EXHIBIT 102

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 REPORT OF DR. CHRISTOPHER D. CORCORAN, Sc.D.

EVALUATION OF GLYPHOSATE EXPOSURE AND CANCER RISK IN RATS AND MICE

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I. SUMMARY

This report examines the rodent studies of glyphosate and cancer risk, particularly the seven feeding experiments using rats and five using mice that were reviewed in the expert report prepared by Dr. Chris Portier. The overarching question is whether these animal experiments provide a scientific basis to opine that glyphosate causes cancer in rats and mice. A few critical characteristics of these studies require careful consideration in addressing this question. Most crucially, the hundreds of individual tumor types evaluated within each experiment across both male and female rodents make it virtually certain that apparent "statistically significant" results will be observed for individual tumors that are in fact due to nothing more than chance. This necessitates the use of common statistical methods that account for multiple tests applied repeatedly to the same data. In addition, most of the tumor types are relatively uncommon, which warrants additional prudence in choosing appropriate statistical methods. In this report, I outline these issues, discussing in Section III how they are managed in everyday statistical practice. In Section IV, I apply the appropriate methods to the glyphosate rodent data and find no evidence whatsoever of a glyphosate effect on the risk any of the tumors evaluated across these studies after accounting for multiple tests. In Section V, I consider the discussion and results in Sections III and IV in the context of Dr. Portier's expert report. Dr. Portier suggests that the glyphosate experiments do provide some evidence of tumor risk among rodents. However, his statistical approaches are deeply flawed, leading him to overstate his findings and seriously misrepresent the data in aggregate. These flaws would prove fatal in any peer review. Most significantly, the results from the animal experiments that were highlighted by Dr. Portier were handpicked because of their "statistical significance", without appropriately accounting for the large number of tests for other tumors that demonstrated no evidence of a glyphosate effect. In addition, Dr. Portier violated conventional statistical practice in his use of historical controls and in combining or "pooling" data from across several sources – using experiments carried out during different years and in different laboratories under different conditions – without appropriately accounting for these studies' unique characteristics. In Section V we illustrate these flaws and their impact on Dr. Portier's conclusions.

II. RESUME AND QUALIFICATIONS

I am a professor of Statistics, and head of the Department of Mathematics and Statistics at Utah State University (USU) in Logan, Utah. I joined the faculty as an Assistant Professor at USU in 1999, after

receiving a B.S. in Statistics from USU in 1995 and a doctorate in Biostatistics from Harvard University in 1999. I was tenured and promoted to the rank of Associate Professor in 2005, and then promoted to the rank of Professor in 2011.

My research interests as a biostatistician focus largely on statistical methods for categorical data analysis, including the analysis of proportions and counts. My dissertation and much of my subsequent work has focused particularly on so-called exact methods for categorical data, developing software tools for researchers that allow them to analyze proportions and counts using exact tests for previously unaddressed study designs, including settings in which data are clustered or correlated (e.g., gestational or developmental toxicology studies using rats or mice), or for large-scale studies of genetics and disease. Much of this work has been funded by the National Institutes of Health, and implemented in the software packages StatXact and LogXact through Cytel Software Corporation (Cambridge, MA). These packages have long been considered the industry standard for exact statistical analysis.

I have also served as a senior biostatistician for a number of large interdisciplinary research projects focused on the epidemiology and genetic causes of complex disease, including Alzheimer's disease, cognitive decline among the elderly, hip fracture, autism, birth defects, and cancer. I have advised collaborators about study design, data management, and data analyses and the appropriate application of statistical methods, and I have either led or assisted with numerous manuscripts and presentations to disseminate research results. This work has likewise largely been funded by the NIH. In all, the collective extramural funding for these efforts has exceeded $25 million.

I have been asked examine data from the rodent glyphosate feeding experiments, and to assess any evidence of potential compound-related effects on the incidence of mouse and rat tumors, and have been compensated for this work at a rate of $250/hour. Unless otherwise stated, all of my opinions are expressed to a reasonable degree of scientific certainty. I reserve the right to amend or supplement my report in response to any rebuttal by plaintiffs' experts or as new information becomes available. I have not testified as an expert witness over the past 4 years. My curriculum vita is included as an attachment to this report.

III. STATISTICAL BACKGROUND

The fields of health and medicine abound with questions that likewise often appear straightforward: What is the best diet for a healthy heart? Are men or women at higher risk for a particular disease? Does a new drug lengthen life for cancer patients? In collaboration with other scientists, a biostatistician's role is to design experiments that address these questions, and to contribute to the analysis of the resulting experimental outcomes or data. Proper statistical methodology has assumed an increasingly important role in health and medicine as research has become more evidence-based. This is largely because (1) data

are generally full of uncertainty and variation, particularly when we study complex diseases or other phenomena in humans or animals; (2) many questions in health and medicine have strong statistical overtones (e.g., How common is a disease? Who is most likely to contract it?); and (3) the comparison of different treatments or potential risks relies heavily on statistical concepts – especially probability – in both designing and analyzing experiments.

As an example, suppose we pose the simple question: Does a flu vaccine work? This could be answered in part by considering a study of people who are randomly assigned to two groups, one receiving the treatment and the other some sort of placebo. At the study's end, the flu rates between the groups would be compared to assess whether the treated subjects experienced less flu than those on placebo. To continue the illustration, suppose such a study was designed with 20 patients in treatment and 20 in control (i.e., given placebo). Suppose further that we subsequently observe 0 flu cases (a 0% flu rate) among those who are treated and 20 cases (a 100% flu rate) among controls. With such a dramatic difference between the respective flu rates, common sense and intuition would strongly suggest that the treatment prevents flu.

On the other hand, suppose that this experiment alternatively results in 5 flu cases within the treatment group versus 10 in control (25% flu rate for treatment versus 50% for control). While the observed flu rate in this scenario is likewise lower within the treatment group, we are clearly *less* certain about declaring that the treatment works more generally. Why? Because it is more difficult to discern whether this result demonstrates an advantage for treatment, or if it could be simply due to chance variation between the people participating in the study. In other words, assuming that the vaccine does not work at all, we would expect that the observed flu rates within the two groups would differ by chance, much as we would expect that the number of heads we observe with 20 flips of a coin would be different than the number we observe if we flipped the same coin an additional 20 times.

How can we quantify the possibility that an experimental result is due simply to chance? The role of probability and statistics is especially critical in providing insight into this question. Common scientific and statistical practice involves designing an experiment with two competing hypotheses in mind. For a study comparing different treatments or groups, the primary hypothesis – generally referred to as the *null hypothesis* – is that there is *no difference* between the groups. The competing or *alternative hypothesis* is that there is a difference between the groups. At the end of the experiment, a probability is computed that measures the evidence against the null hypothesis. This probability, called a *p-value*, represents the likelihood of having observed the experimental result or data given that the null hypothesis is true. A relatively smaller p-value therefore indicates that there is evidence *against* the null hypothesis, since it tells us that the data are unlikely, assuming that the null is correct. On the other hand, a relatively larger p-

value provides no evidence against the null. The process of determining hypotheses and computing and interpreting a p-value based on resulting data is called a *hypothesis test*.

