Porter Exhibit 2

My comments are primarily focused on the Statewide Plant Pest Prevention and Management Programmatic Environmental Impacts Report (Pest PEIR), Volume 2-Appendix A, Human Health and Ecological Risk Assessment (HHRA), SCH #20110657, prepared by Horizon Water and Environment, LLC for the CDFA. Page numbers refer to the HHRA unless otherwise indicated.

My first several comments address some of the overarching assumptions on which the risk assessment relies.

My first comment relates to the statements on page 4, "Pesticide Illness Surveillance Program". The authors state, "If health effects appear to derive from exposure to any component of a pesticide product, including inert ingredients, impurities, and breakdown products, the surveillance program attributes those health effects that pesticide product. Similarly, reporting includes but is not limited to toxic effects similar to those seen in tests." Unfortunately, later in the report it is noted that only pathologies are considered to be problematic, e.g., on page 26: "If endpoints such as blood parameter measurements, body weight, organ weight, or measured enzyme levels were not associated with pathology, these endpoints were considered not of concern." The document also states, on page 146, that "endocrine disruption was not explicitly assessed in this HHRA." This means that subtle effects of pesticide exposure, such as learning disabilities (Levin et al., 2002), attention deficit disorders (Shelton et al., 2014), alterations of immune (Olson et al. 1987) and endocrine function (Cavieres et al., 2002), and potential epigenetic effects, which have been documented (Skinner et al. 2010), are not considered to be "health effects".

Further in that same paragraph from page 4 cited above, the authors use an example, "…a product designed to disrupt nerve function may, at excessive levels, cause neurologic symptoms in humans." This reasoning, referencing "excessive levels," reflects the 16th-century paradigm under which the EPA is still officially functioning, a linear dose response assumption (see the reference to Paracelsus at http://www.epa.gov/pesticides/factsheets/riskassess.htm#step%2020), which has been repeatedly refuted by modern science, especially in the realm of endocrine research (Vandenberg et al., 2012) documenting how the lower the dose, the greater the effect that can occur down into the parts per trillion. These are referred to as "inverse dose responses" and have been documented in neurological (Levin et al. 2002), immune (Olsen et al. 1987), and endocrine effects (Cavieres et al., 2002) of pesticides. The reasons and mechanisms for this are explained in Vandenberg et al., 2012.

Page 6, section 2.2 on hazard identification, "Inactive and Inert Ingredients Assessed" states: "Pesticide manufacturers are not required to report other chemicals or their concentrations if they are determined to be a trade secret or are in small quantities, as allowed under pesticide labeling regulations. Since that information about these other chemicals was available, it was included in the HHRA; a total of 79 pesticide products (including adjuvants or other formulations used in conjunction with pesticides), containing 91 different active or inert ingredients were assessed." However, several papers document added toxicity when "other" ingredients are present in a pesticide mixture, e.g., Bolognesi et al., 1997, where they report that the genotoxic activity of Roundup is significantly greater than the active ingredient glyphosate by itself. Genotoxicity is

not evaluated as part of the registration process of a pesticide. How can our federal or state government allow undisclosed agents contributing to a toxic product to be sprayed on the landscape and contaminate air, food, water and/or soil? No pesticide mixture should be allowed for use unless its composition is fully declared.

At this point it is also important to notice that the HHRA states: "Some of chemicals were determined to be 'not of concern' for the following reasons: The chemical showed no endpoints of concern from an oral, inhalation, and or dermal routes of exposure in toxicity tests where dose levels near or above testing limits were employed in experimental animal studies. If endpoints such as a blood parameter measurements, body weight, organ weight, or measured enzyme levels were not associated with pathology, these endpoints were considered not a concern" (page 6). Based on these criteria, genotoxic or epigenetic effects would not have been included as of concern, since there would typically not be obvious pathologies that would be observed over the short experimental durations typically involved in registering pesticides, see e.g., the recently republished Seralini et al. study (2014).

