Office of Environmental Health Hazard Assessment

George V. Alexeeff, Ph.D., D.A.B.T., Director
Headquarters • 1001 I Street • Sacramento, California 95814
Mailing Address: P.O. Box 4010 • Sacramento, California 95812-4010
Oakland Office • Mailing Address: 1515 Clay Street, 16th Floor • Oakland, California 94612

Matthew Rodriquez
Secretary for
Environmental Protection

Edmund G. Brown Jr.
Governor

MEMORANDUM

TO: Gary T. Patterson, Ph.D., Chief
Medical Toxicology Branch
Department of Pesticide Regulation
P.O. Box 4015
Sacramento, California 95812-4015

FROM: Anna M. Fan, Ph.D., Chief
Pesticide and Environmental Toxicology Branch
1515 Clay Street, 16th Floor
Oakland, California 94612

DATE: November 19, 2013

SUBJECT: COMMENTS ON THE DRAFT RISK CHARACTERIZATION DOCUMENT FOR PHOSPHINE

The Office of Environmental Health Hazard Assessment (OEHHA) has reviewed the draft Risk Characterization Document (RCD) for occupational and ambient air exposure to phosphine, prepared by the Department of Pesticide Regulation (DPR), dated February 15, 2013. Our comments are provided in the attachment. OEHHA has provided comments on the Exposure Assessment Document for Phosphine separately. OEHHA reviews risk assessments prepared by DPR under the authority of Food and Agricultural Code section 11454.1.

OEHHA has provided a number of comments on the risk characterization methodology and conclusions of the draft RCD. These comments are contained in the attachment. Thank you for providing this draft document for our review. If you have any questions regarding OEHHA's comments, please contact Dr. Charles Salocks at (916) 323-2605 or me at (510) 622-3200.

Attachment

cc: Charles B. Salocks, Ph.D., D.A.B.T.
Chief, Pesticide Epidemiology Section
Pesticide and Environmental Toxicology Branch
Office of Environmental Health Hazard Assessment

OEHHA's Comments on DPR's Draft
Risk Characterization Document for Phosphine
(Occupational and Ambient Air Exposures)

The Office of Environmental Health Hazard Assessment (OEHHA) is responding to a request from the Department of Pesticide Regulation (DPR) to comment on the February 15, 2013 draft Risk Characterization Document (RCD) for phosphine. The document addresses occupational and ambient air exposures. OEHHA reviews risk assessments prepared by DPR under the authority of Food and Agricultural Code Section 11454.1, which requires OEHHA to conduct scientific peer reviews of risk assessments conducted by DPR.

SUMMARY OF COMMENTS

The RCD addressed the fumigation product phosphine (PH_3), which is used as a rodenticide and insecticide for stored agricultural products such as grain, tobacco, processed foods and animal feed. The RCD was comprehensive and well-written with a thorough presentation of the toxicological studies, analysis of weight of evidence, and approaches used to identify the critical endpoints and derive No Observed Effect Levels (NOELs) to calculate margins of exposure (MOEs). From a public health perspective, this is a very important RCD because the usage of phosphine in California is increasing. Our principal comments and suggestions are as follows:

- Acute toxicity: Because of its severity, OEHHA does not recommend using lethality as a critical endpoint when determining an acute exposure advisory level. However, in this case because of data availability, OEHHA agrees with the selection of the Newton (1990) study in the RCD, but suggests adding an uncertainty factor (3-fold or higher) because the NOEL (5 parts per million [ppm]) is based on lethality. OEHHA also suggests an additional 3-fold uncertainty factor to protect infants and children as sensitive bystander subpopulations, as discussed below.

- Subchronic toxicity: OEHHA agrees with the use of the subchronic study selected for the subchronic exposure determination, and with the use of the observed NOEL (1 ppm) from the Schaefer (1998b) study as a point of departure for calculating the subchronic exposure advisory level. However, OEHHA suggests incorporating an additional 3-fold uncertainty factor to protect infants and children as sensitive bystander subpopulations, as discussed below.

- Chronic toxicity: OEHHA agrees with the use of the subchronic study for the chronic exposure determination, since an adequate chronic study was not identified, and with the point of departure (Schaefer 1998b). OEHHA suggests the use of an additional 3-fold uncertainty factor when using a NOEL from a subchronic study to assess hazard associated with chronic exposure. Furthermore, OEHHA suggests incorporating an additional 3-fold uncertainty factor to protect infants and children as sensitive bystander subpopulations, as discussed below.

