Assessment (2008) at page 19, it should not be included in the Action Plan as "quality data." As noted above in the discussion of the inaccuracies in Statement 7, Environment Canada's assessment was a screening level assessment that did not provide an evaluation of many of the valid and robust studies available and did not use a weight-of-the-evidence analysis. In fact, as discussed under Statements 1 and 7, the overall conclusion for the pelagic environment in the Environment Canada assessment is founded on the Lahnsteiner study, a study that is highly flawed, and on a single unrepresentative, maximum concentration of BPA in sewage treatment plant effluent as the predicted environmental concentration, rather than on a weight of evidence analysis of the many environmental monitoring data points.[49] Such an approach does not produce the "accurate, reliable, and unbiased" conclusion that must underlie influential information to be used by EPA.

Recommendation for Corrective Action: Remove the quoted passage from the Environment Canada risk assessment; failing to do so mistakenly treats that passage as accurate, reliable, and quality data.

Statement 9:

> *"Limited information is available for BPA concentrations in U.S. water and other environmental media (Table 4, providing values from all of the studies cited in this discussion)."*[50]

Explanation of Inaccuracy: There is a significant body of monitoring data available for concentrations of BPA in various environmental media. In a thorough review of existing monitoring data by Klecka et al. (2009), a total of 1,068 samples in North America and 848 samples in Europe from representative and unique surface water locations were analyzed and reported[5]. In North America, 80% of the sampling locations had no detectable BPA while in Europe 49% had no detectable BPA. Some of the North American surface water data is a result of a U.S. Geological Survey study that focused largely on areas that are susceptible to contamination and thus are weighted toward finding BPA in the environment if it exists. The median surface water concentration in North America, reported by Klecka et al. (2009) is 0.081 µg/L with a 95th percentile concentration of 0.47 µg/L. Clearly, substantial data exists for characterizing surface water concentrations.

Recommendation for Corrective Action: Remove the word "limited" from the statement as a significant amount of high quality monitoring data exists for BPA in North American waters.

Statement 10:

> *"E-FAST2 modeling of BPA releases in the 2007 TRI showed the most conservative estimates of the potential acute dose rate for ingestion of BPA in drinking water by children ages 1-2 ranged from 0.0000531 to 16.5 µg/kg/day, and the most conservative*

[49] Klecka et al. (2009) reported 1,068 surface water samples in North America; see also discussion under Statements 9 and 10.

[50] Bisphenol A Action Plan at 10.

estimates of the surface water concentration ranged from 0.000574 to 232 µg/L. The E-FAST2 model is intended to be used for screening level exposure characterization. E-FAST2 is based on numerous assumptions that are designed to be conservative; for example, E-FAST2 does not account for the half life of a chemical in surface water. The inputs selected for the E-FAST2 modeling of BPA were also selected to be conservative; for example, the bioconcentration factor was selected to be at the high end of the range of values reported for BPA in the literature."[51]

Explanation of Inaccuracy: Again, a weight-of-the-evidence analysis is needed to support influential information such as the BPA Action Plan. EPA cannot rely on the highly conservative E-FAST2 modeling of BPA releases in the 2007 TRI to estimate the amount of BPA in drinking water or surface water when there exists peer-reviewed assessments of BPA in groundwater[52] and drinking water[53] of which EPA is aware (referenced in the Action Plan as an unpublished USGS report[54]), as well as a peer reviewed analysis of available surface water data by Klecka et al. (2009)[5].

In the summer of 2001, USGS collected samples from 74 sources of raw, untreated, drinking water in 25 states and Puerto Rico and analyzed for 100 organic wastewater contaminants. These sources comprise 25 ground water and 49 surface-water sources of drinking water serving populations ranging from one family to more than 8 million people. Site selection for both studies focused on areas known or suspected to contain sources of animal and/or human wastewater. The samples ranged from < 1 µg/L to 2.55 µg/L with 80% being below the detection limit of 1 µg/L. These actual samples are in contrast with the estimated values from the overly conservative E-FAST2 modeling. Moreover, reliance on the conservative E-FAST2 modeling which does not take into account the short half-life of a readily biodegradable chemical such as BPA, does not reflect a weight-of-the-evidence analysis to assure quality data is used in influential information such as the Action Plan.

