Exhibit 5

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION	MDL No. 2741 Case No. 16-md-02741-VC
This document relates to: ALL ACTIONS	

EXPERT REPORT OF DR. CHARLES W. JAMESON, Ph.D.

IN SUPPORT OF GENERAL CAUSATION

ON BEHALF OF PLAINTIFFS

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Independent Consultant
May 12, 2017

Statement of Purpose

I have been asked to provide my expert opinions regarding the carcinogenic potential of glyphosate and glyphosate-based formulations. As a chemist and toxicologist, I evaluated the association of cancer, including non-Hodgkin's lymphoma ("NHL"), with exposure to glyphosate and/or glyphosate-based formulations. In performing my analysis, I relied on standard methods used in toxicology. I reviewed published, peer-reviewed scientific literature, publically available Government and Industry documents, and internal company documents and studies provided to me. All my opinions expressed in this report are based on a reasonable degree of scientific certainty. I reserve the right to supplement this report if additional information becomes available that are relevant to my opinions.

Qualifications

I am a private consultant in environmental toxicology specializing in carcinogenesis. I received my undergraduate degree in chemistry in 1970 from Mount Saint Mary's College, Emmitsburg, Maryland, and my Ph.D. in Organic Chemistry in 1975 from the University of Maryland, College Park. I started my career in 1965 where, as a rising high school senior, I spent the summer at a bioassay research laboratory first as a mouse room tech cleaning cages and later as an assistant in the chemistry lab mixing pesticides in rodent feed for the bioassay studies. Upon completion of my Ph.D. and a brief post-doc at the University of Maryland, I began working in 1976 as a contractor to the National Institutes of Health's (NIH) National Cancer Institute (NCI), serving as a senior chemist in support of NCI's Rodent Bioassay Program. In this capacity I was responsible for helping to monitor and evaluate the chemistry performed at the NCI's contract bioassay laboratories. In addition, I also provided support to the NCI staff for the identification of new substances to be studied in the NCI Bioassay Program. This

support included preparing and providing the background data from the scientific literature concerning exposure and the carcinogenic potential of the substance of interest. I was recruited by, and joined, the NCI in 1979 to serve as the chief chemist for their Rodent Bioassay Program and was responsible for directing and monitoring all chemistry activities, participating in the development of experimental protocols for the 2 year rodent bioassays conducted at the contract laboratories, and doing on-site inspections of all bioassay contract labs to insure they were following our protocols. In addition, I took over the responsibility as secretary for the NCI's Chemical Selection Working Group (CSWG) where I coordinated all activities for the identification of new substances to be studied in the Bioassay Program, including the oversight of the scientific literature searching, gathering and summarization process, documentation of the CSWG's review of the data and recommendations for study by the NCI, and the forwarding of the recommendation to the Director of the NCI Bioassay Program.

Following the formation of the National Toxicology Program (NTP) in 1978, the NCI Rodent Bioassay Program was transferred to the NIH's National Institute of Environmental Health Sciences (NIEHS) in 1980 where I transferred to and assumed the responsibility for all chemistry aspects of the NIEHS Division of Toxicology Research and Testing. I served as the program leader for chemistry in the National Toxicology Program (NTP) from 1978 until 1990. While chemistry program leader, I developed chemistry standards for bioassay studies that were widely accepted as an integral part of many toxicology-testing programs. I am listed as a contributor for the evaluation, interpretation and reporting of results for more than 100 chemicals studied in chronic two-year bioassay studies by the National Toxicology Program as published in the Technical Report Series (1980-1990). These bioassay studies were peer reviewed by the NTP Board of Scientific Counselors.

In 1990, I transferred to the NIEHS Director's Office and became involved with the NTP's Report on Carcinogens (RoC), working on it for more than 18 years, serving as its Director for 13 years before retiring from the NIEHS in February of 2008. The RoC is prepared in response to Section 301(b)(4) of the Public Health Service Act, which stipulates that the Secretary of the Department of Health and Human Services (DHHS) shall publish a report which contains a list of all substances which either are known or may reasonably be anticipated to be human carcinogens; and to which a significant

number of persons residing in the United States are exposed. This responsibility has been delegated by the Secretary to the Director, NTP. As Director of the RoC, I was responsible for the report's overall preparation, review and approval for the Director, NIEHS/NTP. In this capacity, I coordinated all review activities related to the RoC, which is one of the most visible and highly scrutinized activities of the NTP and the DHHS. I oversaw the identification and review of all new nominations for listing and delisting in upcoming editions of the RoC. I served as Chairman of the NIEHS RoC Review Committee, Chairman of the NTP Executive Committee's Interagency Working Group for the RoC, and Advisor to the NTP's Board of Scientific Counselors' Subcommittee for the RoC. I supervised the review of each nomination to the RoC, insuring all relevant information and data for each nomination was available for the review committees and managed the reviews by the three scientific review committees. Shortly after I became Director of the RoC in 1995, the Director, NTP, ordered that a review of the RoC be done to broaden input into its preparation, broaden the scope of scientific review associated with the Report, and provide review of the criteria used for inclusion of substances in the RoC. I coordinated this activity, which lead to revised criteria for the RoC being approved by the Secretary, DHHS in July of 1996. I served as Project Officer for the resource support contract for the preparation of the RoC, which included providing technical direction and coordination of the preparation of the documents prepared for each new nomination to the RoC as well as the preparation of 4 editions of the RoC for submission to the DHHS Secretary for approval.

I am the Senior Author for 69 NTP Report on Carcinogens Background Documents, which contained all available data concerning the exposure and potential carcinogenic activity of the substance being reviewed for possible listing in the RoC. I maintained a continuing liaison with other government agencies, private industries, other non-government research organizations and international organizations to keep abreast of work being done in chemical carcinogenesis, priorities for the listing of substances in the RoC, and resources available for the review of substances nominated for listing in the RoC. I served as the point of contact and focus for all RoC activities which included interacting with stakeholders from national and international government, industry, legal, consumer advocate, and other private concerns. I responded to requests for information from both the national and international press and private individuals on a

routine basis. Upon my retirement in 2008, I established CWJ Consulting LLC as a vehicle for providing expert consulting services in environmental toxicology specializing in carcinogenesis.

During my career, I participated as a Working Group Member for the United Nations' World Health Organization (WHO) International Agency for Research on Cancer (IARC). On several occasions, I served as either overall Chair of the Working Group or Chair of the Subgroup for Cancer in Experimental Animals evaluating cancer data and publishing monographs of the evaluation. I served as a consultant to the WHO, serving as a Task Group member to develop Environmental Health Criteria documents for partially halogenated chlorofluorocarbons (freons).

I am the author or co-author of over 80 peer reviewed scientific publications and nine book chapters. The vast majority of these publications relate to studies conducted in support of animal carcinogenesis bioassay programs. As mentioned above, I was the editor of four editions of the RoC, senior author for 69 NTP RoC Background Documents for substances reviewed for listing in the Report and listed as a contributor for the evaluation, interpretation and reporting of results for more than 100 chemicals studied in chronic two-year bioassay studies by the NTP as published in the Technical Report Series (1980-1990). I co-edited two books: "Chemistry for Toxicity Testing" and "Health and Safety for Toxicity Testing." A copy of my current curriculum vitae is attached as Exhibit A.

International Agency for Research on Cancer (IARC)

As an introduction, I would like to explain the International Agency for Research on Cancer's (IARC) review of glyphosate to assess its potential carcinogenicity, and the development of Monograph 112. The Working Group classified glyphosate as "probably carcinogenic to humans" (Group 2A) at their meeting in March of 2015. Following this meeting, there have been a number of publications (including, but not limited to, Williams et al.[1, 2]; Chang and Delzell[3], Solomon[4]) criticizing the IARC review process and conclusions.

The purpose of the *Monographs* is to render critical reviews and evaluations of carcinogenicity evidence of a wide range of human exposures.[5] The *Monographs*

represent a hazard identification that involves examination of all relevant information to assess the strength of the available evidence that an agent can cause human cancer. Identifying carcinogens is a key step in cancer prevention, and this activity represents an important international activity towards improving public health. The IARC Preamble[5] states that a "cancer 'hazard' is an agent that can cause cancer under some circumstances, while a cancer 'risk' is an estimate of the carcinogenic effects expected from exposure to a cancer hazard. The *Monographs* are an exercise in evaluating cancer hazards, despite the historical presence of the word 'risks' in the title. The distinction between hazard and risk is important, and the *Monographs* identify cancer hazards even when risks are very low at current exposure levels, because new uses or unforeseen exposures could engender risks that are significantly higher." In other words, hazard assessment determines whether an agent can cause cancer.

For the review of glyphosate as it relates to Monograph 112, IARC perfomed a search for all relevant biological and epidemiological data from publically available sources and sent copies of the materials found to the Working Group participants approximately six months prior to the start of the meeting. In addition to the materials sent from IARC, Working Group participants perform their own independent search of the scientific literature. As the IARC Preamble notes, "with regard to epidemiological studies, cancer bioassays, and mechanistic and other relevant data, only reports that have been published or accepted for publication in the openly available scientific literature were reviewed."[5] IARC also considers relevant and publically available material from US Environmental Protection Agency ("EPA"). Studies determined to be irrelevant, inadequate, or published too late to be adequately evaluated were cited but were not summarized. This process of data collection is typical of all IARC *Monographs* and is the body of literature used by the Working Group participants during each Monograph anaylsis.

The IARC Working Group meeting takes places at its headquarters in Lyon, France and lasts for approximately seven to eight days, where the Working Group will then finalize the texts and formulate its final evaluations. Participants are assigned to one of four subgroups covering either exposure data, cancer in humans, cancer in experimental animals, or mechanistic and other relevant data. Working Group participants are also assigned individual chemicals or agents being evaluated and asked to prepare preliminary

working papers for their specific subgroup that are then distributed prior to the meeting. The subgroups prepare joint drafts and summaries in breakout sessions during the first few days. The entire Working Group meets in brief plenary sessions every day to get updates on the progress of each individual subgroup and to discuss any issues the subgroups may have identified. The final days of the meeting consists of plenary session meetings to discuss all relevant data, review the subgroup drafts and develop the final evaluations. The entire Monograph volume is considered the joint product of the Working Group, and there are no individually authored sections.[5]

For Monograph 112, I served as Chairman of the subgroup for Cancer in Experimental Animals to assess the carcinogenicity of several organophosphate pesticides that included glyphosate, the active ingredient in Roundup®. This meeting was held March 3-10, 2015 and the Working Group classified glyphosate as "probably carcinogenic to humans" (Group 2A). This classification was based on limited evidence in humans for the carcinogenicity of glyphosate where a positive association has been observed for NHL, sufficient evidence in experimental animals for the carcinogenicity of glyphosate and that mechanistic and other relevant data support the classification of glyphosate in Group 2A. To provide a better understanding of this, I will: discuss the process used by the Working Group to arrive at this classification, define terms, explain the types of evidence considered, explain the scientific criteria that guide the evaluations, and explain how conclusions were reached throughout the process.

The following summary of the Working Group's evaluation of the available literature is offered here, but also found in the IARC's Preamble[5]:

•Exposure Data: The Working Group concluded there is wide spread exposure to glyphosate based on its use as the active ingredient in Roundup® which is a broad-spectrum herbicide. Glyphosate is the most heavily used herbicide in the world[6] and can be found in soil, air, surface water, groundwater, and food. According to several studies, glyphosate has also been detected in urine from persons around the world.[7-10] The general population is mainly exposed to glyphosate through diet and from use as a household weed control.