- Carcinogenicity: OEHHA agrees with the approach in the RCD not to calculate cancer risk values. The weight of evidence for carcinogenicity is based on a single study in male and female rats (Newton 1998), which observed no carcinogenicity. However studies were not performed in a second species and this should be noted in the RCD. In addition, there were 99 unscheduled deaths in the study. DPR stated that these deaths were unrelated to phosphine exposure. The results of the Newton (1998) study are hard to interpret, but OEHHA believes there is not enough information in the RCD to justify stating that the deaths were unrelated to phosphine exposure.

- Sensitive subpopulations: As noted above, OEHHA is concerned that the exposure values used for occupational and residential bystanders, including infants and children, may not be sufficiently health-protective. Some of these bystanders such as office workers or nearby residents may not be aware that fumigation is taking place near them. Therefore, they would not be expected to use air-purifying respirators or other protective equipment. OEHHA suggests that infants and children may be more susceptible to the adverse health effects of phosphine and phosphine-generating products due to their higher susceptibility to airborne toxicants, higher breathing rates on per kilogram body weight basis and higher incidence of asthma. A recent report cited in the RCD stated the possibility of children being more susceptible to phosphine-induced or mediated death (O'Malley et al., 2013). OEHHA suggests considering an extra 3-fold uncertainty factor to account for increased susceptibility in children.

- Uncertainty factors: In summary, OEHHA is recommending additional uncertainty factors for the acute, subchronic and chronic exposure advisory levels. For the acute point of departure, OEHHA suggests an additional 3-fold uncertainty factor because the NOEL was based on lethality and a 3-fold factor to protect infants and children. For the subchronic point of departure, OEHHA suggests an additional 3-fold uncertainty factor to protect infants and children. Finally, for the chronic point of departure, OEHHA suggest a 3-fold uncertainty factor because the key study utilized subchronic exposure and a 3-fold factor to protect infants and children that are bystanders.

- Usage: Regarding the usage of phosphine and phosphine-generating products, a trend of increasing agricultural use of phosphine gas, aluminum phosphide and magnesium phosphide is apparent, although year-over-year data are quite variable. Given the high toxicity of phosphine and the low MOEs calculated in the RCD, this trend has possible public health implications. Occupational and residential bystanders may not be aware that fumigation is taking place near them and therefore would not be expected to use air-purifying respirators or other protective equipment. For similar reasons, residential bystanders may be exposed if they live close to grain elevators or close to other places where fumigation occurs. Increased usage of phosphine could result in increased exposure for these groups.

ACUTE TOXICITY

The RCD provides a clear review of human exposures (accidental, occupational and suicidal) to phosphine. These descriptions indicate the acute toxicity of phosphine resulting in severe illness and death following exposure. OEHHA suggests providing a summary table of the individual cases of human poisoning and observed adverse effects to improve this section.

- Study and Endpoint Selection
    - A study conducted by Newton (1990) was identified as the critical study supporting the point of departure for acute toxicity. The critical effect in this study was lethality based on the deaths of 4/10 female rats within 3 daily exposures to 10 parts per million (ppm) (6 hours/day, 5 days/week). The NOEL was 5 ppm (internal dose 1.7 milligrams/kilogram). As a policy, OEHHA does not use lethality as a critical endpoint when determining an acute exposure level. In addition, studies conducted by Misra et al. (1988) and Schaefer (1998a) suggest that neurological effects may occur following acute sub-lethal exposure. Therefore lethality may not represent the most sensitive acute toxicity endpoint for phosphine. However, in this case OEHHA supports identification of the 1990 Newton study as the critical acute toxicity study and lethality as the endpoint, but believes that incorporation of a 3-fold additional uncertainty factor is warranted due to the severity of the critical effect.

    - The discussion of acute toxicity endpoints was supported by several studies as presented on pages 12-18, 23-27, and 44 in the RCD. As noted above, the critical effect was lethality. Phosphine has a steep dose-response curve and there is a rapid transition from toxicity to lethality within a narrow exposure range. The RCD (page 63, paragraph 1) states, "The steep dose-response relation between air concentrations which cause little or no toxicity and those which kill animals must therefore be seriously considered when assessing human health risks of phosphine." This further supports OEHHA's recommendation to incorporate a 3-fold additional uncertainty factor due to the severity of the critical effect.