Two generally crucial issues with regard to testing a given hypothesis are (1) how the p-value is computed, and (2) how the p-value is used to make a decision about the null hypothesis. With regard to (1), even for relatively straightforward experiments, such as our hypothetical flu vaccine trial, there may be multiple approaches available for computing a p-value, each of which has certain advantages or disadvantages – these characteristics often depend on a specific study setting, and a biostatistician's role is to evaluate the strengths and weaknesses of competing methods for any given experiment to ensure that the data analysis is as accurate and reliable as possible. With regard to (2), the primary question is: How small does a p-value need to be in order to determine that there is sufficient evidence against a null hypothesis? A decision rule generally provides a cutoff against which the p-value is compared. For a single hypothesis test, the scientific community over time has settled on a threshold of 5%, meaning that a p-value less than 5% indicates sufficient evidence against the null, whereas a p-value greater than 5% provides insufficient evidence. This threshold is called a *significance level*, and p-values below this level are referred to as "statistically significant". Another important role of a biostatistician is to ensure for any given data analysis that the significance level is preserved. Any violation or inflation of the significance level can result in greater likelihood of spurious conclusions, especially in declaring "significant" treatment effects based on experimental results that are only due to chance.

IV.A *Interpreting p-values in the presence of many hypothesis tests*

The "p-value < 0.05" decision rule is relatively straightforward for a single experiment. However, the role of the p-value has become more complicated in today's data-driven world. The fathomless ocean of available data – generated from billions of dollars spent annually on research in health and medicine, and from the sheer volume of electronic transactions and online activity, among other sources – along with the relative ease of computing software for generating statistical analyses, necessitate some additional prudence in interpreting p-values. Nearly every day, online or other media news sources tout claims about an association between an exposure and an outcome, often with some implication of dramatic or broad consequences for the public. Many of these results often do not hold up under additional scrutiny or attempts at replication. How do these kinds of findings so readily find their way into the scientific literature and popular press? Explanations may sometimes include inadequate study design or poor data, but in our "big data" era the culprit is most often the amount of data available from large studies, or from a large number of smaller studies that are examined simultaneously. The tendency of researchers, along with scientific journals and other media venues, is a bias toward "positive" findings. This has led in turn to an overreliance on p-values and statistical significance, at the frequent expense of context, especially in underreporting or ignoring the large number of additional tests performed resulting in "negative" findings.

This issue is relatively straightforward to illustrate, given the application of hypothesis tests and p-values just described. Researchers can easily draw incorrect conclusions from an analysis of a large data set when many associations are examined across a large number of hypothesis tests that each look for a p-value that is less than the conventional 5% significance level. Simply put, when multiple tests are performed, "p-value < 0.05" outcomes will occur quite often even when there are no real effects. Note that the p-value < 0.05 rule was developed relative to a single test. However, this logic breaks down when multiple comparisons or tests are performed within a single analysis. With a 1-in-20 chance of a false positive for a single test, we would expect to see about one false positive for every 20 tests that we compute. In fact, it is straightforward to show using basic probability that there is a 64% chance of at least one false positive among 20 independent tests, and a 99.4% chance of at least one false positive among 100 tests.

This so-called multiple testing issue and the general overreliance on p-values has been discussed and studied extensively within the statistics and epidemiology professions. These issues have likewise been paid some considerable attention over the past several years in the popular media, especially given the many highly publicized findings that create an initial sensation but then fail to hold up under additional study and experimentation. (As just a small sampling of this coverage, within the scientific literature see "Why Most Published Research Findings Are False" by JPA Ioannidis in *PLoS One*, "Statistical Errors: *P* values, the 'gold standard' of statistical validity, are not as reliable as many scientists assume" by R Nuzzo in *Nature*, and "Evolution of Reporting *P* Values in the Biomedical Literature, 1990-2015" by D Chavalarais, JD Wallach, AHT Li, and JPA Ioannidis in *JAMA*. In the popular press see "Trouble at the lab", "How science goes wrong", and "Metaphysicians" in the *Economist*; "Science Isn't Broken: It's just a hell of a lot harder than we give it credit for" at *538.com*; "Striking results, little reliability" in the *Los Angeles Times*; and "New Truths Only One Can See" in the *New York Times*.)

Statisticians have long warned against the practice of computing a multitude of p-values – especially when applying arbitrary criteria to examine the same data in various ways – in order to identify positive associations. More recently, in response to this growing problem and the attention paid to it, our largest and oldest professional organization, the American Statistical Association (ASA), took the unusual step in 2015 of producing "The ASA's Statement on p-Values: Context, Process, and Purpose" (*The American Statistician*), under the direction of a committee comprised of some of our most respected colleagues. Several underlying principles regarding p-values are briefly emphasized in the document. In particular, the committee crystallizes the ongoing issues with multiple testing by noting that

> *P-values and related analyses should not be reported selectively. Conducting multiple analyses of the data and reporting only those with certain p-values (typically those passing a significance threshold) renders the reported p-values essentially uninterpretable. Cherrypicking promising*

> *findings, also known by such terms as data dredging, significance chasing, significance questing, selective inference, and "p-hacking," leads to a spurious excess of statistically significant results in the published literature and should be vigorously avoided. One need not formally carry out multiple statistical tests for this problem to arise: Whenever a researcher chooses what to present based on statistical results, valid interpretation of those results is severely compromised if the reader is not informed of the choice and its basis. Researchers should disclose the number of hypotheses explored during the study, all data collection decisions, all statistical analyses conducted, and all p-values computed. Valid scientific conclusions based on p-values and related statistics cannot be drawn without at least knowing how many and which analyses were conducted, and how those analyses (including p-values) were selected for reporting.*

Of course, none of this means that all science is unreliable, or that we should give up on experimentation altogether. The problem is not with research, generally, but with the overuse and misapplication of p-values. The good news is that the same statisticians and scientists who have identified potential problems with p-values have often also developed or proposed constructive and accessible approaches for increasing the reliability of research results. In addition to the basic suggestions about disclosure quoted above from the ASA report, a couple of the most common among the recurring recommendations include (1) the use of multiple test corrections or what we call "false discovery rates" to adjust for a large number of hypothesis tests; and (2) the reporting of actual effect sizes (in addition to p-values), along with measures of uncertainty about the effect size.

