EXHIBIT 37

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

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

EXPERT WITNESS REPORT

of

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Table of Contents

1.0 Introduction:

Glyphosate (N-(phosphonomethyl)glycine) is a broad spectrum post emergent herbicide that has been registered for use globally. Though it has been confused with organophosphate pesticides, glyphosate is not an organophosphate but rather an aminophosphonic analogue of the natural amino acid glycine (Greim et al. 2015). Briefly, glyphosate is a highly water soluble (hydrophilic) chemical that is poorly absorbed from the skin (Wester et al., 1991). Oral exposure to glyphosate is considered to be the primary route of exposure (EPA, 2016). Absorption from the gut is estimated to be less than 30% of the oral exposure with negligible tissue accumulation and the majority excreted unchanged in the urine. Inhalation exposure is minimal and dermal penetration is low. The glyphosate that is absorbed from the skin is rapidly excreted mainly via the urine within 24 hrs. (Wester et al., 1991; Chan, 1992). Maximal human exposures of 0.47 mg/kg/day were estimated for children (1-2 years) and up to 0.03-7 mg/kg/day for mixer/loaders assuming that personal protective equipment was not employed (EPA, 2016). Regulatory agencies worldwide have independently assessed the ***human health risk*** for glyphosate, consistently reaching the conclusion that glyphosate is not a carcinogen. In contrast, in March 2015 the International Agency for Research on Cancer (IARC) finalized its ***health hazard*** assessment of glyphosate, a less stringent process than risk assessment, concluding that glyphosate is a probable carcinogen (classification 2A).

I have been retained as an expert to render opinions concerning the experimental rodent carcinogenicity bioassay literature including, but not limited to, those described by IARC and plaintiffs' experts purporting to link glyphosate exposure in rodents and carcinogenicity. Following a review of the available data I have concluded that glyphosate is not a rodent carcinogen. If glyphosate were carcinogenic in rodents, one would expect to see replication of carcinogenic findings of a particular tumor type across multiple studies. Such evidence is absent from this comprehensive and rich data set.

The following sections outline my background and expertise, approach taken in conducting my objective review of the data, assessment of the evidence, and my scientific opinions of the data. The opinions I plan to offer in this matter will include opinions set forth in this report, opinions that may be elicited from me in discussing or elaborating on those areas and/or responding to the testimony of plaintiffs' experts and any opinions formed based upon further literature review and review of any additional materials. My opinions are based on my review of the relevant scientific literature; materials specifically related to this case and related proceedings; and my education, training, research, and experience. A list of materials I have considered in forming my opinions is included in Section 6.0 below.

2.0 Background and Qualifications:

My expertise is in toxicology with a special focus on reproductive toxicology and environmental carcinogenesis. I have continuously carried out research in the field since 1991 conducting primarily animal studies according to established internationally accepted test guidelines

designed to provide data for government regulatory needs. I have also carried out numerous studies designed to assess human exposure and define mechanistic pathways to explain toxic phenomena including cancer. Over the course of my career, my expertise in the field has been recognized as shown by numerous invitations to provide expert technical advice to non-government organizations, government, and industry since 1991. Further detail of my background and expertise are summarized below:

(i) I obtained my undergraduate training (Hon. B.Sc.) in Human Biology from the University of Guelph, Guelph, Ontario, Canada (1979) and a M.Sc. from the University of Guelph in medical sciences (1986). I completed my doctoral training in medical sciences in 1991 at McMaster University.

(ii) I joined the staff of the Environmental Health Directorate at Health Canada in 1990 where I worked as a reproductive toxicologist. I was promoted to Head, Reproductive Toxicology Section and subsequently served as the Acting Division Chief, Environmental Health Directorate. As a Health Canada scientist, I oversaw an active research program consisting of four Sections focused on general, inhalation, reproductive/developmental toxicology and mutagenicity. During my career, I have designed and carried out animal studies to provide data necessary for regulatory assessment of chemicals under the Canadian Environmental Protection Act. Specifically, I designed, executed, and performed relevant data analyses, interpreted the study findings, and published the results of numerous animal studies for the assessment of the general, endocrine, reproductive and developmental toxicity as well as carcinogenesis of a broad range of chemicals including pesticides. I have also designed and executed numerous animal and tissue culture studies designed to elucidate mechanisms underlying the pathophysiology of observed adverse outcomes. I also participated in several epidemiological studies designed to assess human exposure to metals and persistent organic compounds to evaluate the potential impact of these exposures on human health. I have continuously carried out research and am recognized for my expertise in animal models and working at the intersection between animal research and clinical research (translational science). My productivity in the field and impact of my work has led to numerous invitations to present my findings at major international medical and scientific conferences and provide expert advice to government, non-government organizations, and industry since 1991 as detailed in my attached CV.

(iii) I am currently a Professor in the Department of Obstetrics and Gynecology at McMaster University where I lead a productive and well-funded research program designed to assess human exposure and the effect of environmental chemicals on adverse outcome pathways including oxidative stress, inflammation, autophagy, apoptosis, and cell proliferation; all of which are normal physiological processes that have also been associated with the pathogenesis of human disease including cancer. A separate line of inquiry in my laboratory is focused on the identification of clinical markers of endometriosis and novel therapeutic interventions. I am also a voluntary clinical professor in the Department of Reproductive Medicine at the University of California, San Diego where I conduct collaborative research in women's health.

(iv) McMaster University is a leading Canadian research intensive university that trains medical, undergraduate and graduate students in the medical sciences. I am involved in the teaching of undergraduate Bachelor of Health Sciences and Medical Students (Ovarian regulation and physiology of selective estrogen receptor modulators and selective progesterone receptor modulators). I teach several graduate courses (Reproductive Endocrinology and Environmental Toxicology) and directly supervise the training of graduate students and postdoctoral fellows. I mentor residents on their research projects and have served as the resident research coordinator and a member of the resident postgraduate education program as well as the postgraduate evaluation committee. I am also a member of the animal advisory committee and the animal research ethics board in the Faculty of Health Sciences at McMaster University.

(v) I have been continuously supported by the Canadian Institutes of Health Research (CIHR) since 2001. My research has also been supported by grants and contracts from the Natural Sciences and Engineering Research Council, The New York Community Trust, the American Chemistry Council, and Health Canada. I have published greater than 180 total career peer reviewed scientific publications. In addition, I have been invited to give over 92 invited scientific presentations and more than 137 presentations at scientific and medical conferences over the course of my career. My work is frequently cited with over 5,820 citations and an H-Index=45 (Measure of impact on the field).

(vi) I have served on numerous local, national and international expert panels including: National coordinator for the Organization for Economic Cooperation and Development (OECD) Test Chemical Guideline Program (bioassays for toxicology and carcinogenesis); WHO/IPCS Steering Group on Endocrine Disruptors, and Member, Council of Canadian Academies Panel, Integrating Emerging Technologies into Chemical Safety Assessment, and Expert Panel on the Integrated Testing of Pesticides. Recently, I also served as a member of an IARC expert monograph working group. I have also served on numerous, Local, National, and International grant review committees (e.g. Canadian Cancer Society, CIHR, NIH, and EPA-STAR), editorial boards, and have been elected by my peers to serve on the board of several prestigious scientific societies (e.g. Society of Toxicology Canada and the Canadian Fertility and Andrology Society) and recently was elected as a Fellow of the Canadian Academy of Health Sciences (CAHS).

(vii) I am currently an editor of the Journal of Applied Toxicology and a member of the editorial boards of the Journal of Toxicology and Environmental Health: B Critical Reviews, Reproductive Toxicology, Immunology, Endocrine & Metabolic Agents in Medicinal Chemistry, and the Faculty of 1000. Further details of my training and contributions to science are provided in my curriculum vitae included in section 7.0 of this report.

3.0 Statement of Opinions and rationale:

Unless otherwise stated, all the opinions expressed in this report are to a reasonable degree of scientific certainty including my opinion that glyphosate is not a rodent carcinogen. My opinion is based on my interpretation of the experimental animal studies and the following points:

(1) The animal studies have been conducted in accordance with recognized animal test protocols for carcinogenicity;
(2) Appropriate routes of test agent administration and dose ranges were used in these studies;
(3) The dose ranges used cover low concentrations through, and in some studies, well above the limit dose of 1,000 mg/kg/day;
(4) The methods of tissue analysis and data interpretation were appropriate;
(5) There is insufficient reliable scientific evidence of a dose-response relationship;
(6) Tumor incidences are generally within historical control ranges with the order of priority as follows:
    a. concurrent controls;
    b. historical controls from the same lab within 2-3 years; and
    c. historical controls from other labs or the same lab beyond 2-3 years.
(7) In all studies reviewed the data fails to show evidence of tumor progression;
(8) There is a lack of consistency of findings of tumor types across the animal studies (mice and rat) including within the same strain of rodent model; and
(9) Additional factors that further call into question the biological plausibility of putative compound-related changes.

Thus, taken together, these data demonstrate that tumors identified in the animal studies are most likely spontaneously occurring and unrelated to glyphosate exposure. Therefore, there is no scientifically reliable basis to justify a conclusion that glyphosate is a rodent carcinogen. Furthermore, I am not aware of any reliable scientific evidence available that Roundup formulations are a rodent carcinogen. I will use the following paragraphs to provide a detailed description of the literature reviewed, the methods of analysis employed to arrive at my conclusions, and highlight in each case how the study contributed to the above conclusions.

Literature reviewed: A critical review of the literature was carried out in which I reviewed all available case materials, which are summarized in the literature-cited section of this report. I reviewed detailed data reports arising from industry sponsored animal carcinogenicity studies, the IARC report on Glyphosate (IARC 2015), EPA evaluations and correspondence, and other regulatory documents (e.g. EFSA, BFR, and the Science Advisory Panel report). In addition, I also carried out an independent literature search using PubMed on August 18th, 2016 and updated on October 3rd, 2016, May 17th, 2017, and July 7th, 2017 to ensure that all relevant literature was included in my assessment. Bibliographies of each paper were also searched for additional relevant papers. To assess the literature, I assigned greater weight to studies conducted according to internationally accepted test guidelines for carcinogenicity for the following reasons:

(1) Greater weight was assigned to studies using at least three dose levels (low, medium, and high) allowing for assessment of dose-response characteristics. Dose levels that covered broad ranges of concentrations including a high dose that approached or included the limit dose (1,000 mg/Kg/day) were also given greater weight. Studies that exceeded the limit dose with concentrations that fell below 5% of the overall diet were also highly valued in my assessment. It is essential to include in the study a high dose group represented by the limit dose or 5% diet concentration to assure that animals have been exposed to a concentration of the test agent that will markedly exceed human exposure. If the dose is in the 5% range it should be below the maximally tolerated dose (MTD) to mitigate potential frank toxicity that would confound the results. Thus, evidence of adverse health effects was also considered in my assessment in reaching my final conclusions.
(2) It is essential that cancer studies use an adequate number of animals in each treatment group to detect statistically significant increases in neoplastic lesions. For this reason, studies that used at least 50 animals/treatment group were viewed as compliant with regulatory test guidelines. Studies that failed to use an adequate number of animals/groups were assigned lower weight in my analysis.
(3) Study duration for cancer studies typically should be at least two years in length (24 months) in rats or 18 months for certain strains of mice. This is done to capture the majority of life expectancy of the animals. Unless justified by evidence of increased mortality or neoplastic lesions of sufficient size or number to induce unreasonable pain or suffering, experimental animal studies of shorter duration were given less weight.
(4) Tumor progression was considered important in my assessment owing to current understanding of the mechanisms of chemical carcinogenesis. Consequently, if present, I would assign greater weight to studies in which evidence of chemical-induced tumor promotion was demonstrable. Additionally, factors contributing to biological plausibility were evaluated.

4.0 Assessment of Carcinogenicity:

Introduction and definitions **-** To understand how exposure to a chemical can lead to cancer, it is necessary to introduce some definitions and briefly discuss how cancer bioassays are conducted. Cancer refers to a malignant neoplasm (an abnormal growth of tissue that forms a swelling or mass), a lesion resulting from the new or autonomous growth of a tissue. Neoplastic lesions may be either benign (non-cancerous growth of cell mass that does not possess the ability to invade neighboring tissues) or malignant (cell growth that tends to worsen and has the properties of cells that have reverted to a less differentiated form, uncontrolled cell growth, invasiveness, and metastasis). Benign lesions are characterized by slow growth of the tissue that fails to invade surrounding tissues. Examples of benign growths include moles, fibroids in the uterus and endometriosis. In contrast, a malignant neoplasm is characterized by rapid growth,

invasions of surrounding tissue, and metastases (growth of cancer cells in distant tissues). When cancer develops it typically follows the progression from benign to malignant growth.

A chemical carcinogen is any chemical or its metabolite that can induce neoplasia (abnormal growth). Throughout the animal literature I reviewed and the studies cited by plaintiffs' experts (Drs. Jameson, Portier, Neugut, Weisenburger, and Nabhan), reference is made to several different types of tumors. Plaintiffs' experts have noted the different tumors in relevant studies but have failed to carry out a critical appraisal of these studies and the data generated. The evaluation and interpretation of data from such rodent studies is paramount in determining their significance. Plaintiffs' experts merely count several tumors and generate statistical comparisons, often novel and untested, absent further analysis. Critical analyses largely absent from plaintiffs' expert reports include, but are not limited to, biological relevance (e.g. dose response, mechanism, relevant human exposure, and translation from rodents to humans), neoplastic continuum or progression, expected tumor incidences, and replication of tumor types across multiple studies. Therefore, I will describe the different types of tumors here and provide a detailed assessment of each study considered in rebutting the plaintiffs' experts and arriving at my conclusions.

Several different tumor types are discussed in the literature reviewed and therefore definitions are provided. Adenomas are benign tumors in which epithelial cells cluster together and form recognizable glandular structures. Also discussed in one study is an interstitial tumor of the testes. These tumors are also known as Leydig cell tumors (the cells that produce testosterone) a type of tumor that is typically benign although some may progress to malignancy. Another tumor type mentioned in the reports that I reviewed was a hemangioma. A hemangioma is a congenital malformation consisting of a benign tumor made up of newly formed blood vessels. The cause of these benign tumors is unknown and they can appear throughout the body, including the skin, liver, and bones. In contrast, a hemangiosarcoma is a malignant tumor of the blood vessels that has its origin in the vascular endothelium (the cells that line the blood vessel). Finally, also discussed in the animal studies are lymphomas. Lymphoma refers to a group of over a dozen tumors that arise from lymphocytes which are a type of white blood cell.

Multistep pathway to cancer **-** Cancerous growths are thought to arise through activation of the carcinogenic cascade that involves initiation, promotion, and ultimately neoplastic progression (Cohen and Ellwein 1991). A carcinogen is a physical (e.g. radiation) or chemical agent (or one or more of its metabolites) whose interaction with DNA typically induces a mutation in the genetic code and thus acts as an initiator of cancer. Mutations do not always lead to cancer since cells possess DNA repair mechanisms or the damage occurs in a non-coding region of the DNA. In contrast, clonal expansion of the mutated cell can occur if repair of the damaged DNA is unsuccessful.

Tumor growth occurs through the influence of growth factors or hormones that induce cell proliferation and thus tumor promotion. Substances that are only tumor promoters are typically not mutagenic and are unable to induce neoplastic lesions on their own.

Cancer progression refers to the irreversible conversion of a benign pre-neoplastic lesion to a neoplastic lesion. Chemicals that induce tumor progression are usually genotoxic. Tumor progression can occur spontaneously from the accumulation of chromosomal aberrations or instability of the chromosomes.

Hallmarks of carcinogens **-** The impact of chemical agents on gene expression, cell signaling pathways, receptor binding and signaling, inflammation, immune surveillance, cell proliferation and nutrient supply has been recognized and are potentially important in the pathogenesis of cancer (Hanahan and Weinberg 2011). Ten key characteristics of chemical carcinogens have been described and summarized (Smith et al. 2016) and cited by Dr. Portier in his expert report. Although chemical carcinogens are reported to induce one or more of the 10 key characteristics, use of these hallmarks as a "rule in" test for carcinogenicity is unfounded. The concentrations and the experimental conditions needed to induce changes in any of the hallmark pathways must be carefully evaluated for their relevance to human health. Indeed, numerous chemicals have one or more hallmarks of carcinogens but the effects can only be produced at concentrations that far exceed potential human exposure.

While rodent carcinogenicity assays are used for identification of potential hazards, it is important to note that not all chemicals that have been shown to be rodent carcinogens have been found to be human carcinogens. Potential reasons for this lack of relationship include: different mechanisms of action in rodents compared to humans; differences in absorption, distribution, metabolism, and excretion; and the concentration of test chemical needed to induce cancer in rodents exceeds concentrations that can effectively be achieved in humans. The assessment of carcinogenic potential and health risk is a complex task involving more than simply counting lesion types and assigning a label. Critical to the process is assessment of the biological relevance of all adverse outcomes enumerated in a study and ultimately evaluation of human exposure relative to doses needed to induce an adverse outcome in the most sensitive animal model accounting for uncertainties in translation of results from animals to humans.

Overview of cancer bioassays **-** In recognition of the long-term serious health consequences represented by chemical carcinogens, chemicals are routinely screened for their potential to induce cancer. Beginning in the early 1960's the National Cancer Institute began to develop animal test methods for the assessment of potential chemical carcinogenicity. Within a decade the two rodent species, two sex, and two year cancer bioassay was in wide use. Regulatory agencies from multiple countries subsequently standardized, validated, and harmonized rodent carcinogenicity test protocols. Standardized cancer bioassays are regulatory requirements for chemical registration. The World Health Organization (WHO) under the leadership of the Organization for Economic Cooperation and Development (OECD) oversees the International Program on Chemical Safety that is mandated with the development and validation of test guidelines for the assessment of chemical hazards. The OECD Test Guideline Program is responsible for a collection of the most relevant internationally agreed upon testing methods used by governments, industry and independent laboratories to assess the safety of chemical products. Test Guidelines are updated on a regular basis to keep pace with progress in science and countries' regulatory needs. The objectives of OECD carcinogenicity studies (OECD-TG-

451/452) and the combined chronic toxicity and carcinogenicity study (OECD-TG-453) are designed to observe test animals for a major portion of their life span for the development of neoplastic lesions with exposure to at least three doses of a test substance and a vehicle control using an appropriate route of administration (typically oral). Carcinogenicity bioassays are conducted over a period of 24 months for rodents; however, for specific strains of mice a duration of 18 months may be more appropriate, to account for the shorter life-span for these strains (e.g. CD-1 mice). A tumor incidence of 57% was found in Sprague Dawley (SD) rats allowed to live out their entire life-span (Davis et al. 1956). The average life-span of SD rats in this laboratory was approximately 760 ± 21 days and 87% of all tumors appeared after the animals were 540 days of age. Consequently, the study duration of 24 months (730 days) in cancer bioassays has been selected to account for the recognized long latency between initiation of cancer and the detection of tumors that follows tumor promotion and progression. This study design also accounts for compensatory mechanisms (e.g. DNA repair) as well as tissue repair providing an adequate time for tumors to appear. Note that a study may be terminated early should the number of survivors in the lower dose groups or the control group fall below 25 per cent. The study may also be terminated early if the tumor volume attains a size or number of tumors that seriously compromises the health of the animals. It is important to note that two-species, two-sex, rodent cancer bioassays conducted according to test guidelines usually generate reproducible results (Gold et al. 1987; Gold et al. 1989; Haseman and Huff 1987) that are valuable for human risk assessment. It is expected that studies adhering to standard operating procedures and following regulatory study guidelines would produce reproducible findings. Failure of studies to generate replicable data causes concern and calls into question the relevance of the data because of the probability the finding might be due to random chance, normal variation of the outcome, or methodological issues. Scientists faced with divergent results are compelled to explore the reasons for the lack of consistency in the data. Common reasons for lack of reproducible findings include: differences in study methodology; animal model used; duration or amount of test chemical exposure; analytical methods employed; time frame of the study; and normal variation in the outcome being observed.

Interpretation of bioassay results – Evaluation of bioassay results and determination of their importance involves several steps, including assessment of adherence to study protocol, statistical methods, data analysis, and interpretation. When reviewing any study, the first step involves understanding the question being addressed and the appropriateness of the animal model employed. For cancer bioassays, the main points to be addressed are the use of an appropriate rat or mouse strain, randomization to treatment groups, appropriate route of test chemical administration, dose levels that cover a broad range up to and including the limit dose, duration of study that allows for development of tumors, assessment of systemic toxicity, and accepted histopathological methods. In reviewing study reports it is important to consider the statistical methods employed to ensure that prescribed methods have been followed or if alternative methods have been employed, their use has been adequately justified.

Detection of statistically significant differences among and between treatment groups is important to note but their biological relevance must also be determined. Specifically, it is

possible to have statistical significance in the absence of biological relevance and thus the investigator must evaluate whether the changes detected are internally consistent based on the totality of the animal data collected and current understanding of underlying biological mechanism(s). This may involve consideration of the normal range for a given outcome. To do this it is best to make comparisons with concurrent controls or alternatively appropriate historical controls. I agree with Dr. Portier that it is best to compare data with contemporary controls. However, historical controls generated in the same lab within 2-3 years of the study may also provide useful information. If such historical control data is not available then it may be necessary to compare with data outside the preferred time period. However, that data should be used with extreme caution for quantitative analysis due to differences in the genetic makeup of animals of the same species/sex/strain over time and between laboratories.

In assessing rodent bioassay data, evidence that could indicate systemic toxic effects as shown by changes in body weight, stereotypical behaviors, abnormal vocalizations, porphyria, lacrimation, ruffled coat, barbering of the hair or changes in clinical chemistry and hematology should also be considered. The interpretation of bioassay outcome is based on the integration of what is known of study conduct, data produced, statistical analysis, and understanding of the underlying biology for each outcome. Finally, bioassays are carried out with the goal of identifying adverse outcomes for hazard identification. A properly conducted hazard identification, taking into account all of the available relevant evidence, is but one step in the process of determining risk. Risk assessment is a protective process used in regulatory toxicology that is the product of hazard, human exposure, and recognition of translational differences between rodents and humans. Furthermore, the determination of a risk does not necessarily mean causation has been established. Evidence of scientific causation involves the integration of additional issues such as demonstration that human relevant concentrations of the test chemical can reproducibly induce the adverse effects, evidence of human exposure, knowledge of internal dose, target tissue exposure, understanding of mechanism of action, and its applicability to humans. Consequently, these rodent bioassays on their own do not generate data that can establish causal relationships between external exposure and human health. However, these bioassays may generate data that suggests a compound is unlikely to be carcinogenic in humans.

The case of glyphosate is unusual in that there are multiple rodent (mouse and rat) study results from cancer bioassays. As a result, there are individual study results to evaluate. Additionally, results of the data generated can be assessed across studies. Dr. Portier uses a novel statistical approach for generating a test statistic for comparing current results with those of historical controls. I have not seen this approach applied elsewhere in the toxicology literature or in my profession, and thus I am not aware that this is a validated method that has been assessed by scientific peers or achieved general acceptance by the scientific community. In addition, Dr. Portier employs pooling of data across studies which again is a novel statistical approach that has not been peer reviewed, validated, and to my knowledge has not been tested to reach the level of a generally accepted approach in regulatory toxicology. Indeed, Dr. Portier admits that his methodology utilized in his report is novel and untested (Portier report, pg. 21). While pooling

such data has not yet been evaluated, validated, and thus is not generally accepted in the scientific community or ever even done to my knowledge, comparing data from rodent studies is a standard practice and paramount in assessing carcinogenicity.

Further, Dr. Portier acknowledges that "simple evaluation of the positive versus negative findings fails to resolve the issue of which findings are driving the overall responses in these data." (Portier report, p.47). A central concept of science is reproducibility of data both within a laboratory, across laboratories, and over time. The lack of reproducible effects, as revealed by the glyphosate data set, indicates that these effects are not compound-related.

Where statistically significant results are lacking, Dr. Portier repeatedly relies upon so-called "marginal trends" to ascribe significance to statistically insignificant data. In my field of expertise, typical research activity outcomes are either statistically significant at the $p<0.05$ level or they are not. The concept of "marginally significant trends" is not typically used in the biomedical literature.

In contrast to the approach of Dr. Portier I, like other scientists in the field, compare the results from multiple studies mindful of differences in animal strain, doses of test chemical employed, experimental conditions, analytical methods employed, data analysis, and interpretation of the study results. This comparison is undertaken utilizing a qualitative evaluation, with statistical significance forming just one piece of the overall evaluation. The generally accepted scientific methodology, which I employed, involves careful consideration of study design, conduct, analytical methodology, data analysis and interpretation to ensure that the results are not over or under interpreted. Moreover, to fully appreciate the data, a critical and qualitative appraisal of the data is necessary and this cannot be achieved through statistical analysis alone.

5.0 Assessment of experimental animal literature:

Animal studies are essential in regulatory toxicology, particularly in the absence of evidence of human exposure and epidemiological data. Animal studies allow scientists to address important questions that are either unethical or cannot be carried out in human studies. For example, in human studies, exposure to test chemicals and developmental life-stage of exposure are often unknown. Moreover, many target tissues (e.g. brain, liver, and kidney) are not easily accessible and thus cannot be examined in human studies. Thus, surrogate markers are frequently used to detect effects in these tissues. In contrast, experimental animal studies allow for the administration of known quantities of a test chemical of known purity under carefully controlled environmental conditions (temperature, humidity, time of day, lighting conditions, and unlimited access to water and food of controlled nutritional composition) in genetically similar animals of known age, developmental stage and health. Experimental animals are monitored over the course of the study for changes in biochemistry and hematology, allowing for the early detection of changes in health status. At study conclusion, a full necropsy is performed and all organ and

tissue samples undergo careful gross and histopathological assessment by board certified veterinary pathologists. Thus, these studies provide robust data for the detection of potential adverse outcomes for regulatory purposes that cannot be achieved through alternative means. Of additional importance, the concept of multiple comparisons increases the probability of finding differences through chance alone (Squire 1989). Dr. Portier also recognizes the problems multiple comparisons pose with a data set of this magnitude (Portier report, p. 40), though we disagree on the remedy for that problem (see pp. 21 and 47). Dr. Portier further acknowledges that one expects a certain number of positive findings due to chance alone (Portier report, p. 50). Thus, the finding of a statistically significant difference cannot be interpreted by itself to indicate a compound-related effect. Therefore, results of animal studies may elucidate statistically significant effects of treatments that must be evaluated further for their biological relevance.