- Neurotoxicity
    - The RCD (page 50) noted the proximity of the no-effect and lethal levels and suggested the possibility that other effects, including subtle neurologic effects, may have been overlooked by Newton (1990). The studies that reported non-lethal effects at sub-lethal doses are discussed below. The risk appraisal section of the RCD noted that a functional observational battery (FOB) to assess neurotoxicity was not performed in the key study (Newton 1990). Had an FOB been conducted, it may have helped identify more sensitive adverse effects. This data gap further

supports the addition of an uncertainty factor to account for other potentially more sensitive adverse effects that occur prior to lethality.

- Supporting Studies
    - The Schaefer (1998a) study showed acute neurotoxic effects of phosphine gas on Sprague-Dawley rats after a 4-hour exposure. The lowest dose tested was 21 ppm, and similar neurotoxic effects may have occurred, if tested, at a lower exposure concentration and/or shorter duration. In a second study, Schaefer (1998b) used exposure concentrations of 0, 0.3, 1 and 3 ppm, but the results from the FOB were inconclusive.

    - The study published by Misra *et al.* (1988) showed some important respiratory and neurological effects in humans at non-lethal doses after acute exposure that support increasing the uncertainty factor. This study investigated phosphine-induced toxicity in workers at an Indian facility where stacks of bagged grain were treated with aluminum phosphide tablets. The breathing zone phosphine concentrations ranged between 0.17 and 2.11 ppm. Though no attempt to correlate symptoms with exposure was reported, many acute adverse health effects short of lethality were observed at these doses such as cough (18.2% incidence), dyspnea (31.8%), tightness around chest (27.3%), headache (31.8%), giddiness (13.6%), numbness /paresthesia (13.6%), lethargy (13.6%), irritability (9.1%), anorexia (18.2%), epigastric pain (18.2%), nausea (9.1%) and dry mouth (13.6%). Other symptoms included a bad taste in the mouth and loss of appetite.

    - Newton (1991) reported acute lethality after a single 6-hour exposure of Sprague-Dawley rats to phosphine at 28 ppm, but no lethality at doses ranging from 0 to 18 ppm. Mean body weight decreases were noted in the 10 ppm and 18 ppm groups. An acute NOEL of 6 ppm was identified in this study based on the body-weight decreases in the 10, 18 and 28 ppm groups. OEHHA again notes the steep dose-response curve and how close these mildly acutely toxic doses are to lethal concentrations. In addition, the Newton (1991) study only looked at a 6-hour exposure. The RCD should point out that if the study duration had been longer, adverse effects may have been observed at lower doses.

- <u>Uncertainty Factors</u>
    - DPR divided the critical NOELs by a total uncertainty factor of 100 using a 10-fold for interspecies extrapolation and a 10-fold for intraspecies variability. OEHHA suggests adding an additional uncertainty factor (3-fold or higher) because the NOEL is based on lethality, which is a severe

acute endpoint, and because of the proximity of the values of the NOELs for acute (5 ppm) versus subchronic/chronic (1 ppm) exposure. In cases where the point of departure is based on a severe endpoint such as lethality, an additional uncertainty factor is warranted. In addition, as noted above, neurological effects have been observed at acute sub-lethal doses. OEHHA also suggests adding a 3-fold uncertainty factor for the protection of sensitive bystander subpopulations such as infants and children (see section on sensitive subpopulations below).

SUBCHRONIC TOXICITY

This section of the RCD provided a thorough and well-written summary of the subchronic studies performed in laboratory animals.

- Study and Endpoint Selection
    - OEHHA agrees with DPR's identification of the study conducted by Schaefer (1998b) as the critical study for the subchronic exposure determination, and supports the conclusion that the observed NOEL in this study was 1 ppm. A NOEL of 1 ppm was identified from this study based on palpebral closure (sleeping behavior, week 4), slowed respiration (weeks 8 and 13) and lowered body temperatures (week 13) in rats at 3 ppm (6 hours/day, 5 days/week). OEHHA agrees with the use of these endpoints and the 1 ppm NOEL as a point of departure for calculating the subchronic exposure guidance level.

- Uncertainty Factors
    - DPR divided the critical NOELs by a total uncertainty factor of 100 using a 10-fold for interspecies and a 10-fold for intraspecies. OEHHA suggests adding a 3-fold uncertainty factor for the protection of sensitive bystander subpopulations such as infants and children (see below).