With regard to (1), how does a biostatistician make sure that p-values < 0.05 for an analysis involving many tests are not merely due to chance? This is generally accomplished by first assessing the number of tests that need to be carried out, and then by computing the individual p-values using a method that accounts for the number of tests. This kind of multiple testing method will yield a set of p-values that can then be individually compared to the 0.05 testing level to identify truly significant findings. Such multiple testing methods are readily available in any one of the most widely-used statistical analysis software packages, and are illustrated in the large number of dedicated multiple testing textbooks and manuals. These methods are taught as a matter of course within many university statistics curricula. In particular, so-called stepwise or closed testing procedures can be readily applied to a set of many p-values computed in a given analysis, adjusting the p-values to preserve the false positive rate not only for the individual tests but for any combination or subset of null hypotheses under consideration. While several options are available, the so-called False Discovery Rate (FDR) approach has been increasingly recommended and used in statistical practice.

IV. ASSESSING THE GLYPHOSATE FEEDING EXPERIMENTS

Section III broadly outlined some of the crucial statistical issues that are highly relevant to the rodent glyphosate feeding experiments and to the analysis provided in Dr. Portier's expert report. Most

importantly, given the dozens of tumor types evaluated for both male and female rodents across the twelve studies we are considering, some sort of multiple comparison correction is imperative to avoid a very serious problem with false positives. In this section, we consider these issues in analyzing the rodent data, and summarize the results. In the next section we discuss these results in light of Dr. Portier's conclusions.

The available data come from 12 different experiments (7 using rats and 5 using mice) in which rodents were randomized – males and females, respectively – to increasing doses of glyphosate, then examined after their natural lifespan or at a pre-specified limit and evaluated for the presence of many

			Glyphosate Doses (mg/kg bw/day)		# Types w/ ≥1 observed		# Types w/ ≥3 observed	
Study	Year	Strain	MALE (M)	FEMALE (F)	M	F	M	F
Lankas	1981	SD	0, 3, 10, 32	0, 3, 11, 34	51	68	19	28
Stout	1990	SD	0, 89, 362, 940	0, 113, 457, 1183	45	44	17	14
Atkinson	1993	SD	0, 11, 112, 320, 1147	0, 12, 109, 347, 1134	46	35	15	11
Enemoto	1997	SD	0, 104, 354, 1127	0, 115, 393, 1247	53	37	20	12
Suresh	1996	Wistar	0, 6, 59, 595	0, 9, 89, 886	50	41	15	11
Brammer	2001	Wistar	0, 121, 361, 1214	0, 145, 437, 1498	45	44	14	15
Wood	2009	Wistar	0, 86, 285, 1077	0, 105, 349, 1382	52	40	16	14

Table 1: Summary of 7 rat glyphosate feeding experiments.

			Glyphosate Doses (mg/kg bw/day)		# Types w/ ≥1 observed		# Types w/ ≥3 observed	
Study	Year	Strain	MALE (M)	FEMALE (F)	M	F	M	F
Knezevich	1983	CD-1	0, 157, 814, 4841	0, 190, 955, 5874	57	98	16	32
Atkinson	1993	CD-1	0, 98, 297, 988	0, 102, 298, 1000	25	27	9	13
Sugimoto	1997	CD1	0, 165, 838, 4348	0, 153, 787, 4116	23	31	6	10
Kumar	2001	Swiss	0, 15, 150, 1453	0, 15, 151, 1467	19	30	8	10
Wood	2009	CD-1	0, 71, 234, 810	0, 98, 300, 1081	21	34	9	11

Table 2: Summary of 5 mouse glyphosate feeding experiments.

specific tumor types. These studies are summarized in Tables 1 and 2 (the totals given in these tables exclude cases where proportionally few mice or rats were apparently evaluated for a given tumor type, as the limited number of animals evaluated may reduce the interpretive value of the results to such a degree as no conclusions may be drawn). There are two critical questions to address in evaluating the collective evidence of a possible glyphosate effect on tumors among rodents. First, how do we evaluate the dose-response effect of glyphosate on a single tumor type? Second, how do we account for many dose-response analyses across multiple tumor types?

In answer to question 1, the most commonly used tool for assessing a dose-response effect is the Cochran-Armitage trend test. In the context of the rodent feeding experiments, this approach provides a p-value to test the null hypothesis of *no dose effect on tumor rate* versus the alternative hypothesis that the *tumor rate increases with increasing dose*. This trend test is generally applicable to any experimental data where subjects are randomized to increasing doses of some drug or other intervention, and then observed to experience (on average) an increasing or decreasing percentage of subjects who experience the outcome of interest. As with much of the analysis provided by Dr. Portier, these results are based on a one-sided exact trend test – "one-sided" in that we are testing the trend in only one direction for a given tumor, and "exact" in that we are using the actual probability distribution under the null hypothesis, instead of a normal or bell-curve approximation (also called the "approximate" or "asymptotic" trend test). The exact test is recommended when outcomes of interest are not common, which is often the case across the glyphosate experiments.

What motivates this recommendation, and why does it matter whether we use the exact or approximate p-value? While it may seem like a statistical technicality, the choice turns out to be germane to the glyphosate rodent carcinogenicity question. The International Agency for Research on Cancer (IARC) monograph on glyphosate used the approximate p-value to conclude that results from the Knezevich experiment (included in Table 2) implicated glyphosate as a cause of kidney adenomas among male mice, based on their reported approximate trend test p-value of 0.034 (without adjusting for multiple tests). However, the exact one-sided test – subsequently reported by Dr. Portier in other material and ultimately his expert report – yields a p-value of 0.062 for these same data. The discrepancy between the approximate and exact p-values in this case illustrates why the former should be avoided when tumor incidence is low. It turns out that the approximate p-value is an estimate of the actual or exact p-value, and tends to be more accurate when the overall sample size is relatively larger and when relatively more investigative events (in this case, tumors) are observed. In general, approximate p-values tend to *underestimate* the exact p-values they are supposed to estimate. When sample sizes and numbers of observed outcomes (such as tumors) are relatively large, this underestimation may not be consequential. However, in cases like the glyphosate feeding experiments – where tumors are relatively less common –

the inaccuracy of approximate p-values when they are used can lead to a significant increase in the number of false positives. In other words, because the approximate test tends to underestimate the exact, we will see more "p-value < 0.05" results with the approximate test when there is actually no dose-response effect. This can lead to serious exaggeration of the evidence in favor of trend effects.

Given the large relative error of this normal approximation for the Knezevich data, one might wonder why anyone would ever use it. Normal approximations in applied research had much greater utility before the widespread availability of powerful computing tools. Without some sort of special calculator or software, a normal probability is relatively much easier to compute than an exact probability. Even now, some analyses of counts and proportions rely on more sophisticated statistical models for which the exact distribution is prohibitively difficult to compute, and so some form of normal approximation can still be useful. However, for many experiments – particularly controlled experiments such as the glyphosate mouse studies – exact p-values can be computed instantaneously with a desktop computer, and no approximation is needed, even in cases where the sample sizes and counts are sufficiently large to justify such an approximation.