Recommendation for Corrective Action: Remove the paragraph about E-FAST2 modeling. Failing to do so mistakenly implies that the underlying data reflects a weight-of-the-evidence analysis of all relevant data.

Statement 11:

"Workers may be exposed to BPA by inhalation or skin contact during the manufacture of BPA and BPA-containing products. No data were available for dermal exposures, and limited data were available for inhalation exposures. Table 5 summarizes EPA's estimates for occupational exposures that may occur during manufacturing. These estimates were derived using models developed by EPA/OPPT for use in preparing

[51] *Id.* at 12.

[52] Barnes, K. K., Kolpin, D. W., Furlong, E. T., Zaugg, S. D., Meyer, M. T., and Barber, L. B. 2008. A national reconnaissance of pharmaceuticals and other organic wastewater contaminants in the United States - I) Groundwater. Science of the Total Environment. 402(2-3):192-200.

[53] Focazio, M. J., Kolpin, D. W., Barnes, K. K., Furlong, E. T., Meyer, M. T., Zaugg, S. D., Barber, L. B., and Thurman, M. E. 2008. A national reconnaissance for pharmaceuticals and other organic wastewater contaminants in the United States - II) Untreated drinking water sources. Science of the Total Environment. 402(2-3):201-216.

[54] U.S. Geological Survey, *Water-Quality Data for Pharmaceuticals and other Organic Wastewater Contaminants in Ground Water and Untreated Drinking Water Sources in the United States*, 2000-01 (USGS 2008).

screening-level exposure assessments of chemicals. These models do not take into account the effect of any personal protective equipment that may be used.

Table 5. Inhalation and Dermal Exposure Estimates to BPA During BPA Lifecycle Stages		
Lifecycle Stage	***Exposure Type***	***BPA Exposure Dose (mg/day)***
Manufacturing	*Inhalation*	*0 – 9.6*
	Dermal (liquids and solids)	*882 – 3,100 a*
USE 1: Polycarbonates	*Inhalation*	*0.7 – 2.7 b*
	Dermal (solids)	*0.31 – 3,100 c*
USE 2: Epoxy Resins	*Inhalation*	*0 – 28*
	Dermal (solids)	*3,100 c*
USE 3: Flame Retardants	*Inhalation*	*0 d*
	Dermal (solids)	*3,100 c*

a – Exposure is in milligrams per event. Events can include sampling of solutions containing BPA or solid BPA and loading/unloading of BPA from containers.

b – Exposure is to polycarbonate dust.

c – Exposure is in milligrams per loading/unloading of BPA from containers, which is the only identified potential exposure during this stage of the lifecycle.

d – Inhalation exposure to BPA during the production of flame retardants is not expected."[55]

Explanation of Inaccuracy: EPA's Action Plan does not take into account occupational exposure data developed in the EU Risk Assessment (2008)[8], which includes detailed measured and estimated data by task and shows levels considerably lower than those estimated by EPA. Moreover, it does not take into account the use of personnel protective equipment recommended for use in the manufacture of BPA.

Recommendation for Corrective Action: Where relevant exposure data exists that meet EPA's quality standards, they should be utilized instead of less accurate estimated values. EPA should remove the estimated data.

Statement 12:

"Connecticut, Minnesota, Wisconsin, Washington, Chicago and Suffolk County, N.Y., have banned the sale of polycarbonate baby bottles, food containers and cups that contain BPA. The Connecticut ban also applies to infant formula cans and all reusable food and beverage containers. The Suffolk County ban (County of Suffolk, 2009) went into effect in July 2009. The Minnesota ban (Minnesota, 2009) went into effect on 1/1/2010, and the Chicago ban (Chicago, 2009) on 1/31/2010. The Wisconsin ban (Wisconsin 2010) will go into effect on 6/15/2010, and the Connecticut ban (Connecticut, 2009) will take effect on 10/1/2011. The Washington state ban (Washington, 2010) will take effect on 7/1/2010 concerning food and drink containers for children three years old

[55] Bisphenol A Action Plan at 12.