•Cancer in Humans: The Working Group identified seven reports from the Agricultural Health Study (AHS) cohort and numerous reports from case-control studies

in the evaluation of the epidemiological studies reporting on cancer risks associated with exposure to glyphosate. This Working Group applied the Bradford Hill criteria in its analyses and determined that in several case–control studies there was an increased risks for NHL due to glyphosate exposure.[11-18] The Working Group further noted that the increased risk for NHL persisted in the studies that adjusted for exposure to other pesticides. The Working Group concluded a positive association has been observed for exposure to glyphosate and NHL and that there is "limited evidence" in humans for the carcinogenicity of glyphosate. IARC determines limited evidence of carcinogenicity for an agent when "a positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence."[5]

•Cancer in Experimental Animals: The Working Group reviewed scientific literature and reports including two studies in which glyphosate was reported to be tested for carcinogenicity in male and female mice by dietary administration, five studies that tested glyphosate in male and female rats by dietary administration and in drinking-water in one study. Studies of a glyphosate-based formulation tested in drinking-water in one study in male and female rats and by skin application in one initiation–promotion study in male mice were also reviewed. They observed that in one feeding study in male CD-1 mice,[19-22] glyphosate induced a positive trend in the incidence of kidney renal tubule carcinoma, a rare tumor in this strain of mice. A second feeding study[23] reported a positive trend for hemangiosarcoma (a blood vessel tumor) in male mice. Glyphosate also increased pancreatic islet-cell adenoma in male rats in two feeding studies.[24-26] The Working Group concluded there is "sufficient evidence" in experimental animals for the carcinogenicity of glyphosate. IARC defines "sufficient evidence" in experimental animals is as "a causal relationship has been established between the agent and an increased incidence of malignant neoplasms or of an appropriate combination of benign and malignant neoplasms in (a) two or more species of animals or (b) two or more independent studies in one species carried out at different times or in different laboratories or under different protocols."[5]

•Mechanistic and Other Relevant Data: The Working Group reported the mechanistic data literature contained strong evidence that glyphosate causes genotoxicity

and oxidative stress. The strong evidence of genotoxicity came from studies conducted in human cells in vitro,[27-32] in mammalian model systems in vivo[27,32] and in vitro,[33,34] and from studies in other non-mammalian organisms[29,35,36,37], all of which yielded largely positive results. The Working Group also found strong evidence for genotoxicity caused by glyphosate-based formulations. There were three studies of genotoxicity end-points in community residents exposed to glyphosate-based formulations, two of which reported positive associations.[38,39] Strong evidence for oxidative stress was determined by studies conducted in human cells in vitro[28,40,41] and in many rodent tissues in vivo.[32,42,43] The Working Group found weak evidence that glyphosate or glyphosate-based formulations induce receptor-mediated effects,[44,45] may affect cell proliferation or death,[44,46] and may also affect the immune system in rodents[47] and fish.[48,49] The Working Group considered the body of evidence described above as a whole and reached an overall evaluation of Group 2A: glyphosate is probably carcinogenic to humans. IARC uses this category when evidence of carcinogenicity in humans is limited and evidence of carcinogenicity in experimental animals is sufficient.[5]

IARC uses the hazard identification process for its review, and this was done for Monograph 112. Hazard identification reflects the toxicological "law" of specificity of effects[50]. Hazard identification uses a strength of the evidence approach. As applied, the Working Groups for Monograph 112 rigorously assessed the toxicological, mechanistic, and epidemiological data to form a judgment regarding the likelihood that the agent produces cancer.

Information Reviewed

During the course of work on this case, I reviewed the following materials:

- scientific literature relating to the carcinogenicity of glyphosate and/or glyphosate-based formulations;
- government documents relevant to assessing the carcinogenic hazard and risks associated with glyphosate and/or glyphosate-based formulations; and,
- various studies and documents produced in the litigation.

For a list of additional materials I reviewed, please see Exhibit B.

Description of the Methodology Used to Assess Carcinogenic Potential Associated with Exposure to Glyphosate and/or Glyphosate-Based Formulations.

Toxicologists routinely assess the hazards to human health related to exposure to chemicals in the everyday environment using a process called hazard identification. A hazard is any agent that can cause harm or damage to humans, property, or the environment.[51] In other words, a hazard is any agent that can cause a specific damage. In this case, the hazard being examined is glyphosate and/or glyphosate-based formulations, the specific damage is NHL, and the hazard assessment I am making is to determine whether or not glyphosate and/or glyphosate-based formulations can cause NHL. The terms hazard and risk are often used interchangeably; however, these are two distinct terms. Risk is defined as the probability that exposure to a hazard will lead to a negative consequence, or more simply, risk = hazard x dose (exposure).[52]

Toxicology is the basis on which hazard identification is established. Hazard assessment has been used for over four decades by a wide variety of governmental and nongovernmental organizations to evaluate the potential adverse health effects from chemical exposures. Hazard identification is a standard tool used by toxicologists when they are trying to determine if exposure to a chemical(s) can cause an adverse health effect in humans and is the first step in risk analysis. Hazard identification is performed by identifying the chemical someone has been exposed to and then reviewing the available toxicity data to outline the spectrum of adverse effects that would be associated with exposure to that particular chemical.[53] The toxicity data could be from studies in humans, in whole animals, or in cells, or could be data collected on chemically-similar substances when data on the chemical of interest are limited.

I used the following criteria for my hazard based assessment of glyphosate and/or glyphosate-based formulations, that is based on the criteria I developed for the Report on Carcinogens[54] and is the same as defined and characterized by IARC[5]:

• Cancer in Humans – Numerous case-control studies and the Agricultural Health Study (AHS) cohort reporting on possible associations of cancer and exposure to glyphosate were evaluated for any evidence of a causal relationship between glyphosate and human cancer.

- "Sufficient" evidence is defined as when a causal relationship was established between exposure to glyphosate and cancer and that chance, bias and confounding could be ruled out.[5]
- "Limited" evidence is defined as a positive association has been observed between exposure to glyphosate and cancer and a causal interpretation is credible but alternative explanations such as chance, bias or confounding could not be ruled out.[5]
- "Inadequate" evidence is defined as available studies are of insufficient quality, consistency or statistical power to permit a conclusion regarding a causal association between glyphosate exposure and cancer.[5]

• Cancer in Experimental Animals – the experimental animal studies reporting on possible associations of cancer and exposure to glyphosate were evaluated for any evidence of a causal relationship between glyphosate and cancer.

- "Sufficient" evidence is defined as a causal relationship between exposure to glyphosate and an increased incidence of malignant and/or a combination of malignant and benign tumors, in multiple species or at multiple tissue sites or from multiple studies, or by multiple routes of exposure, or to an unusual degree with regard to incidence, site, or type of tumor, or age at onset.[5]
- "Limited" evidence is defined as the data suggest a carcinogenic effect but are limited for making a definitive evaluation because, e.g. the evidence of carcinogenicity is restricted to a single experiment; there are unresolved questions regarding the adequacy of the design, conduct or interpretation of the studies; or the agent increases the incidence only of benign neoplasms or lesions of uncertain neoplastic potentials.[5]
- "Inadequate" evidence is defined as studies that cannot be interpreted to show either the presence or absence of a positive carcinogenic effect because of major qualitative or quantitative limitations such as inadequate numbers of animals, lack of adequate pathology, poor survival, major impurities in the test agent, too low a dose to see an effect, etc. It should be noted that although animal testing is routinely used to identify cancer hazard, the sites

of cancer observed in animals do not always correlate directly with the sites of cancer that would be observed in humans[55]. This can be due to the differences in metabolism in laboratory animals and humans, differences in pharmacokinetics, or differences in tissue reactivity (pharmacodynamics) between species. Animal studies, instead, are used to identify a threat of cancer that is applied to human health hazard assessment[55]. All chemicals known to induce cancer in humans, that have been studied under adequate experimental conditions, also cause cancer in laboratory animals[55] and underscores the concept that chemicals found to cause cancer in laboratory animals must be considered capable of causing cancer in humans.[5]

•Mechanistic and other data – studies containing data relevant to the possible mechanim(s) of glyphosate carcinogenesis (genetic toxicity, epigenetic effects, etc.) were also evaluated. Mechanistic data may provide evidence of carcinogenicity and help in assessing the relevance and importance of findings of cancer in animals and humans.[5]

Hazard Assessment of the Human Data for Glyphosate and/or Glyphosate-Based Formulations

Before discussing the human data for glyphosate and/or glyphosate-based formulations, I will define the type of epidemiology studies that were reviewed:

• Case-Control Study - In a case-control study, investigators start by enrolling a group of people with disease. As a comparison group, the investigator then enrolls a group of people without disease (controls). Investigators then compare previous exposures between the two groups. The control group provides an estimate of the baseline or expected amount of exposure in that population. If the amount of exposure among the case group is substantially higher than the amount you would expect based on the control group, then illness is said to be associated with that exposure. The key in a case-control study is to identify an appropriate control group, comparable to the case group in most respects, to provide a reasonable estimate of the baseline or expected exposure.[56]

• Cohort Study - According to Centers for Disease Control and Prevention (CDC),[57] in a cohort study the epidemiologist records whether each study participant is exposed or not, and then tracks the participants to see if they develop the disease of interest. After a

period, the investigator compares the disease rate in the exposed group with the disease rate in the unexposed group. The unexposed group serves as the comparison group or control, providing an estimate of the baseline or expected amount of disease occurrence in the community. If the disease rate is substantively different in the exposed group compared to the unexposed group, the exposure is said to be associated with illness.

• Meta-Analysis – A meta-analysis is an important component of systematic review procedure that combines and analyzes quantitative and qualitative data from several separate but similar experiments or studies to test the pooled data for statistical significance. Combining the results of multiple studies produces a weighted average of the included study results and leads to a conclusion with greater statistical power and point estimate than would be possible from any individual study.

Case Control Studies

• Cantor et al. (1992)[14] evaluated the incidence of NHL among males located in Iowa and Minnesota. A total of 622 men and 1245 population-based controls were included in the study. The association with farming occupation and specific agricultural exposures were evaluated. When compared with non-farmers, the positive associations (odds ratios) for NHL were significant at 1.2 (95% CI, 1.0–1.5) for men who had ever farmed, and not significant at 1.1 (95% CI, 0.7–1.9) for 26 exposed cases for ever handling glyphosate and adjusted for confounders (vital status, age, state, cigarette smoking status, family history of lymphohaematopoietic cancer, high-risk occupations, and high-risk exposures).

•DeRoos et al. (2003)[11] pooled the data from three case–control studies[12-14] to study pesticide exposures as risk factors for NHL in men. Of a total study population of 870 cases and 2569 controls, there were 650 cases and 1933 controls included for the analysis of 47 pesticides that also controlled for potential confounding by other pesticides. A positive association (odds ratios) for the association between exposure to glyphosate and NHL in the 36 cases exposed was reported to be significant at 2.1 (95% CI, 1.1–4.0) in the logistic regression analyses but not in the hierarchical regression analysis (which uses a more conservative adjustment estimate) at 1.6 (95% CI, 0.9–2.8).

•The effect of asthma as a modifier of the association between pesticide exposure and NHL was reported on by Lee et al. (2004)[58]. The study contained 872 cases diagnosed

with NHL, 45 of which had been told they also had asthma and 2381 matched controls, 132 reporting to have asthma. Individuals in the study group with a history of asthma had a non-significantly lower risk of NHL than non-asthmatics and no effect was seen with pesticide exposure. A positive associations (odds ratio) for NHL associated with glyphosate use were reported but were not significant at 1.4 (95% CI, 0.98–2.1; 53 exposed cases) among non-asthmatics and 1.2 (95% CI, 0.4–3.3; 6 exposed cases) for asthmatics, when compared with non-asthmatic non-exposed farmers.

•The associations between exposure to pesticides and NHL was studied by McDuffie et al. (2001)[15] in a multicenter population-based study that included 517 cases and 1506 controls among men of six Canadian provinces. A non-significant positive association (odds ratios) of 1.26 (95% CI, 0.87–1.80; 51 exposed cases; adjusted for age and province) and 1.20 (95% CI, 0.83–1.74, adjusted for age, province, high-risk exposures) were observed for exposure to glyphosate. In an analysis by frequency of exposure to glyphosate, participants with more than 2 days of exposure per year had a statistically significant positive association (odds ratio) of 2.12 (95% CI, 1.20–3.73, 23 exposed cases) compared with those with some, but less than 2 days of exposure.

•Nordstrom et al (1998)[59] conducted a study in Sweden on hairy cell leukemia (considered to be a subtype of NHL). There were 121 cases in men and 484 controls matched for age and sex. A non-significant age-adjusted positive association (odds ratio) of 3.1 (95% CI, 0.8–12; 4 exposed cases) was reported for exposure to glyphosate.

•Hardell and Eriksson (1999)[60] reported on the results of the incidence of NHL in men associated with pesticide exposure in four northern counties in Sweden and included 404 cases and 741 controls. The authors reported a non-significant positive association (odds ratio) for ever-use of glyphosate of 2.3 (95% CI, 0.4–13; 4 exposed cases) in an analysis of glyphosate only, and 5.8 (95% CI, 0.6–54) in a multivariable analysis.