- Supporting Study:
    - DPR commented that the results reported by Newton (1990) did not follow Haber's Law (page 23, paragraph 4, and line 4). DPR postulated that there is a threshold for death at or above 5 ppm, since it was anticipated under Haber's Law that the 5 ppm group should have died after six exposures. The RCD concluded that a "short term" lowest observed effect level (LOEL) of 5 ppm was justified based on the Newton (1990) study. Histological effects in the kidney (pelvic and tubular mineralization) and decreases in absolute and relative liver weights were observed at 3 ppm. Although Newton (1990) was not used as the key study in determining the MOE, OEHHA suggests a LOEL of 3 ppm appears to be justified due to the histological effects observed.

CHRONIC TOXICITY

- Study Selection:
  - OEHHA agrees with the use of the subchronic study conducted by Schaefer (1998b) for the chronic exposure determination since an adequate chronic study was not identified.

- Point of Departure: OEHHA agrees with the use of the subchronic NOEL (1 ppm) as a point of departure (Schaefer, 1998b) to assess chronic exposure since the chronic toxicity study conducted by Newton (1998) did not fully assess all potential toxicity endpoints, particularly neurotoxicity.

- Uncertainty Factor: OEHHA suggests the use of an additional 3-fold uncertainty factor when using a subchronic study to determine a chronic exposure level. OEHHA also suggests adding a 3-fold uncertainty factor for the protection of sensitive bystander subpopulations such as infants and children, as discussed below.

CARCINOGENICITY

In the chronic 2-year study (Newton 1998), no carcinogenicity was observed in male and female Fischer 344 rats. Therefore, no cancer risk values were calculated in the RCD. The weight of evidence for carcinogenicity is based on these findings. The study in male and female rats was well-conducted with a sufficient number of animals and doses. However, there were 99 unscheduled deaths in the study. DPR stated that these deaths were unrelated to phosphine exposure. Although the results of the Newton (1998) study are difficult to interpret, OEHHA believes there is not enough information in the RCD to justify stating that the deaths were unrelated to phosphine exposure. The deaths in the study also reduced study power to detect carcinogenicity. Studies in male and female mice were not conducted. Studies in a second species would provide a more robust data set for carcinogenicity determination. The rat study with unscheduled deaths and the lack of a study in mice constitutes limited data available to judge carcinogenicity. OEHHA agrees with DPR that the available *in vivo* data do not provide evidence of carcinogenicity and are insufficient to calculate a cancer potency value.

GENOTOXICITY

DPR provided well-written descriptions of the genotoxicity studies in the RCD, and Table III-5 provided an excellent summary of these studies. OEHHA agrees with DPR's assessment of the genotoxicity studies and the conclusion that phosphine is potentially clastogenic.

REPRODUCTIVE / DEVELOPMENTAL TOXICITY

No male or female reproductive toxicity studies on phosphine were available for analysis. One developmental toxicity study was conducted in rats (Schroeder, 1989), but the investigators observed no developmental effects at sublethal doses (up to 4.9 ppm). OEHHA agrees with the RCD's characterization of the limited data available to judge reproductive toxicity, which represents a significant data gap in the toxicity dataset for phosphine.

SENSITIVE SUBPOPULATIONS

- Occupational and Residential Bystanders
    - OEHHA is concerned that the exposure values used for occupational and residential bystanders may not be sufficiently health-protective as some of these bystanders such as office workers or nearby residents may not be aware that fumigation is taking place near them. Therefore, they would not be expected to use air-purifying respirators or other protective equipment.

    - OEHHA agrees with the primary conclusion in the report, "*Many acute, seasonal and annual use scenarios produced MOEs of under 100, indicating insufficient health protection for workers and bystanders under those scenarios. Moreover, some acute MOEs for occupational bystanders were as low as 17, including those adjacent to farm bins, flat storage facilities or warehouses during fumigation or aeration. In addition, residential or occupational bystanders under most occupational scenarios showed MOEs of 50. In light of the severity of the acute endpoint (death) and the proximity of the critical acute and subchronic/chronic NOELs, these low MOEs are cause for concern and mitigation measures should be considered.*"

    - The RCD states that exposure to the general public is not anticipated. However, there are currently no restrictions on how close homes can be to structures where phosphine is used. No buffer zones are required between the fumigated structure (e.g., a grain-elevator) and a residence. However, a buffer zone of 100 feet must be established between the fumigated burrow opening(s) and a structure potentially occupied by humans and/or domestic animals (as noted in DPR's EAD). Due to lack of buffer zones and the high toxicity of phosphine at low doses, OEHHA does not believe it is justified to rule out the possibility of significant phosphine exposures for residents living adjacent to structures being treated with aluminum or magnesium phosphide.