Given appropriate computation of the trend test p-value, the second necessity is accounting for the many dose-response analyses across multiple tumor types. As discussed earlier in Section III, the False Discovery Rate (FDR) approach recommended by Ioannidis and others is particularly useful for these data. It is a less conservative adjustment that is recommended in settings where there are hundreds or even thousands of p-values under consideration. Of all multiple testing options available in this setting, the FDR approach minimizes the chance that we would fail to detect an actual glyphosate-related effect. It should be noted that an FDR adjustment could and should be used for any set of p-values computed to assess potential glyphosate effects on tumor incidence, including any pairwise comparisons made between the tumor rates of two dose groups. Such two-group comparisons are not reported here as Dr. Portier's conclusions do not appear to rely upon them, but the same multiple testing problem applies, and even more so: in an experiment with four dose groups, respective comparisons of the three treatment groups to control can yield up to three p-values – as opposed to one trend test p-value – for each tumor type.

Trend test results for the 7 rat studies are summarized by the tables shown in Appendix A, and results for the 5 mouse studies are similarly summarized in Appendix B. Each table contains exact one-sided p-values for each study, reported by tumor type and sex, testing specifically for evidence of increasing tumor probability. In addition, for those p-values < 0.05 reported and highlighted in Appendix A and Appendix B, a multiple testing FDR adjustment is applied and reported in the table shown in Appendix C.

As shown in Tables 1 and 2, of the hundreds of individual tumor types evaluated across all 12 experiments, 1,016 were observed in at least one mouse or rat. Among rats, there were 13 trend test p-

values < 0.05 when testing for increasing incidence of each tumor, without accounting for the false discovery rate. Among mice, there were 7 such trend test p-values < 0.05 without accounting for the false discovery rate. All of these are highlighted in blue for easy identification in the tables contained in Appendices A and B. Note that – assuming no effect of glyphosate on tumor incidence – we would conservatively expect about 5% of all individual trend tests to yield p-values < 0.05 only by chance. This would represent about 51 p-values < 0.05 out of the 1,016 individual cancer types for which at least one tumor was observed. However, it makes sense to consider those cancer types for which three tumors were observed. Given the typical study design of four dose groups with approximately 50 animals per dose, about 3 tumors in total are necessary for an exact one-sided p-value no greater than 0.05. Given 345 tumor types across the 12 rodent studies with at least 3 observed tumors (as summarized in Tables 1 and 2), assuming no compound effects we would expect roughly 17.3 p-values < 0.05. In other words, given the 20 observed p-values < 0.05, the overall results are entirely consistent what we should observe given no compound-related effect on tumor incidence. This is analogous to flipping a coin 345 times that has a 5% probability of heads, and observing 20 heads with an expected number of 17.3. This result is highly likely: there is actually about a 62.5% chance of observing this many independent p-values < 0.05 relative to the expected proportion, given no compound-related effects.

In addition, when computing the trend test p-values to account for the false discovery rate, not one of the 1,016 tests is statistically significant. FDR-adjusted p-values for all tumor types with individual trend test p-values < 0.05 are summarized in Appendix C, and not one has a value even marginally close to 0.05. (Note that adjusting for multiple tests always increases the p-value, so that there is no need to report FDR adjustments for any individual trend test results with p-values > 0.05.) There is no statistical evidence whatsoever that glyphosate increases the risk of any of the tumors examined across these 12 studies.

I would emphasize that the results summarized above correspond to a one-sided test that only evaluates the hypothesis that increased glyphosate exposure is associated with an increased rate of tumors – what we would refer to as a *positive* association. However, the data may also be analyzed to evaluate a *negative* association – that is, a decreased tumor rate as glyphosate exposure increases. In fact, it turns out that the one-sided p-values for testing negative effects can simply be computed as 1.0 minus the one-sided p-values reported in Appendices A and B. In other words, any p-value reported in Appendices A and B that is larger than 0.95 represents a p-value < 0.05 for testing for a negative association. There are 13 such outcomes, as summarized in Appendix D (additionally adjusted for the false discovery rate). Again, as with the tests for positive associations, we would expect 5% of all 345 tumor types with at least three observed tumors to likewise yield one-sided p-values < 0.05 when testing for *negative* associations. The 13 such results are again entirely consistent with this expected proportion: there is actually about a 24.5%

chance of observing this many independent p-values < 0.05 relative to the expected proportion, assuming no compound-related effect.

Finally, I have also been made aware of a statistical reanalysis carried out by Dr. Klaus Weber of data from Kumar mouse study. I have evaluated the reported data used by Dr. Weber. Some of the reported tumor counts differ slightly from the data reported in Greim. My own analysis indicates that utilizing the data tables reported by Dr. Weber does not substantively change my conclusions. I have included my results both based on the Kumar data as reported in Greim, et al, and the data reported by Dr. Weber (see Appendix B, Tables B.5 and B.6).

V. RESULTS FROM DR. CHRIS PORTIER'S EXPERT REPORT

Given that the 1,016 p-values computed across all 12 studies yield nothing more than the expected pattern of false positives given no effect from glyphosate exposure, Dr. Portier nevertheless most recently asserts that there is sufficient evidence glyphosate increases the risk for a handful of cancers, including liver adenomas, thyroid C-cell adenomas and carcinomas, skin keratocanthomas, and kidney adenomas in male rats; mammary gland adenomas and adenocarcinomas in female rats; hemangiosarcomas, kidney tumors, and lymphomas among male mice; and hemangiomas among female mice. His analysis unfortunately would certainly not pass the scrutiny of any meaningful peer review, and could actually be used as an excellent case study in any university statistics course to illustrate the misappropriation of p-values. Most critically, virtually any experienced statistician reviewing Dr. Portier's work with the animal data would see immediately that his approach has led to a very serious multiple testing problem. Dr. Portier's analysis is entirely dependent on p-values, arising from three types of computations: those for individual tumor types by gender across each specific study (handpicked from among the more complete results contained in Appendix A and Appendix B of this report), those that incorporate additional "historical control" data, and those that "pool" data from across studies for a given tumor type. Dr. Portier provides a patchwork of p-values from across these three sources, reporting significant findings (for increased risk, only) wherever and in whatever manner they are found in order to manufacture a pattern implicating glyphosate.

While the multiple testing problem overarches all of these p-values, there are additional chronic flaws with his use of historical controls and pooling procedures that need to be illustrated separately. These three issues – multiple testing, historical controls, and pooling of data sets – are correspondingly addressed in Sections V.A–V.C. Section V.D subsequently summarizes how the conclusions in Dr. Portier's report have evolved from his prior work.