•Hardell et al. (2002)[17] performed a pooled analysis of two case–control studies, one on NHL[60] and another on hairy cell leukemia.[59] These pooled analyses were based on 515 cases and 1141 controls. A significant positive association was found for exposure to glyphosate compared to controls (odds ratio, 3.04; 95% CI, 1.08–8.52; 8 exposed cases), but the positive association (odds ratio) decreased to a non-significant 1.85 (95% CI, 0.55–6.20) when study area, and vital status were considered.

•A large population based case–control study of exposure to pesticides as a risk factor for NHL in Sweden was conducted by Eriksson et al. (2008)[18]. There were 910 cases and 1016 controls included in the study. The association (odds ratio) for exposure to glyphosate to NHL was positive and significant at 2.02 (95% CI, 1.10–3.71) compared to controls, but positive and non-significant at 1.51 (95% CI, 0.77–2.94) when confounders that included exposure to other pesticides, age, sex, and year of diagnosis or enrolment were included in the analysis. When exposure to glyphosate for more than 10 days per year was considered, the positive association (odds ratio) was significant at 2.36 (95% CI, 1.04–5.37). Considering a latency period of greater than 10 years gave a positive association (odds ratio) that was also significant at 2.26 (95% CI, 1.16–4.40). The authors also reported an association with exposure to glyphosate and lymphoma subtypes. Positive associations were reported for most of the cancer forms, including B-cell lymphoma (odds ratio of 1.87; 95% CI, 0.998–3.51, non-significant) and the subcategory of small lymphocytic lymphoma/chronic lymphocytic leukemia (odds ratio of 3.35; 95% CI, 1.42–7.89, significant). These odds ratios were not adjusted for other pesticides.

•Orsi et al. (2009)[61] reported the results of a case–control study conducted in France. The study included 491 cases (244 cases of NHL, 87 cases of Hodgkin lymphoma, 104 of lymphoproliferative syndrome, and 56 cases of multiple myeloma), and 456 age- and sex-matched controls. Positive, non-significant associations (odds ratios) for any exposure to glyphosate were reported: 1.2 (95% CI, 0.6–2.1; 27 exposed cases) for all lymphoid neoplasms combined, 1.0 (95% CI, 0.5–2.2; 12 exposed cases) for NHL, 0.6 (95% CI, 0.2–2.1; 4 exposed cases) for lymphoproliferative syndrome, 2.4 (95% CI, 0.8–7.3) for multiple myeloma, and 1.7 (95% CI, 0.6–5.0; 6 exposed cases) for Hodgkin lymphoma, after adjusting for age, and socioeconomic category.

•Cocco et al. (2013)[62] performed a pooled analysis of case–control studies from six European countries to investigate the role of occupational exposure to specific groups of chemicals in the causation of lymphoma overall, B-cell lymphoma, and its most prevalent subtypes. A total of 2348 incident cases of lymphoma and 2462 controls were included in the study. Analyses were conducted for lymphoma and the most prevalent lymphoma subtypes and adjusted for age, sex, and education. A positive, non-significant association (odds ratio) of 3.1 (95% CI, 0.6–17.1) was reported for exposure to glyphosate and B-cell lymphoma.

I would note that the findings in the McDuffie et al. (2001)[15]; and Eriksson et al.[18] studies is significant because their results are supported by the results reported for micronucleus formation studies in the bone marrow of mice by Rank et al. (1993)[63] where a single dose caused no effect while Bolognesi et al. (1997)[32] and Manas et al. (2009)[27] reported that two daily doses of glyphosate did cause micronucleus formation in the bone marrow of mice in their studies. This implies that level of exposure is an important consideration in the formation of NHL from exposure to glyphosate.

Cohort Studies

The Agricultural Health Study (AHS)[64] is a large prospective study of cancer and other health outcomes in a cohort of licensed pesticide applicators and their spouses from Iowa and North Carolina. The AHS began in 1993 with the goal of answering important questions about how agricultural, lifestyle and genetic factors affect the health of farming populations. More than 89,000 farmers and their spouses in Iowa and North Carolina have participated in the study. It is the only cohort study to date to have published findings on exposure to glyphosate and the risk of cancer at many different sites. My summary of the 7 papers available evaluating cancer incidence associated with pesticide use in the AHS cohort follows:

•No risk estimates and no significant exposure-response associations with cancer of the prostate and exposure to glyphosate were reported by Alavania et al (1996).[65]

•DeRoos et al. (2005)[66,67] evaluated associations between glyphosate exposure and the incidence of cancer at multiple sites in this cohort including lung, melanoma, multiple myeloma, and NHL, oral cavity, colon, rectum, pancreas, kidney, bladder, prostate, and leukemia. No significant exposure–response association with cancer at any of these sites was found.

•Flower et al.,[68] reported the results of the analyses of risk of childhood cancer associated with pesticide application by the parents of 17,357 children of Iowa pesticide applicators from the AHS cohort. For all the children of the pesticide applicators, the risk of cancer was increased for all childhood cancers combined, for all lymphomas combined, and for Hodgkin lymphoma, compared with the general population. A non-significant association (odds ratio) for use of glyphosate and risk of childhood cancer was reported to be 0.61 (95% CI, 0.32–1.16; 13 exposed cases) for maternal use and 0.84 (95% CI, 0.35–

2.34; 6 exposed cases) for paternal use.

• The incidence of cancer of the breast among farmers' wives in the AHS cohort, which included 30,454 women with no history of cancer of the breast before enrolment was reported by Engel et al.,[69]. There was no difference in incidence of breast cancer for women who reported ever applying pesticides compared with the general population. A non-significant association (relative risk) for cancer of the breast was reported to be 0.9 (95% CI, 0.7–1.1; 82 cases) among women who had personally used glyphosate and a non-significant positive association (relative risk) of 1.3 (95% CI, 0.8–1.9; 109 cases) among women who never used pesticides but whose husband had used glyphosate.

•Lee et al.,[70] studied the relationship between exposure to agricultural pesticides and incidence of cancer of the colorectum in the AHS cohort. Non-significant positive associations (relative risks) with exposure to glyphosate was reported to be 1.2 (95% CI, 0.9–1.6) for cancers of the colorectum, and 1.6 (95% CI, 0.9–2.9) for cancers of the rectum. A non-positive association of 1.0 (95% CI, 0.7–1.5) was reported for cancers of the colon.

•Andreotti et al.,[71] used a case–control analysis nested in the AHS cohort to study associations between the use of pesticides and cancer of the pancreas. For pancreatic cancer, a positive association (odds ratio) for ever- versus never-exposure to glyphosate was found but not significant at 1.1 (95% CI, 0.6–1.7; 55 exposed cases) and for highest category of level of intensity-weighted lifetime days was also found but not significant at 1.2 (95% CI, 0.6–2.6; 19 exposed cases).

•Dennis et al.,[72] reported that exposure to glyphosate was not associated with cutaneous melanoma within the AHS cohort but did not report a risk estimate.

Meta-Analyses

•Schinasi & Leon[73] conducted a systematic review and meta-analysis of NHL and occupational exposure to agricultural pesticides, including glyphosate. The meta-analysis for glyphosate included six studies (McDuffie et al.,[15] Hardell et al.,[17] DeRoos et al.,[67,11] Eriksson et al.,[18] and Orsi et al.[61]) and yielded a significant positive asso ciation (meta risk-ratio) of 1.5 (95% CI, 1.1–2.0) for exposure to glyphosate and NHL.

•IARC[74] conducted an additional meta-analysis of NHL and occupational exposure to agricultural pesticides, including glyphosate using data from Schinasi & Leon[73] and

included the fully adjusted risk estimates from the studies published by Hardell et al.,[17] and Eriksson et al.[18] After considering the adjusted estimates of the two Swedish studies in the meta-analysis, the positive association (meta risk-ratio) was still significant at 1.3 (95% CI, 1.03–1.65).

•Chang and Delzell[3] also conducted a systematic review and meta-analysis to examine the relationship between glyphosate exposure and risk of lymphohematopoietic cancer including NHL, Hodgkin lymphoma, multiple myeloma, and leukemia. Their analysis showed a positive association (meta-relative risks or meta-RRs) and was statistically significant for the association between any versus no use of glyphosate and risk of NHL (meta-RR=1.3, 95% confidence interval (CI)=1.0–1.6, based on six studies) and multiple myeloma (meta-RR =1.4, 95% CI=1.0–1.9; four studies). The authors conducted four meta-analyses for NHL, all reporting to have a significant positive association (meta-RR) of 1.3 or 1.4 with 95% CIs ranging from (1.0-1.6) to (1.0-1.8). The authors concluded "we found marginally significant positive meta-RRs for the association between glyphosate use and risk of NHL."

Summary for Human Data

I have evaluated available epidemiology data. Based on my experience doing hazard assessments, I learned that epidemiologists consider case–control studies particularly valuable for determining the carcinogenicity of an agent because their design facilitates exposure assessment and reduces the potential for certain biases. My review of the literature finds that the two case-control studies from the United States and Canada, and the two case–control studies from Sweden indicated statistically significant positive associations between exposure to glyphosate and NHL. The Canadian study, McDuffie (2001)[15], reported a positive association between glyphosate exposure and NHL for those case subjects with more than two days/year of exposure (odds ratio of 2.12(95%CI,1.20–3.73) when compared to those with less than two days exposure. Three studies reported excesses for NHL associated with exposure to glyphosate, after adjustment for other pesticides, De Roos (2003) reported a significant positive association (odds ratio) for a pooled US study[11] at 2.1 (95% CI, 1.1–4.0).; and the two Swedish studies (Hardell (2002)[17], Eriksson (2008)[18]) reported significant positive associations of 3.04; 95% CI, 1.08–8.52

and 2.36(95%CI, 1.04–5.37). The positive association from Hardell (2002)[17] decreased to non-significance (1.85 (95% CI, 0.55–6.2)) when study area, and vital status were considered. Subtype-specific analyses in a Eriksson (2008)[18] indicated positive associations for total NHL, as well as all subtypes, but this association was statistically significant only for the subgroup of lymphocytic lymphoma/chronic lymphocytic leukemia (odds ratio, 3.35; 95% CI, 1.42–7.89). A European study[62] based on few cases also indicated an elevated risk (OR, 3.1; 95% CI, 0.6–17.1) for B-cell lymphoma. A French hospital-based case–control study[61] did not find an association between exposure to glyphosate and NHL (OR, 1.0; 95% CI, 0.5–2.2) based on few exposed cases. For the evaluation of glyphosate, the Agricultural Health Study (AHS) is currently the only cohort study available providing information on its potential carcinogenicity and did not show an excess of NHL. There were three groups that did meta-analyses of the human data for an association between glyphosate use and NHL. Schinasi and Leon[73] reported a significant positive association (meta-RR) of 1.5 (95% CI, 1.1–2.0). The IARC study[74] showed a positive association (meta-RR) of 1.3 (95% CI, 1.03-1.65). Chang and Delzel[3] provided four separate meta-analyses, all of which are reported as having a significant association (meta-RR) of either 1.3 or 1.4 with CIs ranging from (1.0-1.6) to (1.0-1.8). When the data across all epidemiological studies are combined, results indicate a positive association between glyphosate exposure and NHL in humans.

Interpreting the epidemiology findings requires one to properly weight studies according to quality rather than simply count the number of positives and negatives. The pooled case–control analysis from the USA[11] contained 650 cases of NHL. It follows that the case-control studies provide a stronger assessment of the potential carcinogenicity of glyphosate. The case-control studies in the US[11], Canada[15] and Sweden[17,18] indicate a significant positive association for NHL with exposure to glyphosate. This positive association was also observed in the studies that adjusted for other pesticides. The AHS cohort did not show an excess of NHL; however it reports on only 92 NHL cases in the unadjusted analysis.[64] The three meta-analyses I reviewed are good examples of objective evaluations and show a consistent positive association between glyphosate and NHL. Drawing on the Bradford-Hill criteria[75] for causality, I would state that the observations are consistent (relative risks and meta analyses are positive for the case control studies), significant, not specific, temporally observed, shows a biological gradient, and is coherent

with the animal evidence (discussed below). Using my stated criteria, I conclude there is "Limited" evidence for the carcinogenicity of glyphosate in humans, because a positive association has been observed between exposure to glyphosate and NHL, and a causal interpretation is creditable but alternative explanations such as chance, bias or confounding could not be completely ruled out.