and under, and will ban BPA in sports water bottles effective 7/1/2012. Similar bills banning BPA in children's food and drink containers passed both houses in Maryland (Maryland, 2010) in February 2010, and if they are signed into law by the governor, would take effect on 1/1/2012. California bill (California, 2009) to ban the use of BPA in baby bottles and cups and infant formula cans failed to pass in September 2009 and was moved to the inactive file. A similar bill failed to pass in Oregon (Oregon, 2010) in February 2010."[56]

Explanation of Inaccuracy: Enactment of legislation to ban BPA in certain applications is a political act, not a scientific assessment. Such legislative action cannot be considered quality scientific data on which EPA could rely on to support any regulatory action on BPA.

Recommendation for Corrective Action: Remove the paragraph in its entirety, as it has no place in a scientific assessment of BPA.

Statement 13:

"Although there is disagreement in interpreting the novel low-dose studies and some of the effects observed in the many aquatic toxicity studies performed thus far with BPA, a comparison of the range of predicted no effect concentration (PNEC) values used in the three international regulatory risk assessments (0.175 to 1.6 µg/L, Table 3) with measured concentrations in U.S. waters and sediments, which included values as high as 12 µg/L (surface water), 2.55 µg/L (ground water), and 140 µg/kg sediment (freshwater sediment) (Table 4), raises concern about possible risk of injury to aquatic organisms. However, limited information is available for BPA concentrations in U.S. water, and most available environmental monitoring results show that the concentrations of BPA in water bodies are lower than 1 µg/L (median concentration of 0.14 µg/L, below any calculated PNEC). These environmental measurements represent only isolated snapshots in time and do not provide an indication of how many areas may exceed PNEC values or concentrations of concern, how often or how long such concentrations may be exceeded, or the pathways leading to BPA presence in the environment from manufacturing, processing, distribution in commerce, use, or disposal. Additional information would help to resolve these uncertainties."[57]

Explanation of Inaccuracy: In its concluding paragraph, EPA summarizes its analyses and in each instance selects the most conservative data to support its conclusion. Because EPA has relied on earlier flawed portions of the BPA Action Plan as the foundation for EPA's ultimate conclusion, the ultimate conclusion also does not meet EPA's data quality standard, including the rigor of a weight-of-the-evidence analysis.

Specifically, EPA's reliance on "a comparison of the range of predicted no effect concentrations (PNEC) values used in three international regulatory risk assessments (0.175 to 1.6 µg/L, Table 3)" includes inappropriate, wholesale reliance on the Canadian Screening Risk Assessment's PNEC. First, the conclusion of the Canadian Screening Risk Assessment is not a weight-of-the-evidence analysis but is based on the precautionary principle, which is not

[56] *Id.* at 13.
[57] Bisphenol A Action Plan at 15.

incorporated in U.S. law. Second, before including conclusions from another regulatory assessment, EPA must independently examine the data and analysis to determine that it meets EPA's quality standards. That assessment apparently was not performed because the Canadian risk assessment is founded on the flawed Lahnsteiner study that does not meet EPA's requirements for quality data.

The reference to environmental concentration "*values as high as 12 µg/L (surface water), 2.55 µg/L (ground water), and 140 µg/kg sediment (freshwater sediment)"* as support for EPA's planned actions related to pelagic species is flawed. Rather than reflect a weight-of-the-evidence analysis critical to making a reliable determination of environmental concentrations, the references cited reflect the most conservative values found by EPA.

- The proposal to consider 12 µg/L as the relevant surface water concentration for risk assessment purposes ignores EPA's own acknowledgement that the 12 µg/L value is an "outlier" because it was take from a river that is essentially 100% effluent. (See discussion under Statement 1 above). It also ignores a peer-reviewed weight of evidence analysis of 1068 surface water monitoring endpoints in North America reviewed in Klecka et al. (2009), which reported a more reliable prediction of a median concentration of 0.081 µg/L and a 95th percentile of 0.47 µg/L[5].

- The ground water value chosen is also the high point of the range.