Glyphosate has been assessed using regulatory toxicology studies in mice and rats. Sufficiently high doses of glyphosate were used in most studies, approaching or exceeding the limit dose as described in regulatory cancer test guidelines (e.g. OECD TG 451). Results from over a dozen regulatory studies revealed spontaneous tumors in several different tissues but all unrelated to glyphosate treatment. In view of the relatively large number of studies that have been carried out, it is remarkable that although incidental tumors unrelated to glyphosate exposure were found in individual studies, there is a consistent absence of evidence via replicated results for carcinogenicity across studies. The lack of reproducible tumor findings in animal studies designed specifically to produce reliable and reproducible results is compelling evidence for a lack of association between glyphosate and carcinogenicity. One should be mindful that rodent bioassays are not hypothesis driven, and require both gross and histopathological evaluation of all tissues thus increasing the probability of false positive results. Indeed, these studies are designed to maximize the potential of detecting compound-related effects at the expense of detecting false positive results. Detection of a statistically significant positive result is not the end of the study analysis but rather only the beginning of the scientific assessment as it is necessary to determine if the findings are spurious or represent biologically important findings. Consequently, I will describe each study in detail in the following sections.

Rat studies

Studies judged as inadequate

In combination, Drs. Neugut, Portier, and Jameson excluded six studies (Reyna, Chruscieleska, Excel, Seralini, Burnett; Pavkov and Wyland), all of which were reported to be negative studies with the exception of one (Seralini et al. 2014). While in total, plaintiffs' experts excluded six studies, they were not in complete agreement. However, given the methodological concerns and the lack of reliance of plaintiffs' experts, I give the six studies referenced above and the plaintiffs' expert opinions based on those same studies minimal weight in my causation analysis and will not discuss these studies further.

Studies judged as adequate

Of the studies judged as adequate, Dr. Portier provides a statistical assessment of the data only whereas Dr. Jameson provides a summary of the results analogous to the listing of data in the IARC report for glyphosate. The entirety of Dr. Neugut's discussion of the animal data is comprised of two paragraphs and an accompanying table. Similarly, Dr. Weisenburger, though acknowledging the existence of numerous negative glyphosate rodent studies, concludes without engaging in generally accepted toxicological analysis that "positive studies listed above cannot be dismissed, and provide sufficient evidence for the carcinogenicity of glyphosate in experimental animals … ." (Weisenburger Report at p. 8). Plaintiffs' experts fail to provide a critical appraisal of the studies or interpretation of the data. Further they fail to discuss potential explanations for lack of consistency of study findings. Hence, the rationale for their conclusions is in my view unsupported. The results of each study and its' biological relevance are described in detail below.

Bio/Dynamics Inc. 1981 Study BDN-77-2062 (Lankas et al., 1981 discussed in Greim et al., 2015 - Study 1): In 1981 Bio/Dynamics Inc. conducted a combined chronic toxicity and carcinogenicity study of glyphosate for the sponsor. Sprague-Dawley rats were exposed to 30, 100 and 300 ppm glyphosate in the diet over the course of their lifetime (26 months). The dose was adjusted according to changes in body weight such that the rats were exposed to 3.05, 10.30, and 31.49 mg/Kg/day for males and 3.37, 11.22, and 34.02 mg/kg/day for females. Body weight, food consumption and clinical laboratory studies were conducted over the course of the study.

Neoplastic lesions were documented in the pituitary and pheochromocytomas in males and females and mammary tumors in females; however, the incidence was similar across all treatment groups and thus is not considered to be treatment related. Interstitial tumors of the testis (Leydig cell tumors) were detected in this study with an incidence of 0, 3, 1, and 6 in the controls, low, medium, and high dose groups, respectively. While the incidence of testicular tumors in the low and medium dose groups was within the highest incidence found for historical control (7%), the incidence of these tumors in the highest dose group (12%) was greater than that for the historical control group examined for this research laboratory. Consequently, neoplastic changes in the testis of the high dose group were evaluated to better elucidate their importance.

The testes received thorough histopathological evaluation. The study pathologists did not report any evidence of any dose related changes but did report a notable absence of compound-related hyperplasia. Hyperplastic changes would be expected to be present in the case of compound-related tumors and indeed to be coincident with the development of lesions. Moreover, there are several additional issues noted in the conduct of the study that impact the relevance of the testicular tumors. Specifically, the number of animals surviving to the end of the study was greater in the highest dose group compared to the control group. This is an important point because it creates the scenario where there were more animals in this group that had the opportunity to develop spontaneous neoplasms and thus could artificially increase the likelihood

of detecting a neoplastic change in this group compared to the control group. Had animals in the control group lived for the same length of time as those in the highest dose group (this is referred to as survival bias), it is possible that no effect may have been found. Furthermore, the absence of testicular tumors in the control group is below the historical range noted for this strain of rat and studies conducted by this laboratory. A lower response rate than expected in the control group could therefore increase the likelihood of detecting a false positive increase in testicular neoplasms.[1] Finally, interstitial testicular tumors have not been replicated in any other study including those in which much higher doses of glyphosate were employed. Moreover, it is relevant to note that, in the studies considered acceptable by plaintiffs' experts, doses similar to Lankas et al., along with much higher doses were employed, and no relationship between glyphosate and interstitial testicular tumors was found.

This study also revealed the presence of spontaneous occurrence of thyroid C cell tumors in females. A statistically significant trend for thyroid C cell carcinomas in the female animals only was observed (1/47, 0/49, 2/50, and 6/47). However, after combining thyroid C cell adenomas and carcinomas, which is appropriate (McConnell 1986), the statistical significance disappears. Although Dr. Portier agrees that combining these tumors is appropriate, he raises concern about these tumors in female rats even though none of the other studies revealed a statistically significant increased incidence of these tumors. I therefore conclude that the presence of thyroid C cell tumors in female rats in this study is not compound-related.

Although pairwise comparisons revealed a statistically significant increase in the number of pancreatic islet cell tumors for the lowest dose group (3.05 mg/kg/day) in male animals only, there was no evidence of a dose response (0/50, 5/50, 2/50, and 3/50). The low incidence of tumors, lack of evidence for tumor progression, absence of a dose response, and consistency with historical controls all support the conclusion that these tumors are not compound-related.

In summary, both the decreased survival in the control group compared to the highest dose group of rats together with the lower incidence of testicular tumors in the control group compared to historical controls increases the likelihood of finding a statistically significant increase in tumors amongst the other dose groups whose biological relevance is questionable. Dr. Portier speculates that the 26-month duration of the study offers unique insights that may be missed in a study lasting only 24 months. However, no evidence is offered and I am not aware of any data demonstrating that a 26-month study would detect interstitial tumors at any different rate than in a 24-month study in dose groups compared to control groups. I therefore conclude that the absence of a dose related increase in incidence of interstitial tumors, the absence of changes that

[1]An example might be illustrative here. If one expects a background rate of 6% for a given tumor in a study with four arms of 50 rats each, one would expect 3 out of 50 rats in each group to have that tumor (or 12 out of 200 overall). However, if the control group has 0% by chance, then one would expect 4 out of 50 rats in the remaining groups to have that tumor by chance because one would still expect 12 rats out of 200 to have that tumor.

would be indicative of tumor progression, and statistical limitations suggests that these tumors are incidental findings unrelated to glyphosate exposure.

Strengths of this study include an appropriate study design, adequate number of animals/dose group, use of three dose groups plus a negative control, appropriate methodology for tissue assessment and statistical analyses of the data. The study was conducted prior to the introduction of standardized and internationally accepted OECD test guidelines for carcinogenicity although much of the methodology is consistent with those guidelines. However, the use of a high dose that was substantially below the limit dose is a weakness of the present study.

MSL-10495 (Stout and Ruecker, 1990; discussed in Greim et al., 2015 - Study 2): This was a chronic study conducted by Monsanto in albino Sprague-Dawley rats (n=60/group) following a two-year cancer bioassay (consistent with the current OECD carcinogenicity TG-453). Rats were fed glyphosate (96.5% pure) in the diet for 24 months. Target doses were 2,000, 8,000, and 20,000 ppm. Using food consumption data, the authors calculated that the rats were exposed to 89, 362, 940 mg/Kg/day for the males and 113, 457, and 1,183 mg/Kg/day for the females of the low, medium, and high dose groups.

There was a treatment-related significant decrease in body weight among the female rats in the highest dose group in the absence of any change in food consumption. Significantly increased liver weight was also found for males in the highest dose group. The number of animals surviving to the conclusion of the study was 29, 38, 34, and 34 for males and 44, 44, 34, and 36 for females. Taken together, these data suggest that the dose selection was considered adequate for a carcinogenicity study. However, signs of toxicity in the highest dose group could confound interpretation of neoplastic changes. Regardless, non-statistically significant neoplastic changes were noted in pancreatic islet cells. Specifically, pancreatic islet cell adenomas (benign lesion) were found in 1/58 (2%), 8/57 (14%), 5/60 (8%), and 7/59 (12%) in the control, low, medium, and high dose group males. In females the incidence was 5/60 (8.33%), 1/60 (1.67%), 4/60 (6.67%), and 0/59 in the control, low, medium and high dose group females, respectively.

I conclude that the pancreatic islet cell adenomas are not treatment-related for several reasons. First, a dose related increase in the incidence of these lesions was not demonstrated in the males and the incidence of these tumors was higher in the control females compared to those treated with glyphosate. Furthermore, while pancreatic islet cell tumors are more common in male than female rats (Majeed 1997), these tumors are known to occur spontaneously in aged Sprague-Dawley rats (Chandra et al. 1992) and the incidence of these lesions in the current study was within the range of historical controls (0-17%)[2]. Moreover, there was no evidence of neoplastic

[2]I have used the range of historical controls as opposed to the mean, which is the common and standard practice in interpreting toxicological data. The range is more relevant compared to the mean because it provides the reviewer with a better appreciation for the spread of the data, which can be variable for many tumors and is thus important to take into consideration (Baldrick 2005; 2007).

progression noted in any of the pancreatic specimens examined and the only carcinoma that was found occurred in a male animal from the control group. Hence, the neoplastic changes documented in this study are in my opinion spontaneously occurring pancreatic islet cell tumors and unrelated to glyphosate treatment.

To complete its review, the United States Environmental Protection Agency (US-EPA) requested additional data on the historical controls for the following lesions: (1) thyroid C-cell adenomas, carcinomas, and hyperplasia; (2) hepatocellular adenomas, carcinomas, and hyperplasia; and (3) keratoacanthomas. A review of the data submitted on incidence of historical controls led to a conclusion that, for all three neoplasms, the incidences for all lesions fell within the range of historical controls and therefore, I conclude that these lesions were spontaneously occurring and unrelated to glyphosate treatment. Dr. Portier reports that there was a statistically significant trend for liver adenomas; however, this disappears when these lesions are combined with hepatocellular adenocarcinomas. I further noted that there was no compound-related replication of these tumors across multiple studies, there was no progression to carcinoma, no evidence of a dose response, and finally no significance when combining adenomas/carcinomas. Accordingly, I conclude within a reasonable degree of scientific certainty that glyphosate was not carcinogenic in this study.

Strengths of this study include use of an appropriate study design, number of animals/dose group, three dose groups and a negative control, appropriate route of exposure, use of a high dose that reached the limit dose, and adequate study duration.

MRID (Brammer, 2001 – discussed in Greim et al., 2015 - Study 7): This study was conducted by Syngenta and included Wistar rats of both sexes (52/group) treated with vehicle or glyphosate (121, 361, and 1214 mg/kg/day for males and 145, 437, 1498 mg/kg/day for females) in the diet for 24 months in a standard cancer bioassay. Strengths of this study include use of an appropriate study design, number of animals/dose group, three dose groups and a negative control, appropriate route of exposure, use of a high dose that exceeded the limit dose, and adequate study duration.

Results of this study revealed the presence of liver adenomas with an incidence of 0/52, 2/52, 0/52, and 5/52 in the control, low, medium, and high dose animals, respectively. It is important to note that in this study, males of the highest dose group were more robust as reflected in a better survival compared to the control group (26/52 vs. 16/52, respectively). Increased survival to the end of the study is relevant because it allows the animals longer exposure time for tumors to spontaneously emerge and thus increases the likelihood of detecting tumors in the high dose group compared to the control group. Furthermore, according to the historical control data cited by Dr. Portier, it is relevant to note that the range of liver adenomas in Wistar rats is 0 to 17.5% (Giknis and Clifford, 2011). Results of the present study are within the range of these historical controls. Thus, the findings of a 10% incidence in adenomas in rats of the highest dose group in

the present study are not in my opinion treatment related. Beyond just a statistical comparison of the numbers, there was no evidence of a dose response and evidence of progression from adenomas to adenocarcinomas was also lacking. Hence, for multiple reasons this is considered a negative carcinogenicity study.

MRID 49987401 (Wood et al., 2009a – discussed in Greim et al., 2015 - Study 8): Adult male and female Wistar rats were treated with vehicle of 95.7% pure glyphosate (95, 317, and 1230 mg/kg/day) in the diet. This was a combined chronic toxicity and carcinogenicity study and conducted according to regulatory guidelines. Strengths of this study include use of an appropriate number of animals/dose group, three dose groups and a negative control, appropriate route of exposure, use of a high dose that exceeded the limit dose, and adequate study duration. Combining a chronic toxicity study with a two-year cancer bioassay is seen as an additional strength of the study owing to the incorporation of additional outcome measures of general toxicity.

Results of this study failed to reveal a statistically significant increase in the incidence of mammary gland adenomas whilst there was a significant trend for increased incidence of mammary gland adenocarcinomas (2/51, 3/51, 1/51, and 6/51). Combination of adenomas and adenocarcinomas was significant using a pair wise test for the highest dose group only (2/51, 3/51, 1/51, and 8/51) and trend analysis was also significant. However, it is noted that there was no evidence of a dose response. Moreover, I note that in the Brammer study discussed above there was no evidence of a statistically significant trend for an increase in the number of mammary gland adenomas and adenocarcinomas. Furthermore, the incidence of adenomas and adenocarcinomas was higher in the controls of the Brammer study than in the treated animals. The same results were found in the Suresh study discussed in detail below.

Taken together these considerations lead me to conclude that mammary gland tumors in this study were not compound-related. In the summary of the study, there was no mention of proliferative changes. Dr. Portier asserts without reference that mammary adenocarcinomas can arise without the presence of adenomas. It is unclear where this comment derives from and it is inconsistent with the generally accepted view of cancer pathobiology. Mammary gland tumors are common in rats with a prevalence of 57% in female Sprague-Dawley rats allowed to live out their natural life-span (Davis et al. 1956) and within the historical control range for these tumors in Wistar rats (Giknis and Clifford, 2011). I note that there were no statistically significant mammary tumors detected in other well conducted studies in Wistar (Brammer, 2001; and Suresh, 1996) or any Sprague Dawley (Lankas, 1981; Enemoto, 1997; Atkinson et al., 1993; Stout and Ruecker, 1990) rats as summarized in Table 8 from Dr. Portier's report (p. 33).

A non-significant increase in skin keratoacanthomas (2/51/ 3/51/ 0/51, and 6/51) was noted but trend analysis was significant in the males. The dose response was not detected and the lack of pair-wise statistical significance suggests that this pattern of tumors is not compound-related. Furthermore, the lack of reproducibility of these findings - in other well conducted studies - further indicates that these findings were not compound-related.

In summary, this was a well-designed carcinogenicity bioassay that employed an appropriate route of exposure, number of animals, dose groups, and study duration. The maximal dose exceeded the regulatory requirement for a limit dose. Absence of evidence of a significant dose response for the mammary tumors, a common spontaneously occurring tumor, and lack of findings of tumor progression lead me to conclude that the mammary tumors were unrelated to glyphosate treatment. Therefore, it is my assessment that this is a well-conducted negative study.

Atkinson et al. 1993 (discussed in Greim et al., 2015 - Study 3): A combined chronic toxicity and carcinogenicity study was carried out using dietary exposure of glyphosate (98.9% pure) administered to 50 Sprague Dawley rats of both sexes/dose group. Five dose groups including a vehicle control were used. The males received 0, 11, 112, 320, and 1,147 mg/Kg BW/day while the females received 0, 12, 109, 347, and 1,134 mg/Kg BW/day for 104 weeks. Interim sacrifices were carried out at one-year in an additional 35 rats from each sex and dose group.

There were no adverse effects of treatments and histopathological assessment of tissues failed to reveal any morphological abnormalities in this study. Furthermore, there was no increase in the incidence of neoplasia in any tissue studied. Although there were no significant glyphosate effects on thyroid tumor incidence, Dr. Portier notes a statistically significant trend for follicular thyroid tumors based on the assumption that the unexamined mid-dose groups would not have any tumors. Thyroid follicular tumors are relatively common in rats with an average incidence of 3.79% and a range of 0-14% in control animals studied over 24 months (Giknis and Clifford, 2004). Thus, the high dose tumor incidence falls within the range of historical controls suggesting that these tumors are not compound-related. Moreover, the lack of reproducibility of these tumors in other studies further suggests that these are incidental findings that are not related to glyphosate exposure.

This study has a number of strengths including the study design employed, use of an appropriate route of administration, inclusion of four dose groups in addition to a vehicle control, use of an appropriate high dose, appropriate number of animals/dose group, two-year duration of the study, and inclusion of interim analysis of animals for signs of toxicity. No weaknesses were detected with the study design, execution or interpretation of the study results. Dr. Portier considers this a weak study because it failed to examine the pathology of all animals in mid dose groups. However, Dr. Portier's own report (p. 27) showed that adding in additional animals to the mid-dose groups did not produce a meaningful change in the p-trend unless one assumes that all animals in the mid dose groups would not have had tumors. However, as noted above, these are common tumors and thus this maybe an unreasonable assumption. Therefore, I assign little

weight to Dr. Portier's criticism especially because this study was consistent with the state of the art of the time that it was conducted. Accordingly, this study was considered a strong negative study that lends strength to the view that glyphosate is not a carcinogen.

Suresh, 1996 (discussed by Greim et al., 2015 – Study 4): In this study, 50 Sprague Dawley rats of each sex were assigned to one of three dose groups or a control. The animals were administered glyphosate (96-96.8% pure) at doses of 0, 6.3, 59.4, and 595.2 mg/Kg BW/day (males) and 0, 8.6, 88.5, and 886 mg/Kg BW/day (females) in the diet for 24 months. Results of this study did not elicit any evidence of adverse health effects and no morphological abnormalities were demonstrated in the histopathological assessment of the animal tissues studied. Furthermore, there was no evidence of a statistically significant increase the trend in tumor incidence in any of the tissues studied.

Overall, this is a well-designed study with numerous strengths including an appropriate study design for the assessment of carcinogenicity, use of three dose groups plus a control, route of glyphosate administration, and appropriate study length. A weakness of this study is the highest dose group falls below the limit dose.

Enemoto, 1997 (discussed in Greim et al., 2015 – Study 6): This was a combined chronic toxicity and carcinogenicity study carried out by Arysta Life Sciences in 50 Sprague Dawley rats for each sex and dose group. Rats were administered glyphosate in the diet at dose levels of 0, 104, 354, and 1127 mg/kg BW/day (males) and 0, 115, 393, and 1247 mg/kg BW/day (females) for two-years. Interim sacrifices were carried out at 26, 52, and 78 weeks in 10 rats of each sex/dose group. This study did not reveal any signs of adverse effects and histopathological assessment of the tissues did not demonstrate any evidence of any morphological abnormalities. However, although pair wise comparisons failed to reveal a statistically significant effect of treatments, Dr. Portier reports that there was a statistically significant trend for kidney adenomas (p. 30). Histopathological assessment of the tissues failed to reveal any evidence of hyperplasia and thus there was no evidence of tumor progression. The lack of evidence of morphological abnormalities (hyperplasia), absence of significant differences by pair wise comparisons, absence of dose response, and failure to replicate these findings in other well-conducted studies leads me to conclude that this observed trend is not glyphosate related.

This study has a number of strengths including the study design employed, use of an appropriate route of administration, inclusion of three dose groups in addition to a vehicle control, use of an appropriate high dose, appropriate number of animals/dose group, two-year duration of the study, and inclusion of interim analysis of animals for signs of toxicity. No weaknesses were detected with the study design, execution or interpretation of the study results. Accordingly, this negative study lends strength to the view that glyphosate is not a carcinogen.

Synthesis of rat studies

In summary, while potential neoplastic changes were documented in these studies, the totality of the evidence leads me to conclude that these tumors were not glyphosate related. This is especially because, as Dr. Portier readily admits, statistically significant results are to be expected given the high number of tests performed across these studies. These studies all failed to demonstrate any evidence of statistically significant compound-related increased incidence of tumors. These studies are highly relevant and were generally conducted according to well-established and internationally harmonized regulatory carcinogenicity test guidelines. The conclusions in the current report diverge from those of Dr. Portier. Reasons for our divergent conclusions can in part be explained by differences in the methodology and analyses carried out. Specifically, in the current assessment of the data, I have assessed the overall scientific quality of the studies in addition to evaluating neoplastic changes in the context of the totality of scientific knowledge rather than limiting discussion to just a statistical analysis of the data. The absence of compound-related tumor findings in each of these individual negative studies indicates that in total glyphosate is not carcinogenic in rats. Therefore, I conclude that there is no scientifically reliable evidence of glyphosate carcinogenicity in rats.

Mouse studies

Studies judged as inadequate

The doses of glyphosate used in the Reyna and Gordon (1974) and Pavkov and Turner (1987) studies were inadequate and thus were excluded from my analysis. Dr. Portier similarly considered these studies and excluded them from his analysis. These studies were all negative; however, in view of their limitations and consistency of opinions on these studies they will not be discussed further here.

Studies judged as adequate

Knezevich and Hogan, 1983 (discussed by Greim et al., 2015 - Study 10): The results of this mouse study conducted by Monsanto have received considerable attention owing to the pattern of kidney tubule lesions. Briefly, CD-1 mice (50/dose group for both sexes) were administered 99.8% pure glyphosate (161, 835, and 4,945 mg/Kg/day for male mice and 195, 968, and 6,069 mg/Kg/day for female mice) in the diet for 24 months. Results of this study revealed an 11% loss in body weight of the male animals in the highest dose group compared to controls. In addition, this study revealed that glyphosate treatment had no statistically significant effect on survival; however, kidney adenomas were found in 0/49, 0/49, 1/50, and 3/50 male mice. A reanalysis of

the tissue blocks identified one additional adenoma in the control group changing the dose response to 1/49, 0/49, 1/50 and 3/50.

Since it is typical of large cancer bioassays to only grossly examine the whole organ and to cut a single paraffin section for histopathological analysis, it is possible that even more tumors could be identified and further insight gained from examination of additional tissue sections in all four dose groups. Therefore, additional sections were cut for histopathological assessment and further insight was sought. Subsequently, a Pathology Working Group (PWG) met to review the kidney adenoma and carcinoma data. It is noteworthy that the PWG unanimously agreed that the control mouse tumor was present and should be considered. As Dr. Portier notes, with the tumor in the control mouse included there are no statistically significant differences. Moreover, I note Dr. Portier's statistical analysis indicated that none of the other mouse cancer bioassays to assess the carcinogenicity of glyphosate generated statistically significant incidences of compound-related kidney tumors using the guideline Fisher test or Cochran-Armitage trend analysis. The high dose of glyphosate used gives me confidence that that the absence of significant changes in kidney tumors is a genuine negative finding.

From my review of the data and subsequent analyses together with a review of the relevant correspondence, I conclude that the absence of a dose response together with lack of evidence of tumor progression supports the conclusion that these tumors are spontaneously occurring and unrelated to treatment. Drs. Neugut and Jameson use the *p* trend from the IARC assessment to justify their conclusions of a statistically significant trend yet Dr. Portier disagrees with this conclusion. Moreover, the dose levels employed were at or near the limit dose for the medium dose group whereas in the highest dose group they are 5 times above the limit dose for the male high dose group. While an 11% loss in body weight in the highest dose group of males was detected no compound-related tumors were demonstrated. Finally, the step sectioning employed makes it even more unlikely that the tumors were a compound-related effect, as extra sections failed to identify any new tumors in any dose group.

Atkinson, 1993b – (discussed in Greim et al., 2015 - Study 11): In this carcinogenicity study, 50 mice/dose group of each sex were administered 97% pure glyphosate (98, 297, and 988 mg/Kg/day for males and 102, 298, and 1,000 mg/Kg/day for females) in the diet for 24 months. No pre-neoplastic or related neoplastic lesions were detected. Incidental pituitary adenomas were noted and there was a non-significant increase in bronchioalveolar adenomas of the males only. Dr. Portier suggests that some of these tumors are marginally significant. However, as previously noted in my report, the concept of marginal significance would not typically be accepted in any credible scientific publication. There was a significant trend in hemangiosarcomas with 4/45 (8.9%) lesions found in the high dose group of males only. While these tumors are rare in humans (Weiss 2008) they are common neoplasms of mice (Elwell 2004), suggesting a potential different underlying mechanism for their development in mice compared to humans. Also note that the two-year hemangiosarcoma incidence data reported for historical controls in CD-1 mice ranges up to 12% (Giknis 2000). This incidence is well within the range of the historical controls

cited by Dr. Portier[3]. Moreover, since these tumors were not detected in a statistically significant trend in male mice in other appropriately conducted cancer bioassays, the lack of replication weighs against considering these tumors to be treatment related. Finally, I note that kidney adenomas in this study were found in a dose pattern of 2, 2, 0, and 0. This pattern contrasts with that reported previously in mice (Knezevich and Hogan, 1983) with a pattern of 1, 0, 1, and 3. These conflicting data further illustrate the lack of consistency of data across studies and further indicate the lack of compound-related effects. This is a strong study owing to the study design, and the dose levels used, including a dose representative of the limit dose, and absence of confounding systemic toxicity. Hence, this study is considered a negative study.