Hazard Assessment of the Experimental Animal Data for Glyphosate and/or Glyphosate-Based Formulations

Before discussing the experimental animal data for glyphosate and/or glyphosate-based formulations, I will define what is involved in a cancer bioassay in experimental animals. The basic cancer bioassay design has remained relatively constant for more than 40 years and consists of groups of 50 male and female mice and rats in each dose and control group. Treatment traditionally lasts for 24 months and commences when the animals are 6–8 weeks of age. Early bioassay studies involved two treatment groups plus a control group. The first treatment group was a high dose, referred to as a maximally tolerated dose (MTD), and the second treatment group was half that dose. More recent studies typically include three (and sometimes up to five) treatment groups plus the control group.

In the bioassays, I reviewed the nature and extent of impurities or contaminants, the animal species, strain, sex, numbers per group, age at start of treatment, route of exposure, dose levels, duration of exposure, survival and information on tumors. With regard to the tumors, I evaluated the incidence, latency, severity or multiplicity of neoplasms or preneoplastic lesions. Studies in experimental animals that I determined to be inadequate for evaluation (e.g. too short a duration, too few animals, poor survival) can be found at the end of my reference list.

Cancer Bioassays in Mice

•Knezevich and Hogan[76] (1983) were the authors of a report submitted to the Environmental Protection Agency (EPA)[77] by Monsanto in support of the registration of glyphosate as an herbicide. This report was also discussed in the paper by Greim[78] (referred to as Study 10). For 24 months, groups of 50 male and 50 female CD-1 mice received diets containing glyphosate (purity, 99.7%) at a concentration of 0, 1000, 5000,

or 30,000 ppm, ad libitum. The study observed no treatment-related effect on body weight in male and female mice at the lowest or intermediate dose, but a slight reduction in body weight in the male and female mice at the highest dose compared with controls. Survival in all dose groups was similar to controls. (It does not appear that a MTD was reached). There was a positive trend[79] (p = 0.016, trend test) in the incidence of renal tubule adenoma in dosed male mice: 0/49, 0/49, 1/50 (2%), 3/50 (6%). Renal tubule adenoma is a rare tumor in CD-1 mice. Historical control data from 14 studies conducted between 1977 and 1981 at the testing laboratory indicated that the mouse renal tumors ranged from 0 to 3% and the incidence in the current study (3/50; 6%) exceeded the upper limit of the historical control range by a factor of two. The rarity of this tumor in CD-1 mice is documented in a publication by Chandra and Frith[80] that reports only 1 out of 725 [0.14%] CD-1 male mice in their large historical database had developed renal cell tumors (one carcinoma). No tumors of the kidney were observed in the female mice. No other tumor sites were identified.

A re-evaluation of the original renal section was conducted by a Monsanto consulting pathologist who reported a small renal tubule adenoma in one control male mouse, which was not diagnosed as such in the original pathology report.[81] This finding was contrary to the initial findings of Bio/dynamics lab, the lab commissioned to complete this report. Following Monsanto's submission of the consulting pathologist's report, the EPA reported there was no difference in diagnoses between his and other pathologists' diagnoses with respect to kidney tumors in mid- and high-dose groups (i.e. 0/49, 0/49, 1/50 (2%), 3/50 (6%)). The EPA pathologist also indicated in his report[79] this data also shows a positive trend (p = 0.016, trend test) in the incidence of renal tubule adenoma in the dosed male mice. Regarding the questionable male control kidney, it was his opinion that the presence of a tumor cannot definitely be established. Nonetheless, the EPA requested additional renal sections be cut and evaluated from all male mice in the control and treated groups; this additional review found no additional tumors.[81] The EPA also requested that a pathology working group (PWG) be convened to evaluate the tumors of the kidney observed in male mice treated with glyphosate, including the additional renal sections.[82] Monsanto sponsored a PWG that reported the incidence of adenoma of the renal tubule was 1/49 (2%), 0/49, 0/50, 1/50 (2%)(not statistically significant); the incidence of carcinoma of the renal tubule was 0/49, 0/49, 1/50 (2%),

2/50 (4%) (which gives a significant p = 0.037, trend test for carcinoma); and the incidence of adenoma or carcinoma (combined) of the renal tubule was 1/49 (2%), 0/49, 1/50 (2%), 3/50 (6%) (which gives a significant p = 0.034, trend test for combined). The PWG did not discuss their finding of an adenoma in the control male mice or address the previous opinion that the presence of a tumor in the control male mice cannot definitely be established and concluded the kidney tumors were not compound related.[83] It is important to note that the renal tumor identified in the controls by the PWG after re-evaluation of the original slides was not seen in the re-sectioned kidney slides. My conclusion of the results discussed above is that there was a significant increase in the incidence of these rare kidney tumors in the CD-1 mouse, with a dose-related trend, which is caused by glyphosate. For the purpose of this hazard identification the increase the incidence of carcinoma of the renal tubule and the incidence of adenoma or carcinoma (combined) of the renal tubule in male mice is due to treatment with glyphosate that caused a significant, dose related increase of these rare tumors in male CD-1 mice.

•Atkinson et al.[84] (1993) were the authors of a report submitted to the EPA in support of the re-registration of glyphosate as an herbicide. This study was also discussed in the paper by Greim[78] (Study 11). Groups of 50 male and 50 female CD-1 mice were given diets containing glyphosate (purity, 98.6%) at a concentration that was adjusted weekly for the first 13 weeks and every 4 weeks thereafter to give doses of 0, 100, 300, or 1000 mg/kg bw, ad libitum, for 104 weeks. There was no treatment-related effect on body weight or survival in any of the dosed groups indicating a maximum tolerated dose was not achieved. The EPA reported[77] a statistically significant increase in the incidence of hemangiosarcoma (blood vessel tumor) in males – 0/47, 0/45, 0/50, 4/45 (9%) ($p < 0.01$, trend test), and non-significant increase in females – 0/50, 2/50 (4%), 0/50, 1/50 (2%). The EPA pointed out that the incidence in the high dose males was near the upper limit (0-8%) for the performing laboratory. However, if one looks at excerpts from the full report,[84] Table 15 (page 97) indicates that as few as 2 animals per dose group were examined histologically for this tumor. This would lead one to consider that the incidence of this tumor could have been higher in this study as more of these tumors could have been found if all 50 animals per dose group were examined. There was also reported a non-significant increase in the incidence of histiocytic sarcoma in the lymphoreticular/haemopoietic tissue in males – 0/50, 2/50 (4%), 0/50, 2/50 (4%), and

in females – 0/50, 3/50 (6%), 3/50 (6%), 1/50 (2%). The EPA stated[77] that for their risk analysis, the increase in hemangiosarcomas in male mice was not considered to be treatment-related. For the purpose of this hazard identification, I determined the increased incidence of hemangiosarcomas in male mice is due to the treatment with glyphosate that caused a significant dose related increase in the incidence of hemangiosarcoma in male CD-1 mice. This association may have been stronger if all the animals in this study had been examined histologically for this tumor.

•Greim[78] (Study 12, Sugimoto, K.) reported on a study submitted by Arysta Life Sciences to the EPA in support of the re-registration of glyphosate as an herbicide. Groups of ICR-CD-1 mice (50/sex/group received diets containing glyphosate (94.6–97.6% pure) at 0, 1600, 8000 or 40,000 ppm for 18 months. Parameters evaluated included clinical signs, body weight, food consumption, hematology, clinical chemistry, and urinalysis, organ weights, gross necropsy and histopathological examination. The EPA reported[77] no adverse effects on survival were observed in either sex across the doses tested and there were no statistically significant increases in any tumor type in this study based on details provided by Greim[78]. A review of the tumor tables for this study (Sugimoto[85]) shows that there was a significant trend for the development of hemangiosarcomas in male mice (0/50; 0/50; 0/50; 2/50 (4%)) with a p-value for trend of 0.008, Chi-Square test; a significant trend for the development of malignant lymphomas in male mice (2/50 (4%); 2/50 (4%); 0/50; 6/50 (12%)) with a p-value for trend of 0.008, Chi-Square test; and a significant trend for the development of renal adenomas (0/50; 0/50; 0/50; 2/50 (4%)) with a p-value for trend of 0.008, Chi-Square test seen in male mice. The EPA also reported[86] that hemangiosarcomas in female mice were found to occur with a statistically significant trend in this study (0/50; 0/50; 2/50, (4%); 5/50, (10%) p=0.002, Trend test), and the tumor incidence in the high-dose female mice was statistically significant with p=0.028 as compared to concurrent controls. I also reviewed the Tier II Summaries for Glyphosate Carcinogenicity Studies from Greim, et al.[87] for Study 12, Sugimoto, which showed a reported statistically significant increase in malignant lymphoma in high dose male mice – 0/26, 0/34, 1/27(4%), 5/29(17%) ($p<0.05$ Fisher's exact test); however I could not resolve the difference in the tumor incidence between the Greim Tier II Summary[87], the published Greim et al, 2015[78] and the Sugimoto[85] tumor tables. These appear to be low response rates but this is only an 18-month study where low rates of

tumors are not unusual. For the purpose of this hazard identification there was an increased incidence of malignant and/or a combination of malignant and benign tumors, at multiple tissue sites in male and female CD-1 mice in this study. The significant increase in malignant lymphoma in high dose male mice, and the significant trend in the development of hemangiosarcomas, malignant lymphomas, and renal adenomas in male mice is due to treatment with glyphosate that caused these cancers in male CD-1 mice. The significant trend in the development of hemangiosarcomas in female mice is also related to treatment with glyphosate that caused this cancer in female CD-1 mice.

•Greim[78] (Study 14, Wood, et al. 2009b) reported on a study submitted by Nufarm to the EPA in support of the re-registration of glyphosate as an herbicide. Groups of 51 male and 51 female CD-1 mice were given diets containing glyphosate (purity, 94.6–97.6%) at a concentration of 0, 500, 1500, or 5000 ppm for 18 months. Parameters evaluated included clinical signs, body weight, food consumption, organ weights, gross necropsy and histopathological examination. There was no treatment-related effect on survival. In male mice at the high dose there was a significant increase in the incidence of malignant lymphomas (0/51, 1/50(10%), 2/51(4%), 5/51(10%) $p<0.05$, pair-wise comparison, $p<0.01$ for trend) and a significant increase in the trend of formation of adenocarcinomas of the lung (5/51(10%), 5/51(10%), 7/51(14%), 11/51(22%) $p<0.01$ for trend[86]). For the purpose of this hazard identification, I determined the formation of malignant lymphomas and the formation of adenocarcinomas of the lung in male mice in this study is due to treatment with glyphosate that caused a significant increase in the incidence of malignant lymphoma in high dose male CD-1 mice and an increase in the trend of formation of the adenocarcinomas of the lung and malignant lymphomas in male CD-1 mice.

•Greim[78] (Study 13, Kumar) reported on a study submitted by Feinchemie Schwebda to the EPA in support of the re-registration of glyphosate as an herbicide. Groups of 50 male and 50 female Swiss albino mice [age at start not reported] were given diets containing glyphosate (purity >95%) at a concentration of 0, 100, 1000, or 10,000 ppm for 18 months. There were no treatment-related effects on clinical signs, behavior, body weight, body weight gain, food consumption, and differential white blood cell counts in both sexes. There was a slightly higher mortality rate observed in the high dose groups. There was a significant increase in malignant lymphoma reported in high dose male mice

(10/50, 20%; 15/50, 30%; 16/50, 32%; 19/50, 38%, p<0.05 pair wise) and female mice (18/50, 36%; 20/50,40%; 19/50, 38%; 25/50, 50%, p<0.05 pair wise). There was also a significant increased trend (one-sided p-value for trend=0.05) for the formation of this tumor in males. The incidence of malignant lymphoma in the high dose male was double the historical rate, reported to be 18%[87] for males, and for high dose female mice the incidence was well above the historical rate of 41%[87]. There was also a significant increased trend in the incidence of kidney renal cell adenomas reported[88] in males (0/50; 0/26; 1/26 (4%); 2/50 (4%); one-sided p-value for trend p=0.04). I would note that the EPA stated[77] this study was not included in their review due to the report by Greim (2015)[78] that there was possibly a viral infection within the colony, which confounded the interpretation of the study findings. EPA also stated although the incidences in this study were within or near the normal variation of background occurrence. It is not clear whether or not ther viral component may have contributed to incidence value reported or the lower survival seen at the high dose in the study.[89] An internal Monsanto email among the authors of Greim would indicate there was no viral infection in the mouse colony during this study. Further, Greim[78] (table 18, p. 201) considers this study GLP and OECD compliant. For the purpose of this hazard identification, I determined formation of malignant lymphoma in the male and female mice and the renal cell adenomas in males in this study is due to treatment with glyphosate that caused a significant increase in the incidence of malignant lymphoma in high dose male and female Swiss albino mice and renal cell adenomas in male Swiss albino mice.