(V.A) *The Use and Interpretation of P-Values*

Given the large number of animal tumors under investigation here, any analysis should consider the concerns and recommendations of statisticians and researchers about the use and misuse of p-values, as discussed in Section III. Unfortunately, Dr. Portier does not consider or apply even one of the common or recommended remedies for this problem. In his tables summarizing results for individual rat and mouse studies, he includes only what he terms as "Tumors of Interest", which appear to be selected primarily on the basis of their statistical significance within at least one of the several studies. His report makes no effort to directly adjust p-values for multiple comparisons, for example by using the false discovery rate approach recommended by experts in the profession. This is in spite of Dr. Portier's brief comment on page 40 of his expert report that "an adjustment for multiple comparisons is indeed warranted in evaluating the outcomes of these studies."

The only other mention of the multiplicity problem is the inclusion of Table 15, which Dr. Portier constructed in response to comments submitted last year to the EPA by Dr. Joseph Haseman. Dr. Portier has used Haseman's tally of the expected number of false positives as a basis for demonstrating that there are more significant results among male CD-1 mice than would be expected by chance, given no glyphosate effects. A couple of critical differences in Dr. Portier's approach account for his findings. First, Haseman bases his own expected false positive number on the number of tumors for which there are at least 3 observed cases (roughly the number required for a possibility of a p-value < 0.05). Haseman confined his estimate to sites with three tumors based on the use of an exact one-sided p-value, given that the study designs used for the glyphosate feeding experiments generally cannot yield a p-value < 0.05 unless at least three rodents are observed with a given tumor type. However, Dr. Portier is including his historical control test, which (while not validated, as illustrated in the following section) can yield p-values < 0.05 for *observed* tables that contain only two tumors. For example, the Sugimoto hemangiosarcoma figures in male mice (0/50, 0/50, 0/50, 2/50) generates an exact one-sided trend test p-value of 0.062, which is > 0.05. When reanalyzed by Dr. Portier using historical controls his resulting p-value (what he refers to as "P_{Hist}") is 0.004, which is < 0.05. In other words, when he incorporates historical controls he is able to generate a p-value < 0.05 for smaller numbers of tumors in the observed table. In addition, since he appears to be counting either trend test result with a p-value < 0.05, or a "P_{Hist}" result < 0.05, as "positive," he is at least doubling the number of observed tests among those tumor types for which historical control data are available. These uses of historical controls explain the disparity in Dr. Portier's Table 15 between what is observed and what is expected relative to statistically significant findings among male CD-1 mice.

However, in addition to that, such a comparison of observed and expected – while interesting for exploratory purposes – does not directly address the more pressing question: is there evidence of a compound-related effect with respect to any *specific* cancer type? The answer in part requires multiple

testing adjustments to individual p-values, such as the false discovery rate approach we use for both the rat and mouse studies. As reviewed in Section III, other recommendations for balancing the overuse of p-values include full disclosure of all tests performed, and the estimation of actual effect sizes along with measures of effect size variability (such as confidence intervals). Dr. Portier uses neither of these approaches.

(V.B) ***P-values Using Historical Controls***

The quantitative use of historical controls for the sake of establishing treatment effects within a given statistical analysis is not universally accepted in experimental research. Many researchers view historical controls at best as a means of laboratory quality control (to check consistency of outcome rates) or as a qualitative measure before reaching any determination of causation. However, even if the historical data are judged by study toxicologists to be comparable and potentially useful for inclusion with new experimental data, any statistical analysis needs to be carefully planned and conducted to ensure that p-values are computed appropriately. Dr. Portier's expert report helps to illustrate why. He argues that we can compare prior experimental results for unexposed rats or mice to what we observe among treated rodents in a given experiment. Particularly for rare or uncommon events, such as the cancer types investigated for the glyphosate experiments, it may appear compelling or interesting when the number of tumors observed in a treatment group is markedly higher than what we would expect given the average control rate in prior experiments. However, the approach not only is not helpful for this particular analysis, but is fundamentally inaccurate and is moreover applied inconsistently by Dr. Portier.

Most critically, underlying response rates almost always vary across different experiments, even when those experiments are studying the same outcomes but using different samples at different times and in different settings or laboratories. Even for the best or most consistently controlled studies, there are underlying factors inherent in the sampling, the methods, the environment, and so forth, that can significantly affect the likelihood of response. This is why, for example, statisticians account for study differences or heterogeneity when combining data from different experiments or study sites (as discussed more extensively in the following section – Section V.C – in the context of Dr. Portier's "pooled" analyses).

Dr. Portier illustrates this with an example on page 28 of his report. In this case, he uses historical controls to assess hepatocellular adenoma in the Wistar rats studied by Brammer, and cites results from 16 historical control groups with an underlying range in adenoma rates of 0% to nearly 18%. This relatively wide range in adenoma rates, across studies using the same genetic strain of rat, is a perfect example of how significantly these outcome rates can vary between experiments. However, Dr. Portier's solution is simply to apply the average rate of 4.3% across the 16 studies to the results of the Brammer

experiment, which yielded 0/53, 2/53, 0/52, and 5/52 male rats with liver adenomas across the four respective dose groups. Although Portier appears to dismiss the possibility, it is entirely possible that the Brammer sample actually *did* have an underlying liver adenoma rate nearer to 18% than to 0%. In that case, observing 7 liver adenomas out of 210 mice would not be at all remarkable. Because Dr. Portier failed to formally account for the potential range of historical control tumor rates when generating his test statistic, his resulting p-value is flawed.

Even assuming justification for including historical controls in his analysis (i.e., the historical controls are sufficiently consistent with the given feeding experiment data), Dr. Portier's approach is deeply flawed, and alarmingly inconsistent even with the recommended statistical methods cited within his own sources. He appeals on page 21 of his expert report to four references as "guidelines" (numbers 30, 33, 34, and 66 in his citation list). The first three provide an exceptionally thin foundation for such a key aspect of Dr. Portier's analysis: the first is somewhat of a self-reference (the preamble to the IARC glyphosate monograph, written by a group chaired by Dr. Portier), and the second and third are regulatory references specific to the EPA and the European Chemicals Agency. The fourth is an expository article authored by Dr. Joseph Haseman in an environmental health journal – the only one of the four references that outlines specific statistical methodology for incorporating historical controls. The Haseman paper describes the heterogeneity problem described above – the tendency of different study samples to have significantly different tumor rates – and proposes a sensible modeling method that accounts for these differences. Dr. Portier offers no explanation for why he fails to use this approach, in spite of his citing the paper in which it was suggested. Moreover, there are other references in the statistical literature that specifically address the problem of incorporating historical controls. For example, Fung et al (*Canadian Journal of Statistics*, 1996), Greim et al (*Human & Experimental Toxicology*, 2003), and Peddada et al (*Journal of the American Statistical Association*, 2007), among others, all offer overviews and options for a proper analysis using historical controls – none of them mentioned or utilized by Dr. Portier, in spite of his citing these articles in a recently published commentary (Portier and Clausing, 2017). The common principle underlying all of these methods is the need to account for differences in underlying tumor rates for controls drawn from a variety of experiments. As explained more fully in the following section, the general consequence of not properly adjusting for such differences is underestimation of p-values, which leads to inflation of p-values < 0.05 and "statistically significant" findings due to nothing more than chance. Given the hundreds of tumor types under consideration across the glyphosate rodent experiments, this is a problem that should be meticulously avoided.