Sugimoto, 1997 (discussed by Greim et al., 2015 - Study 12): A chronic study in ICR-CD-1 mice (50/dose group) treated with 97.6% pure Glyphosate (165, 838, and 4,438 in mg/kg/day in males and 153, 787, and 4,116 mg/Kg/day for female animals) in the diet for 18 months was performed consistent with an OECD carcinogenicity TG. In this study, malignant lymphomas were reported 2/50, 2/50, 0/50, and 6/50. The absence of a dose response, lack of statistical significance by Fisher pair wise comparison, and absence of compound-related effects on lymphomas in other well conducted mouse cancer bioassays lead me to conclude that these tumors are not compound-related. Furthermore, the incidence of these tumors falls within the range of historical controls in the Giknis (2000) report (0-14%) cited by Dr. Portier and the range of historical controls (3-19%) from contemporaneous studies conducted at the same laboratory (BFR, 2015).

A statistically significant increased incidence of hemangiomas (a non-malignant tumor) was found in the female mice only with a pattern of 0/50, 0/50, 2/50, and 5/50. However, effects seen at the high dose are potentially confounded by signs of systemic toxicity as revealed by liquid stool, retarded growth, and reduced food consumption (Greim et al. 2015). These are non-malignant lesions of unknown cause that generally appear early in life. The absence of evidence of tumor progression in an 18-month study is reassuring that glyphosate is not carcinogenic. In addition, I note that there were no compound-related hemangiomas found in other well designed carcinogenicity bioassays. Thus, potential compound-related effects in the high dose group of this study are considered less reliable.

In summary, this study has numerous strengths including study design including multiple dose groups, dose range and duration of the study and is therefore regarded as a strongly negative study.

[3]Dr. Portier appears to confuse historical control data for "whole body" hemangiosarcomas as capturing all hemangiosarcomas. Thus, he does not count hemangiosarcomas reported in the Giknis data at other sites, like the liver. In fact, hemangiosarcomas are a vascular tumor that appears throughout the body and thus it may be best to report these tumors as the number of mice with these tumors regardless of site.

Wood et al., 2009b – (discussed in Greim et al., 2015 - Study 14): In this chronic toxicity study, CD-1 mice (50/sex/treatment group) were given free access to food containing 95.7% pure glyphosate (71, 234, and 810 mg/Kg/day for male mice and 97.9, 300, and 1,081 mg/kg/day for female mice) for 80 weeks. Lung tumors and malignant lymphomas were detected in the male mice. While Dr. Portier argues that rodent and human NHLs are similar, it is unclear what he means. Specifically, are they similar in the biological processes of their origin, their pathobiology, incidence, progression, or ultimate impact on health? Contrary to Dr. Portier's suggestion, clear differences in the biological development of lymphomas in rodents and humans have been described (Morse, 2003) leading me to question whether the connection between lymphoma in mice and NHL in humans can be definitively established. Consequently, the published evidence, along with the known difficulty of directly extrapolating animal findings to humans, suggests that these tumors cannot be considered similar as suggested by Dr. Portier.

In this study, there was a statistically significant increase in the trend for the incidence of malignant lymphomas in the male mice. Historical controls from the same lab were available. The historical background incidence was 12% in an 18-month study (SafePharm) and thus the incidence in the high dose group in the current study is consistent with historical controls. Furthermore, lymphomas are common tumors in mice (Haseman et al. 1998; Ward 2006). The historical control data cited by Dr. Portier (Giknis and Clifford, 2005) indicate that it is unusual to have zero lymphomas in a control group. This observation, together with the absence of malignant lymphomas reported for the control group of the current study and considering the historical control rate of 12% in the same laboratory, suggests that the finding of statistically significant differences is most likely a statistical artifact. Therefore, I conclude that there were no treatment-related tumors in this study.

Treatment had no effect on lung adenomas but a trend towards an increase in lung adenocarcinomas was detected although statistical significance could not be demonstrated for the high dose group. The lack of agreement between the Fisher's comparison test and assessment of a trend is less convincing than if both are significant and prompts further analysis. Furthermore, the combined incidence of lung tumors (14/51, 12/51, 16/51, and 15/51) in this study lacks any evidence of a dose response and statistically significant changes could not be demonstrated. Moreover, absence of evidence of preneoplastic changes and tumor progression was noted indicating lack of compound-related effects. Dr. Portier speculates that tumors could arise by alternative mechanisms that do not involve pre-neoplastic lesions but fails to provide any citation to support his opinion. This notion runs contrary to widely accepted understanding of tumor pathobiology and thus is speculative at best. Therefore, I conclude that these are spontaneously occurring lesions unrelated to treatment.

This study has numerous strengths including study design including multiple dose groups, dose range, and number of animals/dose group. The 80-week duration of the study is shorter than the 24 months used for rat studies but is consistent with regulatory requirements for this strain of mice. Consequently, this study is regarded as a negative study.

Kumar et al., 2001 – (discussed in Greim et al., 2015 - Study 13): This study was conducted in Swiss Albino mice and failed to show evidence of morphological abnormalities although there was a statistically significant pairwise increase in the number of malignant lymphomas seen in the highest dose group; however, a statistically significant trend was not detected.

This study was noted to be of questionable value in a prior review (Greim et al., 2015) owing to a possible viral infection amongst the study animals. I note that this study was excluded from consideration by EPA and Dr. Jameson but that Dr. Portier considered this study. Furthermore, in Dr. Portier's revised report (p. 43), he cites a recent memo from Martens (2017) asserting that the incidences for malignant lymphomas and kidney adenomas described in Greim et al., (2015) and BFR (2013) are incorrect. I am troubled by the potential existence of multiple data sets for the same study and the lack of explanation or evidence of data verification. Consequently, the existence of two different data sets and lack of data validation together with the questionable value of this study owing to the potential viral infection leads me to conclude that this study is unreliable and thus was excluded from my analysis.

Synthesis of mouse studies

In summary, a statistically significant trend towards an increase in incidental tumors in varying tissues was reported in four studies (Knezevich and Hogan, 1983; Atkinson, 1993b; Wood et al., 2009b; and Sugimoto, 1997). However, looking beyond a simple statistical analysis of the data to include a comparison of incidence with concurrent controls, historical controls or the relevant literature suggests that these tumors did not depart from normal variation for the individual tumor types. The lack of consistent evidence of dose response further indicates that glyphosate is not carcinogenic. Furthermore, the lack of evidence for tumor progression and absence of statistically significant effects when benign and malignant tumors were combined adds further strength to the conclusion that any effects were not compound-related. I also found that there was no replication of potential compound-related effects across these studies.

I note that these studies are highly relevant and were conducted consistent with well-established test guidelines. The conclusions in the current report diverge from those of Dr. Portier. Reasons for our divergent conclusions can in part be explained by differences in the methodology and analyses carried out. Specifically, in the current assessment of the data, I assessed the overall scientific quality of the study in addition to evaluating neoplastic changes in the context of the totality of scientific knowledge. My analysis extends beyond that of Dr. Portier who limits discussion to a statistical analysis of the data only, utilizing novel statistical procedures without

scientific validation supported only by unreferenced speculation about potential causes. The absence of compound-related tumor findings in each of these individual negative studies indicates that in total glyphosate is not carcinogenic. Therefore, I conclude that there is no scientifically reliable evidence of glyphosate carcinogenicity in mice.

Synthesis of animal data

My review of the animal literature on the potential carcinogenicity of glyphosate revealed a robust data set. These studies were conducted over a wide span of time including introduction of regulatory test guidelines for carcinogenicity testing. I note that the majority of the studies reviewed followed regulatory guidelines for or were consistent with carcinogenicity testing guideline requirements.

Dr. Portier in his report argues (p. 52) that you cannot compare data across animal studies if you also argue that the data cannot be combined in a pooled analysis of the kind he carries out. However, it is a very different thing to look at results across studies you know are similar but have some important differences as opposed to treating them as if they are one big study suitable for combining in an unvalidated pooled analysis. Specifically, important differences between studies include a) different time periods, b) different labs, c) different doses, d) different rodent stocks, and e) known genetic drift. Animal studies are designed to control for or minimize the effect of other sources of variation that would confound the detection of a compound-related effect within the study. However, if you combine the data from multiple animal studies then you cannot exclude the influence of the above noted variables on the outcomes of interest (e.g. tumors). If you have a bias in time going one way (i.e. an increase in stomach tumors in that particular strain from 1970 – 2000), then that might produce significance when in fact there is no compound-related effect. Similarly, if there is a trend towards a decrease in incidence for a particular tumor over time, then combining data would increase the odds of not detecting tumors when they were in fact present. It is routine for toxicologists to compare the results of studies whereas combining studies in a single statistical analysis is not the current scientific standard. Although all studies detected tumors and some detected statistically significant increases of tumors, it is my opinion that the occurrences of these tumors were, within a reasonable degree of scientific certainty, incidental findings unrelated to glyphosate treatment. Glyphosate does not induce rodent tumors and thus, animal carcinogenicity experiments do not support the hypothesis that glyphosate poses a ***health risk to humans***. The basis for my conclusions arise from multiple lines of consideration including the lack of consistency of the data across studies, weakness of the reported associations, lack of dose response characteristics, and absence of biological plausibility. Details of my considerations are described in the following paragraphs.

The animal studies reviewed were conducted according to or consistent with standardized and internationally recognized carcinogenicity test guidelines. Test guidelines are designed to produce robust and reproducible data. However, in the studies that I reviewed, I found no

consistency of tumor findings across the studies and tumors identified in the individual studies are, in my opinion, nothing more than statistical anomalies arising from multiple comparisons, unusually low prevalence of tumors in the control groups for common tumor types, decreased survival of control animals relative to treated animals, or evidence of systemic toxicity in the high dose group. Thus, there is a lack of consistency for any association of glyphosate exposure and tumor development. It was noted that a statistically significant trend was detected for hemangiosarcomas, a relatively common neoplasm in mice (Elwell 2004), in one mouse study (Atkinson, 1993) and was not replicated in other mouse studies. If glyphosate was acting as a carcinogen then I would expect the data to be reproducible with neoplastic changes in one study being replicated by other carefully conducted studies as previously described (Gold et al. 1987; Gold et al. 1989). Although there may be small variations between animals and studies, the mechanism of compound action is expected to remain constant and thus tumors in a given target tissue should be reproducible across animal studies. Indeed, I agree with Dr. Portier when he states that replication of studies "is critical in most scientific debates." (p. 5). Furthermore, I note that these regulatory studies typically assessed a broad spectrum of outcome measures including histopathological assessment of major organs. Therefore, as designed the regulatory studies favor the detection of false positive adverse outcomes (finding non-treatment related tumors) in preference to false negatives (missing detection of a treatment-induced tumor). In view of the lack of reproducibility of the tumor data between studies I conclude that these tumors are not compound-related. This view is further supported by similarity of incidence with concurrent and historical controls. While I appreciate that it is best to compare results with contemporaneous historical controls, this is not always possible especially with retrospective data. However, I note that the incidence of the tumors is generally in agreement with the published literature on tumor incidence in rats and mice (Chandra et al., 1992; Davis et al., 1956; Haseman et al., 1998; Majeed 1997; Prejean et al., 1973; Ward 2006; Giknis 2000; and Giknis and Clifford, 2011) providing further assurance that the observed tumor findings are spontaneously occurring and not attributable to glyphosate treatment. Thus, I conclude that the observed tumors are false positives and cannot be attributed to glyphosate treatment.

In addition to the lack of consistency across the studies, I further note that there is a general lack of a dose response for the tumor data. The dose response patterns reported in the reviewed studies can be characterized as inconsistent and in most cases statistically non-significant. Thus, the statistically significant dose-response curves when observed were of questionable biological relevance. It is important to acknowledge that, in almost all the cases, when tumor incidence became an observation of interest, it was almost exclusively a result of increased incidence in the highest dose group. Several studies used doses that were slightly to well above the limit dose (Brammer, 2001; Wood et al., 2009a; Knezevich and Hogan, 1983; Sugimoto, 1997). While this is acceptable when dosing through the diet, it remains important to ensure that the dose used is not inducing systemic toxicity that could confound interpretation of the study results. Moreover, the translational importance of potential adverse findings in these studies to humans is questionable. Therefore, I conclude that there is no scientifically reliable evidence from the studies reviewed that glyphosate treatment, under the conditions of these animal studies, can be considered carcinogenic.

In the context of assessing the animal carcinogenicity of glyphosate, I also assessed the biological plausibility that glyphosate is carcinogenic. Specifically, carcinogenesis is widely acknowledged to be a multistep process involving tumor initiation, promotion, and progression. Central to the process of carcinogenesis is the process of tumor initiation and tumor progression arising from genotoxic effects of a test chemical or its metabolites. While my task was to assess the animal literature, I note that the genotoxicity studies with glyphosate were both numerous and almost all (98%) were negative (Greim et al., 2015).

I further searched the literature for any evidence that glyphosate could act as a tumor promoter. One study suggested that glyphosate could act as a tumor promoter in a skin carcinogenicity test (George et al. 2010); however, any such mechanism remains elusive. While plaintiffs' expert Dr. Portier cites the preceding study as evidence of glyphosate's potential to act as a tumor promoter, this study has numerous shortcomings including but not limited to: no positive vehicle control, no defined source of substances or purity, low numbers of animals, and lack of pathological analysis. Therefore, I conclude that this poorly defined study does not provide convincing (or reliable) support for the notion that glyphosate acts as a tumor promoter.

From my review of the literature, I conclude that the carcinogenicity of glyphosate has been thoroughly evaluated according to validated and internationally recognized carcinogenicity test guidelines. Dr. Portier is correct when he notes, "it is clear that not every tumor shows a positive trend with glyphosate exposure." (pg. 46). In fact, the vast majority do not. Multiple well designed and executed studies conducted in rats and mice reveal that tumors are not more common in glyphosate treated animals than in the concurrent control groups. When perceived differences in tumor incidence were detected, careful evaluation of animal survival, body weight, historical control incidence, dose-response, and assessment for signs of tumor progression showed that tumors did not occur with higher incidence in the treatment groups compared to controls and thus glyphosate was not carcinogenic. Assessment of animal survival and body weight, as well as comparison with historical controls and histopathological assessment of proliferation and tumor progression are standard and expected procedures carried out routinely by scientists in regulatory, government, and academic laboratories worldwide. Thus, as an end-point, carcinogenesis does not meet the standard required for the establishment of a no effect level in the hazard identification step of the risk assessment process. Moreover, the lack of consistency of effects across well designed regulatory carcinogenicity studies, weakness of the evidence for any association in the animal studies, flat or non-linear dose-response, and absence of credible or reproducible evidence for biological plausibility further add confidence to the conclusion that glyphosate is not a carcinogen, a conclusion that is consistent with independent reviews from multiple expert government regulatory panels from around the globe (EFSA, US-EPA, BFR etc.).

In summary, I conclude that, on the basis of multiple carcinogenicity studies conducted in rats and mice, there is no scientifically reliable evidence of glyphosate-induced increased tumor incidence. I disagree with the conclusions of Drs. Portier, Jameson, Weisenburger, Neugut, and Nabhan that any of the individual glyphosate rodent studies discussed in their reports demonstrates that glyphosate is a rodent carcinogen, let alone that there are any replicated findings of tumor-related effects. Accordingly, I disagree with Dr. Portier's statement that in the rodent carcinogenicity studies "there is clear evidence of a biological gradient" (Portier report at p. 75). Moreover, any extrapolation of the incidental tumors found (all of which are unrelated to glyphosate) to humans is scientifically implausible given the differences in relevant dosing, as well as the translational challenges in extrapolating from animals to humans. Additionally, pooling of these studies is an unproven approach that makes no toxicological sense. The hallmark signs of carcinogenicity are absent from the robust glyphosate data set. The glyphosate data does not demonstrate: a dose-response relationship between glyphosate exposure and tumors; tumor progression (neoplastic continuum); or a biologically plausible mechanism of action. Moreover tumor incidences are generally what one would expect when considering pairwise and historical controls, and the expected number of positive findings due to chance alone. The absence of glyphosate-induced tumor incidence in any one study, given the large number of studies, is compelling evidence that glyphosate is not carcinogenic. With such a robust data set, if glyphosate were carcinogenic, one would expect to see evidence of carcinogenicity not only in an individual study, but replication of carcinogenic findings in a particular tumor type across multiple studies. Accordingly, I conclude that, within a reasonable degree of scientific certainty, glyphosate is not a rodent carcinogen.

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110. Letter from Bernhard Url, Exec. Dir. EFSA, to Christopher Portier, Sr. Contributing Scientist EDF (Jan. 13, 2016) (Available at https://www.efsa.europa.eu/sites/default/files/EFSA_response_Prof_Portier.pdf).

111. Letter from Christopher Portier, Sr. Contributing Scientist EDF, to Vytenis Andriukaitis, Comm'r Health and Food Safety, European Comm'n (Nov. 27, 2015) (Available at https://www.efsa.europa.eu/sites/default/files/Prof_Portier_letter.pdf)

112. Letter from Frank S. Serdy, Manager, Federal and State Registration Affairs, Monsanto Co. on Resectioning of Kidneys – Glyphosate Chronic Mouse Study (BD-77-420; EPA Registration Number 524-308) to the Director, Registration Division, Office of Pesticide Programs, U.S. EPA (Aug. 7, 1985).

113. Letter from Frank S. Serdy, Manager, Federal and State Registration Affairs, Monsanto Co. on Roundup® Herbicide EPA Reg. Nos. 524-308, 524-330, 524-339, 524-332, 524-343 Chronic Mouse Study With Glyphosate to Douglas D. Campt, Director, Registration Division, Office of Pesticide Programs, U.S. EPA (Mar. 13, 1985).

114. Letter from Frank S. Serdy, Manager, Federal and State Registration Affairs, Monsanto Co. on Roundup Herbicide EPA Reg. No. 524-308 Additional Information Relating to Chronic Mouse Study, BD-77-420 to the Director, Registration Division, Office of Pesticide Programs, U.S. EPA (Feb. 5, 1985).

115. Letter from Frank S. Serdy, Manager, Federal and State Registration Affairs, Monsanto Co. on Roundup® Herbicide EPA Reg Nos. 524-308, 524-330, 524-332, 524-339, 524-343. Chronic Mouse Study with Glyphosate to the Director, Registration Division, Office of Pesticide Programs, U.S. EPA (May 21, 1985).

116. Letter from George B. Fuller, U.S. and International Registration Director, Monsanto Co. on Glyphosate Registration Standard: Request for Meeting with EPA to the Director, Registration Division, Office of Pesticide Programs, U.S. EPA (Oct. 5, 1988).

117. Letter from Klaus Stemmer, M.D., Institute of Environmental Health, Univ. of Cincinnati Medical Center on Roundup® Chronic Mouse Study with Glyphosate to Timothy Long, Ph.D., Senior Product Toxicologist, Monsanto Co. (Oct. 17, 1985).

118. Letter from Marvin Kuschner, M.D. on mouse kidney slides to Timothy J. Long, Ph. D., Senior Product Toxicologist, Monsanto Co. (May 11, 1985).

119. Letter from Marvin Kuschner, M.D., Dean, School of Medicine, SUNY Stony Brook on kidney adenomas found in mice exposed to glyphosate to Timothy Long, Ph.D., Senior Product Toxicologist, Monsanto Co. (Oct. 3, 1985).

120. Letter from Robert A. Squire, D.V.M., Ph.D. on chronic toxicity data on Glyphosate in mice to Timothy Long, Ph.D., Senior Toxicologist, Monsanto Co. (Sept. 29, 1985).

121. Letter from Robert Olson, M.D., Ph.D. Professor of Medicine, Professor of Pharmacological Sciences, SUNY Stony Brook on glyphosate mouse kidney adenoma study to Timothy Long, Ph.D., Senior Product Toxicologist, Department of Medicine & Environmental Health, Monsanto Co. (Oct. 7, 1985).

122. Letter from Robert W. Street, Manager, Product Health and Safety Information, Monsanto Co. on Roundup® Herbicide, EPA Reg. No. 524-308, Additional Information Relating to Chronic Mouse Study BD-77-420 to the Director, Registration Division, Office of Pesticide Programs, U.S. EPA (Mar. 20, 1984).

123. Letter from Thomas F. Armstrong, Registration Manager, Monsanto Co. on Roundup® Herbicide EPA Reg. Nos. 524-308, 524-330, 524-332, 524-339, 524-343, 524-351 Addendum to Chronic Mouse Study with Glyphosate: Additional Evaluations to the Director, Registration Division, Office of Pesticide Programs, U.S. EPA (Oct. 28, 1985).

124. Levine, S. et al., *Disrupting mitochondrial function with surfactants inhibits MA-10 Leydig cell steroidogenesis*, 23 Cell Biology Toxicology 385 (2007).

125. Li, Q. et al., *Glyphosate and AMPA inhibit cancer cell growth through inhibiting intracellular glycine synthesis*, 7 Drug Des. Devel. Ther. 635 (2013).

126. Lin, N. and V. Garry, *In vitro studies of cellular and molecular developmental toxicity of adjuvants, herbicides, and fungicides commonly used in Red River Valley, Minnesota*, 60 J. Toxicol. Environ. Health A 423 (2000).

127. Majeed, S., *Studies of the incidence of spontaneous pancreatic tumours in ageing cd rats*, 47 Arzneimittelforschung 879 (1997).

128. McConnell, E. et al., *Guidelines for Combining Neoplasms for Evaluation of Rodent Carcinogenesis Studies*, 76 Journal of the National Cancer Institute 283 (1986).

129. McConnell, R., *A Chronic Feeding Study of Glyphosate (Roundup® Technical in Mice): Pathology Report on Additional Kidney Sections Addendum to Final Report Dated July 21, 1983* (Sept. 26, 1985).

130. Mecchi, M., *Mutagenicity Test with MON 59117 in the Salmonella – Escherichia Coli/Mammalian-Microsome Reverse Mutation Assay with a Confirmatory Assay* (June 23, 2000).

131. Memorandum from R.W. Street, Manager, Product Health and Safety Information, Monsanto Co. on *Chronic Rat Study with Glyphosate BD-77-416* to Arthur Chin (Mar. 27, 1985).

132. Mink, P. et al., *Epidemiologic studies of glyphosate and cancer: a review*, 63 Regul. Toxicol. Pharmacol. 440 (2012).

133. Mink, P. et al., *Epidemiologic studies of glyphosate and non-cancer health outcomes: a review*, 61 Regul. Toxicol. Pharmacol. 172 (2011).

134. Morse, H. et al., *B Lymphoid Neoplasms of Mice: Characteristics of Naturally Occurring and Engineered Diseases and Relationships to Human Disorders*, 81 Advances in Immunology 97 (2003).

135. Morse, H. et al., *Mouse models of human B lymphoid neoplasms, in* The Lymphoid Neoplasms 281 (Ian T. Magrath et al. eds., 3rd ed. 2010).

136. Murli, H., *Chromosomal Aberrations in Cultured Human Peripheral Blood Lymphocytes wit MON 59117* (Apr. 28, 2000).

137. Murli, H., *Measuring Chromosome Aberrations in Human Whole Blood Lymphocytes With and Without Exogenous Metabolic Activation With a Confirmatory Assay With Multiple Harvests With MON 58121*, (Sept. 3, 1997).

138. Myers, J. et al., *Concerns over use of glyphosate-based herbicides and risks associated with exposures: a consensus statement*, 15 Environmental Health 19 (2016).

139. Myhr, B., *In Vivo Mouse Micronucleus Assay with MON 59117* (May 31, 2000).

140. Myhr, B., *Mutagenicity Test on MON 59112 in the In Vivo Mouse Micronucleus Assay* (May 31, 2000).

141. NTP, *Technical Report on the Toxicology and Carcinogenesis Studies of Sodium Dichromate Dihydrate (CAS No. 7789-12-0) in F334/N Rats and B6C3F1 Mice*, National Institutes of Health (2008).

142. OECD, *Carcinogenicity Studies, OECD Guideline for the Testing of Chemicals, No. 451*, OECD (2009).

143. OECD, *Combined Chronic Toxicity\Carcinogenicity Studies, OECD Guidelines for Testing of Chemicals, No. 453*, OECD (2009).

144. OECD, *Guidance Document 116 on the Conduct and Design of Chronic Toxicity and Carcinogenicity Studies, Supporting Test Guidelines 451, 452 and 453*, OECD (2012).

145. Parajuli, K. et al., *Aminomethylphosphonic acid and methoxyacetic acid induce apoptosis in prostate cancer cells*, 16 Int. J. Mol. Sci. 11750 (2015).

146. Parajuli, K. et al., *Aminomethylphosphonic acid inhibits growth and metastasis of human prostate cancer in an orthotopic xenograft mouse model*, 7 Oncotarget 10616 (2016).

147. Pattengale, P. & C. Taylor, *Experimental Models of Lymphoproliferative Disease: The Mouse as a Model for Human Non-Hodgkin's Lymphomas and Related Leukemias*, 113 American J. Pathology 237 (1983).

148. Portier, C. & D. Hoel, *Optimal design of the chronic animal bioassay*, 12(1) J Toxicology Envtl. Health 1 (1983).

149. Portier, C. et al., *Age-specific models of mortality and tumor onset for historical control animals in the National Toxicology Program's carcinogenicity experiments*. 46 Cancer Res. 4372 (1986).

150. Portier, C. et al., *Differences in the carcinogenic evaluation of glyphosate between the International Agency for Research on Cancer (IARC) and the European Food Safety Authority (EFSA)*. 70 J Epidemiology Community Health 741 (2016).

151. Portier, C., *Comments of C. Portier on USEPA (EPA-HQ-OPP-2016-0385-0094)*, Regulations.gov (Oct. 4, 2016) https://www.regulations.gov/document?D=EPA-HQ-OPP-2016-0385-0371.

152. Prejean, J. et al., *Spontaneous tumors in Sprague-dawley rats and swiss mice*, 33 Cancer Res. 2768 (1973).

153. Sauer, R., *Pathology Working Group Report on Glyphosate in CD-1 Mice* (Oct. 10, 1985).

154. Schinasi, L. and M. Leon, *Non-Hodgkin lymphoma and occupational exposure to agricultural pesticide chemical groups and active ingredients: a systematic review and meta-analysis*, 11 Int. J. Environ. Res. Public Health 4449 (2014).