Cancer Bioassays in Rats

•Greim[78] reported on a Bio/dynamics study (Study 1, Lankas, et al.) submitted by Monsanto to the EPA in support of the registration of glyphosate as an herbicide. Groups of 50 male and 50 female Sprague-Dawley rats were fed diets containing glyphosate (98.7%, pure) at concentrations of 0, 30, 100 or 300 ppm for 26 months. These concentrations were adjusted during the course of the study so that actual doses of 0, 3, 10, and 31 mg/kg/day in males and 0, 3, 11, and 34 mg/kg/day in females were maintained. There were no treatment-related effects on body weight or survival at any dose level. An MTD was not achieved. There was a significant increase reported in the incidences of interstitial cell tumors in the testes of male rats: controls 0/50, 0%; low dose

3/5, 6%; mid dose 1/50, 2%; high dose 6/50; 12%; p=0.013 by pairwise comparison[77]. The incidence of interstitial cell tumors in the testes in the high dose animals in this study is almost twice that seen in the range of this tumor (3.4% to 6.7%) in control animals (historical controls) from 5 contemporary studies[87]. There was also a significant increase in the incidence of pancreatic islet cell adenoma reported in males at the low dose: controls, 0/50; low dose 5/49, 10% ($p < 0.05$ Fisher exact test); mid dose 2/50, 4%; high dose 2/50, 4%. For the purpose of this hazard identification, I determined the increase in the incidence of interstitial cell tumors in the testes and pancreatic cell tumors in male rats is due to the treatment with glyphosate that caused a significant increase in the incidence of interstitial cell tumors in the testes and pancreatic islet cell tumors in male Sprague-Dawley rats.

•Greim[78] reported on a study (Study 2, Stout, et al.) submitted by Monsanto to the EPA in support of the registration of glyphosate as an herbicide. Groups of 60 male and 60 female Sprague-Dawley rats were given diets containing glyphosate (technical grade; purity, 96.5%) at a concentration of 0 ppm, 2000 ppm, 8000 ppm, or 20,000 ppm, ad libitum, for 24 months. No compound-related effect on survival was observed. There was no statistically significant decreases in body-weight gain in male rats. The study reported significant decreases in body-weight gain in females at the highest dose, beginning on day 51. There was a statistically significant increase in the incidence of pancreatic islet cell adenoma in males at the lowest dose compared with controls: control 1/58, 2%; low dose 8/57, 14% ($p \leq 0.05$ Fisher exact test); mid dose 5/60, 8%; high dose 7/59, 12%. The EPA[77] did additional analysis of this data for pancreatic islet cell adenoma by excluding rats that died or were killed before week 55 and then using statically analyses (Cochran–Armitage trend test and Fisher exact test) that gave a statistically significant higher incidence of these tumors in males at the lowest and highest doses compared with controls: control 1/43, 2%; low dose 8/45, 18% ($p = 0.018$; pairwise test); mid dose 5/49, 10%; high dose 7/48, 15% ($p = 0.042$; pairwise test). The incidence of these adenomas in the low (18%) and high (15%) dose males was almost twice that seen in historical controls. The range for historical controls for pancreatic islet cell adenoma reported in males at this laboratory was 1.8–8.5%[77]. One should note that there was no statistically significant positive trend in the incidence of these tumors, and no apparent progression to carcinoma. There was also a statistically significant positive trend ($p = 0.016$) in the

incidence of hepatocellular adenoma observed in male rats[86] and a statistically significant positive trend of thyroid follicular cell adenomas (p = 0.031) and thyroid follicular cell adenomas and carcinomas combined (p=0.033) observed in female rats[86] reported in this study. For the purpose of this hazard identification, I determined that the increase in the incidence of pancreatic islet cell adenoma in male rats is due to the treatment with glyphosate that caused a significant positive increase in the incidence of pancreatic islet cell adenomas of male Sprague-Dawley rats. Glyphosate also caused a significant increase in the trend for formation of hepatocellular adenomas in male Sprague-Dawley rats and of thyroid follicular cell adenomas and follicular cell adenomas and carcinomas combined in female Sprague-Dawley rats.

•Greim[78] reported on a study (Study 3, Atkinson, et al.) submitted by Cheminova to the EPA in support of the registration of glyphosate as an herbicide. Groups of 50 male and 50 female Sprague-Dawley rats were given diets containing glyphosate, purity, 98.7–98.9%, at a concentration that were adjusted to provide doses of 0, 10, 100, 300, or 1,000 mg/kg bw/day, ad libitum, for 104 weeks. Decreased body-weight gain was observed in males and females at the highest dose. There was no significant decrease in survival reported at any dose level. Neoplasms were noted in control and treated groups, but dose-responses were not evident, and no statistically significant increases versus controls were noted for any tumor type. Additionally, EPA's evaluation[86] of this study indicated there were no treatment-related increases in the occurrence of any tumor type in this study.

•Greim[78] reported on a study (Study 7, Brammer) submitted by Syngenta to the EPA in support of the re-registration of glyphosate as an herbicide. Groups of 52 male and 52 female Wistar rats received diets containing 0, 2,000, 6,000, and 20,000 ppm glyphosate (97.6% pure), ad libitum, for 24 months. Survival in the high dose group males was significantly better than the other dose groups throughout the study while survival in the females was similar across all dose groups. The bodyweights of the high dose males and females were statistically significantly lower than controls throughout the study. The study's author reported no significant increase in turmor incidence in any of the treated groups. The EPA's evaluation[77] of this study indicated there was a significant increase in the incidence of hepatocellular adenomas in male rats at the high dose when compared to controls (control 0/52, 0%; low dose 2/52, 4%; mid dose 0/52, 0%; high dose 5/52, 10%, p=0.03). There was also a significant trend (p=0.008) in the formation of this tumor in

male rats. The EPA goes on to state the incidences observed were within the range (0–11.5%) of historical controls for this strain of rats in 26 studies conducted during the relevant time period (1984–2003) at the testing laboratory indicating this increase was not considered to be related to treatment with glyphosate. For the purpose of this hazard identification, I determined the increase in the formation of hepatocellular adenomas in male Wistar rats could not be attributed to exposure to glyphosate in this study despite the fact that there was an observation of increased incidence of hepatocellular adenomas in male rats.

•Greim[78] reported on a study (Study 4, Suresh) submitted by Feinchemie Schwebda to the EPA in support of the registration of glyphosate as an herbicide. Groups of 50 male and 50 female Wistar rats received diets containing 0, 100, 1,000, and 10,000 ppm glyphosate (97.6% pure), ad libitum, for 24 months. There were no treatment-related deaths or clinical signs in any of the dose-groups and there were no treatment related effects on body weight gain or food consumption noted. This suggests that the MTD was not reached, and this study is inadequate for the evaluation of the carcinogenicity of glyphosate.

•Greim[78] reported on a study (Study 6, Enomoto) submitted by Arista Life Sciences to the EPA in support of the registration of glyphosate as an herbicide. Groups of 50 male and 50 female Sprague-Dawley rats received diets containing 0, 3,000, 10,000, or 30,000 ppm glyphosate (94.6–97.6% pure) for 24 months. Decreases in body weight were observed in both sexes in the mid and high dose group along with a lower food consumption. Survival in the high dose males was lower than controls while there was no compound-related effect on survival in any other dose group. There were no statistically significant increases in any tumor type reported for this study.

•Greim[82] reported on a study (Study 8, Wood 2009a) submitted by Nufarm to the EPA in support of the registration of glyphosate as an herbicide. Groups of 51 male and 51 female Wistar rats received diets containing 0, 3,000, 10,000, or 15,000 ppm glyphosate (95.7% pure) for 24 months, the highest dose level was progressively increased to 24000 ppm by week 40. There were no treatment-related deaths or clinical signs in any of the dose-groups. No significant treatment-related effects on mortality were observed during the study. This suggests that the MTD was not reached, and this study is inadequate for the evaluation of the carcinogenicity of glyphosate.

•Chruscielska et al.[90] gave groups of 55 male and 55 female Wistar rats drinking-water containing an ammonium salt of glyphosate (purity not given) that was used to make drinking water solutions of 0, 300, 900, and 2700 mg/L, for 24 months. The authors reported that survival and body-weight gain were similar in treated and control animals and that no significant increase in tumor incidence was observed in any of the treated groups. There was limited information provided on dosing regimen, histopathological examination method, and tumor incidences that makes this study inadequate for the purpose of this hazard assessment.

Summary for Experimental Animal Data

I reviewed a total of five dose feed bioassays of glyphosate in mice. Four of these studies (Study 12 and Study 14 in Greim[78], Knezevich and Hogan (1983)[76], and Atkinson[84]) were in male and female CD-1 mice, and one study[78(Study13)] was in male and female Swiss albino mice. Glyphosate caused a significant increase in the incidence of adenoma or carcinoma (combined) and a significant positive trend for the formation of adenoma or carcinoma (combined) of the renal tubule in male CD-1 mice in one study[76], and a significant positive trend for the formation of adenomas of the renal tubule in male CD-1 mice in another study[78(Study 12)]. Glyphosate also caused a significant increase in the incidence of renal cell adenomas in male Swiss albino mice[78(Study13)]. Adenoma and carcinoma of the renal tubule constitutes a morphological continuum in the development and progression of renal neoplasia in mice[91,92]. It is important to note that renal tubule carcinoma is a very rare tumor in CD1 mice[80] and that this tumor was caused by exposure to glyphosate in two different strains of mice (CD-1 and Swiss). Glyphosate caused a significant increase in the incidence of malignant lymphoma in male CD-1 mice in two studies[78(Study 12, Study 14)] and in male and female Swiss albino mice in another study[78 (Study 12)]. Glyphosate also caused a significant positive trend for the formation of malignant lymphoma in one of these studies in male CD-1 mice[78(Study 12)] and caused a significant positive trend for the formation of hemangiosarcomas in 2 separate studies in male CD-1 mice[78(Study 12),84]. There was also a significant positive trend for the formation of adenocarcinomas of the lung in male CD-1 mice in one study[78(Study 14)] and hemangiosarcomas in female CD-1 mice in another study[82(Study 12)].

I reviewed a total of 7 dosed feed and 2 drinking water bioassays of glyphosate in rats. Four of the feed studies and one drinking water study were in male and female Sprague-Dawley rats and three feed studies and one drinking water study were in male and female Wistar rats. Glyphosate caused a significant increase in the incidence of pancreatic islet cell adenoma in two feeding studies in male Sprague-Dawley rats[78(Study 1 and Study 2)]. Glyphosate caused a significant increase in the incidence of thyroid tumors in male Sprague-Dawley rats in one feeding study[78(Study 1)] and a significant positive trend for the formation of thyroid tumors in female Sprague-Dawley rats in another feeding study[78(Study 2)]. Glyphosate caused a significant increase in the incidence of interstitial cell tumors in the testes of male Sprague-Dawley rats in one feeding study and a significant positive trend for the formation of hepatocellular adenomas in male Sprague-Dawley rats in another feeding study[78(Study 1)].

To state my findings more concisely, I determined that in CD-1 mice, glyphosate expsoure causes kidney tumors in males in two separate studies[76,78(Study 12)], hemangiosarcomas in males in two separate studies,[78(Study 12),84] malignant lymphoma in males in two separate studies[78(Study 12, Study 14)], adenocarcinomas of the lung in males in one study[78(Study 14)], and hemangiosarcomas in females in one study[78(Study 12)]. In one study[78(Study 13)] in Swiss albino mice, exposure to glyphosate causes malignant lymphoma in males and females and kidney tumors in males.