Aside from his completely incorrect analysis, the p-values computed by Dr. Portier using historical controls do not change any of the substantive conclusions of the analysis, since Dr. Portier neglected to account for the enormous multiple testing problem. Even when the corresponding trend test p-values in

Tables A1–A7 and B1–B5 of this report were replaced by Dr. Portier's historical control-based results and then adjusted with respect to the false discovery rate, none of them was significant. In addition, Dr. Portier neglects to explain why he selectively highlights tests using historical data – there were apparently many other tumor types for which historical control data were available but not used. It appears that such results were reported by Dr. Portier primarily if they resulted in a p-value < 0.05.

(V.C) ***P-values From "Pooled" Analyses and Interpretation of Results Across Studies***

Given the multiple testing problem and the relative rarity of most all of the cancer types, there would seem to be some impetus to attempt combining data from across studies. Aggregating the sample size and tumor counts could potentially increase the likelihood of observing a compound-related effect, if any such an effect exists. Dr. Portier's attempts to accomplish this through his "pooled" analyses are nevertheless completely unreliable. His analysis and comparative interpretations across the various experiments disregard conventional statistical practice in several fundamental and egregious respects, and his approach is ad hoc and inconsistently applied, without any kind of systematic analysis plan across the available studies or tumor types.

First and most critically, Dr. Portier's "pooled" procedures flout statistical standards by making no adjustments at all for differences between experiments or for the similarities among mice within each study. Dr. Portier simply aggregates data across various subsets of rat and mouse studies, treating rodents born and raised in different environments, fed from different sources, measured using different tools by different researchers over a 30-year span as though they were all included within a single experiment at the same time. This is an astonishing violation of accepted practice that would serve as an example in any relevant college class of how not to combine data from different sources.

Generally speaking, any combining of data across experiments such as those considered here requires that (1) the experiments are comparable enough in terms of their measurements and conditions to justify their inclusion in a combined analysis; and (2) if the studies are sufficiently comparable, some adjustment is made for similarities or correlation of subjects within each study, as well as for differences in treatment effects that are often observed. Addressing (1) is a primarily qualitative first step that usually relies on some consensus among collaborating investigators with complementary expertise, who assess the admissibility of available studies in terms of their comparability (e.g., that they consistently measured outcomes and administered treatment doses). Without such strong justification, any attempt to quantitatively combine the data from the individual studies can be unreliable. Dr. Portier has provided very little information in his report about how he conducted such a review – for example, describing consultations with other collaborators or sources about whether pathologies were examined consistently for the handful of tumors types that he selected for his pooled analyses.

Putting aside the lack of a qualitative review, the "pooling" approach used by Dr. Portier to simply combine data from different studies – as though they arose from the same experiment – is completely inappropriate and incorrect. The underlying principle in any analysis that combines data from independent studies is that the studies themselves – carried out at different times and in different settings – may be distinct in ways that may or may not be measurable. These differences, often referred to in experimental research as sample or study *heterogeneity*, need to be considered within the statistical analysis in order to avoid bias when computing p-values. Why is this so crucial? There are two reasons to account for study differences. First, ignoring them often leads to an increased chance of a false positive result. To illustrate, consider an example where we have access to data for a flu vaccine that was administered to 10 large nuclear families, with 5 family members in each home. For the sake of illustration, suppose that the members within each of these families – for one reason or another – have the exact same response to the vaccine. In other words, if one member of a given family responds to the vaccine, then *all* family members respond. If one does not respond, then neither to any of the other family members. Although there are 50 total individuals enrolled in this study, our *effective* sample size is only 10. In other words, one family member from each home is sufficient. The other 4 give us no additional information about the treatment effect, and are statistically redundant. From a statistical standpoint, naively assuming that all 50 individuals are somehow independent could lead to significant underestimation of the p-value testing the vaccine effect, making a false positive much more likely.

This example is obviously extreme. In practice, we would seldom (if ever) observe that kind of perfect correlation among data from a given study site or experimental source. However, in any analysis of data from multiple sites or experiments, some appreciable correlation within each will exist due to variations in the different sampling populations or experimental conditions. This heterogeneity will result in at least some effective reduction of the sample size, in proportion to the strength of the correlation between subjects within each study. Suppose that we ignore those study differences by simply aggregating the data and analyzing them as though they all came from the same experiment, as Dr. Portier has done. Then p-values computed to test overall treatment effects will be inaccurate. Generally speaking, they will be too small, leading researchers to overstate any evidence of a treatment effect.

The second reason to assess and account for study differences is that the treatment effect often differs between the individual studies, both with respect to the size and even the direction of the effect (e.g., increasing or decreasing trend). Another crucial step in combining datasets is to compare the effects across studies, to understand how they are either alike or different with respect both to their direction and magnitude. This typically involves some estimation of effect sizes, along with additional formal statistical comparisons to ensure that the effects are consistent before any data from across the various studies are pooled. Treatment effects in dose-response experiments are often summarized using an odds ratio or

relative risk, which in the case of the glyphosate experiments would estimate the relative increase or decrease in the odds or risk of tumor for some given increase in glyphosate dose. For example, the so-called logistic regression model that is used extensively by researchers across numerous fields (particularly in biomedicine) allows researchers to estimate such odds ratios in ways that can examine whether the estimated odds ratios are consistent across experiments. A logistic regression can help an investigator to make a reasonable judgment about whether the observed results – expressed as odds ratios relative to glyphosate dose – in two or more different experiments are significantly different. This is a crucial assessment in any combined analysis that statisticians use to decide whether they are justified in estimating a "common" or averaged effect across all of the studies. The important point is that such methods are generally applied as a matter of course in this kind of analysis, although they are not used at all by Dr. Portier.