155. Seralini, G. et al., *Answers to critics: Why there is a long term toxicity due to a Roundup-tolerant genetically modified maize and to a Roundup herbicide*, 53 Food and Chemical Toxicology 476 (2013).

156. Seralini, G. et al., *Conflicts of interests, confidentiality and censorship in health risk assessment: the example of an herbicide and a GMO*, 26 Environ. Sci. Eur. 13 (2014).

157. Seralini, G. et al., *Long term toxicity of a Roundup herbicide and a Roundup-tolerant genetically modified maize*, 50 Food and Chemical Toxicology 4221 (2012).

158. Seralini, G. et al., *Republished study: long-term toxicity of a Roundup herbicide and a Roundup-tolerant genetically modified maize*, 26 Envtl. Scis. Eur. 14 (2014).

159. Smith, M. et al., *Key characteristics of carcinogens as a basis for organizing data on mechanisms of carcinogenesis*, 124 Envtl. Health Persp. 713 (2016).

160. Solomon, K., *Glyphosate in the general population and in applicators: a critical review of studies on exposures*, 46 Critical Reviews in Toxicology 21 (2016).

161. Son, W. & C. Gopinath, *Early occurrence of spontaneous tumors in CD-1 mice and Sprague-Dawley rats*, 32 Toxicologic Pathology 371 (2004).

162. Sorahan, T., *Multiple myeloma and glyphosate use: a re-analysis of US Agricultural Health Study (AHS) data*, 12 Int. J. Environ. Res. Public Health 1548 (2015).

163. Squire, R., *The Interpretation of Equivocal Or Marginal Animal Carcinogenicity Tests*, 5 Cell Biology and Toxicology 371 (1989).

164. Stankowski, L., *Ames/Salmonella-E. coli Reverse Mutation Assay on Test Article 6933-22-20* (July 02, 1996).

165. Stegeman, S. & A. Li, *Ames/Salmonella Mutagenicity Assay of MON 0818* (Oct. 26, 1990).

166. Stegeman, S. & L. Kier, *Mouse Micronucleus Screening Assay of MON-0818* (Mar. 26, 1998).

167. Stout, L. & F. Ruecker, *Chronic Study of Glyphosate Administered in Feed to Albino Rats* (Sept. 26, 1990).

168. Sugimoto, Y et al., *Failure of parturition in mice lacking the prostaglandin f receptor*, 277 Sci. 681 (1997).

169. Tarazona, J. et al., *Glyphosate toxicity and carcinogenicity: a review of the scientific basis of the European Union assessment and its differences with IARC,* Archives of Toxicology (2017).

170. Tarone, R., *On the International Agency for Research on Cancer classification of glyphosate as a probable human carcinogen*, European Journal of Cancer Prevention (2016).

171. Thongprakaisang, S et al., *Glyphosate induces human breast cancer cells growth via estrogen receptors*, 59 Food & Chem. Toxicology 129 (2013).

172. *Tier II Summaries for Glyphosate Carcinogenicity Studies from Greim et al., 2015,* (Nov. 9, 2015).

173. Tuck, M. et al., *Standard operating procedures for serum and plasma collection: Early detection research network consensus statement standard operating procedure integration working group*, 8 JProteomeRes 113 (2009).

174. Uno, H., et al., *A versatile test for equality of two survival functions based on weighted differences of Kaplan-Meier curves*, 34 Statistics In Medicine 3680 (2015).

175. Vandenberg, L. et al., *Is it time to reassess current safety standards for glyphosate-based herbicides?*, 71 J. Epidemiol. Community Health 613 (2017).

176. Videotaped Deposition of Charles W. Jameson, In re: Roundup Products Liability Litigation, Case No. 16-md-02741-VC (N.D. Cal. May 3, 2017) (with all exhibits).

177. Ward, J., *Lymphomas and leukemias in mice*, 57 Experimental & Toxicology Pathology 377 (2006).

178. Weber, K., *Statistical Evaluation of Pre-Neoplastic and Neoplastic Lesions from Study: Study No. TOXI: 1559.CARCI-M Carcinogenicity Study with Glyphosate Technical in Swiss Albino Mice* (Jan. 23, 2017).

179. Weiss, S. & J. Goldblum, *Malignant vascular tumors, in* Ezinger and Weiss's Soft Tissue Tumors 703 (5th ed. 2008).

180. Wester, R. et al., *Glyphosate Skin Binding, Absorption, Residual Tissue Distribution, and Skin Decontamination*, 16 Fundamental and Applied Toxicology 725 (1991).

181. Wester, R. et al., *In Vitro Percutaneous Absorption of Model Compounds Glyphosate and Malathion from Cotton Fabric into and through Human Skin*, 34 Food and Chemical Toxicology 731 (1996).

182. Williams, G. et al., *A review of the carcinogenic potential of glyphosate by four independent expert panels and comparison to the IARC assessment*, 43 Critical Revs. Toxicology 3 (2016).

183. Williams, G. et al., *Glyphosate rodent carcinogenicity bioassay expert panel review*, 46 Critical Reviews in Toxicology 44 (2016).

184. Williams, G. et al., *Safety Evaluation and Risk Assessment of the Herbicide Roundup and Its Active Ingredient, Glyphosate, for Humans*, 31 Regulatory Toxicology and Pharmacology 117 (2000).

185. Wood, E. et al., *Observations on the Development of Spontaneous Neoplasms in Male and in Female Crl: CD-1 (ICR) CR Strain Mice Following 18-Months on Control Diet* (July 24, 2008).

186. Wratten, S. et al., *MSL 0025540: Amended Report updating MSL 0023134 A Review and Discussion of Glyphosate Toxicology Test Materials* (Mar. 04, 2014).

7.0 Summary of qualifications:

Curriculum Vitae

WARREN G. FOSTER, Ph.D., FCAHS
July 31, 2017

PERSONAL:

Business Address:
McMaster University
Department of Obstetrics & Gynecology
1280 Main Street West, HSC-3N52D
Hamilton, Ontario, Canada
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E-mail: [redacted]

Citizenship: [redacted]

EDUCATION:

1991	Ph.D.	McMaster University, Hamilton, ON	Health Sciences
1986	M.Sc.	University of Guelph, Guelph, ON	Biomedical Sciences
1979	B.Sc.(Hon.)	University of Guelph, Guelph, ON	Human Biology

MEMBERSHIP IN PROFESSIONAL SOCIETIES:

1. Society of Toxicology, 1999 – present.
    - Councilor, Lake Ontario Regional Chapter, 2016 – present.
    - Past President, Lake Ontario Regional Chapter, 2014 – 2015.
    - President, Lake Ontario Regional Chapter, 2012 – 2014.
    - Secretary Treasurer, Reproductive Developmental Toxicology Specialty Section, 2010 – 2012.
    - Member, Scientific Program Committee, 2010 – 2012.

2. Society for the Study of Reproduction, 1994 – present.
    - Member, Membership Committee, 2012 – 2015.
    - Member, Scientific Program Committee, 2010 – 2012.
    - Chair, Committee on Reproduction and the Environment (CoRE), 2010 – 2012.
    - Member, Committee on Reproduction and the Environment (CoRE), 2009 – 2012.
    - Member, Animal Care Committee, 1995.

3. The Canadian Fertility and Andrology Society, 1991 – present.
    - Member, Scientific Program Committee, 2007 – 2011.
    - Past President 2008 – 2009.
    - Chair, Scientific Program Committee, 2007 – 2008.
    - President 2007 – 2008.
    - Vice-President, 2006 – 2007.
    - Industrial Liaison Committee, 2005 – 2006.

4. World Endometriosis Society, 2010 – present.
    - Member, Scientific Organizing Committee, World Congress of Endometriosis, Vancouver BC, 2012 – present.

5. European Society for Human Reproduction and Embryology, 2007 – present.

6. American Society for Reproductive Medicine, 2004 – present.

7. Association of Professors of Obstetrics & Gynaecology, 2003 – present.

8. Society of Obstetricians & Gynaecologists of Canada, 2001 – present.

9. Society of Toxicology of Canada, 1992 – present.
    - Vice-President (President Elect), 1998 – 1999.
    - Chairperson, Scientific Program Committee, 1997.
    - Member, Scientific Program Committee, 1996.

10. American Association for the Advancement of Science, 1988 – present.

11. International Federation of Placenta Associations, 2007.
    - Abstract Reviewer, 2007.

HONOURS AND AWARDS:

1. Elected to Fellowship in the Canadian Academy of Health Sciences, 2016, with citation for "Demonstrated leadership, creativity, distinctive competencies and commitment to advance academic health sciences." *The Canadian Academy of Health Sciences recognizes the full breadth of academic health science including all of the medical and allied health sciences and ranging from fundamental science to social science and population health. Members elected to the Academy will be well recognized by their peers nationally and internationally for their contributions to the promotion of health science and will have demonstrated leadership, creativity, distinctive competencies and commitment to advance academic health sciences. Such individuals are elected to the organization after a nominating and rigorous peer review procedure, which seeks to recognize those who are marked by a record of substantial accomplishment. Election to the Academy is considered one of the highest honours for members of the Canadian health sciences community and carries with it a covenant to serve the Academy and the future well-being of the health sciences irrespective of the member's specific discipline.*

2. Award of Excellence in Reproductive Medicine, 2016. *An honour reserved for members of the Canadian Fertility and Andrology Society for an outstanding contribution to, and leadership in, the field of Reproductive Medicine and Science. The recipient must demonstrate tremendous dedication to advancing the field in Reproductive Medicine and Science in research, clinical activity, teaching and consulting activities. Recognized as an innovator and scholar by peers—as shown by invitations to participate in professional societies and associations, providing service to the field in international, national or provincial public advisory groups, scholarly activities, such as symposia or workshops; applied experimental and clinical results to the benefit of society; shared research and clinical experience willingly with other members of the*

medical and scientific community through various activities, such as offering expert advice, demonstrating new techniques, communication with lay support groups and related public relations and, conveying original research and clinical results lucidly and in a time manner to peers via journals and scientific meetings.

3. Postdoctoral Fellow Supervision Award, Faculty of Health Sciences, McMaster University, 2016.

4. Graduate Student Supervision Award, Faculty of Health Sciences, McMaster University, 2016.

5. Postdoctoral Fellow Supervision Award, Faculty of Health Sciences, McMaster University, 2015.

6. Senior author of the best CFAS paper (oral presentation) in basic science category. Wessels JM, Leyland NA, Agarwal SK, **Foster WG**. Estrogen regulation of brain-derived neurotrophic factor in the uterus and the link to endometriosis. 60th Annual Meeting of the Canadian Fertility & Andrology Society, Quebec City, QC, 2014.

7. Graduate Student Supervision Award, Faculty of Health Sciences, McMaster University, 2014.

8. Mid-career Award, CIHR/Ontario Women's Health Council. In recognition of excellence and contributions to women's health and reproductive toxicology. Five-year salary award, ($75,000/year) 2006 – 2010.

9. Career Award, Ontario Women's Health Council. In recognition of expertise and scientific contributions to the field of reproductive toxicology. One-year award, ($100,000/year) 2005.

10. Co-author of the best CFAS paper (oral presentation) in basic science category and Alpha Award. Neal MS, Petrik J, **Foster WG**, Holloway AC. *In utero* and lactational exposure to nicotine: ovarian effects. Conjoint American Society for Reproductive Medicine and the 51st Annual Meeting of the Canadian Fertility & Andrology Society, Montreal, QC, 2005.

11. Co-author of the best CFAS paper (oral presentation) in basic science category. Van Vugt DA, Krzemien A, Roy BN, **Foster W**, Lundhal S, Marcus S, and Reid RL. Photodynamic ablation in non-human primates. 42nd Annual Meeting of the Canadian Fertility & Andrology Society, Lake Louise, AB, 1996.

12. Senior author of the best CFAS paper (oral presentation) in basic science category. **Foster WG**, Rice DC, McMahon A. Suppression of luteal function in the chronically lead exposed cynomolgus monkey (*Macaca fascicularis*). 40th Annual Meeting of the Canadian Fertility & Andrology Society, St. John, NB, September 7 – 10, 1994.

13. Medical Research Council of Canada Studentship, 1988 – 1990.

14. Medical Sciences Programme Scholarship, 1987 – 1990.

15. Ontario Graduate Scholarship, 1985 – 1986; 1987 – 1988.

EMPLOYMENT EXPERIENCE:

1. Voluntary Clinical Professor, Department of Reproductive Medicine, University of California, San Diego, Health Sciences, San Diego, CA, June 2016 – present.

2. Research Director, Center for Endometriosis Research and Treatment (CERT), UC San Diego Health, San Diego, CA, September 2014 – present.

3. Adjunct Professor, Herbert Wertheim College of Medicine, Florida International University, Miami, FL, April 2013 – present.

4. Professor, Reproductive Biology Division, Department of Obstetrics & Gynecology, McMaster University, Hamilton, ON, June 2005 – present.

5. Affiliate Scientist, Institute of Population Health, University of Ottawa, Ottawa, ON, 2003 – 2014.

6. Director, Reproductive Biology Division, Department of Obstetrics & Gynecology, McMaster University, Hamilton, ON, 2002 – March 2010.

7. Coordinator, Resident Research Program, Department of Obstetrics & Gynecology, McMaster University, Hamilton, ON, 2003 – 2010.

8. Medical Director, Centre for Reproductive Care, Hamilton Health Sciences, Hamilton, ON, April 2005 – March 2008.

9. Adjunct Assistant Professor, Department of Obstetrics & Gynaecology, Foothills Hospital, University of Calgary, Calgary, AB, 1993 – 2008.

10. Associate Professor, Reproductive Biology Division, Department of Obstetrics & Gynecology, McMaster University, Hamilton, ON, June 2001 – June 2005.

11. Senior Science Advisor, Bureau of Chemical Hazards, Health Canada, Ottawa, ON, September 2000 – June 2001.

12. Adjunct Assistant Professor, Department of Obstetrics & Gynecology, McMaster University, Hamilton, ON, 1997 – 2001.

13. Associate Director/Director of Research, Center for Women's Health, Cedars-Sinai Medical Center, Los Angeles, CA, January 1999 – September 2000.

14. Acting Division Chief, Environmental & Occupational Toxicology Division, Environmental Health Directorate, Health Protection Branch, Health Canada, Ottawa, ON, May – October 1997; June 1998 – January 1999.

15. Head, Reproductive Toxicology Section, Environmental & Occupational Toxicology Division, Environmental Health Directorate, Health Protection Branch, Health Canada, Ottawa, ON, 1992 – June 1998.

16. Reproductive Toxicologist, Reproductive Toxicology Section, Environmental & Occupational Toxicology Division, Environmental Health Directorate, Health & Welfare Canada, Ottawa, ON, 1990 – 1992.

SCHOLARLY, AND PROFESSIONAL ACTIVITES:

i) **editorial boards** –
Reproductive Toxicology 2004 – 2008; 2015 – present.

BioMed Research International, 2014 – present.
Guest Editor, Obstetrics and Gynecology, 2014 – present.

Journal of Clinical Toxicology, 2013 – present.

Journal of Environmental & Analytical Toxicology, 2012 – present.

ISRN Toxicology, 2011 – present.

Journal of Toxicology and Environmental Health: B Critical Reviews, 2010 – present.

Journal of Applied Toxicology, 2008 – present.
Editor, Journal of Applied Toxicology, 2010 – present.

Immunology, Endocrine & Metabolic Agents in Medicinal Chemistry, 2009 – present.

Faculty of 1000, 2008 – present.

ii) **grant panels and committees** –

International Agency for Research on Cancer (IARC). Pentachlorophenol, Aldrin and Dieldrin; Volume 117. Lyon, France. October 1 – 12, 2016.

Canadian Institutes of Health Research (CIHR), College of Reviewers, 2015 – present.

Ontario Council on Graduate Studies, Ontario Women's Health Scholars Awards Selection Committee, elected membership, 2015 – present.

Canadian Breast Cancer Fund Grants Review Committee, 2014 – present.

Canadian Institutes of Health Research (CIHR), Operating Grant, Gender & Health Peer Review Committee, 2013 – present.

Development and Reproductive Toxicology (DART) Technical Committee, International Life Science Institute (ILSI), Health and Environmental Sciences Institute (HESI), Scientific Advisor, 2012 – present.

The Anti-NMDA Receptor Encephalitis Foundation, Inc, Board of Directors, 2012 – present.

Faculty of Health Sciences, McMaster University, Graduate Curriculum Committee, 2006 – present.

US-National Toxicology Program, Center for the Evaluation of Risk to Human Reproduction, Member of Expert Registry, 2005 – present.

CIHR Strategic Training Program in Tobacco Research (CIHR-STPTR), University of Waterloo, Mentor, 2005 – present.

College of Reviewers, Canada Research Chairs Program, Member, 2000 – present.

FIFRA Scientific Advisory Panel, US-Environmental Protection Agency (EPA), Ad hoc Member, 1999 – present.

CIHR Training Program in Reproduction, Early Development, and the Impact on Health (REDIH), Program Advisory Committee Member, 2009 – 2016.

CIHR Training Program in Reproduction, Early Development, and the Impact on Health (REDIH), Strategic Training Initiative in Health Research, Member and Mentor, 2009 – 2016.

CIHR Gender, Sex & Health, Committee for the Transitional Operating Grant, Member, 2013 – 2015.

National Institute of Environmental Health Sciences (NIEHS), National Toxicology Program (NTP) and the NTP Toxicology Branch, NTP Board of Scientific Counselors (BSC) meeting, Ad hoc Reviewer, December 9 – 10, 2014.

CIHR Clinical Investigation 'A', Grants Committee Panel, Member, 2013 – 2014.

Canadian Institutes of Health Research (CIHR) Catalyst Grant Committee, Genes and Chronic Disease, July 17 – 18, 2013.

National Sciences and Engineering Research Council (NSERC), Undergraduate Research Student Awards (URSA) Scholarship Review Committee, Member, March 13, 2013.

R. Samuel McLaughlin Centre for Population Health Risk Assessment, Affiliate, 2008 – 2013.

National Center for Environmental Assessment, US Environmental Protection Agency, Improving the risk assessment of persistent, bioaccumulative and toxic (PBT) chemicals in breast milk, Invited participant, Expert Workshop, October 24 – 26, 2012.

National Institute of Child Health and Human Development (NICHD) site visit, Division of Epidemiology, Statistics & Prevention Research, Expert Panel Member, October 22 – 24, 2012.

Council of Canadian Academies Panel, Integrating Emerging Technologies into Chemical Safety Assessment, Member, 2010 – 2012.

Council of Canadian Academies, Integrated Testing of Pesticides, Expert Panel Member, 2009 – 2012.

Centre for Disease Control, Infertility Research Working Group, Member, 2009 –2012.

Society for the Study of Reproduction, Committee on Reproduction and the Environment, Member, 2008 – 2012.

Institute of Medicine of the National Academies, "Veterans and Agent Orange: 8th Biennial Update", Expert Reviewer, 2011.

Strategic Training in Research in Reproductive Health Sciences (STIRRHS), University of Montréal, Mentor, 2006 – 2011.

ASRM ERSIG Advisory Board Member, June 2005 – 2011.

Canadian Fertility & Andrology Society, Strategies Planning Meeting, Member, November 19 –

20, 2010.

Health Canada, Assisted Human Reproduction Canada, The Environment and Reproductive Health: A Scientific Roundtable Steering Committee, Organizing Committee and Expert Panel Member, 2010.

National Institutes of Health/National Institute of Environmental Health Sciences – National Toxicology Program (NIH/NIEHS-NTP), Reproductive & developmental effects of soy products and genistein, Expert Panel Member, 2009 – 2010.

College of Physicians and Surgeons of Ontario, Obstetrics & Gynaecology Task Force - *In vitro* Fertilization, Mentor, 2008 – 2010.

Canadian Breast Cancer Fund Grants Review Committee, Member, 2007 – 2010.

CIHR Clinical Investigation 'A', Grants Committee Panel, Member, 2006 – 2010.

Environment and Health, Collaborations for Health, McMaster University, Co-theme Team Leader, 2005 – 2010.

US Environmental Protection Agency, the National Institute of Environmental Health Sciences, and the California Breast Cancer Research Program, Mammary Gland Evaluation and Risk Assessment Workshop, Invited Participant, November 16 – 17, 2009.

NIH Study Section, Integrative and Clinical Endocrinology and Reproduction Study Section, Member, October 5 – 6, 2009.

Health Canada and CIHR sponsored, Canadian Children's Environmental Health Research Workshop, Invited Participant and Session Chair, Ottawa, ON, February 9 – 11, 2009.

Preimplantation Genetic Diagnosis and Related Activities in Canada, Steering Committee Member, 2008 – 2009.

Assisted Human Reproduction Research Workshop: Developing a National Research Agenda, Scientific Planning Committee Member, Montreal, QC, October 15 – 16, 2008.

National Institute of Child Health and Human Development (NICHD) site visit, Team Member, September 24 – 26, 2008.

National Institute of Child Health and Human Development (NICHD), National Children's Study (NCS), Member of RFP Review Panel, April 2008.

CIHR Institute of Human Development and Child and Youth Health Workshop – Environmental Toxicology, Invited Participant, February 2008.

The Society of Obstetricians and Gynaecologists of Canada (SOGC), Research Committee, Member, 2003 – 2008.

Ontario Tobacco Research Unit, Small Grants Committee, Member, 2007.

National Institute of Child Health and Human Development (NICHD), Effects of Aspirin on Gestation and Reproduction Panel, Study Section Member, 2006.

US Environmental Protection Agency – Star program, Grant Review Panel Member, 2004; 2005.

National Institute of Child Health and Human Development (NICHD), Child Study; Study Section Member, 2005.

National Cancer Institute of Canada, Canadian Tobacco Control Research Initiative Peer Review Panel Member, 2004.

Toxic Substances Research Initiative, Health Canada, Healthy Environments and Consumer Safety Branch, 1999 – 2003.

- Chairman, Endocrine Disrupters Technical Review Committee, 1999 – 2000.

CIHR-Institute of Human Development, Child and Youth Health sponsored Pre and Post Implantation Consensus Workshop, Invited Participant, Niagara-on-the-Lake, ON, April 5 – 7, 2002.

Health Canada sponsored, Canadian Children's Environmental Health Research Workshop, Invited Participant and Session Chair, Ottawa, ON, March 17 – 19, 2002.

WHO/IPCS Steering Group on Endocrine Disruptors, 1998 – 2002.

- Global Inventory of Endocrine Disruptor Research.
- International Assessment of the State of Knowledge on Endocrine Disruptors.

Natural Sciences and Engineering Research Council (NSERC) Grant Selection, 1996 – 2001.

- Past Chair, 2001
- Committee Chair Person, 1999 – 2000.
- Committee Member, 1996 – 2000.

National Institutes of Environmental Health Safety, National Institutes of Health, and National Toxicology Program sponsored expert meeting on low-dose effects of endocrine disruptors,

Invited Panelist, October 2000.

US Environmental Protection Agency, Atrazine, Science Advisory Panel Member, June 2000.

SETAC-SOT co-sponsored Workshop on Environmental-Human Interconnections, Snowbird, UT, Invited Participant, June 10 – 15, 2000.

US Environmental Protection Agency – Endocrine Disruptor Screening and Testing Standardisation Committee, Mammalian Test Working Group Member, 1999 – 2000.

Joint US/EU Endocrine Disruptor Research, Expert Panel Member, 1999.

US-EPA ORD Strategy for Research on Environmental Risks to Children, Peer Reviewer, 1999.

Health Canada and Environment Canada, Interdepartmental Research Committee on Endocrine Disruptors, Co-Chair, 1998 – 1999.

Organisation for Economic Co-operation and Development (OECD), Member, 1997 – 1999.

- Working Group on Endocrine Disruptor Testing and Assessment, 1997 – 1999.
- Health Canada Endocrine Disruptor Committee, 1997 – 1999.
- National Co-ordinator, Test Guideline Program, 1997 – 1998.

National Sanitation Foundation - International, Health Effects Task Group, Member, 1996 – 1998.

Health Canada, Health Protection Branch, Animal Care Committee, Member, 1993 – 1998.

iii) **executive positions** –
Councillor, Society of Toxicology, Lake Ontario Regional Chapter, 2016 – present.

Board Member, The Anti-NMDA Receptor Encephalitis Foundation, Inc, 2012 – present.

Past President, Society of Toxicology, Lake Ontario Regional Chapter, 2014 – 2015.

Member, Membership Committee, Society for the Study of Reproduction, 2012 – 2015.

President, Society of Toxicology, Lake Ontario Regional Chapter, 2012 – 2014.

Member, Scientific Program Committee, Society for the Study of Reproduction, 2010 – 2012.

Chair, Core Committee, Society for the Study of Reproduction, 2010 – 2012.

Secretary Treasurer, Reproductive Developmental Toxicology Specialty Section, Society of

Toxicology, 2010 – 2012.

Member, Scientific Program Committee, Canadian Fertility & Andrology Society, 2006 – 2010.

Member of the Association of Professors of Obstetrics & Gynaecology, Science Committee, 2003 – 2009.

President, Canadian Fertility & Andrology Society, 2007 – 2008.

Chair, Scientific Program Committee, Canadian Fertility & Andrology Society, 2007 – 2008.

Vice-president, Canadian Fertility & Andrology Society, 2006 – 2007.

Chair, Science Panel, EM-COM web site, www.EMCOM.ca, 2002 – April 2005.

Member, Board of Directors, Infertility Awareness Association of Canada, 1998 – 2004.

Vice-president, President-elect, Society of Toxicology of Canada, 1998 – 1999.

Chair, Scientific Program Committee, Society of Toxicology of Canada, 1997.

Member of Scientific Program Committee, Society of Toxicology of Canada, 1996.