The other reason given for excluding from the PEIR risk assessment chemicals determined to be "not of concern" was that, "The only *available* toxicity data show that the chemical was not known to be harmful to humans and had a history of safe use" (page 6, emphasis added). There are two problems in this statement. The first is the word "available", since frequently there are no available toxicity data as mentioned throughout this document (see for example: "The quality and depth of information available on inert ingredients in pesticide products was highly variable; in some instances, full disclosure of ingredients was given, others offered partial disclosure, and some offered none. In instances where inert ingredients were not disclosed and no information was available to estimate risk, the extent of risk, if any, remains unknown" (page 144)). When there are no data, the chemical is assumed to be safe, a fatal logical flaw. The second problem is that there are no tests of impacts of pesticides on learning abilities (Levin et al., 2002) or immune (Olson et al., 1987) or endocrine functionality (Jaeger et al., 1999), all of which are associated with exposure to pesticides, and there are no studies of altered immune function caused by pesticide exposure, that are now known to be associated with a variety of diseases, such as obesity, type II diabetes and atherosclerosis (Dietert and Dietert, 2007). By the measures defined in the PEIR HHRA, none of these fundamental biochemical or biological processes would qualify as indicating adverse effects. Furthermore any such tests that would be conducted as part of standard pesticide registration process would be conducted only with the active ingredient, not with the mixture. The HHRA repeatedly uses the word "pathological" to define the limited range of potential health effects that that it assesses.

On page 7 under "Step Two: Toxicity Dose-Responses Assessment", the HHRA states (paragraph 2): "Often adequate human scientific studies are not available for a specific chemical and its health effects to derive a toxicity value based on a dose-response model. In these situations a hierarchy of alternative scientific studies is used to derive an appropriate toxicity value. For instance, often scientific studies are available for various animal species that exhibit similar effects as humans would on exposure. In other cases a specific chemical may not be available, but a related chemical that is expected to behave in a similar manner does have adequate studies available. In such instances, a toxicity value is derived using these data while applying safety and uncertainty factors to account for extrapolation of the studies and to reflect population variation."

This toxicity dose response assessment process is seriously flawed. New epigenetic data and biochemical pathways research are both suggesting that each different chemical has a unique biochemical signature in terms of biomarkers and pathway alterations it induces. These pathway alterations can be precursors to chronic long-term disease (Skinner et al, 2010). None of this would show up under the evaluation procedures described in this PEIR.

In the last paragraph on page 7 of the Step 2 process, the authors state, "the toxicity values used in an HHRA are intended to protect identifiable sensitive individuals from harm. However, the toxicity values may not necessarily be protective for hypersensitive individuals who do not exhibit a dose-response reaction with chemical exposure. In a typical HHRA, the chances of an adverse health effects are assumed to escalate with increasing exposure to a specific chemical. The health effects of an individual who may have an allergy to a specific chemical do not follow a dose-response mechanism, rather the person gets the same effect regardless of the amount of chemicals to which he/she is exposed."

There are two things of note here. The first is that it is admitted that *there is no safe dose for individuals with an allergy to a specific chemical*. There are many such individuals in our society today and the number of them is growing (http://www.webmd.com/allergies/multiple-chemical-sensitivity). The PEIR specifically excludes individuals with multiple chemical sensitivity (MCS) from its assessment: "the Human health Risk Assessment that was prepared for this Draft PEIR does not include a quantitative analysis of MCS" (page 6.5-14). The second thing to note is that the chances of an adverse health effect are assumed to escalate with increasing exposure to a specific chemical, i.e., a linear dose response with increasing concentration. As mentioned above, the open scientific peer-reviewed literature for neurological, endocrine and immune effects has demonstrated that *there can be greater effects at lower doses than higher doses*, especially in very low concentration ranges where physiological responses typically occur to biological molecules like estrogen, such as the parts per trillion to parts per billion ranges. These concentrations are typically not part of the standard registration process for pesticides. Thus, large effects of low doses can be completely missed during registration processes. Moreover, the registration process typically concerns only the active ingredient and not the mixture, which is sold to consumers and found in air, food and/or water.

The last paragraph on page 7, "Step Three: Exposure Assessment", states: "The next step in determining human exposure after the concentrations in the environment were identified was to estimate how much the human body takes up. Exposure was determined by combining the concentration in the environment with specific exposure factors. Exposure factors took into account the amount that would be taken into the body, the amount of time exposure would occur, and the frequency of exposure." This method of assessment does not take into account the amount that may be stored because it is fat-soluble or the molecules that are broken down into other molecules that may be more toxic than the parent compound. Such substances are not part of the exposure evaluation process. The assessment process does not take into account the possibility of epigenetic effects that can be transgenerational up to four generations beyond the individual (Skinner et al., 2010; partial Skinner lab references for 2014).