- Similarly, the third, of 140 µg/kg in fresh water sediment, chose the highest point in the range and likewise ignores the Klecka et al. (2009) weight of evidence analysis of 71 observations in freshwater sediments that resulted in a more reliable prediction of media freshwater sediment concentration of 0.6 ng/g-dw and a 90th percentile concentration of 3.4 ng/g-dw[5].

EPA again claims that "limited information is available for BPA concentrations in U.S. waters", but as discussed in detail under Statements 9 and 10 there is ample U.S. monitoring data, much of which has been collected by the U.S. Geological Survey. Klecka et al. (2009) reviewed 1068 observations in North American fresh surface water and 71 observations in North American fresh water sediments[5].

Finally, EPA's claim that *"these [limited] environmental measurements represent only isolated snapshots in time and do not provide an indication of how many areas may exceed PNEC values or concentrations of concern, how often or how long such concentrations may be exceeded, or the pathways leading to BPA presence in the environment from manufacturing, processing, distribution in commerce, use or disposal*" shows that EPA has not, as it is required to do before issuing influential information, independently examined the data. While environmental monitoring data is by its nature a snapshot in time, there have been over a thousand snapshots taken at different points in time; taken all together they provide a good picture of the environmental concentrations of BPA.

For example, Klecka et al. (2009) evaluated 100 papers and reports published between 1991 and 2007 for quality; of the 89 papers retained for analysis, 31 papers focused on North America and 58 papers focused on Europe. Moreover, EPA's stated concern that the data may

underestimate environmental concentrations is belied by the fact that most of the studies reviewed by Klecka et al (2009) *"characterized the sample locations as being downstream of water discharges, receiving waters for industrial facilities, areas susceptible to contamination, urban waterways or industrial ports"* – in short, places where BPA concentrations were likely to be higher than average.

Recommendation for Corrective Action: The foundations of the statement and the specifics of the text are so flawed that it should be removed in its entirety.

V. Conclusion: Immediate Amendment or Withdrawal of the BPA Action Plan Is Essential

ACC respectfully requests that this request for correction be granted and that the BPA Action Plan be amended immediately as detailed above. If that is not possible, the Action Plan should be withdrawn entirely. We ask that this be done quickly so as to minimize risk of further harm to the public and to the epoxy resin and polycarbonate plastic industries from inaccurate conclusions drawn from the information listed above. This action does not prejudge EPA's decisions regarding BPA or the end result of EPA's own review process, but assures that the public is not misinformed about BPA and whether or not it has potential health or environmental effects.

More importantly, ACC believes existing relevant data is sufficient to assess BPA in a weight-of-the-evidence analysis and EPA should complete such an analysis before pursuing any further actions. If EPA believes insufficient data exists, EPA should work with industry to develop necessary data under TSCA § 4. ACC stands by to assist EPA in this data development. Until that time, EPA should not and cannot rely on data in a manner that is not supported by the standards of the Data Quality Act.

Appendix A

Chapel Hill Group Members Receiving Recovery Act NIH Grand Opportunities Grants

Grant Recipients	Grant Title	Grant Amount
Scott Belcher (U. of Cincinnati)	Defining the impact of dietary bisphenol A on heart health in the C57BL/6 mouse	$0.8M
Gail Prins (U of Illinois - Chicago) Shuk-Mei Ho (U of Cincinnati) Kevin White (U of Illinois - Chicago)	Developmental exposure to low-dose bisphenol A and human prostate cancer susceptibility	$0.9M
Beverly Rubin (Tufts) Andrew Greenberg (Tufts)	Defining the role of BPA in promoting obesity and associated metabolic complications	$0.8M
Ana Soto (Tufts)	Does breast cancer start in the womb? BPA, mammogenesis and neoplasia	$0.92M
Frederick Vom Saal (U of Missouri) William Allen Ricke (U of Rochester)	Bisphenol A: Urine flow disorder and prostate pathology	$0.45M
Cheryl Walker (U of Texas) Shuk-Mei Ho (U of Cincinnati) Michael Mancini (Baylor)	Developmental reprogramming of prostate carcinogenesis by BPA	$0.95M
Gail Prins	Carcinogenesis following early estrogenic exposures	$0.56
Ana Soto (Tufts)	Mechanism of developmental toxicity of bisphenol A	$0.34M