Member of Animal Care Committee, Society for the Study of Reproduction, 1995.

iv) **journal referee** –

Biology of Reproduction

British Journal of Obstetrics and Gynaecology

Comparative Biochemistry & Physiology

Critical Reviews in Toxicology
Endocrinology

Environmental Health Perspectives

Environmental Research

Environmental Toxicology

Fertility & Sterility

Food & Chemical Toxicology

Human Reproduction

Immunology, Endocrine & Metabolic Agents in Medicinal Chemistry

International Journal of Cell Biology

Journal of Applied Toxicology

Journal of Clinical Investigation

Journal of Toxicology & Environmental Health

Pesticide Biochemistry & Physiology

Placenta

Regulatory Toxicology and Pharmacology

Reproductive Biomedicine Online

Reproductive Toxicology

Systems Biology in Reproductive Medicine

The Journal of Urology

Toxicological Sciences

Toxicology and Industrial Health

v) **external grant review agencies –**

Canadian Institutes of Health Research

European Commission – Health Research

Natural Sciences and Engineering Research Council of Canada

Canadian Tobacco Control Research Initiative

Hospital for Sick Children, Toronto, ON

College of Reviewers for Canadian Research Chairs

Research Grants Council of Hong Kong

Puerto Rico Life Sciences Research Fund

Canterbury Medical Research Foundation

Netherlands Organization for Scientific Research - Earth and Life Sciences

AREAS OF RESEARCH INTEREST:

My research interests fall primarily into three categories as follows: (1) reproductive epidemiology and biomonitoring; (2) reproductive and development toxicity and carcinogenicity of environmental and dietary chemicals; and (3) the cellular and molecular mechanisms of endometriosis. My team of postdoctoral fellows and graduate students are assigned to each area carrying to better define the relationship between exposure to exogenous chemicals and adverse reproductive outcomes including estrogen related cancers.

COURSES TAUGHT:

i) **undergraduate –**

Pharmacology 4C03 Principles of Toxicology, Lectures on Endocrine Disruption

Undergraduate Medicine MF3 – Reproductive Biology

ii) **graduate –**

MS712 Reproductive Endocrinology

MS714 Industrial and Environmental Toxicology

MS720 Tobacco and Health: From Cells to Society

MS799 Independent Study in Reproductive Biology

SUPERVISORSHIPS:

i) **master** –

- Maria Haikalis, Identification of microRNA species in endometriosis. (Supervisor, 2015 – present).
- Eli Crapper, Characterization of a novel clinical marker of endometriosis. (Supervisor, 2015 – 2016). Medical School.
- Anne Doedée, Visiting Student (Netherlands), Neurotrophins and trks – novel reproductive tract proteins. (Supervisor, 2010). PDF, Germany.
- Alex Lagunov, Mechanisms regulating oocyte activation. (Supervisor, 2008 – 2010). Lab Director.
- Dana Anger, Tyrosine kinase receptor B expression in human endometrium. (Supervisor, 2005 – 2007). Cornerstone Research Group.
- Katie Stys, Mechanisms of AhR-ligand induced changes in inappropriate estrogen production in the endometrium. (Supervisor, 2003 – 2005). Health Law.
- Katie Edmunds, Proliferative effects of dietary isoflavones in the human endometrium. (Supervisor, 2002 – 2004). Health Law.
- Megan Miller, Effects of benzo[a]pyrene on matrix metalloproteinase expression and activity in breast cancer. (Supervisor, 2002 – 2004). High School Science Teacher.

ii) **doctoral –**

- Jocelyn Wessels, Brain derived neurotrophic factor (BDNF), a novel diagnostic marker for endometriosis. (Supervisor, 2011 – 2015). PDF, McMaster Unviersity.

- Mahmoud Aarabi, Queen's University, Characterization of post-acrosomal sheath protein PAWP. (Co-supervisor, 2008 – 2013). PDF, McGill, University.

- Anne Gannon , M.Sc., The effect of cigarette smoke constituents on ovarian follicle growth and apoptosis. (Supervisor, 2006 – 2013). PDF, Health Canada.

- Nakpangi A Johnson, Duquesne University – Does DDE shorten time to tumor formation in MMTV-neu mice? (Co-supervisor, 2008 –2011). Charles River Laboratories, USA.

- Mike Neal, M.Sc., The effect of environmental agents on follicle dynamics and oocyte quality. (Supervisor, 2002 – 2006) *incomplete*. Lab Directory, One Fertility.

- Ebrahim Nasir, Reproductive toxic effects of bilirubin on testicular function. (Co-supervisor, 2002 – 2003).

iii) **post-doctoral –**

- Hayley Furlong, Ph.D., Mitochondrial homeostatis and ovarian follicle development. (2014 – 2016).

- Marina Guerra, Ph.D., Ovarian toxicity of parabens. (2014 – 2015).

- Harry Peery, Ph.D., Mitochondrial oxidative stress following toxic insult. (2014 – 2015).

- Jean Clair Sadeu, Ph.D., Effect of environmental toxicants on folliculogenesis. (2009 – 2013).

- Heather Cameron, Ph.D., Effects of environmental toxicants on estrogen dependent mammary tumor development in mice. (2006 – 2008).

- Rocio Monroy, M.D., Cigarette smoking during pregnancy and glucose transport protein expression in the human placenta. (2006 – 2008).

- Gentao Liu, Ph.D., The reproductive effects of dietary galactose in the rat. (1999 – 2000).

- Jack Yang, Ph.D., The role of environmental pollutants in the pathophysiology of endometriosis in rodents and non-human primate models. (1996 – 1999).

- Michael Wade, Ph.D., The effect of environmentally relevant concentrations of priority contaminants on ovarian follicle differentiation, steroidogenesis and ovulation. (1996 – 1998).

- Daniel Cyr, Ph.D., The effects of methyl mercury on male reproduction. (1994 – 1995).

iv) **professional –**

- Andrea Mosher, M.D., Ph.D., OBS & GYN Resident research project, McMaster University, 2015 – present.
- Miguel Dominguez, D.V.M., Ph.D., Visiting Scientist, from Mexico, 2006 – 2009 & 2013.
- Sandra Gregorovich, M.D., Research Coordinator, 2009 – 2011.
- Heather Cameron, Ph.D., Research Associate, 2009 – 2010.
- Mehrnoosh Faghih, M.D., OBS & GYN Resident research project, McMaster University, 2006 – 2008.
- Myoung-seok Han, M.D., OBS & GYN, Visiting Scientist, from Korea, 2006 – 2007.
- Greg Athaide, M.D., OBS & GYN Resident research project, McMaster University, 2005 – 2006.
- Julie Francis, M.D., OBS & GYN Resident research project, McMaster University, 2004 – 2005.
- Pezhman Mirshokraei, D.V.M., Ph.D., Visiting Scientist, from Iran, 2003 – 2004.
- Alison Holloway, Ph.D., Cellular and molecular mechanisms of inappropriate estrogen production in endometriosis. (2001 – 2004).
- Anna Chomej, M.D., OBS & GYN Resident research project, McMaster University, 2003.

v) **supervisory committees –**

- Lucas Greville, 2017 – present, Ph.D. candidate, McMaster University.
- Tyler Pollock, 2014 – present, Ph.D. candidate, McMaster University.
- Evan Borman, 2013 – present, Ph.D. candidate, McMaster University.
- Jennifer Fazzari, 2012 – present, Ph.D. candidate, McMaster University.
- Michael Tsoulis, 2013 – 2015, M.Sc. candidate, McMaster University.

- Stephanie Zantinge, 2012 – 2014, M.Sc. candidate, McMaster University.
- Jonathan Lockwood, 2012 – 2013, M.Sc. candidate, McMaster University.
- Robert Berger, 2007 – 2010, M.Sc. candidate, McMaster University.
- Ayesha Khan, 2006 – 2009, Ph.D. candidate, McMaster University.
- Jenny Bruin, 2005 – 2009, Ph.D. candidate, McMaster University.
- Carolyn Cesta, 2007 – 2009, M.Sc. candidate, McMaster University.
- Jordan Shaw, 2007 – 2008, M.Sc. candidate, McMaster University.
- Rochelle Fernandez, 2004 – 2005, M.Sc. candidate, McMaster University.

vi) **others (summer/co-op students) –**

- Mona Kahlid, (January 2016 – present) Life Sciences, McMaster University. Project Title: Autophagy markers in ovarian follicular fluid.
- Allana Simon, (September 2015 – present) Health Sciences, McMaster University. Project Title: RANTES clinical marker of endometriosis, or not?
- Marina Bockaj, (September 2014 – present) Biomedical Engineering, McMaster University. Project Title: Miniaturized diagnostic test system for endometriosis.
- Nicholas Stalteri, (January 2016 – May 2016) Engineering, University of Waterloo. Project Title: Cigarette smoke-induced changes in autophagy gene expression and target tissues of the mouse.
- Yosef Ellenbogen, (September 2015 – July 2016) Health Sciences, McMaster University. Project Title: Fallopian tube endometriosis.
- Allegra Drumm, (September 2015 – September 2016) Biomedical Sciences, University of Guelph. Project Title: Interleukin 6 expression in the endometrium. Medical School.
- Thuy Linh Do, (September 2015 – May 2016) Health Sciences, McMaster University. Project Title: Toxicant-induced mitochondrial dysregulation in ovarian granulosa cells. Medical School.
- Aamer Somani, (January 2014 – September 2016) Biochemistry, McMaster University.

Project Title: Dietary chemical effects on endometrial epithelial cell aromatase activity. Medical School.

- Garima Aryal, (January 2014 – May 2016) Health Sciences, McMaster University. Project Title: Human developmental exposure to BPA: developmental effects.

- Sal Vivona, (September 2014 – 2015) Health Sciences, McMaster University. Project Title: Endometriosis markers. Medical School.

- Anna Parackal, (January 2014 – May 2015) Biopharmacology, McMaster University. Project Title: Effects of cigarette smoke on markers of autophagy in the mouse ovary. AstraZenica, Canada.

- Alia Tewari, (January – December 2014) Health Sciences, Western University, Project Title: Environmental obesogens, what's the skinny?

- Trevor Patch, (September 2013 – May 2014) Psychology, McMaster University. Project Title: Mechanisms of stress induced pregnancy loss in the mouse. Ph.D. student, University of Guelph.

- Piraveena Sivapatham, (September 2013 – May 2014) Life Sciences, McMaster University. Project Title: Connexin-26 expression in the endometrium and spontaneous abortion. BScN student, University of Toronto.

- Kabir Toor, (Summer 2013) Bachelor of Health Sciences, McMaster University. Project Title: Clinical markers in endometriosis. M.Sc. student, University of British Columbia.

- Alia Tewari, (Summer 2013) Bachelor of Health Sciences, University of Western Ontario. Project Title: Neurotrophins in endometriosis.

- Vanessa Kay, (Summer 2011; September 2011 – May 2013) Thesis student, McMaster University. Project Title: Measurement of urinary phthalate metabolites. M.D./Ph.D student, Queen's University.

- Vanessa Kay, (Summer 2010) Bachelor of Health Sciences, McMaster University. Project Title: Identification of a novel diagnostic marker for endometriosis.

- Vanessa Kay, (Summer 2009) Bachelor of Health Sciences, McMaster University. Project Title: Identification of a novel diagnostic marker for endometriosis.

- Natalie Cho, (Summer 2009) Bachelor of Health Sciences, McMaster University. Project

Title: Ovarian effects of Bisphenol A exposure. Resident of Anesthesiology, University of Ottawa.

- Mary Peric, (Summer 2009) Bachelor of Health Sciences, McMaster University. Project Title: Placenta change induced by in utero nicotine treatment in the rat. Naturopath.

- Melissa Coubrough, (Summer 2009) Midwifery, McMaster University. Project Title: Neurotrophic expression in breast cancer cell lines. Midwife.

- Otis Kryzanauskas, (Summer 2009) Midwifery, McMaster University. Project Title: Effects of maternal smoking on placenta glucose transport. Midwife.

- Mary Peric, (Summer 2008) Bachelor of Health Sciences, McMaster University. Project Title: Developmental effects of nicotine exposure in the rat.

- Vivian Ho, (Summer 2008) Bachelor of Health Sciences, McMaster University. Project Title: Developmental effects of nicotine exposure in the rat.

- Rami Elias, (Summer 2007) Bachelor of Health Sciences, McMaster University. Project Title: Neurotrophin and cognate receptor expression in endometriosis associated ovarian cancer.

- Mary Peric, (Summer 2007) Gr. 12 Hon. Student. Project Title: Brain derived neurotrophic factor and Tyrosine receptor kinase B expression in the reproductive tract of sexually mature BALB/c mice.

- Derek Chaves, (Fall 2004) Pharmacology & Toxicology Thesis Project, McMaster University. Project Title: Cyclooxygenase-II and matrix metalloproteinase expression in breast cancer cell lines. Architect.

- John Agzarian, (Summer 2004) Bachelor of Health Sciences, McMaster University. Project Title: Environmental toxicant mixture effects on thyroid gland morphology. (Funded by a scholarship from the Thyroid Foundation of Canada). Community Physician.

- Alex Petre, (Summer 2004) Bachelor of Science, McMaster University. Project Title: Toxicant induced changes in tissue remodelling enzyme expression in granulosa cells. (NSERC scholarship). M.D./Ph.D., University of Toronto.

- Dana Anger, (Summer 2004) Pharmacology & Toxicology Thesis Project, McMaster University. Project title: Mechanisms of methylchloranthene-induced changes in ovarian apoptosis. Cornerstone Research Group.

- Gareth Lim, (Summer 2003) Pharmacology & Toxicology Thesis Project, McMaster University. Project Title: Developmental toxicity of in utero exposure to drinking water disinfection by-products in the rat. PDF, University of British Columbia.

- Sarah Sinasac, (Summer 2002) Bachelor of Health Sciences, Medical Student, McMaster University. Project Title: Toxicant effects on granulosa cell steroidogenesis. Physician.

- Donna Grant, (Summer 1994) Biochemistry/Toxicology, University of Guelph. Project Title: Ovarian apoptosis in the PMSG primed immature rat ovary.

- Carmen Mertineit, (Winter 1992) Biology Thesis Project, McMaster University. Project Title: The effect of Hexachlorobenzene on the ovariectomized rat. Ph.D., Research Scientist, Astra Pharmaceuticals.

- Pete Ecclestone, (Winter 1991) Biology Thesis Project, McMaster University. Project Title: The effect of Lead intoxication on serum radioimmunoreactive vs. bioactive levels of pituitary gonadotropins and serum testosterone levels in the male cynomolgus monkey. Pharmaceutical sales.

- Greg Major, (Summer 1991) Biology, University of Western Ontario. Project Title: Modification of an enzyme fluorescent method for quantification of DNA in subcellular fractions. Orthopedic Surgeon, University of Colorado.

- Julie Pentick, (Fall 1990) Biochemistry, University of Waterloo. Project Title: Tissue distribution and subcellular localization of Hexachlorobenzene in the rat ovary. Contract researcher, Health Canada.

- Greg Major, (Summer 1990) Biology, University of Western Ontario. Project Title: Mating induced changes in the distribution of immunoreactive GnRH neural elements in the female rabbit.

vii) **graduate examining committees –**

- Tyler Pollock, Ph.D., Comprehensive Examiner, McMaster University, 2015.

- Evan Borman, Ph.D., Comprehensive Examiner, McMaster University, 2015.

- Jennifer Fazzari, M.Sc., Transfer Examiner, McMaster University, 2014.

- Stephanie Ondovcik, Ph.D., External Thesis Examiner, University of Toronto, 2013.

- Kristy Roth, Ph.D., Comprehensive Examiner, McMaster University, 2011.
- Jessica Kafka, Ph.D., Comprehensive Examiner, McMaster University, 2011.
- Robert Berger, M.Sc., Thesis Examiner, McMaster University, 2010.
- Ayesha Khan, Ph.D., Thesis Examiner, McMaster University, 2009.
- Jenny Bruin, Ph.D., Thesis Examiner, McMaster University, 2009.
- Arkadiusz (Eric) Hul, Ph.D., Thesis Examiner, McMaster University, 2009.
- Carolyn Cesta, M.Sc., Thesis Examiner, McMaster University, 2009.
- Jordan Shaw, M.Sc., Thesis Examiner, McMaster University, 2008.
- Navkiran Gill, Ph.D., Thesis Examiner, McMaster University, 2008.
- Anne Ellis, M.D., M.Sc., Thesis Examiner, McMaster University, 2008.
- Sudha Bhavanam, M.Sc., Thesis Examiner, McMaster University, 2008.
- Lorna Ryan, Ph.D., Thesis Examiner, McMaster University, 2007.
- Sherri Fernandez, M.Sc., Thesis Examiner, McMaster University, 2007.
- Alexandera Kollara, Ph.D., External Thesis Examiner, University of Toronto, 2006.
- Caleb Zavitz, M.Sc., Transfer Examiner, McMaster University, 2006.
- Rochelle Fernandez, M.Sc., Thesis Examiner, McMaster University, 2006.
- Tamara Lee Jocelyn, Ph.D., External Thesis Examiner, McGill University, 2005.
- Michael Cyr, M.Sc. Thesis Examiner, McMaster University, 2005.
- Erin McDonald, M.Sc., Thesis Examiner, McMaster University, 2005.
- Anthony Wood, Ph.D., External Thesis Examiner, University of Guelph, 2004.

- Julang Li, Ph.D., External Thesis Examiner, University of Ottawa, 1998.

RESEARCH FUNDING:

1. **Foster WG**, Leyland NA, Agarwal SK, Villeneuve P. Characterization of a novel clinical marker of endometriosis. CIHR Institute of Gender and Health $705,752. (***Awarded***). 2015 – 2020.

2. **Foster WG**, Allen-Vercoe E, Cyr D, Haddad S, Haines J, Hooks G, Kubwabo C, Langille M, Venners S. Developmental origins of toxicant-induced obesity and insulin resistance. Canadian Institutes of Health Research (CIHR) Team Grant: Developmental Origins of Health and Disease – Implications for Men, Women, Boys and Girls – LOI $10,000. (***Awarded***) 2015 – 2016.

3. **Foster WG** and Zhu, J. Mechanism(s) of cigarette smoke-induced ovarian follicle loss. Canadian Institutes of Health Research (CIHR) $873,835. (***Awarded***) 2011 – 2016.

4. Tayade C and **Foster WG**. A novel anti-angiogenic therapy for endometriosis. CIHR $483,410. (***Awarded***). 2011 – 2016.

5. **Foster WG**, Leyland NA, Agarwal SK, Villeneuve P. Characterization of a novel clinical marker of endometriosis. CIHR Institute of Gender and Health $100,000. (***Awarded***). 2014 – 2015.

6. Fraser W, Arbuckle T, **Foster WG**, et al., (9 co-applicants). Maternal-Infant Research on Environmental Chemicals – Child Development Plus (MIREC-CD+). Health Canada $149,165. (***Awarded***). 2013 – 2015.

7. Baltz JM, **Foster WG**, et al., (30 co-applicants). Training Program in Reproduction, Early Development, and the Impact on Health (REDIH). CIHR Human Development, Child and Youth Health $1,787,598. (***Awarded***) 2009 – 2015.

8. Cameron R, **Foster WG**, et al., (58 co-applicants). Population Intervention for Chronic Disease Prevention: A Pan-Canadian Program. CIHR $1,950,000. (***Awarded***) 2009 – 2014.

9. **Foster WG**. Evaluation of a dietary treatment for endometriosis. Concourse Health Sciences LLC $68,525 USD. (***Awarded***). 2012 – 2013.

10. **Foster WG**. Neurotrophins and Trks: Novel reproductive tract proteins. Natural Sciences and Engineering Research Center (NSERC) $185,000. (***Awarded***). 2008 – 2013.

11. Fraser W, Arbuckle T, **Foster WG**, et al., (9 co-applicants)**.** Maternal-Infant Research on Environmental Chemicals : A National Profile of *In Utero* and Lactational Exposure to Environmental Contaminants. CIHR $1,248,126. (***Awarded***) 2006 – 2012.

12. Fraser W, Arbuckle T, **Foster WG,** et al., (9 co-applicants). Maternal-Infant Research on Environmental Chemicals – Infant Development (MIREC-ID). Health Canada $2,084,968. (***Awarded***). 2008 – 2011.

13. **Foster WG** and Cameron H. Dieldrin increases breast cancer metastasis via dysregulation of neurotrophin expression. CIHR $100,000. (***Awarded***) 2009 - 2010.

14. **Foster WG**, Yauk C, Quinn J, Robaire B, and McCarry B. Urban air particulate pollution & genetic instability. CIHR Team Grant LOI $10,000. (***Awarded***) 2009 - 2010.

15. **Foster WG**. Cellular and molecular mechanisms of toxicant-induced changes in ovarian follicular atresia. CIHR $616,408. (***Awarded***) 2006 – 2010.

16. **Foster WG**. Cellular and molecular mechanisms of toxicant-induced changes in follicular dynamics and ovarian regulation. Ontario Women's Health Council/CIHR Institute of Gender and Health Mid-career Award $375,000. (***Awarded***) 2005 – 2010.

17. Oko R and **Foster WG**. Improvement of ICSI treatment by co-injection of recombinant PAWP protein. CIHR RxND $100,000. (***Awarded***) 2007 – 2008.

18. **Foster WG**. Toxicant-induced resistance to ANOIKIS in estrogen sensitive target tissues. NSERC $35,136. (***Awarded***). 2007 – 2008.

19. **Foster W**, Holloway A, Krewski D, Kourti T. Surrogate biomarkers of *in utero* exposure to xenobiotics. American Chemistry Council $878,418. (***Awarded***) 2003 – 2007.

20. **Foster WG**. Toxicant induced tissue remodelling in estrogen sensitive target tissues. NSERC $144,000. (***Awarded***) 2003 – 2007.

21. **Foster W**, Holloway A, Krewski D, Muller W. Biomarkers of breast cancer. American Chemistry Council $941,751. (***Awarded***) 2002 – 2007.

22. Casper R and **Foster WG**. Identification of early pathogenetic events leading to endometriosis and discovery of novel therapeutic strategies. CIHR – Operating $415,794. (***Awarded***) 2003 – 2006.

23. **Foster WG**. AhR ligands and endometriosis: towards understanding their mechanism of

action. CIHR – Operating $345,601. (***Awarded***) 2002 – 2006.

24. **Foster WG**. Ontario Women's Health Council Career Award $100,000. (***Declined***) 2005.

25. **Foster W**, Holloway A. Effect of binary mixtures on estrogen sensitive target tissues. Canadian Network of Toxicology Centres $55,000. (***Awarded***) 2003 – 2004.

26. **Foster WG**. Hormonally active chemicals: cellular and molecular mechanisms of action. Canadian Foundation for Innovation-Ontario Innovation Trust (CFI-OIT) $422,096. (***Awarded***) 2002 – 2003.

27. Holloway A, and **Foster WG**. Effects of *in utero* chemical insult on postnatal health. Canadian Chlorine Coordinating Council $48,000. (***Awarded***) 2002 – 2003.

28. **Foster WG**, Hughes CL, and Chan S. Human developmental exposure to endocrine disruptors. New York Community Trust Fund $105,000 USD. (***Awarded***) 2002 – 2003.

29. **Foster WG**. Dietary factor modulation of endometrial tissue production of IL-6 and IL-6sR and angiogenic factors *in vitro*. Dow Chemical $25,000 USD. (***Awarded***) 2001 – 2002.

30. Davis V, **Foster WG**, and Hughes CL. Influence and localized DDT exposure on breast cancer. California Breast Cancer Research Program $305,989 USD. (***Awarded***) 2000 – 2002.

PEER REVIEWED PUBLICATIONS:

i) **books** –

1. Ritter L, Austin CP, Bend JR, Brunk CG, Caulfield T, Dellarco VL, Demers P, **Foster W**, Infante-Rivard C, Jumarie C, Kacew S, Kavlock RJ, Krewski D, Mezey PG, Shultz T. (2012) Integrating Emerging Technologies into Chemical Safety Assessment. Council of Canadian Academies. Ottawa, Canada.

ii) **contributions to books** –

1. Dominguez MA, Sadeu JC, Guerra MT, Furlong HC, Bains S, **Foster WG**. (2016) Chapter: Ovarian toxicity on environmental contaminants: 50 shades of grey. In: Translational Toxicology: Defining a new therapeutic discipline, Molecular Integrative Toxicology, Hughes C and Waters MD, Editors. Springer International Publishing, Switzerland.

2. Valez MP, Monnier P, **Foster WG**, Fraser WD. (2015) Chapter 7:The impact of phthalates on women's reproductive health: Current state-of-the-science and future directions. In: Our Chemical Selves – Gender, Toxics, and Environmental Health, Dayna Nadine Scott, Editor. UBC Press. Part III, Hormones as the 'Messengers of Gender'? 231-254.

3. Gannon AM, Sadeu JC, Agarwal SK, Hughes CL, **Foster WG**. (2013) Chapter 10:Cigarette smoking and ovarian function. In: Ovarian Toxicology, Patricia Hoyer, Editor. CRC Press. Part II, Ovotoxic Chemical Classes. 231-250.

4. Peery HE, Day GS, Doja A, Xia C, Fritzler M, **Foster W**. (2013) Chapter 129:Anti-NMDA receptor encephalitis in children: the disorder, its diagnosis, and treatment. In: Handbook of Clinical Neurology, Vol. 112, Pediatric Neurology Part II. 1229-1233.

5. Rier S and **Foster WG**. (2003) Environmental dioxins and endometriosis. Semin. Reprod. Med. 21(2):145-154.

6. **Foster W** and Hughes C. (2002) Chapter 2:Review of Normal Human Reproduction. In: Principles for Evaluating Human Reproductive Effects of Chemicals. International Programme on Chemical Safety of the World Health Organization.

7. Van Vugt DA, Krzemien A, **Foster W**, Lundhal S, Marcus S, Reid RL. (2000) Photodynamic endometrial ablation in non-human primates. In: Photomedicine in Gynecology and Reproduction. (P. Wyss, Tadir Y, Tromberg BJ, Haller U, eds.) Karger, Basal, Switzerland. 213-218.

iii) **journal articles** –

1. Guerra MT, Sanabria M, Leite GAA, Borges CS, Cucielo MS, Anselmo-Franci JA, **Foster WG**, Kempinas WG. (2016) Maternal exposure to butylparaben impairs testicular structure and sperm quality on male rats. Environ. Res. (*In-press*).