In short, Dr. Portier has apparently made no reasonable effort to address study heterogeneity, either with respect to the correlation of rodents within study or to differences in dose-response effects across studies. The seriousness of this flaw cannot be overstated. In addition to his failure to account for the way that mice and rats are correlated within the individual studies, Dr. Portier has combined data from different sources without regard for the magnitude or direction of observed effects within groups. At a minimum, by failing to account for within-study correlation, Dr. Portier has underestimated the actual p-values – hence overstating the evidence (and increasing the chance for a false positive result) – for those tumor types that he has selected for "pooled" analyses. Moreover, relying only on p-values for these "pooled" analyses, even if they correctly account for study heterogeneity, masks study differences in ways that can seriously undermine any possible understanding of potential compound-related effects. I know of no available applied statistical text or handbook that touches on this topic that even entertains the possibility that an analyst would simply combine data from various experiments as Dr. Portier has done, without carefully examining and accounting for study differences. His approach can only be described as naïve at best, and deliberately misleading at worst.

Interestingly, Dr. Portier provides two citations (numbers 92 and 93 in his report) that he uses to justify his combined analyses, and that provide some guidance about how he should conduct them. They are expository articles from epidemiological journals, and – while not statistical sources, strictly speaking – both provide general information about how to analyze data from different sources, consistent with the principles summarized above. Both emphasize the importance of evaluating and accounting for study heterogeneity to avoid bias in statistical inference, and both recommend the use of logistic regression models to estimate treatment effects between studies and to assess whether those effects differ significantly. Neither of these sources mention the option of aggregating data in the way that Dr. Portier has done. On the contrary, one of them suggests that study heterogeneity should conservatively be

assumed even if there is statistical evidence that it does not exist. Dr. Portier astonishingly and inexplicably ignores all of this information within his own sources.

How does this enormous oversight specifically compromise Dr. Portier's conclusions? As just one example among his several pooled analyses, consider Dr. Portier's assessment of liver adenomas among rats. Relying only on his personal qualitative judgment, and without any formal statistical justification, Dr. Portier chose to focus only on studies using Wistar rats (including the Brammer, Suresh, and Wood studies in Table 2), and to ignore female rats altogether. (If a logistic regression model was used, the analysis could readily include the other four rat studies as well as all female rats, easily accounting for any possible differences between the genetic strains and genders.) The Brammer study observed counts of 0/53, 2/53, 0/53, 5/52, with a trend test p-value of 0.008 and an FDR-adjusted trend p-value of 0.370. Note that the Suresh study resulted in 24/50, 22/50, 10/48, and 21/50 liver adenomas across the male dose groups, an increasing trend that was not statistically significant (trend p–value = 0.391; FDR-adjusted trend p-value = 0.715). The Wood study resulted in 0/50, 2/51, 1/51, and 1/51 liver adenomas across the male dose groups, a weak increasing trend that was also not statistically significant (trend p–value = 0.418; FDR-adjusted trend p-value = 0.839). Even after excluding the other rat studies, along with any results for females, in an argument spanning pages 32 and 33 of his report, Dr. Portier first suggests pooling the Brammer, Wood, and Suresh liver adenoma data for male rats, and then arbitrarily excludes the Suresh study because of its higher overall rate of liver adenomas (based again only on personal judgment, without any formal statistical analysis). Dr. Portier then combines the data from Brammer and Wood into a single table to produce a single trend test p-value, that he concludes demonstrates evidence that glyphosate increases incidence of liver adenomas. Dr. Portier takes the same approach with mammary tumors, combining only the data from Brammer and Wood, in order to generate a p-value < 0.05. However, he then elects to combine data from all three studies in order to obtain a p-value < 0.05 with respect to skin keratocanthomas. Notably, he reports that using only Brammer and Wood for skin keratocanthoma does not generate a "statistically significant" p-value. This appears to be a straightforward case of the "p-hacking" phenomenon discussed earlier in Section III, as he offers no empirical justification for how he chooses to include or exclude the Suresh study from these additional analyses.

Unfortunately, Dr. Portier's arbitrary and incorrect analysis renders his resulting "pooled" p-value entirely meaningless. Accounting for heterogeneity and estimating study-specific effects, as recommended by Dr. Portier's own sources, my own analysis of the liver adenoma data first demonstrated definitively that there is highly significant correlation among rats within each study (using an exact test for correlation in the StatXact software package). In addition, using a logistic regression model to estimate observed effects, I found that the Brammer study indicates an odds ratio (OR) of 1.21 with

respect to an increased dose of 100 mg/kg (meaning a 21% increase in odds of liver adenoma for every additional 100 mg/kg bw/day). The Wood study resulted in an estimated OR of 1.01 (only a 1% increase in the odds of liver adenoma for an increased dose of 100 mg/kg), and the Suresh study also resulted in an estimated OR of 1.01. Moreover, the logistic regression revealed that there is a highly statistically significant difference in observed effects between the three studies – specifically, the effect observed in Brammer is higher than the effects observed in the Wood and Suresh data. In other words, Dr. Portier included two of the three studies in his "pooled" analysis that actually are demonstrably different with respect to glyphosate. This further invalidates Dr. Portier's "pooled" p-value for evaluating a common potential effect across studies, which he computed using the Brammer and Wood data. Nevertheless, aggregating the two datasets, without accounting for these potentially serious differences between the underlying adenoma findings, Dr. Portier reports a significant "pooled" finding that is entirely driven by the Brammer data. He implies that this somehow makes the result more convincing, which is a logical leap equivalent to combining a gallon of paint with a gallon of paint thinner, and then selling the product as two gallons of paint.

In addition to this conspicuous and fatal problem, Dr. Portier takes a highly inconsistent approach with his "pooled" analyses that appears to focus primarily on achieving statistical significance. He "pools" and "re-pools" rat and mouse data (always ignoring study heterogeneity), using different combinations of studies without any predefined strategy or logical criteria. Dr. Portier's "Joint Analysis" of the mouse studies on pages 45-47 of his expert report is a particularly confusing and ad hoc jumble. To summarize the arbitrary and incongruous nature of his approach:

- Dr. Portier proposes that the only neoplasms that he needs to examine for combined or "pooled" analyses are the five for which at least one of the four CD-1 studies resulted in a statistically significant finding. Why the dozens of others should be ignored is not explained. At the very least, Dr. Portier is compounding the grievous multiple testing problem discussed earlier, since the significance of the "pooled" trend test p-values that he reports are driven entirely by the five individual statistically significant results. A more systematic analysis would combine data from across studies for each tumor type (assuming that the tumor types are consistent, and appropriately accounting for study heterogeneity); estimate a common observed effect for each tumor type, along with measures of statistical significance (including p-values and confidence intervals), assuming that the effect is consistent across studies; and finally account for multiple comparisons (e.g., adjust for the false discovery rate) among the set of resulting p-values. However, conducting such a systematic analysis would still need to be preceded by a sound qualitative toxicological analysis to ensure that the studies are comparable, as discussed at the beginning of Section V.C.