- Phosphine is designated as a restricted use pesticide (RUP) in recognition of its acute inhalation hazard. Page 6 of the RCD detailed the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Value (TLV) of 0.3 ppm and the Short-Term Emergency Limit (STEL) of 1 ppm based on Jones et al. (1964). In that study, workers were exposed intermittently to phosphine at concentrations up to 35 ppm, but averaging below 10 ppm in most cases (ACGIH, 2001). Commenting on the methodology used in this study, the RCD referenced O'Malley et al. (2013), who pointed out, "*Most of the phosphine measurements reported were area samples...it was difficult to identify the level of exposure associated with individual cases of illness and consequently difficult to identify levels of exposure that were tolerated without symptoms.*" Based on this comment, OEHHA suggests that the TLV and STEL may not be health protective values, especially for bystanders. Bystanders may be exposed for longer periods of time than workers and would not be expected to be wearing respiratory protection equipment. OEHHA notes that the STEL is equivalent to the NOEL identified for the subchronic and chronic calculations, which supports the need for an additional uncertainty factor to assess the health hazards associated with longer duration exposures.

- OEHHA suggests that infants and children may be more susceptible to the adverse health effects of phosphine and phosphine-generating products. A recent report cited in the RCD stated the possibility of children being more susceptible to phosphine-induced or mediated death (O'Malley et al., 2013). This may be due to their higher susceptibility to airborne toxicants, higher breathing rates on per kilogram body weight basis, and higher incidence of asthma. OEHHA suggests an additional 3-fold uncertainty factor to account for the intraspecies toxicokinetic and toxicodynamic differences in infants and children to account for increased susceptibility.

MISCELLANEOUS

- Usage
  - The annual agricultural use rates for phosphine from 2001-2010 are very well detailed in Table II-1, which includes pesticide application rates to parks, golf courses, cemeteries, rangeland, and pastures. Total pounds sold (which includes agricultural uses as well as home, urban-commercial, industrial, and other non-agricultural scenarios) are indicated in separate rows of the table. OEHHA suggests that data from 2011 be incorporated into this table, as total use of phosphine gas increased more than ten-fold from 2010 to 2011, and total use of aluminum phosphide increased nearly 50 percent over the same period. Year-over-year data are quite variable,

although a trend of increasing agricultural use of phosphine gas, aluminum phosphide and magnesium phosphide is apparent. Given the high toxicity of phosphine, and the low MOEs calculated in the RCD, this trend has possible public health implications. Occupational and residential bystanders may not be aware that fumigation is taking place near them and therefore would not be expected to use air-purifying respirators or other protective equipment. For similar reasons, residential bystanders may be exposed if living close to grain elevators or close to other places where fumigation occurs. Increased usage of phosphine could result in increased exposure for these groups.

- Oral Toxicity of Aluminum Phosphide
    - The RCD discusses the acute oral toxicity/lethality of a specific "test article" called Celphos in three places in the RCD (pages 12, 19, 22). This product was not listed in Table 1 ("Aluminum Phosphide Products") of DPR's 2013 Exposure Assessment Document (EAD), and Table III-1b of the RCD notes that the exact composition of this material was not stated in the oral toxicity study published by Batra et al (1994). Page 19 of the RCD notes that this product contains "56% aluminum phosphide along with ammonium compounds, binding and lubricating agents, fillers, etc." However, as an imprecisely characterized test agent, Celphos may not provide the best understanding of aluminum phosphide's toxicity. OEHHA recommends that DPR insert a caveat to this effect in the RCD. Given the uncertainties regarding the test article composition in the Batra et al. study and a second oral toxicity study conducted by Okolie et al. (2004), which evaluated the oral toxicity of a similarly uncharacterized product referred to as "phostoxin," OEHHA agrees with DPR's decision to not calculate an acute oral Reference Dose for aluminum phosphide.

- Environmental Fate
    - DPR included a separate analysis of the environmental fate of phosphine as an appendix to the RCD. The main body of the RCD provides brief descriptions of phosphine's fate in air, soil, water and wildlife that are clear and concise. The relevance to real world applications of the disappearance rate of phosphine gas measured in dry sealed tubes is unclear and an analysis of the relevance of these studies should be provided in the RCD. In addition, there is no citation for these studies in the RCD text (page 10). In the Environmental Fate document, attached as an appendix to the RCD (page 88), a study by Hilton and Robison (1972) was cited. If this is the same study as the one discussed on page 10, it should be referenced on page 10 as well.