2. Yao C, **Foster WG**, Sadeu JC, Siddique S, Zhu J, Feng YL. (2016) Screening for DNA adducts in ovarian follicles exposed to benzo[a]pyrene and cigarette smoke condensate using liquid chromatography-tandem mass spectrometry. Sci. Total Environ. (*In-press*).

3. **Foster WG**, Evans JA, Little J, Arbour L, Moore A, Sauve R, León JA, Luo W. (2016) Human exposure to environmental contaminants and congenital anomalies: A critical review. Crit. Rev. Toxicol. (*In-press*).

4. Fisher M, Arbuckle TE, Liang CL, LeBlanc A, Gaudreau E, **Foster WG**, Haines D, Davis K, Fraser WD. (2016) Concentrations of persistent organic pollutants in maternal and cord blood from the maternal-infant research on environmental chemicals cohort study. Environ. Health. 15(1):59.

5. Guerra MT, Furlong HC, Kempinas WG, **Foster WG**. (2016) Effects of in vitro exposure to butylparaben and di-(2 ethylhexyl) phthalate, alone or in combination, on ovarian function. J. Appl. Toxicol. 26(9):1235-1245.

6. Furlong HC, Wessels JM, Guerra MT, Stämpfli MR, **Foster WG**. (2016) Hydroxychloroquine attenuates cigarette smoke induced autophagic signaling in the mouse ovary. Reprod. Toxicol. 61:105-113.

7. Kubwabo C, Kosarac I, **Foster WG**. (2016) Quantitative determination of nine urinary metabolites of organophosphate flame retardants using solid phase extraction and ultra performance liquid chromatography coupled to tandem mass spectrometry (UPLC-MS/MS). J. Chromatogr. B. Analyt. Technol. Biomed. Life Sci. 1014:24-30.

8. **Foster WG**. (2016) Diagnosing endometriosis: CA125 rules in, but not out. BJOG. 123(11):1769.

9. Perreault M, Xu VY, Hamilton S, Wright D, **Foster W**, Atkinson S. (2016) Validation of a food frequency questionnaire for bone nutrients in pregnant women. CJDPR. 77(3):133-139.

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2. **Foster WG**. (2015) Human exposure to environmental contaminants and congenital anomalies: a systematic review. Public Health Agency of Canada.

3. Lamb JC 4th, Boffetta P, **Foster WG**, Goodman JE, Hentz K, Rhomberg LR, Staveley J, Swaen G, Van Der Kraak G, Williams AL. (2014) Critical review of WHO-UNEP state of the science of endocrine disrupting chemicals – 2012.

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9. Vélez MP, Monnier P, **Foster WG**, Fraser WD. (2008) The impact of phthalates on women's reproductive health: State-of-the science and future directions. National Network on Environment and Women's Health.

10. **Foster WG** and Rousseaux C. (1995) The reproductive toxicology of great lakes contaminants. In: Proceedings of the State of the Lakes Environment Conference.

11. **Foster WG**, Singh A, Rice DC, McMahon A. (1991) Reproductive effects of chronic Lead-exposure in the male cynomolgus monkey (*Macaca fascicularis*). Symposium On Lead In Adults, Durham, NC. December 9 – 11, 1991.

12. Inskip MJ, Yagminas A, Franklin CA, **Foster W**, Wandelmaier F, Haines D, Blenkinsop J. (1991) Maternal-fetal transfer of Lead in a non-human primate *Macaca fascicularis*: Preliminary studies using stable isotope tracers. In: Proceedings of International Conference on Heavy Metals in the Environment. CEP Consultants.

NON-PEER REVIEWED PUBLICATIONS:

i) **journal articles** –

1. **Foster WG** and Moore E. (2008) Chemical exposures and infertility. J. Infertility Awareness Association of Canada.

2. Neal MS and **Foster WG.** (2005) Applications for *in vitro* follicle culture assays. Fertility World. 3:10-11.

3. **Foster WG**. (2004) Chemical exposures and human fertility. Fertility Magazine.

4. YoungLai EV and **Foster WG**. (2004) Dichlorodiphenylchloroethylene and human fertility. A.R.T. & Science. 3(3):6-8.

5. **Foster WG** and Beecroft ML. (2003) Chemical exposures and human fertility. Infertility Awareness 4:30-31.

6. Hughes CL Jr. and **Foster WG.** (2001) Some potential health effects of endocrine-disrupting chemicals across the lifespan of adult women. ASRM Menopausal Medicine 9(2):7-12.

7. Safe S, **Foster W**, Lamb J, Newbold R, Van Der Kraak G. (2000) Estrogenicity and Endocrine Disruption. CAST. 16:1-16.

8. Yang JZ and **Foster WG**. (1997) Causes of endometriosis: Do environmental contaminants play a role? Infertility Awareness 13:10-13.

ARTICLES IN PREPARATION:

1. Furlong HC, Stampfli MR, Gannon AM, **Foster WG**. (2016) Is cigarette smoke induced autophagy unique to the ovary – A tissue comparison study.

2. Aryal G, Kubwabo C, **Foster WG**. (2016) Free Bisphenol A (BPA) concentrations in maternal and cord blood.

UNPUBLISHED DOCUMENTS:

1. Provisional Patent No. 61889085: An assay for inflammatory disease progression and response to treatment.

2. Provisional Patent No. 796896: Method to predict pregnancy potential of an oocyte.

3. International PCT Application No. PCT/CA2007/002114: Trkβ: a diagnostic tool in endometriosis.

PAPERS GIVEN AT SCIENTIFIC MEETINGS:

i) **invited presentations** –

1. **Foster WG**. Life-style and environmental effects on ovarian function: Myths or real concerns? 62nd Annual Meeting of the Canadian Fertility & Andrology Society. Toronto, ON. September 22 – 24, 2016.

2. **Foster WG**. Environmental toxicant exposure and dysregulation of ovarian function. Environment & Reproductive Science Summit 2016 – Environmental, Nutritional, and Genetic Factors Affecting Reproduction. Sponsored by the American Society for Reproductive Medicine, the Society for the Study of Reproduction and the Society of Reproductive Biologists and Technologists. The ONLY Canadian invited speaker. Dallas/Fort Worth TX. March 5 – 6, 2016.

3. **Foster WG**. Environmental toxicant exposure induced dysregulation of ovarian function. 48th Annual Meeting of the Society for the Study of Reproduction. San Juan, Puerto Rico. June 18 – 22, 2015.

4. **Foster WG**. Toxicant-induced changes in ovarian function and follicle development: From clinic to bench and back. Texas A & M University. College Station, TX. February 2 – 3, 2015.

5. **Foster WG**. Endometriosis: clinical and experimental aspects. Female Reproductive Tract session. International Conference on Reproductive Biology and Toxicology, Auditorium of the University of São Paulo Law School (FDRP). Ribeirão Preto, São Paulo, Brazil. November 10 – 11, 2014.

6. **Foster WG**. Research: making tomorrow better today. Round table discussion, Translation of Basic Science to Real-World Practice. International Conference on Reproductive Biology and Toxicology, Auditorium of the University of São Paulo Law School (FDRP). Ribeirão Preto, São Paulo, Brazil. November 10 – 11, 2014.

7. **Foster WG**. The Jekyll and Hyde tale of neurotrophin expression in the endometrium. Escola Paulista de Medicina, Universidade Federal de São Paulo, Centro de Pesquisa em Urologia, Ribeirão Preto, São Paulo, Brazil. November 10 – 11, 2014.

8. **Foster WG.** Clinical markers of endometriosis. University of São Paulo, São Paulo, Brazil. November 5, 2014.

9. **Foster WG**. Cigarette smoke exposure induced dysregulation of mitochondrial homeostasis in granulosa cells. University State de São Paulo (UNESP), Botucatu, Brazil. November 6, 2014.

10. **Foster WG**. The role of phthalates in obesity: what's the skinny? City Wide Endocrine Rounds, University of Toronto. October 2014.

11. **Foster WG.** Publish or Perish: The pitfalls and tricks to getting your scientific article published. XIII Workshop da Pós-Graduação, Publicação, Pesquisa e Ensino, Salão Nobre da FMB, UNESP, Botucatu/ São Paulo, Brazil. June 4 – 7, 2014.

12. **Foster WG.** Endometriosis: Animal models and the role of toxicants. XIII Workshop da Pós-Graduação, Publicação, Pesquisa e Ensino, Salão Nobre da FMB, UNESP, Botucatu/ São Paulo, Brazil. June 4 – 7, 2014.

13. **Foster WG.** The ovarian effects of environmental toxicants: Clinical implications. The Society of Obstetricians and Gynaecologists of Canada, Ontario CME Program, Toronto, ON. November 28 - 30, 2013.

14. **Foster WG**. Characterization of a novel clinical marker of endometriosis. Grand Rounds, Department of Obstetrics & Gynecology, McMaster University, Hamilton, ON. September 4, 2013.

15. **Foster WG.** Exposure to environmental toxicants and ovarian dysfunction. International Workshop in Neuroendocrinology – Brazilian International Symposium on Integrative Neuroendocrinology. Dourado, Brazil. August 4 – 7, 2013.

16. **Foster WG.** Mechanisms of cigarette smoke induced ovarian follicle loss: The bumpy road to discovery. Graduate Program on General and Applied Biology, Institute of Biosciences of Botucatu, Universidade Estadual Paulista (unesp), Brazil. July 29 – August 2, 2013.

17. **Foster WG**. Clinical markers of endometriosis: what's new? Rounds, Department of Obstetrics & Gynecology, McMaster University. Hamilton, ON. May 29, 2013.

18. **Foster WG**. Strengths and limitations of *in vitro* test methods for reproductive toxicology. 52nd Annual Meeting of the Society of Toxicology. San Antonio, TX. March 10 – 14, 2013.

19. **Foster WG**. Women's reproductive health. Canadian Memorial Chiropractic College (CMCC). Toronto, ON. March 1, 2013.

20. **Foster WG**. Environmental toxicants and reproductive health. Centre INRS-Institut Armand Frappier. Laval, QC. December 7, 2012.

21. **Foster WG.** The ovarian toxic effects of cigarette smoke exposure. Florida International University. Miami, FL. November 29, 2012.

22. **Foster WG.** The ovarian effects of environmental toxicants and clinical implications. 58th Annual Meeting of the Canadian Fertility & Andrology Society. Ottawa, ON. September 6 – 9, 2012.

23. **Foster WG.** Effects of endocrine disruption on reproductive function in the female. Center for Research in Biology of Reproduction (CRBR) Laval University, QC. December 15, 2011.

24. **Foster WG.** Mechanisms of cigarette smoke-induced sub-optimal ovarian follicle development and atresia. Department of Veterinary Medicine and Biomedical Sciences, Texas A&M University. College Station, TX. September 30, 2011.

25. **Foster WG.** Windows of susceptibility: improving understanding of physiological and exposure differences. International Council of Chemical Associations Long-Range Research Initiative (ICCA-LRI) & Health Canada Workshop; Advancing Exposure Science to Improve Chemical Safety. Quebec City, QC. June 22 – 23, 2011.

26. **Foster WG**. Mechanisms of environmental toxicant induced ovarian follicle loss. Department of Reproductive Medicine, Seminars in Reproductive Science and Medicine, University of California San Diego. April 5, 2011.

27. **Foster WG.** The environment and its' implication to cancer and other diseases. The Kiwanis Club of Oakville, monthly meeting. Oakville, ON. November 15, 2010.

28. **Foster WG.** Regulating estrogen production and metabolism in estrogen-dependent diseases. Citywide Rounds. University of Toronto, Mount Sinai Hospital. Toronto, ON. November 27, 2009.

29. **Foster WG.** Bisphenol A (BPA): science, policy options and risk communication. Improving the Public Communications of Chemical-related Health Risks Workshop. University of Ottawa, ON. September 30 – October 1, 2009.

30. **Foster WG**. Current and emerging issues in reproductive medicine and the placenta biology. 2009 Human Placenta Workshop, Opening Address. Queen's University. Kingston, ON. July 19, 2009.

31. **Foster WG.** Exposure to environmental toxicants and consequences for adverse health effects. Bay Area Restoration Council's 17th Annual Community Workshop – "Looking Beyond 2015". Parks Canada Discovery Centre. Hamilton, ON. April 25, 2009.

32. **Foster WG.** Effects of environmental contaminants on human reproductive health. Hamilton's 3rd Annual Health Research in the City. Hamilton, ON. February 11, 2009.

33. **Foster WG.** Breast Cancer and Environmental Toxicants: New Approaches to Animal Studies in Research, Environment & Health Seminar Series, Centre for Environment. University of Toronto, ON. January 29, 2009.

34. **Foster WG.** Impact of cigarette smoke and its constituents on ovarian function, Public Health Symposium on Infertility. Centers for Disease Control and Prevention. Atlanta, GA. September 15 – 17, 2008.

35. **Foster WG.** The animal evidence: Critical effects and dose-response. Dioxin 2008. 28th International Symposium in Halogenated Persistent Organic Pollutants (POPs). Birmingham, England, UK. August 17 – 22, 2008.

36. **Foster WG.** Bisphenol A: a reproductive hazard but what are the public health implications? Ontario Public Health Unit and Medical Officers of Health Webinar. June 24, 2008.

37. **Foster WG**. Hormone mimics and human health. The 5th PCB Workshop. New Knowledge Gained from Old Pollutants. Iowa City, IA. May 18 – 22, 2008.

38. **Foster WG.** Environmental hazards posed by chemicals and the problems associated with trying to understand the true risk. The Probus Club of Hamilton Mountain, meeting. Hamilton, ON. May 1, 2008.

39. **Foster WG**, Neal MS, Mulligan Tuttle A, Dominguez MA. Impact of environmental factors on ovarian function. Preconception Care Research: Improving Birth Outcomes and Reproductive Health Workshop. Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), National Institutes of Health (NIH), U.S. Department of Health and Human Services. Potomac, MD. April 14 – 15, 2008.

40. **Foster WG.** Reproductive toxicity of environmental toxicants: state of the science and data gaps. CIHR Institute of Human Development and Child and Youth Health Workshop. Montreal, QC. February 11, 2008.

41. **Foster WG.** Reproductive toxicology: Potential hazards and risks to human reproductive health from environmental contaminants. San Diego Reproductive Endocrinology Society. San Diego, CA. November 15, 2007.

42. **Foster WG.** Reproductive toxicity of environmental contaminants: Potential hazards vs. risk to human reproductive health. Reproductive Medicine Grand Rounds. University of California. San Diego, CA. November 14, 2007.

43. **Foster WG.** Reproductive effects of endocrine disrupting compounds. Seminars in Reproductive Science and Medicine. University of California. San Diego, CA. November 14, 2007.

44. **Foster WG.** Cellular and molecular mechanisms of cigarette smoke-induced reproductive

toxicity. 2nd Sino-Canada Workshop on Reproductive Medicine. Ottawa, ON. October 19 – 21, 2007.

45. **Foster WG.** Emerging issues in Reproductive Environmental Toxicant Research. UCSF-CHE Summit on Environmental Challenges to Reproductive Health and Fertility. University of California at San Francisco, CA. January 28 – 30, 2007.

46. **Foster WG**. Endocrine Disruption. National Policy Consultation Series on Children's Health and Environment. Ottawa, ON. January 23 – 24, 2007.

47. **Foster WG.** Resistance to Anoikis in estrogen dependent disease. National Institutes of Child Health and Human Development, Division of Epidemiology, Statistics, & Prevention Research. Rockville, MD. January 11, 2007.

48. **Foster WG.** Anoikis in estrogen sensitive target tissues and disease. Department of Anatomy and Physiology, Queen's University. Kingston, ON. December 7, 2006.

49. **Foster WG.** Reproductive effects of endocrine toxicants: From the lab to the clinic. 52nd Annual Meeting of the Canadian Fertility & Andrology Society. Ottawa, ON. November 15 – 18, 2006.

50. **Foster WG.** Environmental toxicants and ovarian function. American Society of Reproductive Immunology. Nashville, TN. June 15 – 17, 2006.

51. **Foster WG.** Ovarian cancer: Mechanisms of action for environmental toxicants and dietary chemicals. National Toxicology Program sponsored Hormonally-induced reproductive tumors: Relevance of rodent bioassays workshop. Raleigh, NC. May 22 – 24, 2006.

52. **Foster WG.** Reproductive effects of environmental endocrine toxicants. First Sino-Canada Bilateral Workshop on Reproductive Health Research. Beijing, China. November 15 – 18, 2005.

53. **Foster WG.** 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) induced increase in endometrial aromatase activity is mediated through altered PGE2 metabolism. Institute of Gender and Health Annual Meeting. Vancouver, BC. November 8, 2005.

54. **Foster WG.** Endocrine toxicants and laboratory evidence. Endocrine Toxicants: Ecological and population health impacts. University of Ottawa. Ottawa, ON. October 28, 2005.

55. **Foster WG.** Relative effects of environmental toxicants on aromatase expression and activity in human endometrial stromal cell cultures. NIEHS sponsored workshop on Atrazine. Iowa City, IA. April 25 – 26, 2005.

56. **Foster WG.** Adverse effects of environmental toxicants on female reproduction. Society of Toxicology of Canada, 37th Annual Symposium. Montreal, QC. December 6 – 7, 2004.

57. **Foster WG**. Current context and need for basic research on environmental effects on reproduction. CIHR Workshop, 50th Annual Meeting of the Canadian Fertility & Andrology Society. Jasper, AB. November 24 – 28, 2004.

58. **Foster WG.** Endocrine disrupters and ovarian function. International Federation of Fertility Societies. Montreal, QC. May 23 – 28, 2004.

59. **Foster WG.** Environmental toxicants and breast cancer. Juravinski Cancer Centre. Hamilton, ON. January 2004.

60. **Foster WG.** *In utero* exposure to endocrine toxicants and dietary phytoestrogens. Linus Pauling Institute, Oregon State University. Corvallis, OR. December 18, 2003.

61. **Foster WG.** Are dioxins involved in the pathogenesis of endometriosis? Department of Pharmacology & Toxicology, and the Department of Obstetrics & Gynecology, Queen's University. Kingston, ON. October 30, 2003.

62. **Foster WG**. Environmental toxicants: ovulation and endometriosis. World Congress on Endometriosis. San Diego, CA. February 24 – 27, 2002.

63. **Foster WG.** and YoungLai EV. Presence of environmental contaminants in human follicular fluid, serum, and seminal plasma of couples undergoing *in vitro* fertilization. 28th Aquatic Toxicology Workshop. Winnipeg, MB. September 30 – October 3, 2001.

64. **Foster W.** Chan S. Platt L. and Hughes C. Human developmental exposure to endocrine active compounds. Key Note lecture, 5th International Symposium on Biological Monitoring. Banff, AB. September 18 – 21, 2001.

65. **Foster W.** Human exposure and potential health effects of endocrine disrupting compounds. Symposium # 2. Effects of Pollutants on Humans. Canadian Federation of Biological Societies, 44th Annual Meeting. Ottawa, ON. June 20 – 23, 2001.

66. **Foster WG** and Agarwal SK. Environmental contaminants and dietary factors in endometriosis. Endometriosis: Emerging Research and Intervention Strategies. National Institute of Child Health and Human Development (NICHD). Bethesda, MD. April 9 – 10, 2001.

67. **Foster WG**. Effects of environmental contaminants on ectopic endometrium in animal

models of endometriosis. University of Laval. Quebec City, QC. November 18, 2000.

68. **Foster WG**. Environmental contaminants - effects upon human reproductive physiology. Grand Rounds. Cedars-Sinai Medical Center. Los Angeles, CA. August 16, 2000.

69. **Foster WG**, Chan S, Platt L, Hughes CL. Detection of organochlorine compounds and phytoestrogens in second trimester human amniotic fluid. Int. Assoc. Great Lakes Res. Cornwall, ON. May 22 – 26, 2000.

70. **Foster WG**. Effects of environmental contaminants on ectopic endometrium in animal models of endometriosis. University of Western Ontario. London, ON. April 20, 2000.

71. **Foster WG**, Hughes CL. An overview of endocrine disruption and human health. Endocrine Disruption Workshop: Establishing a National Science Agenda on the scientific assessment of endocrine disrupting substances. Huntsville, ON. February 17 – 19, 2000.

72. **Foster WG**. Environmental contaminants and dietary factors: Consequence for human health. University of Ottawa. Ottawa, ON. November 18, 1999.

73. **Foster WG**, Hughes CL, Platt L, Chan S. Detection of Endocrine Disrupting Chemicals and Dietary Factors in Samples of Second Trimester Human Amniotic Fluid. Environmental Hormones: Past, Present, Future. Tulane University. New Orleans, LA. October 18 – 20, 1999.

74. **Foster WG**, EU/US Transatlantic Co-operation in Human Environmental Health; Expert Panel Meeting on Opportunities for Collaborative EU/US Research Programmes. Ispra, Italy. April 1999.

75. **Foster WG**. 1999 Special Meeting of the Toxicology Forum: Dose-Response Considerations for Potential Endocrine Active Substances. Washington, DC. April 1999.

76. **Foster WG**. Endocrine Disruptors and Human Health Effects: Health Canada Perspective. Southern Ontario Reproductive Biology Workshop. University of Waterloo. Waterloo, ON. May 8, 1998.

77. **Foster WG**. Endocrine Disruptors Panel Member. Health Conference. Montreal, QC. May 15 – 17, 1997.

78. **Foster WG**. Human health effects of endocrine modulating substances: fact or fiction? Grand Rounds, Department of Obstetrics & Gynecology, McMaster University. Hamilton, ON. May 21, 1997.

79. **Foster WG**. Endocrine Disruptors: Potential Risks to Children. What on Earth Conference sponsored by the Canadian Institute of Child Health. Ottawa, ON. May 26 – 27, 1997.

80. **Foster WG**. White House expert committee on endocrine disrupters. Washington, DC. January 27, 1997.

81. **Foster WG**. Environmental Exposures and Human Reproduction: Women. Reproductive Health and The Environment Symposium. Toronto Department of Health. Toronto, ON. May 1996.

82. **Foster WG**. Endocrine Disruptors Chemicals: The Human Connection. DOE Workshop on Endocrine Disrupter Issue. Hull, QC. May 1996.

83. **Foster WG**. Endocrine Disruptors: Human Health Effects. Canadian Chemical Producers Association. Ottawa, ON. June 1996.

84. **Foster WG**. Animal models in reproductive biology and toxicology. Canadian Association for Laboratory Animal Science. Ottawa, ON. June 2, 1994.

85. **Foster WG**. Steroid actions on Endometriotic Tissue. Canadian Workshop on Human Reproduction and Reproductive Biology "From Bench to Bedside". Miami, FL. April 25 – 29, 1994.

86. **Foster WG**. Environmental contaminants and adverse reproductive health outcomes: Current status and future directions. University of Laval. Quebec City, QC. March 25, 1994.
87. **Foster WG**. Biomarkers in Reproduction: Effects in females. IPCS/Australian workshop on Biomarkers. Adelaide, Australia. October 11 – 15, 1993.

88. **Foster WG**, McMahon A, Rice D. The reproductive effects of chronic Lead exposure male cynomolgus monkey. Loebb Research Institute, Ottawa Civic Hospital, University of Ottawa. Ottawa, ON. January 1993.

89. **Foster WG**, McMahon A, Rice D. The reproductive effects of chronic Lead exposure male cynomolgus monkey. Canadian Wildlife Service. Hull, QC. January 1993.

90. **Foster WG**. Environmental Contaminants and Reproduction. Toxicology Research Division Seminar Series. Ottawa, ON. January 1993.

91. **Foster WG**. Session Moderator: Ovarian Function. 12th Annual Ottawa Reproductive Biology Workshop and 1993 Southern Ontario Reproductive Biology Meeting. May 25 – 26, 1993.

92. **Foster WG**. Environmental Contaminants and Reproduction. Ontario Farm Women's

Association. Guelph, ON. June 1993.

93. **Foster WG**, McMahon A, Rice D. Reproductive toxicity of chronic Lead-exposure in the female and male cynomolgus monkey. Symposium On Lead In Adults. Durham, NC. December 9 – 11, 1991.

ii) Contributions to peer reviewed presentations –

1. Haikalis ME, Wessels JM, Leyland NA, **Foster WG**. Insights into microRNA expression and use as diagnostic markers for endometriosis. 62nd Annual Meeting of the Canadian Fertility & Andrology Society. Toronto, ON. September 22 – 24, 2016.

2. Furlong H, Stämpfli MR, Gannon A, **Foster WG**. Identification of microRNAs as biomarkers for toxic insult in the ovary. 49th Annual meeting of the Society for the Study of Reproduction. San Diego, CA. July 16 – 20, 2016.

3. Haikalis ME, Wessels JM, Leyland NA, **Foster WG**. Preliminary insights on microRNA expression and use as diagnostic markers for endometriosis. 49th Annual Meeting of the Society for the Study of Reproduction. San Diego, CA. July 16 – 20, 2016.

4. Furlong H and **Foster WG**. Novel microRNAs as biomarkers for toxic insult in the ovary. Annual Meeting for the European Society of Human Reproduction and Embryology (ESHRE). Helsinki, Finland. July 3 – 6, 2016.

5. Furlong H and **Foster WG**. Environmental toxicant exposure and dysregulation of ovarian function. The Reproductive Health Summit. London, UK. April 19 – 21, 2016.

6. Guerra MT, Sanabria M, Leite GA, Borges C, Anselmo-Franchi JA, **Foster WG**, Kempinas WG. Maternal exposure to butylparaben impairs testicular structure and function on adult male Wistar rats. 48th Annual Meeting of the Society for the Study of Reproduction. San Juan, Puerto Rico. June 18 – 22, 2015.

7. Wessels JM, Somani A, **Foster WG**. Regulation of brain-derived neurotrophic factor by estrogen, GABA, and melatonin in endometrial epithelial cells. 48th Annual Meeting of the Society for the Study of Reproduction. San Juan, Puerto Rico. June 18 – 22, 2015.