On page 8 under the same heading of exposure assessment the authors state, "An exposure pathway would have to be complete for it to be relevant to the HHRA. For instance, ingestion of

tree leaves at a nursery would not be likely to occur because most people do not eat leaves." This is an excellent example of excluding the most sensitive individuals like babies and small children by simply assuming that they will not consume objects that everyone knows they do consume, such as dirt, leaves, and anything else they can get their hands on and put in their mouth.

The authors go on to admit "Thus, ingestion of tree leaves would not be considered a complete exposure pathway, and this was not evaluated." They further state, "In some instances, the exposure pathway may be complete, but based on low concentrations or a minimal amount of exposure compared to a dominant pathway of exposure, it may not have been fully quantified and was dismissed as discountable." Again, the authors dismiss low concentrations as irrelevant by relying on a 16th-century assumption of linear dose response which is not supported by modern scientific understanding of inverse dose responses for neurological, endocrine and immune functions.

The risk assessment contains many more unfounded or unrealistic assumptions about receptors, for example, omission of babies in the drift exposure analysis (page 52) and of adults over 40 from the category of "post-application resident," (page 53) and undocumented assumptions about exposure durations (page 52 – potential exposure of downwind bystander for 3 years in "CDFA's expert opinion"). All of these assumptions bear on the validity of the risk assessment results.

Step 4: Risk Characterization. The authors state, "For this analysis, it was performed by combining the exposure and dose-response assessments to determine the likelihood that the use of the chemicals can cause harm to the relevant sensitive receptors". In this section, all of the risk assessment is based on an assumption of linear dose effects. This ignores the statements above that for individuals allergic to chemicals there is no safe dose. This would imply that all individuals with allergies to chemicals would be especially at risk from the Proposed Program. This also ignores the fact that fetuses, pre-pubertal children and the elderly, who all have reduced liver-based defense mechanisms that under active sexually mature conditions degrade sex hormones and a variety of xenoestrogens and pesticides (Wright and Welbourn, 2002), are effectively ignored in risk assessments.

Pesticide impacts on neurological function have been demonstrated. For example, the organophosphates chlorpyrifos and malathion, both of which are included on the list of 79 chemicals reviewed in the PEIR (Appendix L, Table L-1), have been demonstrated to appear as metabolites in the urine of children consuming conventionally produced fruits and vegetables (Lu et al., 2006), and observations by Levin and colleagues (2002) using chlorpyrifos for learning experiments in female rats or Rodriguez and colleagues (2005) using atrazine, reveal inverse dose responses for neurotransmitters in the prefrontal cortex of the brain. These types of effects are ignored in this risk assessment. As noted earlier, this risk assessment includes no epigenetic effects or the multigenerational effects that they might engender.

On page 10, the authors state, "EPA has identified five groups of pesticides that each has a common mechanism of toxicity: organophosphates, N-methyl carbamates, triazines, chloroacetanilides and pyrethrins/pyrethroids." When data are not available, data from another related chemical are used as a surrogate, as pointed out on page 7. However, using surrogates is not a valid methodology. For example, chlorpyrifos and malathion are both organophosphates, but are very different in their persistence and excretion from the body (Lu et al., 2006). Thus the

paradigm of assuming similarity of structure and similarity of function is not a responsible or reliable method of extrapolation.

Unfortunately, as the authors state, "… a large number of possible combinations of exposures would be possible, and predicting which combinations would be most likely would be impossible." This statement is evidence that true integrated pest management, which uses chemicals sparingly, rarely, and as a last resort, would be a much safer pathway for CDFA to follow, rather than relying on pesticides. Emerging data clearly show that greater and greater challenges and more and more of them in more and more complex situations are the reality with regard to chemical exposure. The massive lack of data and unfounded assumptions regarding risk are not acceptable for an intelligent society. The repeated failure to exterminate populations of insects when relying on ever more toxic substances to try to control them is clearly indefensible, especially in the context of the health of our children and our fetuses, i.e., our future as a nation. I could spend many more days evaluating and commenting on other parts of this report, but in my view, it needs a fundamental reworking.

Most sincerely,

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