I also determined that in Sprague-Dawley rats, glyphosate exposure causes pancreatic cell tumors in males in one study[78(Study 2)], interstitial cell tumors in the testes in males in one study[78(Study 1)], hepatocellular adenomas in males in two studies[78(Study 2, Study 7)], and thyroid follicular cell tumors in females in one study[78(Study 2)].

Considering all data from the mice and rat studies I reviewed, there is "Sufficient" evidence that shows glyphosate is carcinogenic in experimental animals causing kidney tumors, hemangiosarcomas, malignant lymphoma, adenocarcinomas of the lung, and hemangiomas in mice and pancreatic cell tumors, interstitial cell tumors in the testes, hepatocellular adenomas, and thyroid follicular cell tumors in rats. This statement is based on my stated criteria of a causal relationship between exposure to glyphosate and an increased incidence of malignant and/or a combination of malignant and benign tumors, in multiple species, at multiple tissue sites, from multiple studies, and to an unusual degree with regard to incidence, site, or type of tumor.

Hazard Assessment of the Mechanistic and Other Data for Glyphosate and Glyphosate-Based Formulations

Data on the absorption of glyphosate via intake of food and water in humans could not be found in the published literature. Glyphosate has been found in the urine of agricultural workers. In a study by Acquavella[7], 60% of farmers had detectable levels of glyphosate in 24-hour composite urine samples taken on the day they had applied a glyphosate-based formulation. Wearing protective gear such as rubber gloves reduced the concentrations of glyphosate in the urine. This implies that dermal absorption is a relevant route of exposure. Curwin[8] demonstrated that glyphosate is also present in the urine of non-farm families. No data in humans on the distribution of glyphosate in systemic tissues other than blood were found in the available published literature. In cases of accidental or deliberate intoxication involving ingestion of glyphosate-based formulations, glyphosate was measured in blood.

Strong evidence indicates that glyphosate is genotoxic. As noted in Monograph 112, studies in human cells[27,31,32], mammalian model systems[27,32,33], and in non-mammalian organisms[35,37] have given positive results . The end-points evaluated in these studies included biomarkers of DNA adducts and various types of chromosomal damage. Tests in bacterial assays gave consistently negative results.

The evidence for genotoxicity caused by glyphosate-based formulations is also strong. As noted in Monograph 112, three studies[39,93,94] reported examining genotoxic end-points in community residents exposed to glyphosate-based formulations and two of these studies reported positive associations. One study[39] looked at micronucleus formation in circulating blood cells before and after aerial spraying with glyphosate-based formulations to determined chromosomal damage in exposed individuals. This study revealed a significant increase in micronucleus formation after exposure in three out of four different geographical areas. Additional positive evidence came from in vitro studies with positive results in human cells[32,45], in vivo[27,32] and in vitro[95] studies in mammalian systems, and studies in non-mammalian organisms[35,96] such as fish. Biomarkers of DNA adducts and different types of chromosomal damage were examined in these studies. The pattern of tissue specificity of genotoxicity end-points observed with glyphosate-based

formulations is similar to that observed with glyphosate. Tests of glyphosate-based formulations in bacterial assays gave generally negative results.

There is strong evidence that glyphosate and glyphosate-based formulations induce oxidative stress. As noted in Monograph 112, vidence of oxidative stress comes from in vitro studies in human cells[97,98] and in many in vivo studies[32,42], examining rodent tissues. Studies of oxidative stress and glyphosate in non-human mammalian experimental systems were conducted in rats and mice, and examined a range of exposure durations, doses, preparations (glyphosate and glyphosate-based formulations), administration routes and tissues. In these studies glyphosate caused free radicals and oxidative stress in mouse and rat tissues through alteration of antioxidant enzyme activity, depletion of glutathione, and increases in lipid peroxidation. In at least one of the studies in human cells the oxidative stress caused by glyphosate was ameliorated by co-administration of antioxidants[40]. Similar findings of oxidative stress have been reported in fish and other aquatic species providing additional evidence for glyphosate-induced oxidative stress[99]. Molecular epidemiology studies[100,101] have documented that oxidative stress is a pathway to the formation of NHL in humans. Further, the in vitro studies in humans cells and in vivo and in vitro studies in rodents provides evidence that exposure to glyphosate causes oxidative stress. Logically it follows that there is a positive association between oxidative stress caused by glyphosate and glyphosate-based formulations and NHL observed in humans exposed to glyphosate-based formulations and that a causal interpretation is credible.

Hazard Assessment Conclusion

Based on the significant positive association observed in the studies discussed above, I conclude that there is evidence that glyphosate and glyphosate-based formulations are carcinogenic in humans. First, the human study data supports a positive association between exposure to glyphosate and glyphosate-based formulations and the development of NHL. Second, all the data from the animal bioassay studies provide evidence that glyphosate is carcinogenic in experimental animals. Third, the mechanistic data show that glyphosate and glyphosate-based formulations cause genotoxicity and oxidative stress in humans and animals. Therefore, I conclude to a reasonable degree of

scientific certainty that glyphosate and glyphosate-based formulations are probable human carcinogens. I also conclude to a reasonable degree of scientific certainty that glyphosate and glyphosate-based formulations cause NHL in humans.

Compensation and Testimony

My billing rate is $400/hr plus travel fees and expenses. I have not testified in any case in the last four years.

Charles W. Jameson, Ph.D.

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Studies I reviewed but determined inadequate for use:

Greim[78] reported on a study (Study 4, Suresh): Greim H, Saltmiras D, Mostert V, Strupp C (2015). Evaluation of carcinogenic potential of the herbicide glyphosate, drawing on tumor incidence data from fourteen chronic/carcinogenicity rodent studies. Crit Rev Toxicol, 45(3):185–208.

Greim[78] reported on a study (Study 8, Wood 2009a) : Greim H, Saltmiras D, Mostert V, Strupp C (2015). Evaluation of carcinogenic potential of the herbicide glyphosate, drawing on tumor incidence data from fourteen chronic/carcinogenicity rodent studies. Crit Rev Toxicol, 45(3):185–208.

Chruscielska K, Brzezinski J, Kita K, Kalhorn D, Kita I, Graffstein B et al. (2000). Glyphosate - Evaluation of chronic activity and possible far-reaching effects. Part 1. Studies on chronic toxicity. Pestycydy (Warsaw), 3–4:11–20.

Seralini GE, Clair E, Mesnage R, Gress S, Defarge N, Manuela Malatesta M et al. (2014). Republished study: long-term toxicity of a Roundup herbicide and a Roundup-tolerant genetically modified maize. Environmental Sciences Europe, 26(1):1–14

George J, Prasad S, Mahmood Z, Shukla Y (2010). Studies on glyphosate-induced carcinogenicity in mouse skin: a proteomic approach. J Proteomics, 73(5):951–64.

EXHIBIT A

Name	Charles William Jameson
Mailing Address:	[redacted]
Date And Place Of Birth:	[redacted]
Citizenship:	[redacted]
Marital Status:	Married, four children
Education:	B.S. 1970 Chemistry, Mount Saint Mary's College Emmitsburg, Maryland Ph.D. 1975 Organic Chemistry, Physical Chemistry minor University of Maryland College Park, Maryland

Brief Chronology of Employment:

1965	Chemistry Laboratory Technician, Bionetics Research Laboratories, Falls Church, Virginia
1968 – 1969:	Organic Chemistry Laboratory Assistant, Mount Saint Mary's College, Emmitsburg, Maryland
1969 – 1970:	Organic Chemistry Laboratory Instructor, Mount Saint Mary's College, Emmitsburg, Maryland
1970 – 1973:	Graduate Teaching Assistant, Chemistry Dept., University of Maryland College Park, Maryland
1973 – 1975:	Graduate Research Assistant, Center of Materials Research, University of Maryland, College Park, Maryland
1975 – 1976	Faculty Graduate Assistant, Chemistry Dept., University of Maryland, College Park, Maryland
1976 – 1979:	Senior Chemist, Tracor Jitco, Inc., Rockville, Maryland
1979 – 1980:	Chemist, Carcinogenesis Testing Program, National Cancer Institute, National Institutes of Health (NIH), Bethesda, Maryland

1980 – 1983:	Head, Chemistry Section, Program Resources Branch, National Toxicology Program (NTP), National Institute of Environmental Health Sciences (NIEHS), NIH, Research Triangle Park, North Carolina
1983 – 1985:	Acting Chief, Program Resources Branch, NTP, NIEHS, NIH, Research Triangle Park, North Carolina
1985 – 1989:	Head, Program Resources Group, Carcinogenesis and Toxicologic Evaluation Branch, NTP, NIEHS, NIH, Research Triangle Park, North Carolina
1989 – 1990:	Supervisory Chemist, Experimental Toxicology Branch, NTP, NIEHS, NIH, Research Triangle Park, North Carolina
1990 – 1995:	Senior Chemist, Office of the Senior Scientific Advisor to the Director NIEHS, NIH, Research Triangle Park, North Carolina
1995 – 2008	Director, Report on Carcinogens, NTP, NIEHS, NIH, Research Triangle Park, North Carolina
2008 – present	Principal, CWJ Consulting, LLC, Cape Coral, Florida

Department of Health and Human Services Activities

Chairman, National Toxicology Program's Executive Committee's Interagency Working Group for the Report on Carcinogens, 1995 to 2005

National Institutes of Health Activities

NIEHS Representative to the Deafness and Other Communication Disorders Interagency Coordination Committee, 1990 - 1996.

NIEHS Representative on the Task Force on Aging Research, 1990-1994.

National Institutes of Environmental Health Sciences Activities

Chairman, NIEHS/NTP Review Committee for the Report on Carcinogens, 1995 to 2005

Chairman, Search Committee for NIEHS Tenure / Tenure Track Staff Epidemiologist 1998

Peer-Review Panel Member for Draft Report on Carcinogens Monograph on Cobalt and Certain Cobalt Compounds. July, 2015

Member and Chairman for the Special Emphasis Panel to review proposals responding to RFP ES2015038, "Scientific Information Management and Literature-Based Evaluations for the National

Toxicology Program (NTP)." The objective of this contract is to provide scientific and technical expertise and support for the NTP to compile, review, and analyze information and data from the scientific literature and other sources regarding the effects of environmental substances and other issues that may impact public health. October, 2015

International Activities

Member, WHO Task Group on Environmental Health Criteria for Fully Halogenated Chlorofluorocarbons, Neuherberg, Federal Republic of Germany, November 21 – 25, 1988.

Member, WHO Task Group on Environmental Health Criteria for Partially Halogenated Chlorofluorocarbons (Ethane Derivatives), Carshalton, Surrey, United Kingdom, September 30 – October 5, 1991.

NIEHS representative to the WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 82 on the Carcinogenic Risks To Humans Of Some Traditional Herbal Medicines, Some Mycotoxins, Naphthalene And Styrene, Lyon, France, February 11 – 20, 2002

Member, IARC *Monographs* Advisory Group for Five Year Plan, Lyon, France, 18-21 February 2003

NIEHS representative to the WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 87 on The Carcinogenic Risks To Humans Of Lead And Lead Compounds, Lyon, France, February 8 – 18, 2004

NIEHS representative to the WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 91 on The Carcinogenic Risks To Humans Of Combined Oral Contraceptives And Estrogen-Progestogen Replacement Therapy, Lyon, France, June 4-15, 2005.

NIEHS representative to the WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 93 on The Carcinogenic Risks To Humans Of Carbon Black, Titanium Dioxide And Non-Asbestiform Talc, Lyon, France, February 4 – 15, 2006

Member, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 97 on The Carcinogenic Risks To Humans Of 1,3 –Butadiene, Ethylene Oxide, And Vinyl Halides (Vinyl Fluoride, Vinyl Chloride And Vinyl Bromide), Lyon, France, June 6-15, 2007.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 99 on The Carcinogenic Risks To Humans Of Some Industrial And Cosmetic Dyes And Related Exposures, Lyon, France, February 4-13, 2008.