8. Furlong H, Gannon AM, Stämpfli M, **Foster W**. Autophagic cascade triggered by cigarette smoke-induced activation of the AMPK pathway. 48th Annual Meeting of the Society for the Study of Reproduction. San Juan, Puerto Rico. June 18 – 22, 2015.

9. Furlong HC, Stampfli MR, Gannon AM, Foster WG, Cigarette smoke exposure triggers the

autophagic cascade via activation of the AMPK pathway. CIHR Training Program in Reproduction, Early Development and the Impact on Health (REDIH) meeting. Montreal, QC. June 2015.

10. Kosarac I, Kubwabo C, **Foster W**. Determination of urinary metabolites of organophosphate flame retardants using ultra performance liquid chromatography (UPLC) tandem mass spectrometry (MS/MS). 63rd Annual Conference of the American Society for Mass Spectrometry. St. Louis, MO. May 31 – June 4, 2015.

11. Furlong HC, Stampfli MR, Gannon AM, **Foster WG**. Cigarette smoke exposure triggers the autophagic cascade via activation of the AMPK pathway. Research Plenary, Department of Obstetrics & Gynecology, Faculty of Health Sciences, McMaster University. Hamilton, ON. May 2015.

12. Furlong HC, Stampfli MR, Gannon AM, **Foster WG**. Cigarette smoke exposure triggers the autophagic cascade via activation of the AMPK pathway. 54th Annual Meeting of the Society of Toxicology. San Diego, CA. March 22 – 26, 2015.

13. Furlong HC, Stampfli MR, Gannon AM, **Foster WG**. Cigarette smoke exposure triggers the autophagic cascade via activation of the AMPK pathway. CIHR Training Program in Reproduction, Early Development and the Impact on Health (REDIH) meeting. Ottawa, ON. December 2014.

14. Wessels JM, Leyland NA, Agarwal SK, **Foster WG**. Good proteins gone bad: brain-derived neurotrophic factor and its receptors in endometriosis implants and the role of estrogen. Oral presentation at the 70th Annual Meeting of the American Society for Reproductive Medicine. Honolulu, HI. October 18 – 22, 2014.

15. Wessels JM, Leyland NA, Agarwal SK, **Foster WG**. Estrogen regulation of brain-derived neurotrophic factor in the uterus and the link to endometriosis. 60th Annual Meeting of the Canadian Fertility & Andrology Society. Quebec City, QC. September 11 – 14, 2014.

16. Curren MS, Davis K, Liang CL, Adlard B, **Foster WG,** Donaldson SG, Kandola K, Brewster J, Potyrala M, Van Oostam J. Comparison of persistent pollutants (POPs) and metals in primiparous women from Canada and Mexico. 26th Annual Conference of the International Society for Environmental Epidemiology. Seattle, WA. August 24 – 28, 2014.

17. deCatanzaro D, Thorp J, Rajabi N, Partch T, **Foster W**. Novel male exposure and predator stress similarly disrupt blastocyst implantation while suppressing uterine closure and levels of uterine e-cadherin in mice. International Congress of Neuroendocrinology. Sydney, Australia. August 19, 2014.

18. Wessels JM, Leyland NA, Agarwal SK, **Foster WG**. (2015) The Jekyll and Hyde of estrogen induced changes in uterine brain-derived neurotrophic factor and its receptors. 47th Annual Meeting of the Society for the Study of Reproduction. Grand Rapids, MI. July 19 – 23, 2014.

19. Agarwal SK and **Foster WG**. Medical shrinkage of endometriomas with aromatase inhibition and progestin add-back. 69th Annual Meeting of the American Society of Reproductive Medicine. Boston, MA. October 12 – 17, 2013.

20. Wessels JM, Leyland NA, Agarwal SK, Murji A, **Foster WG**. Can brain-derived neurotrophic factor be a clinical marker for endometriosis? Oral presentation at the 69th Annual Meeting of the American Society of Reproductive Medicine. Boston, MA. October 12 – 17, 2013.

21. Wessels J, Leyland N, **Foster WG**. The brain-uterus connection: Uterine expression of brain-derived neurotrophic factor (BDNF) and its receptor vary over the estrous cycle. 46th Annual Meeting of the Society for the Study of Reproduction. Palais des congress de Montréal. Montreal, QC. July 22 – 26, 2013.

22. Curren MS, Liang CL, Davis K, Thuppal V, Said F, Adlard B, Donaldson S, Kandola K, Brewster J, **Foster WG**, Van Oostdam J. Examination of contaminant exposures for populations from northern and southern Canada. Environmental Health 2013, Boston, MA. March, 2013.

23. Gannon AM, Stämpfli MR, **Foster WG**. Dysregulation of mitochondrial dynamics and activation of the autophagy cascade occur in a mouse model of cigarette smoke-induced ovarian follicle loss. 68th Annual Meeting of the American Society of Reproductive Medicine. San Diego, CA. October 20 – 24, 2012.

24. Sadeu JC and **Foster WG**. Mechanism of benzo[a]pyrene-induced inhibition of follicle growth and dysfunction. 68th Annual Meeting of the American Society of Reproductive Medicine. San Diego, CA. October 20 – 24, 2012.

25. Sadeu JC and **Foster WG**. In vitro exposure to benzo[a]pyrene alters the expression of factors controlling follicle growth. 58th Annual Meeting of the Canadian Fertility & Andrology Society. Ottawa, ON. September 6 – 9, 2012.

26. Gannon AM, Stämpfli MR, **Foster WG**. Cigarette smoke exposure triggers dysregulation of mitochondrial dynamics, leading to autophagy-mediated ovarian follicle loss in a mouse model. 45th Annual Meeting of the Society for the Study of Reproduction. Pennsylvania State University. State College, PA. August 12 – 15, 2012.

27. Wessels J and **Foster WG**. Uterine expression of brain-derived neurotrophic factor (BDNF) and its receptor during the estrous cycle and menstrual cycle. 45th Annual Meeting of the

Society for the Study of Reproduction. Pennsylvania State University. State College, PA. August 12 – 15, 2012.

28. Sadeu JC, Doedée AM, Neal M, Hughes EG, **Foster WG**. Neurotrophins (BDNF and NGF) in ovarian follicular fluid of women with different infertility diagnoses. 57th Annual Meeting of the Canadian Fertility & Andrology Society. Toronto, ON. September 21 – September 24, 2011.

29. Gannon AM, Stämpfli MR, **Foster WG**. Cigarette smoke exposure triggers autophagy-mediated ovarian follicle loss in a mouse model. 44th Annual Meeting of the Society for the Study of Reproduction. Oregon Convention Center. Portland, OR. July 31 – August 4, 2011.

30. Sadeu JC, Doedée AM, **Foster WG**. Localization of ovarian neurotrophins (BDNF, NT-4/5 & NGF) and their receptors (Trk B & p^{75NTR}): Role in follicle growth. 2011 Annual Ottawa Reproductive Biology Workshop. Ottawa, ON. June 17, 2011.

31. Cameron H and **Foster WG.** Endocrine toxicants promote resistance to anoikis and invasiveness of breast cancer cells *in vitro.* 50th Anniversary Annual Meeting & ToxExpo, Society of Toxicology. Washington, DC. March 6 – 10, 2011.

32. Mulligan Tuttle AM, Stämpfli M, **Foster WG**. Cigarette smoke exposure results in significant follicle loss via an alternative ovarian cell death pathway. 50th Anniversary Annual Meeting & ToxExpo, Society of Toxicology. Washington, DC. March 6 – 10, 2011.

33. Sadeu JC and **Foster WG.** Cigarette smoke condensate inhibits follicular development, oocyte maturation and dysregulates steroids synthesis *in vitro*: Implications for human fecundity. 50th Anniversary Annual Meeting & ToxExpo, Society of Toxicology. Washington, DC. March 6 – 10, 2011.

34. Johnson NA, Meng WS, Witt-Enderby PA, **Foster WG,** Davis VL. Localized exposure to DDT congeners influence mammary gene expression. 50th Anniversary Annual Meeting & ToxExpo, Society of Toxicology. Washington, DC. March 6 – 10, 2011.

35. Sadeu JC and **Foster WG**. Cigarette smoke condensate (CSC) inhibits follicular development, oocyte nuclear maturation and disrupts progesterone synthesis *in vitro*: Implications for human fecundity. 56th Annual Meeting of the Canadian Fertility & Andrology Society. Vancouver, BC. September 29 – October 2, 2010.

36. Doedée A, Sadeu JC, **Foster W**G. The effects of bisphenol A (BPA) on the expression of neurotrophins (NTs) and neurotrophin receptors (NTRs) during *in vitro* follicle growth. 56th Annual Meeting of the Canadian Fertility & Andrology Society. Vancouver, BC. September 29 – October 2, 2010.

37. Mulligan Tuttle A and **Foster WG**. Cigarette smoke exposure results in significant follicle loss and decreased pro-survival expression in an *in vivo* mouse model. 56th Annual Meeting of the Canadian Fertility & Andrology Society. Vancouver, BC. September 29 – October 2, 2010.

38. Mallach G, Davidson A, Arbuckle T, Nethery E, Van Ryswk K, You H, Fisher M, **Foster W**, Moore E, Ripley D, Wheeler AJ. Assessing the Value of Including GPS in Personal Exposure Monitoring. ISES-ISEE 2010 Joint Conference of International Society of Exposure Science & International Society for Environmental Epidemiology. Seoul, Korea. August 28 – September 1, 2010.

39. Sadeu JC and **Foster WG.** Benzo[a]pyrene (B[a]P)-treatment at concentrations representative of human exposure attenuates ovarian follicle development and survival. 49th Annual Meeting of the Society of Toxicology. Salt Lake City, UT. March 7 – 11, 2010.

40. Lagunov A, Sadeu JC, Bruin JE, Woynillowicz AK, Anzar M, Khan MIR, Buhr M, Holloway AC, **Foster WG**. Effect of *in utero* and lactational nicotine exposure on the male reproductive tract in pubertal and adult rats. 55th Annual Meeting of the Canadian Fertility & Andrology Society. Montreal, QC. November 18 – 21, 2009.

41. Neal MS and **Foster WG**. Aryl hydrocarbon receptor (AhR) antagonists attenuates the deleterious effect of benzo[a]pyrene on isolated rat follicle growth *in vitro*. 54th Annual Meeting of the Canadian Fertility & Andrology Society. Calgary, AB. November 26 – 29, 2008.

42. Dominguez MA, Zhang B, **Foster WG**. BDNF and Trk B expression in the mouse ovary. 54th Annual Meeting of the Canadian Fertility & Andrology Society. Calgary, AB. November 26 – 29, 2008.

43. Mulligan Tuttle A, Stämpfli M, **Foster WG**. *In vivo* and *in vitro* follicle loss caused by cigarette smoke and benzo[a]pyrene exposure at physiologically relevant concentrations. 54th Annual Meeting of the Canadian Fertility & Andrology Society. Calgary, AB. November 26 – 29, 2008.

44. Boutross-Tadross O, Faghih M, Elias R, Elit L, **Foster WG**. Immunolocalization of tyrosine kinase receptor B (TrkB) expression in endometriosis associated ovarian cancer (EAOC) cells. 3rd Intercontinental Congress of Pathology. Barcelona, Spain. May 17 – 22, 2008.

45. Mulligan Tuttle A and **Foster WG**. Cigarette smoke and benzo[a]pyrene cause follicle loss *in vivo* and *in vitro* at physiologically relevant concentrations. 47Th Annual Meeting of the Society of Toxicology. Seattle, WA. March 16 – 20, 2008.

46. Monroy R, Bourgeois J, Shaw D, Morrison K, Atkinson S, Teo K, **Foster WG**. Effects of cigarette smoking in pregnancy on the placenta vasculosyncytial membrane thickness. 47th Annual Meeting of the Society of Toxicology. Seattle, WA. March 16 – 20, 2008.

47. Mulligan Tuttle A and **Foster WG.** Cigarette smoke causes follicle loss *in vivo* at physiologically relevant concentrations. 4th Annual Invitational Symposium for Research to Inform tobacco Control. A pre-conference symposium at the Society for Research on Nicotine and Tobacco Annual Conference. Portland, OR. February 2008.

48. Davis VL, Johnson NA, Jayo MJ, Hughes CL, **Foster WG**. Localized exposure to *p,p'* DDE accelerates mammary tumor development in MMTV-*neu* transgenic mice. Future Research on Endocrine Disruption: Translation of Basic and Animal Research to Understand Human Disease. Durham, NC. August 27 – 29, 2007.

49. Monroy R, Bourgeois J, Shaw D, Morrison K, Teo K, Atkinson S, **Foster WG**. Effects of maternal smoking on the placenta vasculosyncytial membrane thickness. 13th International Federation of Placenta Associations. Kingston, ON. August 17 – 22, 2007. Poster of Mention and Y. W. Loke New Investigator Award.

50. Cameron HL and **Foster WG.** The organochlorine pesticide dieldrin increases resistance to anoikis and invasiveness of breast cancer cells *in vitro.* American Association for Cancer Research Edward A. Smuckler Memorial Pathobiology of Cancer Workshop, Snowmass Village, CO. July 15 – 22, 2007.

51. Faghih M, Elias R, Boutross-Tadross O, Elit L, **Foster WG**. Immunolocalization of tyrosine kinase receptor B (TrkB) expression in endometriosis associated ovarian cancer (EAOC) cells. Society of Gynaecologists of Canada Annual Clinical Meeting. June 23, 2007.

52. Cameron HL and **Foster WG.** Developmental and lactational exposure to environmentally relevant concentrations of dieldrin in neu/ErB2 transgenic mice. American Association for Cancer Research special conference. Albuquerque, NM. May 30 – June 2, 2007.

53. Neal M, Holloway AC, **Foster WG**. Ovotoxicity of benzo[a]pyrene and the ovarian protective effects of aryl hydrocarbon receptor antagonist. 52nd Annual Meeting of the Canadian Fertility & Andrology Society Meeting. Ottawa, ON. November 15 – 18, 2006. Organon Canada Ltd. Ontario Region Student/Resident Award.

54. **Foster WG**, Monroy R, Morrison K, Teo K, Atkinson S, Kubwabo C. Serum Levels of perfluorinated compounds in human maternal and umbilical cord blood samples. International Council of Chemical Associations. Minneapolis, MN. July 26 – 27, 2006.

55. Neal MS, Petrik J, **Foster WG**, Holloway AC. *In utero* and lactational exposure to nicotine: ovarian effects. Conjoint American Society for Reproductive Medicine and Canadian Fertility & Andrology Society Meeting. Montreal, QC. October 16 – 19, 2005. Best Basic Science Paper and Alpha Award.

56. Neal MS, Zhu J, **Foster WG**. Quantification of benzo-[*a*]-pyrene (B[*a*]P) in serum and follicular fluid and its effects on follicle growth in an isolated follicle culture assay. Conjoint American Society for Reproductive Medicine and Canadian Fertility & Andrology Society Meeting. Montreal, QC. October 16 – 19, 2005.

57. Stys KA, Zhang B, Holloway AC, **Foster WG**. Dioxin-induced changes in endometrial aromatase expression and activity. Society for the Study of Reproduction. Quebec City, QC. July 24 – 27, 2005.

58. Neal MS, Lim GE, YoungLai EV, Daya S, Holloway AC, **Foster WG**. Aromatase activity in granulosa cells as a predictor of pregnancy. International Federation of Fertility Societies. Montreal, QC. May 23 – 28, 2004.

59. **Foster WG**, Agarwal SK, Holloway AC. 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) induced increased endometrial aromatase activity is mediated through altered PGE_2 metabolism. 51st Annual Meeting of the Society for Gynecologic Investigation. Huston, TX. March 24 – 27, 2004.

60. Edmunds KE, Holloway AC, Beecroft ML, Daya SH, Crankshaw DJ, **Foster WG**. The effects of dietary phytoestrogens on aromatase activity in endometrial stromal cell cultures. 51st Annual Meeting of the Society for Gynecologic Investigation. Houston TX. March 24 – 27, 2004.

61. Lim GE, Stals SI, **Foster WG**, Petrik JJ, Holloway AC. Fetal exposure to water disinfection byproducts alters postnatal growth and glucose homeostasis. 51st Annual Meeting of the Society for Gynecologic Investigation. Houston TX. March 24 – 27, 2004.

62. **Foster WG**, Wade MG, Hughes CL, YoungLai EV. Developmental exposure to a complex mixture of environmental toxicants interacts with postnatal genistein to induce changes in reproductive development of female Sprague Dawley rats. 43rd Annual Meeting of the Society of Toxicology. Baltimore, MD. March 21 – 25, 2004.

63. YoungLai E, Holloway A, Lim G, Neal M, **Foster W.** Synergistic effects of FSH & DDE on human granulosa cell aromatase. 49th Annual Meeting of the Canadian Fertility & Andrology Society. Victoria, BC. November 5 – 8, 2003.

64. YoungLai E, Kakuda N, Neal M, **Foster W**, Buhr M. Additive effects of progesterone &

DDE on calcium flux in human sperm. 49th Annual Meeting of the Canadian Fertility & Andrology Society. Victoria, BC. November 5 – 8, 2003.

65. Neal M, Holloway A, Hughes E, **Foster W**. Effect of sidestream and mainstream smoking on IVF outcomes. 49th Annual Meeting of the Canadian Fertility & Andrology Society. Victoria, BC. November 5 – 8, 2003.

66. Chomej A, Holloway A, **Foster W**. Effects of Bisphenol A on tissue remodeling enzymes in human luteinized granulosa cells *in vitro*. 49th Annual Meeting of the Canadian Fertility & Andrology Society. Victoria, BC. November 5 – 8, 2003.

67. Miller M, Holloway AC, **Foster WG**. Benzo-a-pyrene alters invasion in human breast cancer cell lines BT-474 and MDA-MB-231 through altered prostaglandin E_2 (PGE_2) metabolism. 2nd Annual AACR International Conference, Frontiers in Cancer Prevention Research. Phoenix, AZ. October 26 – 30, 2003.

68. Holloway A, Beecroft ML, Sinasac S, YoungLai E, Daya S, Edmunds K, **Foster WG**. Environmental Toxicant Induced Changes in Aromatase Activity in Estrogen Sensitive Target Tissues. Society for the Study of Reproduction, 36th Annual Meeting. Cincinnati, OH. July 19 – 22, 2003.

69. WWW.EMCOM.CA - A risk communication vehicle about endocrine disruption. Phillips KP, Aronson KJ, Brunet P, **Foster WG**, Kacew S, Leiss W, Mehta M, Poirier R, Salem T, Van Der Kraak G, Wade MG, Walker M, Wigle D, Krewski D. Society for the Study of Reproduction, 36th Annual Meeting. Cincinnati, OH. July 19 – 22, 2003.

70. Davis VL, Jayo MJ, Hardy ML, Ho A, Shaikh F, Lee H, **Foster WG**, Hughes CL. Effects of black cohosh on mammary tumor development and progression in MMTV-*neu* transgenic mice. American Association of Cancer Researchers. Washington, DC. July 11 – 14, 2003.

71. Davis VL, **Foster W**, Jayo M, Ho A, Shaikh F, Lee H, Hughes C. Influence of Locally Stored DDT Metabolites on Mammary Cancer Development induced by the *neu* proto-oncogene. American Association of Cancer Researchers. Washington, DC. July 11 – 14, 2003.

72. Hughes CL, Davis V, Shaikh F, Villegas M, Ho A, **Foster WG.** The effects of *in utero* and Lactational Exposure to Genistein or Daidzein on reproductive development in FVB/N mice and occurrence of mammary tumors in MMTV-*neu* transgenic mice. US-EPA Sponsored Endocrine Disruptors Workshop. Research Triangle Park, NC. October 29 – 31, 2002.

73. **Foster W**, Sinasac S, Holloway A. Histopathological changes in endometrial stroma and epithelium of 2,3,7,8-tettachlorodibenzo-*p*-dioxin (TCDD) treated monkeys. 48th Annual Meeting of the Canadian Fertility & Andrology Society. Charlevoix, QC. September 25 – 28,

2002.

74. **Foster WG**, Daya SH, Holloway AC. Inappropriate estrogen production induced by environmental toxicants. 48th Annual Meeting of the Canadian Fertility & Andrology Society. Charlevoix, QC. September 25 – 28, 2002.

75. Davis VL, Villegas M, Shaikh F, Ho A, **Foster WG**, Hughes CL. The Effects of *in Utero* and Lactational Exposure to the Soy Isoflavones, Genistein and Daidzein, on Reproductive Development of Male and Female Mice. Society for Gynecologic Investigation. Los Angeles, CA. March 20 – 24, 2002.

76. Helliwell J, Agarwal SK, **Foster WG**. Endometriosis and rheumatological disease: An observational study using diagnostic criteria from the American Board or Rheumatology. Society of Gynecologic Investigation. Los Angeles, CA. March 20 – 24, 2002.

77. Rodriquez S, Estrada S, **Foster W,** Agarwal SK. What motivates women to take part in clinical and basic science endometriosis research? World Congress on Endometriosis. San Diego, CA. February 24 – 27, 2002.

78. Agarwal SK, Estrada S, **Foster WG**. Pilot study evaluating the efficacy of delorelin with add-back low-dose sex steroids for the treatment of pelvic pain secondary to laparoscopically confirmed endometriosis. World Congress on Endometriosis. San Diego, CA. February 24 – 27, 2002.

79. **Foster WG**. Yang JZ. Fournier M. Immunological effects of 2,3,7,8-tetrachlorodiebnzo-p-dioxin (TCDD) in monkeys with endometriosis. 47th Annual Meeting of the Canadian Fertility & Andrology Society. Whistler, BC. October 3 – 6, 2001.

80. YoungLai EV. Wade MG. **Foster WG**, Hughes CL. Endocrine disrupting effects of a mixture of environmentally relevant pollutants in the pregnant female. 47th Annual Meeting of the Canadian Fertility & Andrology Society. Whistler, BC. October 3 – 6, 2001.

81. Wade MG, **Foster WG**, YoungLai EV, Desaulniers D, Hughes CL. Thyroid and reproductive effects of *in utero*/lactational exposure to a mixture of persistent environmental contaminants. Canadian Federation of Biological Societies, 44th Annual Meeting. Ottawa, ON, June 20 – 23, 2001.

82. Agarwal SK, Surrey MW, **Foster WG**, Harris R. The occurrence of and screening for appendiceal disease in women with endometriosis and right lower quadrant pain. 49th Annual Meeting of the Pacific Coast Reproductive Society. Rancho Mirage, CA. April 2001.

83. **Foster WG**, Chan S, Platt L, Hughes C. Detection of phytoestrogens in samples of second

trimester human amniotic fluid. 46th Annual Meeting of the Canadian Fertility & Andrology Society. St. John's, NL. September 13 – 16, 2000.

84. Liu G, Blas-Machado U, Duong Q, Lee H, **Foster WG**, Davis VL, Magoffin DA, Hughes CL. Long-term exposure to high lactose diet fails to induce ovarian dysfunction in Long Evans rats. Society for the Study of Reproduction. Madison, WI. July 15 – 18, 2000.

85. Shridhar S, Farley A, Reid RL, **Foster W**, Van Vugt DA. Tetrachlorodibenzo-p-dioxin (TCDD) on gene expression of Corticotropin Releasing Hormone, Arginine Vasopressin, and Pro-opiomelanocortin in the hypothalamus of non-human primates. 82nd Annual Meeting of the Endocrine Society. Toronto, ON. June 21 – 24, 2000.

86. YoungLai EV, **Foster WG**, Wade MG, Hughes CL. Effect of a mixture of environmental contaminants on the male rat. American Society of Andrology. Boston, MA. April 7 – 11, 2000.

87. Hughes C, **Foster W**, Platt L, Chan S, Thompson S, Hubbard S, DuBose A, Tyrey L. Midgestation intrauterine exposure of the human fetus to dietary isoflavones in North America: How does this exposure compare to animal studies in late gestation and lactation that alter developmental endpoints. Third International Symposium on The Role of Soy in Preventing and Treating Chronic Disease. Washington, DC. October 31 – November 3, 1999.

88. **Foster WG**, Chan S, Platt L, Hughes C. *In utero* exposure of the human fetus to xenobiotics endocrine disrupting chemicals: Detection of organochlorine compounds in samples of second trimester human amniotic fluid. 81st Endocrine Society Annual Meeting. San Diego, CA. June 12 – 15, 1999.

89. Douglas GR, Gingerich JD, Soper LM, McMahon A, **Foster WG**. Gene mutations in follicular granulosa cells of super-ovulated lacZ transgenic mice. Environmental Mutagen Society Meeting. Washington, DC. March 27 – April 1, 1999.

90. Desaulniers D, Leingartner K, Cooke GM, Wade M, Yang J, Yagminas A, **Foster WG**. Effects of a "Human-milk PCB-DDT-DDE" mixture on MCF-7 cell proliferation *in vitro* and on nitrosomethylurea-induced mammary tumours in the rat. Society of Toxicology of Canada. Montreal, QC. December 2 – 3, 1998.

91. Ruka MP, Gareau PJ, Janzen EG, **Foster WG**. Use of MRI T1/T2 weighted images to evaluate the lesions in surgically induced endometriosis in a mouse model. Royal College of Physicians and Surgeons Annual Meeting. Toronto, ON. September 24 – 27, 1998.

92. Desaulniers D, Leingartner K, Fintelman E, Wade M, Yagminas A, **Foster WG**. Relative sensitivity of several endocrine biomarkers following acute exposure to PCB-126 and 153 in

adult Sprague Dawley male rats. Health Conference '97. Montreal, QC. May 12 – 15, 1997.

93. Buttar HS, Moffatt JH, McMahon A, **Foster WG**. Sperm motility analysis, sperm chromatin strucutre assay and serum hormone assessment in alph-chlorohydrin treated rats. Society of Teratology. Palm Beach, FL. June 21 – 27, 1997.

94. Van Vugt DA, Kizemian A, Roy BN, **Foster W**, Lundahl S, Marcus S, Reid RL. Photodynamic endometrial ablation in non-human primates. 42nd Annual Meeting of the Canadian Fertility & Andrology Society. Lake Louise, AB. November 20 – 23, 1996.