Editorial Comments

Page 1. I. Summary: OEHHA suggests adding to the summary the routes of exposure that will be covered in the report.

Page 4, paragraph 1, line 9 and paragraph 4 line 7: OEHHA suggests not using Wikipedia as a citation as it is not necessarily a reliable source of information. Original reports as opposed to secondary references should be cited in the document.

Page 6, paragraph 3, line 3: "*...headache and dizziness in a **anumber** of workers exposed intermittently to phosphine at concentrations up to 35 ppm.*" This typo should be corrected. (Bold added for emphasis)

Page 6 C. Technical and Product Formulations: The RCD states, "There were two phosphine gas products registered in California as of 2008." OEHHA suggests adding the names of these two phosphine gas products. In addition, there are 18 products containing aluminum phosphide and five products containing magnesium phosphide as the active ingredient. Both compounds generate phosphine on contact with moisture and were evaluated in DPR's Exposure Assessment Document (EAD) for phosphine. OEHHA suggests that DPR refer the reader to the EAD for additional information on phosphine gas and phosphine-generating products registered for use in California, and provide a statement that they are evaluated in the Environmental Fate section at the end of the RCD. OEHHA also suggests adding post-2008 product information, if available.

Page 9 E. Illness Reports: The RCD states that the illness reports and cases for 2005-2009 are detailed in the Exposure Assessment Document (EAD). OEHHA suggests adding additional information in this section summarizing the reported illnesses, since the RCD and EAD are stand-alone documents and the RCD is directed at evaluating risk. These data provide useful information to consider in evaluating risk.

Page 11: III. Toxicology Profile: A. Pharmacokinetics: The RCD provides limited ADME data. Therefore, OEHHA suggests that DPR review the WHO (1988) report that evaluated pharmacokinetic data and provide a brief summary of it in this section.
Page 13, paragraph 1, line 5: Childs and Coates (1971) quoted a 1937 reference from the German literature. OEHHA suggests that DPR provide a citation for the original German report, and indicate whether this is the same study as the O.R. Klimmer study mentioned later (page 14, paragraph 2, line 1).

Page 14, 3. Laboratory animal studies, a. Inhalation:

- This section began by introducing a study by Garry and Lyubimov (2001) that cited a publication by O.R. Klimmer in German; however the year of that German study was not indicated.

- The RCD then described a variety of adverse health effects in laboratory animals. OEHHA suggests including the species of laboratory animals that were tested.

Page 21-22, Table III-1a: The acute/short term toxicity of phosphine was an excellent summary of the data.

- There was a formatting issue with the table and several items in the first column cannot be viewed.
- In addition, it is not clear where footnote "i" is in the table.
- Table II-1b has the same formatting issue (page 22).

Page 30, Table III-3: Please re-format column 1 so the text is completely visible.

Page 40, Table II-3: There is a problem with the table formatting in the first column cutting off the text. It is unclear where footnote c is in the table.

Page 43, Table II-3: Column 1 of this table needs to be reformatted.

Page 70 (and elsewhere): The O'Malley manuscript has been published. OEHHA suggests changing the citation to read 2013 throughout the document and in the References Cited section.

In the RCD, pages 3, 58 and 75 say: "*Many acute, seasonal and annual use scenarios generated MOEs under 100,* ***which indicates*** *insufficient health protection for workers and bystanders under those scenarios.*" OEHHA suggests changing the words "which indicates" to "indicating". (Bold added for emphasis).

Environmental Fate of Phosphine (Appendix II of the end of the RCD)

Page 8: The Environmental Fate Report stated that 27 products contain or produce phosphine gas, with two formulations of phosphine gas, 20 products containing aluminum phosphide and 5 containing magnesium phosphide. These numbers should be reconciled with the numbers on page 6 under Technical and Product Formulations in the Risk Characterization Document (RCD), which stated there are 18 products that contain aluminum phosphide.

Citations

O'Malley M, Fong H, Sánchez ME, Roisman R, Nonato Y, Mehler L. (2013). Inhalation of phosphine gas following a fire associated with fumigation of processed pistachio nuts. J Agromedicine 18(2):151-73.

World Health Organization (1988). Phosphine and selected metal phosphides. Environmental Health Criteria 73:1-77. Geneva. Resource document. Accessed 11 January 2013. http://www.inchem.org/documents/ehc/ehc/ehc73.htm.