Member, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph 100A on A Review Of Human Carcinogens - Pharmaceuticals (Anti-Cancer Drugs – Hormonal Drugs & Therapies – Others), Lyon, France, October 14 – 21, 2008.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 100F on A Review Of Human Carcinogens - Chemical Agents And Related Occupations, Lyon, France, October 20 – 27, 2009.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 103 on Bitumen And Bitumen Fumes, And Some Heterocyclic Aromatic Hydrocarbons, Lyon, France, October 11 - 18, 2011.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 105 on Diesel And Gasoline Exhausts And Some Nitroarenes, Lyon, France, June 5 - 12, 2012.

Member WHO's International Agency for Research on Cancer (IARC) Workshop on Tumour Concordance And Mechanisms Of Carcinogenesis: Lessons Learned From Volume 100 of the IARC Monographs, Lyon, France: April 16-18, 2012 and November 28-30, 2012

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 108 On Some Drugs And Herbal Medicines, Lyon, France, June 4 - 11, 2013.

Member and Chair of Experimental Animal Data Subgroup, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 112 on Some Organophosphate Insecticides And Herbicides, Lyon, France, March 3-10, 2015.

Member and overall Chair, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph Vol. 115 on Some Industrial Chemicals, Lyon, France, February 2-9, 2016.

Member, WHO's International Agency for Research on Cancer (IARC) Workgroup preparing Monograph 116 on Coffee, Mate And Very Hot Beverages, Lyon, France, May 24 – 311, 2016.

Honors and Awards

President, Student Affiliate Chapter of the American Chemical Society, Mount Saint Mary's College, 1969; Vice President, 1968.

National Toxicology Program Representative to American Chemical Society's Committee on Regulatory Affairs 1982 – 1992.

National Institutes of Health Special Achievement Cash Award (Spy Dust Project): 1986.

Merit Pay Cash Award for Sustained High Quality Work Performance, NIEHS: 1982, 1989

Performance Award for Sustained High Quality Work Performance, NIEHS: 1991, 1992, 1993, 1995, 1996, 2001, 2002, 2003, 2004, 2006, 2007.

Special Act or Service Award, NIEHS: 1996 (Review of Report on Carcinogens criteria); 1997 (Publication of 8th Report on Carcinogens); 1998 (Recruitment of NTP Staff Epidemiologist), 1998 (Restructuring of lead biokinetics contract and establishment of new Report on Carcinogens support contract)

Staff Recognition Award, NIEHS: 1999 (Preparation of final draft of 9th Report on Carcinogens)

NIEHS Director's Award, NIEHS: 2000 (Review of nominations for the 9th Report on Carcinogens)

Special Training

American Chemical Society, Short Course: "Chemical Carcinogenesis," 1978.

National Institutes of Health (NIH) Training Course: "Project Officers Civil Rights Contract Compliance," 1979.

Department of Health and Human Services Training (DHHS) Course: "Program Officials Guide to Contracting," 1980.

U. S. Office of Personnel Management (OPM) Training Course: "EEO - Its Place in the Federal Government," 1983.

U. S. OPM Training Course: "Introduction to Supervision," 1984.

NIH Training Course: "Employee Performance Management System Training," 1984.

DHHS Training Course: "Advanced Project Officer Training," 1985.

National Institute of Environmental Health Sciences Training Course: "Care and Handling of Laboratory Animals," 1986.

Rockhurst College Continuing Education Center: "How to Manage Projects, Priorities and Deadlines," 1992.

NIH Training Course: "PHS Animal Welfare Policy for HSA's," 1993.

Fred Pryor Seminars: "Total Quality Management," 1994.

Fred Pryor Seminars: "How to Manage Priorities and Meet Deadlines," 1994.

NIH Training Course: "Workplace Violence," 1994.

NIH Training Course: "NIH Guidelines on the Inclusion of Women and Minorities as Subjects in Clinical Research," 1994.

NIH Training Course: "Workplace Issues Associated with HIV/AIDS," 1994.

The Bookings Institution Course: "Issues in Science and Technology Policy", 1996

Professional Society Memberships and Activities

American Chemical Society

- Division of Analytical Chemistry
- Division of Chemical Health and Safety
- National Toxicology Program Representative to American Chemical Society's Committee on Regulatory Affairs 1982 – 1992
- Overall Co-Organizer and Co-Chairman of a symposium entitled "Chemistry and Safety for Toxicity Testing of Environmental Chemicals," sponsored by the Divisions of Chemical Health and Safety, Analytical Chemistry and Environmental Chemistry at the 183rd National American Chemical Society Meeting, Las Vegas, NV, March 1982.

Society of Toxicology

Research interests:

Chemical Carcinogenesis
Analytical chemistry methods development to support toxicology studies.

Reviewer for Scientific Journals

Analytical Chemistry
Bulletin of Environmental Contamination & Toxicology (Member of Editorial Board)
Environmental Health Perspectives (Contributing Editor)
Fundamental and Applied Toxicology
Journal of the National Cancer Institute
Science

Invited Papers

Invited to be Session Chairman and to present paper entitled "Analytical Chemistry Requirements for Toxicity Testing of Environmental Chemicals" at the Symposium on Chemistry and Safety for Toxicity Testing of Environmental Chemicals, at the 183rd National American Chemical Society Meeting, Las Vegas, NV, March 1982.

Invited to serve as a panelist on the NBC nationally televised series "Health Field" with Dr. Frank Field. A two-day series was filmed on Environmental Chemistry and Chemical Health Concerns, 1982.

Invited to give a seminar entitled "Analytical Chemistry Requirements for Toxicity Testing." Duke University, Durham, NC, July 1982.

Invited to present a paper entitled "Practical Aspects of Analytical Chemistry Support for Toxicity Testing" at the Symposium on the Role of the Analytical Chemist in Animal and Molecular Toxicology, at the Federation of Analytical Chemistry and Spectroscopy Societies Meeting XI, Philadelphia, PA. September 16-21, 1984.

Invited to present a paper entitled "Application of Microencapsulation in Toxicity Testing" at the NIEHS Center Directors Meeting, Research Triangle Park, North Carolina, November 1984.

Invited to be Session Chairman and to present paper entitled "Chemical Quality Assurance Techniques for Toxicity Testing of Environmental Chemicals" at the Symposium on Accurate Measurements of Environmental Pollutants, at the 1984 International Chemical Congress of Pacific Basin Societies, Honolulu, Hawaii, December 16-21, 1984.

Invited to present a paper entitled "Lack of Evidence for Involvement of Cyanide in Methyl Isocyanate (MIC) Toxicity" at the Society of Toxicology Meeting, New Orleans, LA, March 3-7, 1986.

Invited to present a paper entitled "Toxicology From A Chemist's Viewpoint" at the Mount Saint Mary's College Science Alumni Homecoming, Emmitsburg, Maryland, October 23-26, 1986.

Invited to be Session Chairman and to present paper entitled "Application of Microencapsulation for Toxicity Studies" at the Symposium on Techniques for Microencapsulation of Chemicals at the 198th National Meeting of the American Chemical Society, Dallas, Texas, April 10-14, 1989.

Invited to be Session Chairman and to present paper entitled "Application of a Fischer Rat Leukemia Transplant Model as a Screen for the Leukemogenic Potential of Chemicals" at the International Symposium on Toxicology, Beijing, P. R. China, October 16-19, 1990.

Invited to present a paper entitled "Investigation of Alternative Vehicles for Use in Toxicology Research: Use of Microencapsulated and Molecular Encapsulated Chemicals in Toxicity Studies" at the Institute of Pharmacology and Toxicology, Academy of Military Medical Sciences, Beijing, P. R. China, October 20, 1990.

Invited to present a paper entitled "Toxicology and Carcinogenicity Studies of d- Limonene in Male and Female F344 Rats and B6C3F1 Mice" at the Symposium on Food Phytochemicals for Cancer Chemoprevention at the 204th National Meeting of the American Chemical Society, Washington, D.C., August 23-28, 1992.

Invited to be a Faculty Member and to present talk entitled " The National Toxicology Program's Report on Carcinogens " at the Toxicology Forum, Washington, DC, February 1995.

Invited to be a Faculty Member and to present talk entitled " The Report On Carcinogens (RoC): Status Of The Review Of The Criteria For Listing Substances In The RoC " at the Toxicology Forum, Washington, DC, February 1996.

Invited to be a Faculty Member and to present talk entitled " Update of 1997 review of Nominations for the 9th Report on Carcinogens " at the Toxicology Forum, Washington, DC, February 1998.

Invited to be a Faculty Member and to present talk entitled " NTP Report on Carcinogens: History and the Process " at the Toxicology Forum, Aspen, CO, July 1999.

BIBLIOGRAPHY

Publications

1. Mazzocchi PH, Ammon HL, **Jameson CW**. Lanthanide Shift Reagents III: Errors Resulting from the Neglect of Angle Dependence, Tetrahedron Letters, 573, 1973.

2. **Jameson CW**. I. Study of Lanthanide shift Reagent - Substrate Interaction in Solution. II. Competitive Photochemical Type I and Type II Reactions of Amides and Imides. Dissertation Abstracts, 1975.

3. Ennis DM, Kramer A, Mazzocchi PH, **Jameson CW**, Bailey WJ. Synthetic N-Releasing Biodegradable Soil Conditioners I, Hort Science, 10, 505, 1975.

4. Ammon HL, Mazzocchi PH, Colicelli E, **Jameson CW**, Liu L. A Convenient Method for Mixing ^{2}H and ^{13}C Lanthanide Induced Shift (LIS) Calculations, A Technique for Facilitating ^{13}C Assignments, Tetrahedron Letters, 1745, 1976.

5. Ennis DM, Kramer A, **Jameson CW**, Mazzocchi PH, Bailey WJ. Structural Factors Influencing the Biodegradation of Imides, Appl Environ Microbiology, 35, 51, 1978.

6. Murrill EA, Woodhouse EJ, Olin SS, **Jameson CW**. Carcinogenesis Testing and Analytical Chemistry, Analytical Chemistry, 52, 1188A, 1980.

7. Douglas JF, Hamm TE, **Jameson CW**, Mahar H, Stinson S, Whitmire CE. Monitoring Guidelines for the Conduct of Carcinogen Bioassays. US Department of Health and Human Services. DHHS Publication No. (NIH) 81-1774. Washington, DC, US Government Printing Office, 80 pp., 1981.

8. Dieter MP, Luster MI, Boorman GA, **Jameson CW**, Dean JH, Cox JW. Immunological and Biochemical Responses in Mice Treated with Mercuric Chloride, Toxicol Appl Pharmachol, 68, 218, 1983.

9. **Jameson CW**, Dunnick JK, Brown RD, Murrill EA. Chemical Characterization of Psoralens Used in the National Toxicology Program Research Projects, National Cancer Institute Monograph, 66, 103, 1984.

10. Timmons L, Cannon M, Grese D, Brown R, Haile C, Murrill E, **Jameson CW**. Identification of Chlorinated Phenyl and Phenoxy Substituted Dibenzodioxin, Dibenzofuran and Diphenyl Ether Homologs in Commercial Grade Pentachlorophenol, Analytical Letters, 17(A4), 277-296, 1984.

11. Timmons L, Steel D, Cannon M, Grese D, Brown R, Murrill E, **Jameson CW**. Identification of Bromotertrachlorophenol in Commercial Pentachlorophenol Samples, Journal of Chromatography, V 314, 476-481, 1984.

12. Dunnick JK, **Jameson CW**, Benson JM. Toxicology and Carcinogenesis Studies of Nickel Oxide, Nickel Subsulfide and Nickel Sulfate. Annals of Clinical and Laboratory Science. V14.N5. 400-401, 1984.

13. Lamb JC, IV, **Jameson CW**, Choudury H, Gulati D K. Fertility Assessment by Continuous Breeding: Evaluation of Diethylstilbestrol and a Comparison of Results from Two Laboratories. J Amer Coll Toxicol 4, 173, 1985.

14. Thigpen JE, Liu LA, Richter CB, Lebetkin EH, Haseman JK, **Jameson CW**. The Comparative Estrogenic Activity of Semipurified, Certified, Standard and Open Formula Rodent Diets. Laboratory Animal Science, V35, N5, 526-527, 1985.

15. Kline DA, Hanna GR, Kuhn GO, Honaker CB, **Jameson CW**. Preparation and Stability of Animal Feed Mixtures Dosed with Rotenone, J Asso Off Anal Chem, Vol. 69, #4, 660-663, 1986.