95. Yang JZ, **Foster WG**. Repeated exposure to TCDD inhibits growth of surgically induced endometriosis in the mouse. Society for the Study of Reproduction. London ON. 1996.

96. Van Vugt DA, Kizemian A, Roy BN, **Foster W**, Lundahl S, Marcus S, Reid RL. Photodynamic endometrial ablation in rhesus monkeys: 22nd Annual Meeting of the Society of Obstetrics & Gynaecology Canada. 1996.

97. **Foster WG**, Desaulniers D, Chu I, Poon R. Reproductive effects of tris (4-chlorophenyl) methanol in the male rat. 41st Annual Meeting of the Canadian Fertility & Andrology Society. Montebello, QC. September 19 – 23, 1995.

98. Jarrell JF, Gocmen A, **Foster WG**. A long-term review of the human reproductive outcomes of inadvertent exposure to hexachlorobenzene. 41st Annual Meeting of the Canadian Fertility & Andrology Society. Montebello, QC. September 19 – 23, 1995.

99. Yang JZ, Van Vugt DA, Reid RL, **Foster WG**. Intrauterine 5-aminolevulinic acid induces selective endometrial photosensitization in the Rhesus and Cynomolgus monkeys. 41st Annual Meeting of the Canadian Fertility & Andrology Society. Montebello, QC. September 19 – 23, 1995.

100. YoungLai EV, **Foster WG**, Jarrell JF. Relationship between environmental contaminants in human reproductive fluids and results of *in vitro* fertilization. 41st Annual Meeting of the Canadian Fertility & Andrology Society. Montebello, QC. September 19 – 23, 1995.

101. Ruka MP, McCutcheon JL, Gareau PJ, **Foster WG**, Janzen EG. Surgically-induced model of endometriosis in B6C3F1 mice. Royal College of Physicians & Surgeons of Canada, 64th Annual Meeting. Montreal QC, September 13 - 17, 1995.

102. Ruka MP, McCutcheon JL, Gareau PJ, **Foster WG**, Janzen EG. Evaluation of estrogen/progesterone treatment on the functional integrity of the endometrium temporally exposed to warm ischemia prior to its auto-transplantation. Royal College of Physicians & Surgeons of Canada, 64th Annual Meeting. Montreal, QC. September 13 – 17, 1995.

103. **Foster WG**, Rice DC, McMahon A, Reed BL. Altered semen quality in Cynomolgus monkeys with occupationally relevant circulating concentrations of lead. Society for the Study of Reproduction. Davis, CA. July 9 – 12, 1995.

104. Desaulniers DM, Leingartner K, Stoddart K, **Foster WG**. Development of an *in vitro* bioassay to assess the estrogenic potential of xenobiotics using cloned and wild types MCF-7 cells. Society for the Study of Reproduction. Davis, CA. July 9 – 12, 1995.

105. Desaulniers D, Leingartner K, **Foster WG**. Comparison of proliferation indices derived from 3H-thymidine incorporation and the metabolic reduction of alamar blue (Tm) in estradiol-stimulated human breast cancer cells. 14th Annual Ottawa Reproductive Biology Workshop. Ottawa, ON. June 2 – 3, 1995.

106. **Foster WG**, Jarrell JF, YoungLai EV. Preliminary results of a survey of contaminants present in serum and follicular fluid of women participating in fertility clinics in six Canadian cities. Southern Ontario Reproductive Biology Workshop. Kingston, ON. May 5, 1995.

107. **Foster WG**, McMahon A, Reed BL, Rice DC. Flow cytometric and conventional analysis of semen from chronically lead exposed Cynomolgus monkeys (Macaca fasicularis). Southern Ontario Reproductive Biology Workshop. Kingston, ON. May 5, 1995.

108. YoungLai EV, Baillie J, Yie S-M, Hughes EG, Collins JA, **Foster WG**. Acrosin activity in frozen sperm samples does not correlate with *in vitro* fertilization of human oocytes. International Federation of Gynaecology & Obstetrics (FIGO). Montreal, QC. September 24 – 30, 1994.

109. **Foster WG**, Rice DC, McMahon A. Suppression of luteal function in the chronically lead exposed cynomolgus monkey (*Macaca fascicularis*). 40th Annual Meeting of the Canadian Fertility & Andrology Society. St. John, NB. September 7 – 10, 1994.

110. **Foster WG**, Yagminas A, McMahon A. Reverse phase HPLC demonstration of cortisol, corticosterone, progesterone, and 17 a-hydroxyprogesterone in rat serum. 40th Annual Meeting of the Canadian Fertility & Andrology Society. St. John, NB. September 7 – 10, 1994.

111. YoungLai EV, Yie S-M, Hughes EG, Collins JA, **Foster WG**. Seasonal variation in hormones in human follicular fluid and granulosa cell steroidogenesis. 40th Annual Meeting of the Canadian Fertility & Andrology Society. St. John, NB. September 7 – 10, 1994.

112. **Foster WG**, McMahon A, YoungLai EV, Hughes EG. Ovarian toxicity of hexachlorobenzene in the cynomolgus monkey (*Macaca fascicularis*). Society for the Study of Reproduction,

27th Annual Meeting. Ann Arbor, MI. August 24 – 27, 1994.

113. **Foster WG**. Alterations in ovarian function following exposure to hexachlorobenzene (HCB) in the rodent and primate model. 13th Annual Ottawa Reproductive Biology Workshop. Ottawa, ON. June 8, 1994.

114. **Foster WG**, Rice DC, McMahon A. Adverse effects of low circulating lead levels on menstrual cycle characteristics in the monkey. Southern Ontario Reproductive Biology Workshop (Tuck et al.), Hamilton, ON. May 6, 1994.

115. **Foster WG**. Steroid actions on endometriotic tissue. The Canadian Workshop on Human Reproduction and Reproductive Biology. Miami, FL. April 25 – 29, 1994.

116. Singh A, Bourque A, Lakhanpal N, McMahon A, **Foster WG**. Electron microscopy of ovary from the monkey administered hexachlorobenzne. Society of Toxicology. Dallas, TX. March 12 – 18, 1994.

117. Bourque A, Singh A, Dykeman A, McMahon A, **Foster W**. Hexachlorobenzene at low doses produces lesions in nonhuman primate ovary. 26th Annual Meeting of the USGEB/USSBE. University of Bern, Switzerland. March 17 – 18, 1994.

118. **Foster WG**, McMahon A, YoungLai EV, Hughes EG. Ovarian toxicity of hexachlorobenzene in the cynomolgus monkey (*Macaca fascicularis*). Montreal area Reproductive and Developmental Biologists First Annual Research Day. Montreal, QC. November 15, 1993.

119. **Foster WG**. Biomarkers in Reproduction - Effects in females. IPCS/Australian Workshop on Biomarkers. Adelaide, Australia. October 11 – 15, 1993.

120. Cullen C, Singh A, **Foster WG**. Electron microscopy of monkey seminal vesicle. 159th National Meeting of the American Association for the Advancement of Science. Boston, MA. February 11 – 16, 1993.

121. **Foster WG**, McMahon A. Effects of organochlorine contaminants on steroidogenesis in the rat. 12th Annual Ottawa Reproductive Biology Workshop and 1993 Southern Ontario Reproductive Biology Meeting (Tuck et al.). May 25 – 26, 1993.

122. Singh A, Dykeman A, Rice D, **Foster WG**. Electron microscopy of testis from the monkey fed Lead: A 9-year study. American Association of Veterinary Anatomists. July 15 – 18, 1993.

123. **Foster WG**, McMahon A, YoungLai EV, Hughes EG, Rice DC. Reproductive endocrine effects of chronic Lead exposure in the male cynomolgus monkey (*Macaca fascicularis*). 38th

Annual Meeting of the Canadian Fertility & Andrology Society. November 27, 1992.

124. Todoroff EC, Sevcik M, Brännstrom M, Janson PO, **Foster WG**, Villeneuve DC, Jarrell JF. The effect of photomirex on the *in vitro* perfused ovary of the rat. 38th Annual Meeting of the Canadian Fertility & Andrology Society. November 27, 1992.

125. Reid RL, Kennedy JC, Van Vugt DA, Yang JZ, Fletcher A, **Foster W**. Evidence in the human and the non human primate for aminolevulinic acid (ALA) induced selective endometrial photosensitization: A potential agent for photodynamic ablation of the endometrium. Society for Gynecologic Investigation. March 31 – April 3, 1993.

126. Singh A, **Foster WG**, Dykeman A, Villeneuve DC. Hexachlorobenzene toxicity in the rat ovary II. Ultrastructure induced by medium (10 mg/kg) dose exposure. Proceedings of the 50th Annual Meeting, Electron Microscopy Society of America. August 16 – 21, 1992.

127. Singh A, **Foster WG**, Arendz J. Chronic Lead exposure induces ultrastructural alterations in the monkey seminal vesicle. C.V.M.A. July 5 – 8, 1992.

128. Singh A, **Foster W**, McMahon A, Villeneuve DC. Lead-induced alterations in the testis of monkeys: An ultrastructural study. Annual Meeting, Society of Toxicology. February 23 – 27, 1992.

129. Singh A, **Foster WG**, McMahon A, Rice DC, Villeneuve DC. Electron microscopy of seminal vesicles from monkeys exposed to Lead: A 9-year study. 24th Annual Meeting, USGEB/USSBE. March 19 – 20, 1992.

130. Singh A, **Foster W**, Villeneuve D. Hexachlorobenzene-induced alterations in the ovary of rats. 105th Annual Meeting, American Association of Veterinary Anatomists. March 1992.

131. **Foster WG**, McMahon A, Pentick JA. Hexachlorobenzene (HCB) augments circulating progesterone concentration in the female rat. 34th Annual Meeting Canadian Federation of Biological Societies. (Abstract) p. 95. 1991.

132. McMahon A, **Foster WG**, Pentick JA, Lecavalier PR. Tissue distribution and ovarian subcellular localization of Hexachlorobenzene (HCB) in the female rat. 34th Annual Meeting Canadian Federation of Biological Societies. (Abstract) p. 95. 1991.

133. **Foster WG**, McMahon A, Villeneuve DC, Jarrell JF. Hexachlorobenzene (HCB) suppresses progesterone secretion during the luteal phase in the female cynomolgus monkey. 73rd Annual Meeting of The Endocrine Society. (Abstract) P. 118. 1991.

134. **Foster WG**, Jarrell JF, Younglai EV. Sexual maturation in the female rabbit: Effect of

Tamoxifen and Pregnant Mare Serum. 32nd Annual Meeting Canadian Federation of Biological Societies. (Abstract) p. 35. 1989.

135. **Foster WG**, Jarrell JF, Younglai EV. Light and ultrastructural characterisation of gonadotropin hormone-releasing hormone (GnRH) neurones in the rabbit. 31st Annual Meeting Canadian Federation of Biological Societies. (Abstract) p. 114. 1988.

136. **Foster WG**, Jarrell JF, Dolovich J, Younglai EV. IgE mediated hypersensitivity in response to chronic treatment with Gonadorelin-HC_l (Factrel) in a female patient. 44th Annual Meeting Society of Obstetrics & Gynecology Canada. (Abstract) p. 105. 1988.

KNOWLEDGE TRANSLATION:

i) **Consulting – Government & Industry**

2014 – present The Endometriosis Association, USA., Scientific Advisory Panel. *The Endometriosis Association Advisors are selected for their expertise on specific scientific and/or clinical aspects of endometriosis. They advise the Association on important matters related to endometriosis, review research proposals the Association might fund, provide a sounding board for Association board and staff, and help promote the Association's mission.*

2012 – present Development and Reproductive Toxicology (DART), International Life Science Institute (ILSI), Health and Environmental Sciences Institute (HESI), Washington DC. *Elected to serve as one of only two academic science advisors to the International Life Sciences Institute Developmental and Reproductive Toxicology Panel. As a panel member participate in setting panel objectives and budget, contribute to panel sponsored research projects, and authoring technical reports.*

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2014 – 2015 Health Canada, Public Health Agency of Canada (PHAC), Ottawa ON. *I was invited to carry out a systematic review of the literature relating exposure to environmental contaminants and development of congenital anomalies. This report was prepared under contract to PHAC and was used to guide PHAC decisions on inclusion of occupational groups and chemical exposure priorities for inclusion in a new Canadian congenital anomalies surveillance program. Results of this contract have also been accepted for publication in the peer reviewed scientific literature.*

2013 – 2014 Exponent, Inc., Alexandria VA. *Provided expert technical advice for inclusion in government submission on the relevance of exposure to hormonally active chemicals in adverse human health outcomes.*

2011	Institute of Medicine of the National Academies, "Veterans and Agent Orange: 8th Biennial Update". *By invitation provided expert technical advice on the reproductive health implications associated with exposure to Agent Orange.*
2008	Government of Canada: Risk Management of Toxic Substances - Canadian Environment Network, Sound Management of toxic substances. *Invited to provide expert technical advice for chemical substance prioritization, methods for hazard identification, and risk assessment to protect the health of Canadians.*

ii) **Reports** –
Reviewer for Ecojustice, Toronto ON, Special review of Atrazine: Proposed Decision for Consultation, Health Canada, January 12, 2016.

iii) **Media** –

Magazine	Scientia	Searching for new biomarkers of endometriosis http://www.scientiapublications.com/professor-warren-foster-science-diffusion/ May/June 2016.
Newspaper	The Hamilton Spectator	How worried should we be about bisphenol A? April 15, 2016.
Press Release	Science Media Centre	Expert reaction to study on endocrine disrupting chemicals and onset of menopause in women. January 28, 2015.
Radio	CHML AM900 – The Scott Thompson Show	Bisphenol A (BPA) and its' association with exposure and risk for fertility problems, diabetes, and other adverse health conditions. January 16, 2015.
Press Release	Science Media Centre	Expert reaction to phthalates in pregnancy and asthma in children. September 17, 2014.
Public Lecture	McMaster University	Characterization of a novel clinical marker of endometriosis. September 4, 2013.
Press	Science Media Centre	BPA, brain and behavior – experts. May 27, 2013.

Release		
Press Release	Science Media Centre	Expert reaction to research into urinary bisphenol A and arterial stenosis. August 15, 2012.
Newsletter	Plastics Europe	Questioning the latest BPA "scare story". May 16, 2012.
Newsletter	Food Production – Rory Harrington	Study conclusion over bisphenol A breast cancer hazard challenged by scientists. May 9, 2012.
Newsletter	Hamilton Health Sciences	Enlightening girls who 'light up' about reproductive health. September 1, 2011.
Television	BBC News, Europe	EU bans bisphenol A chemical from babies' bottles. November 25, 2010.
Public Lecture	Kiwanis Club of Oakville Ontario Canada	The environment and its' implication to cancer and other diseases. November 15, 2010.
Newspaper	CBC News: The Associated Press	Higher BPA levels in urine tied to fewer sperm. October 28, 2010.
Television	CTV News: The Canadian Press	Higher BPS levels linked to lower semen quality. October 28, 2010.
Press Release	Science Media Centre	Should bisphenol A be banned? December 4, 2009.
Newspaper	The Hamilton Spectator	Researchers tap local mothers-to-be. May 21, 2009.
Newspaper	The Canadian Press	Lead levels in blood plummet after lead in gas, paints phases out. November 19, 2008.
Magazine	Today's Parent – Wendy Haaf	Scent of a baby – The flap over phthalates. December 1, 2008.
Newspaper	The Hamilton Spectator	Lead is common, but high levels pose risks. November 11, 2008.

Television	CTV News: The Canadian Press	Toxic shower curtains? Study overblown, experts say. June 12, 2008.
Public Lecture	The Probus Club of Hamilton Mountain	Environmental hazards posed by chemicals and the problems associated with trying to understand the true risk. May 1, 2008.
Radio	WAMU 88.5 – The Dianne Rehm Show, Washington DC	Bisphenol A exposure and human health concerns. April 29, 2008.
Newspaper	The Canadian Press – Anne-Marie Tobin	Sperm under siege: Though a man makes 100 million sperm every day, a variety of chemicals and workplace conditions put reproduction at risk. April 22, 2008.
Television	CTV News: The Canadian Press	Environment affects how well men's 'boys' can swim. April 21, 2008.
Magazine	Backpacker	Bottle Blues: Canada calls Nalgene plastic toxic. April 16, 2008.
Newspaper	The New York Times – Ian Austen	Canada likely to label plastic ingredient toxic. April 16, 2008.
Newspaper	The News, Healthy Living, Ancaster/ Dundas Edition	Toxic baby bottle scare or simply scary science? February 15, 2008.
Public Lecture	The Hamilton Spectator Auditorium, Science in the City	Hormone mimics and human health. May 9, 2006.

ADMINISTRATIVE RESPONSIBILITIES:

i) **Local** – McMaster University/Hamilton Health Sciences

1. Representative, Animal Research Ethics Board, Faculty of Health Sciences, 2016 – present.
2. Member, Tenure & Promotion Committee, Department of Obstetrics & Gynecology, 2008 – present.
3. Member, Animal Advisory Committee, Faculty of Health Sciences, 2007 – present.

4. Member, Graduate Curriculum Committee, Faculty of Health Sciences, 2006 – present.
5. Member, Undergraduate Hearing Committee, Faculty of Health Sciences, 2005 – present.
6. Member, Finance Management Committee, Department of Obstetrics & Gynecology, 2004 – present.
7. Member, Resident Program Evaluation Committee, Department of Obstetrics & Gynecology, 2003 – present.
8. Member, Postgraduate Education Committee, Department of Obstetrics & Gynecology, 2003 – present.
9. Member, Search Committee for Chair, Department of Obstetrics & Gynecology, 2009 – 2010.
10. Member, Reproductive Endocrinology and Infertility Fellowship Committee, Department of Obstetrics & Gynecology, 2008 – 2010.
11. Member, Undergraduate Student Appeals Committee, Faculty of Health Sciences, 2005 – 2010.
12. Co-theme Team Leader, Environment & Health, Collaborations for Health, 2005 – 2010.
13. Director, Reproductive Biology Division, Department of Obstetrics & Gynecology, 2002 – 2010.
14. Coordinator, Resident Research Program, Department of Obstetrics & Gynecology, 2002 – 2010.
15. Member, Research Advisory Committee, Hamilton Health Sciences, 2006 – 2009.
16. Member, Search Committee for Associate Dean, Graduate Studies, 2007 – 2008.
17. Medical Director, Centre for Reproductive Care, Hamilton Health Sciences, 2005 – 2008.
18. Member, BioSafety Committee, Faculty of Health Sciences, 2003 – 2005.

ii) **National -**

1. Member, Grants Review Committee, Canadian Breast Cancer Fund, 2014 – present.
2. Member, Operating Grant Review Panel, Gender & Health Peer Review Committee, Canadian Institutes of Health Research (CIHR), 2013 – present.
3. Board Member, The Anti-NMDA Receptor Encephalitis Foundation, Inc, 2012 – present.
4. Mentor, CIHR Strategic Training Program in Tobacco Research (CIHR-STPTR), University of Waterloo, 2005 – present.
5. Member of Expert Registry, US-National Toxicology Program, Center for the Evaluation of Risk to Human Reproduction, 2005 – present.
6. Member, Research Advisory Board of the Reproductive and Developmental Origins of Health, Disability and Disease, Queen's University, 2010 – 2016.
7. Member and Mentor, CIHR Training Program in Reproduction, Early Development, and the Impact on Health (REDIH), University of Ottawa, 2009 – 2016.
8. Member, Gender, Sex & Health, Committee for the Transitional Operating Grant, CIHR, 2013 – 2015.
9. Member, Clinical Investigation 'A', Grants Committee Panel, CIHR, 2013 – 2014.
10. Member, Catalyst Grant Committee, Genes and Chronic Disease, CIHR, 2013.
11. Affiliate, R. Samuel McLaughlin Centre for Population Health Risk Assessment, 2008 – 2013.

12. Panel Member, Integrating Emerging Technologies into Chemical Safety Assessment, Council of Canadian Academies, 2010 – 2012.
13. Expert Panel Member, Integrated Testing of Pesticides, Council of Canadian Academies, 2009 – 2012.
14. Mentor, Strategic Training in Research in Reproductive Health Sciences (STIRRHS), University of Montréal, 2006 – 2011.
15. Expert Panel Member, Assisted Human Reproduction Canada, The Environment and Reproductive Health: A Scientific Roundtable Steering Committee, Health Canada, 2010.
16. Organizing Committee Member, Assisted Human Reproduction Canada, The Environment and Reproductive Health: A Scientific Roundtable Steering Committee, Health Canada, 2010.
17. Member, Obstetrics & Gynecology Task Force - In vitro Fertilization, College of Physicians and Surgeons of Ontario, 2008 – 2010.
18. Member, Grants Review Committee, Canadian Breast Cancer Fund, 2007 – 2010.
19. Member, Clinical Investigation 'A', Grants Committee Panel, CIHR, 2006 – 2010.
20. Member, Scientific Program Committee, Canadian Fertility & Andrology Society, 2006 – 2010.
21. Invited Participant and Session Chair, Canadian Children's Environmental Health Research Workshop. Health Canada and CIHR sponsored, Ottawa, ON, February 9 – 11, 2009.
22. Member, Science Committee, Association of Professors of Obstetrics & Gynaecology, 2003 – 2009.
23. Member, Assisted Human Reproduction Canada/CIHR Sponsored Workshop Scientific Organizing Committee, 2008.
24. President, Canadian Fertility & Andrology Society, 2007 – 2008.
25. Chair, Scientific Program Committee, Canadian Fertility & Andrology Society, 2007 – 2008.
26. Member, Research Committee, The Society of Obstetricians and Gynaecologists of Canada (SOGC), 2003 – 2008.
27. Chair, Science Panel, EM-COM web site, www.EMCOM.ca, 2002 – April 2005.
28. Member, Board of Directors, Infertility Awareness Association of Canada, 1998 – 2004.
29. Toxic Substances Research Initiative, Healthy Environments and Consumer Safety Branch, Health Canada, 1999 – 2003.
30. Invited Participant and Session Chair, Canadian Children's Environmental Health Research Workshop. Health Canada sponsored, Ottawa, ON, March 17 – 19, 2002.
31. Chairman, Endocrine Disrupters Technical Review Committee, 1999 – 2000.
32. Vice-president, President-elect, Society of Toxicology of Canada, 1998 – 1999.
33. Co-chair, Interdepartmental Research Committee on Endocrine Disruptors, Health Canada,1998 – 1999.
34. Member, Endocrine Disruptor Committee, Organisation for Economic Co-operation and Development (OECD), Health Canada, 1997 – 1999.
35. Chairperson, Committee on Endocrine Disruptors, Health Protection Branch, Health Canada, 1994 – 1999.
36. Member, Animal Care Committee, Health Protection Branch, Health Canada, 1993 – 1998.

iii) **International -**

1. Councillor, Lake Ontario Regional Chapter, Society of Toxicology, 2016 – present.
2. Science Advisory Board Member, Endometriosis Association, 2014 – present.
3. Scientific Advisor, Development and Reproductive Toxicology (DART) Technical Committee, International Life Science Institute (ILSI), Health and Environmental Sciences Institute (HESI), 2012 – present.
4. Member, Center for Scientific Review (CSR), US Department of Health & Human Services, National Institutes of Health (NIH), 2010 – present.
5. Ad hoc Member, FIFRA Scientific Advisory Panel, US-Environmental Protection Agency (EPA), 1999 – present.
6. Past President, Lake Ontario Regional Chapter, Society of Toxicology, 2014 – 2015.
7. Member, Membership Committee, Society for the Study of Reproduction, 2012 – 2015.
8. President, Lake Ontario Regional Chapter, Society of Toxicology, 2012 – 2014.
9. Ad hoc Reviewer, National Institute of Environmental Health Sciences (NIEHS), National Toxicology Program (NTP) and the NTP Toxicology Branch, NTP Board of Scientific Counselors (BSC) meeting, December 9 – 10, 2014.
10. Invited participant, National Center for Environmental Assessment, US Environmental Protection Agency, Expert Workshop: Improving the Risk Assessment of persistent, bioaccumulative and toxic (PBT) chemicals in breast milk, October 24 – 26, 2012.
11. Expert Panel Member, site visit, Division of Epidemiology, Statistics & Prevention Research, National Institute of Child Health and Human Development (NICHD), October 22 – 24, 2012.
12. Secretary Treasurer, Reproductive Developmental Toxicology Specialty Section, Society of Toxicology, 2010 – 2012.
13. Chair, Core Committee, Society for the Study of Reproduction, 2010 – 2012.
14. Committee on Reproduction and the Environment, Society for the Study of Reproduction, 2008 – 2012.
15. Expert Panel Member, Reproductive & developmental effects of soy products and genistein, National Institutes of Health/National Institute of Environmental Health Sciences – National Toxicology Program (NIH/NIEHS-NTP), 2009 – 2010.
16. Invited Participant, Mammary Gland Evaluation and Risk Assessment Round Robin Workshop, Oakland, CA, November 16 – 17, 2009.
17. Member, NIH Study Section, Integrative and Clinical Endocrinology and Reproduction Study Section, October 5 – 6, 2009.
18. Team Member, National Institute of Child Health and Human Development (NICHD) site visit, September 24 – 26, 2008.
19. Member, WHO/IPCS Steering Group on Endocrine Disrupters, 1998 – 2002.
    - Global Inventory of Endocrine Disrupter Research.
    - International Assessment of the State of Knowledge on Endocrine Disrupters.
20. Expert Panel Member, Joint US/EU Endocrine Disruptor Research, 1999.
21. Member, Organisation for Economic Co-operation and Development (OECD), Working Group on Endocrine Disrupter Testing and Assessment, 1997 – 1999.
    - National Co-ordinator, Test Guideline Program – OECD, 1997 – 1998.

22. Member, National Sanitation Foundation - International, Health Effects Task Group, 1996 – 1998.

8.0 Statement of compensation:

I have been asked by the defendant's lawyer to provide an expert opinion on the carcinogenicity of glyphosate in rodents. I am compensated at a rate of $250 hour, $400 per hour for deposition, and a trial rate of $400 per day plus expenses. I have not testified as an expert witness in the past four years.