16. **Jameson CW**, Moseman RF, Collins BJ, Hooper ND. Spy Dust: Methods for the Detection and Cleanup of a Chemical Tracking Agent. Analytical Chemistry, 58, 915A, 1986.

17. Agarwal DK, Eustis S, Lamb JC, **Jameson CW**, Kluwe WM. Influence of Dietary Zinc on Di(2-ethylhexyl)phthalate-Induced Testicular Atrophy and Zinc Depletion in Adult-Rats. Toxicology and Applied Pharmacology, V84, N1, 12-24, 1986.

18. Boorman GA, Hong HL, **Jameson CW**, Yoshitomi K, Maronpot, RP. Regression of Methyl Bromide Induced Forestomach Lesions in the Rat. Toxicology and Applied Pharmacology, 86, 131-139, 1986.

19. Collins B, Goehl TJ, **Jameson CW**, Kuhn G, Dux T. Analytical Methods for the Analysis of Microencapsulated Trichloroethylene in Corn Oil, Feed Dosage Formulations and Rat Whole Blood. J. of Analytical Toxicology, 10, 236, 1986.

20. **Jameson CW**, NTP Technical Report on the Toxicology and Carcinogenesis Studies of Tetrakis(hydroxymethyl)phosphonium sulfate (THPS) and Tetrakis(hydroxymethyl)phosphonium Chloride (THPC) in F344/N Rats and B6C3F1 Mice (Gavage Studies). NIH Publication No. 296, 1987.

21. Dunnick J K, **Jameson CW**, Montgomery CA. Subchronic Toxicity of Propantheline Bromide Administered in the Feed to Fischer 344/N Rats and B6C3F1 Mice. Fundamental and Applied Toxicology, V9, N3, 496-503, 1987.

22. Germolec DR, Burleson GR, **Jameson CW**, Ackermann MF, Lamm KR, Hayes HT, Luster MI. Depression of Natural-Killer Cell-Activity by Ochratoxin-A. Environmental Health Perspectives, V75, No. 5, 145-145, 1987.

23. **Jameson CW**, Moseman RF, Hooper ND, Collins BJ. Spy Dust - Detecting a Chemical Tracking Agent. Environmental Health Perspectives, V75, No. 5, 143-143, 1987.

24. Melnick RL, **Jameson CW**, Goehl TJ. Application of Microencapsulation for Toxicology Studies - Stability, Bioavailability, and Toxicity of Microencapsulated Trichloroethylene. Environmental Health Perspectives, V75, No. 5, 142-142, 1987.

25. Melnick RL, **Jameson CW**, Goehl TJ, Kuhn GO. Application of Microencapsulation for Toxicology Studies. 1. Principles and Stabilization of Trichloroethylene In Gelatin-Sorbitol Microcapsules. Fundamental and Applied Toxicology, V8, N4, 425-431, 1987.

26. Melnick RL, **Jameson CW**, Goehl TJ, Maronpot RR, Collins BJ, Greenwell A, Harrington FW, Wilson RE, Tomaszewski KE, Agarwal DW. Application of Microencapsulation for Toxicology Studies. 2. Toxicity of Microencapsulated Trichloroethylene in Fischer 344 Rats. Fundamental and Applied Toxicology, V8, N4, 432-442, 1987.

27. Thigpen JE, Lung-An L, Richter CB, Lebetkin, EH, Haseman, JK, **Jameson CW**. The Mouse Bioassay Test for the Detection of Estrogenic Activity in Feeds and Foodstuffs. Part I: A Standardized Method for Conducting the Mouse Bioassay using the CD-1 Mouse. Laboratory Animal Science, V37, N5, 596-601, 1987.

28. Thigpen JE, Lung-An L, Richter CB, Lebetkin EH, **Jameson CW**. The Mouse Bioassay Test for the Detection of Estrogenic Activity in Feeds and Foodstuffs. Part II: The Comparative Estrogenic Activity of Purified, Certified Standard, Open and Closed Formula Rodent Diets. Laboratory Animal Science, V37, N5, 602-605, 1987.

29. Bucher JR, Gupta BN, Adkins B, Thompson M, **Jameson CW**, Thigpen J E, Schwetz BA. The Toxicity of Inhaled Methyl Isocyanate in F344/N Rats and B6C3F1 Mice. I: Acute Exposure and Recovery Studies. Environmental Health Perspectives, V72, 53-61, 1987.

30. Luster MI, Germolec DR, Burleson GR, **Jameson CW**, Ackermann MF, Lamm KR, Hayes HT. Selective Immunosuppression in Mice of Natural Killer Cell Activity by Ochratoxin A. Cancer Research, Vol. 47, 2259-2263, 1987.

31. Dieter MP, **Jameson CW**, Tucker AN, Luster MI, French JE, Hong, HL, Boorman, GA. Evaluation of Tissue Disposition, Myelopoietic and Immunologic Responses in Mice After Long-term Exposure to Nickel Sulfate in the Drinking Water. Journal of Toxicology and Environmental Health, V24, 357-372, 1988.

32. Huff JE, McConnell EE, Haseman JK, Boorman GA, Eustis SL, Schwetz BA, Rao GN, **Jameson CW**, Hart LG, Rall DP. Carcinogenesis Studies Results of 398 Experiments on 104 Chemicals from the U. S. National Toxicology Program. Annuals of the New York Academy of Sciences V534, 1-30, 1988.

33. Shan A, Harben D, **Jameson CW**. Analyses of Two Azo Dyes by High Performance Liquid Chromatography. Journal of Chromatographic Science, V26, 439-442, 1988.

34. Hong HL, Canipe J, **Jameson CW**, Boorman GA: Comparative Effects of Ethylene Glycol and Ethylene Glycol Monomethyl Ether Exposure on Hematopoiesis and Histopathology in B6C3F1 Mice. Journal of Environmental Pathology, Toxicology, and Oncology, V8, N7, 27-38, 1988.

35. Hong HL, **Jameson CW**, Boorman GA. Residual Hematopoietic Effect of Ochratoxin A in Mice Exposed to Irradiation. Toxicology, V53, 57-67, 1988.

36. Dieter MP, **Jameson CW**, French JE, Gangjee S, Stefanski SA, Chan, PC. Development and Validation of a Cellular Transplant Model for Leukemia in Fischer Rats: A Short-term Assay for Potential Anti-leukemic Chemicals. Leukemia Research, V13, 841-849, 1989.

37. Timmons L, Brown R, Arneson DW, **Jameson CW**. Rapid Determination of Low pg/mg Amounts of N-Nitrosodiethylamine in Rodent Body Fluid and Tissue Samples by Isotope-Dilution High Resolution Mass Spectrometry. J. Anal. Tox., V13, N6, 333-336, 1989.

38. Heindel JJ, Lamb JC, Chapin RE, Gulati DK, Hope E, George J, **Jameson CW**, Teague J, Schwetz BA. Reproductive Toxicity Testing by Continuous Breeding Test Protocol in CD-1 Mice. DHHS Publication No. (NIH) 89 Washington, DC, US Government Printing Office, 1989.

39. Cannon JM, Brown D, Murrill EM, **Jameson CW**. Identification of Components in Iodinated Glycerol. Journal of Pharmaceutical Sciences, V78, N1, 48-51, 1989.

40. Morgan DL, **Jameson CW**, Mennear JH, Prejean JD. 14-Day and 90-Day Toxicity Studies of C.I. Pigment Red 3 in Fischer 344 Rats and B6C3F1 Mice. Fd. Chem. Toxic., V27, N12, 793-800, 1989.

41. Morgan DL, **Jameson CW**, Mennear JH, Ulland BM. Thirteen-Week Toxicity Studies of CI Direct Blue 15 and 3,3'-Dimethoxybenzidine in the Fischer 344 Rat. Toxicology, V59, 297-309, 1989.

42. Dieter MP, **Jameson CW**, Maronpot RR, Langenbach RJ, Braun AG. The Chemotherapeutic Potential of Glycol Alkyl Ethers: Structure-Activity Studies of Nine Compounds in a Fischer Rat Leukemia Transplant Model. Cancer Chemother. Pharmacol., 26, 173-180, 1990.

43. Gorski T, Goehl TJ, **Jameson CW**, Collins BJ. Sources of Error in the Determination of Trichloroethylene in Blood. Bull. Environ. Contam. Toxicol., V45, 1-5, 1990.

44. Dieter MP, Boorman GA, **Jameson CW**, Matthews HB, Huff JE. The Carcinogenic Activity of Commercial Grade Toluene Diisocyanate in Rats and Mice in Relation to the Metabolism of the 2,4- and 2,6-TDI Isomers. Toxicology and Industrial Health, V6, No. 6, 599-621, 1990.

45. Morrissey RE, Fowler BA, Harris MA, Moorman MP, **Jameson CW**, Schwetz BA. Arsine: Absence of Developmental Toxicity in Rats and Mice. Fundamental and Applied Toxicology 15, 350-356, 1990.

46. **Jameson CW**, NTP Technical Report on the Toxicology and Carcinogenesis Studies of d-Limonene in F344/N Rats and B6C3F1 Mice (Gavage Studies). NIH Publication No. 347, 1990.

47. Gorski T, Goehl TJ, **Jameson CW**, Collins BJ, Bursey J, Moseman R. Gas Chromatic Determination of 2-Ethylhexanol and 2-Ethylhexanoic Acid as Derivatives suitable for Electron Capture and Nitrogen-

Phosphorus Detection After Single Reaction with Heptafluorobutyrlimidazole. Journal of Chromatography, 509, 383-389, 1990

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3. 2-Amino-3,4-dimethylimidazo[4-5-f]quinoline (MeIQ) - 2002
4. 2-Amino-3,8-dimethylimidazo[4-5-f]quinoxaline (MeIQx) - 2002
5. 2-Amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP) - 2002
6. 2-Amino-3-methylimidazo[4,5-f]quinoline (IQ) - 2002
7. Azacitidine - 1996
8. Beryllium and Beryllium Compounds - 2000

9. 2,2-bis-(bromomethyl)-1,3-propanediol (BBMP) (Technical Grade) - 2000
10. Boot & Shoe Manufacturing - 1998
11. 1,3- Butadiene - 1997
12. Cadmium and Cadmium Compounds - 1997
13. Chloramphenicol - 2000
14. Chloroprene - 1997
15. Chlorozotocin - 1996
16. p -Chloro-o-toluidine and its Hydrochloride Salt - 1996
17. Cobalt Sulfate - 2002
18. Cyclosporin A - 1996
19. Danthron (1,8-Dihydroxyanthraquinone) - 1996
20. Diazoaminobenzene - 2002
21. 2,3-Dibromo-1-propanol - 2000
22. Diesel Exhaust Particulates - 1998
23. Diethanolamine - 2002
24. 1,6-Dinitropyrene & 1,8-Dinitropyrene - 1996
25. Disperse Blue I - 1996
26. Dyes Metabolized to Benzidine (Benzidine Dyes as a Class) - 1997
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30. Estrogens, Steriodal - 2000
31. Ethyl Acrylate - 1998
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38. Lead and Lead Compound - 2003
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41. Naphthalene - 2002
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43. Nickel (Metallic) and Certain Nickel Alloys - 2000
44. o-Nitroanisole - 1996
45. Nitrobenzene - 2002
46. 6-Nitrochrysene - 1996
47. Nitromethane - 2002
48. 1-Nitropyrene - 1996
49. 4-Nitropyrene - 1996
50. Phenolphthalein - 1997
51. Saccharin - 1997
52. Silica, Crystalline (Respirable Size) - 1998
53. Smokeless Tobacco - 1997
54. Solar Radiation & Exposure to Sunlamps or Sunbeds - 1997
55. Strong Inorganic Acid Mists Containing Sulfuric Acid - 1997
56. Styrene-7,8-oxide - 2000

57. Tamoxifen - 1997
58. 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) - 1997
59. Tetrafluoroethylene - 1997
60. 4,4'-Thiodianiline - 2002
61. Thiotepa - 1996
62. Tobacco Smoking - 1997
63. 1,2,3-Trichloropropane - 1996
64. Trichloroethylene - 1997, 2000
65. Ultraviolet (UV) Radiation, Broad Spectrum and UVA, UVB, and UVC - 2000
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Jameson CW Contributor to the following NTP Report on Carcinogens Background Documents:

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