- After confining himself to the CD-1 studies, Dr. Portier alternatively combines the two 18-month studies, the two 24-month studies, and then all four studies together, and then for each tumor type simply bases his conclusions on the one of those three that results in statistical significance. For example, in the summary of his findings, he claims there is evidence that glyphosate "causes" kidney tumors, after pooling all four CD-1 studies. However, he also claims there is evidence that glyphosate "causes" malignant lymphomas, conveniently based on the result from "pooling" only the two 18-month studies, even though there is no statistically significant effect when all four CD-1 studies are used. This is internally inconsistent and another example of "p-hacking."
- Dr. Portier's analysis of hemangiosarcomas in males is especially troubling. After first "pooling" the two 18-month studies (significant result), and then the two 24-month studies (no significant result), *he proposes simply removing the 0/50 count observed in the highest dose group of the Knezevich study*. By excluding the mice in this high dose group – none of whom were observed with any hemangiosarcomas, which would suggest no effect of the test compound – Dr. Portier is then able to manufacture a statistically significant p-value when he pools the 24-month studies, as well as a significant p-value when pooling all four CD-1 studies. This is a breathtaking manipulation that can only be charitably described as statistical malpractice.
- Dr. Portier's summaries of the results for each of the five tumors introduce logical circularities and other redundancies that artificially boost the impact of his findings. For example, consider his discussion of kidney tumors. After alternately pooling the 18-month, 24-month, and all CD-1 studies, Dr. Portier then compares the observed adenoma rates to historical controls. (As an aside, historical controls are not considered by most statisticians or statistical sources as a valid means of establishing causation, as discussed earlier. However, even using Dr. Portier's criterion on page 21 of his report, it is unclear why he uses historical controls in his analysis of the mouse studies that were not "from untreated control groups from studies in the same laboratory within two to three years of the study being evaluated.") His conclusion is that, given historical control rates, the two adenomas observed in each of the highest dose groups of the 24-month studies is highly improbable, and strengthens the evidence of a compound-related effect. However, as discussed earlier in Section III in the context of multiple hypothesis tests, this is self-evident when we are evaluating hundreds of tumor types across 12 studies: while such a result may be improbable for a single analysis, it is nearly certain that we would observe such results for many tumors when we are computing hundreds of p-values. Dr. Portier is merely providing another outstanding explanation for how a false positive arises when we carry out a large number of statistical tests.

- Dr. Portier declares that all five mouse studies, including the four CD-1 studies and the Swiss Albino study, are "useful", but then confines his analysis to the CD-1 studies. No explanation is given for the omission of the Kumar study.

Dr. Portier's joint analysis of the rat studies (under "Summary – Rats" on pages 32-35 in his expert report) is similarly uneven, suffering from inconsistencies similar to his mouse analyses. To highlight:

- As with the mouse studies, for his "pooled" analyses of rats Dr. Portier selects only those tumor types with statistically significant individual p-values (unadjusted for false discovery rates). There is no systematic approach applied to the dozens of other tumor types that were evaluated, and no attempt to make an adjustment for multiple comparisons.
- Dr. Portier carried out "pooled" analyses of both liver adenomas, mammary gland tumors, and skin keratocanthomas among the three studies that used Wistar rats (Brammer, Suresh, and Wood in Table 2). As discussed previously, for his analysis of liver adenomas Dr. Portier eliminated the Suresh study, without any formal statistical justification, based only on his personal judgment that the studies cannot be combined because of differences in underlying tumor rates. He likewise excluded the Suresh study from his "pooled" analysis of mammary gland adenomas, but then included Suresh for testing skin keratocanthomas. For all three tumor types, Dr. Portier's arbitrary exclusion or inclusion resulted in a "pooled" p-value < 0.05. Again, as noted before, an averaged or pooled effect can be estimated even if the underlying average tumor rates differ, provided that the observed effects across the studies are consistent. Dr. Portier made no attempt to evaluate the latter issue, which invalidates his results.

(V.D) Evolution of Dr. Portier's Analyses of Animal Carcinogenicity Studies

In addition to the flaws in Dr. Portier's expert report, there are other serious questions about the consistency of his approach, particularly in light of how his work has evolved. He at times appears to selectively rely on analytic strategies motivated primarily by arbitrarily seeking for "statistical significance" (i.e., computing more p-values < 0.05). A few illustrations:

- The IARC Glyphosate monograph – for which Dr. Portier served as an invited specialist – used approximate trend test p-values to assess potential glyphosate effects for the Knezevich data. As discussed in Section IV, approximate p-values tend to underestimate the corresponding actual p-values, and thus increase the potential for "statistically significant" results that are only due to chance. As outlined in the supplementary material of Dr. Portier's expert report, criticism of the approximate trend test by Dr. Joseph Haseman and others prompted Dr. Portier to rely solely on the exact test in his subsequent work. However, he has resorted again to approximate p-values for some of the p-values he computes using historical controls, arguing that the sample sizes justify their use. Since exact p-values can be computed instantaneously using modern software, there is no good reason to use approximate

tests, particularly when their substantive results disagree with the exact p-values that they are merely estimating.

- It is particularly puzzling that Dr. Portier has previously dismissed the rat feeding studies, declaring that they provide no collective evidence that glyphosate increases cancer risk (for example, on page 11 of Document 9 in the supplementary material of his expert report). He offers no explanation regarding why he has now decided that the statistical evidence supports such an association.
- Dr. Portier reports only results that demonstrate increasing tumor incidence for increasing glyphosate dose, but mentions nothing about tumors that demonstrate decreasing risk of tumor across the treatment groups. When computing one-sided p-values in the absence of any strong prior evidence in favor of either a positive or negative effect, statistical convention dictates that we maintain equipoise about what is observed, even if the result is counterintuitive or in a direction opposite of what we would either hope for or expect.
- Dr. Portier also appears to be inconsistent in his standard for statistical significance. After quoting EPA guidelines on page 20 of his expert report, establishing a significance threshold of 5%, he later (on page 25) fudges somewhat to suggest that we should also consider p-values between 5% and 10%. This is borne out in Tables 8 and 14, where he implies "statistical significance" by highlighting p-values > 0.05 for multiple tumor sites. This further elevates the likelihood of observing false positive results, even assuming his other strategies (i.e., historical controls and "pooled" analyses) were actually valid.

VI. Conclusion

As discussed in Sections III and IV, in the context of the hundreds of tumors evaluated across all 12 rodent glyphosate feeding experiments, it is clear that the individual statistically significant findings closely follow the pattern we would expect given that glyphosate does not increase the risk of cancer. Dr. Portier's own analysis of the rodent feeding studies violates several major foundational principles of statistical practice. His entire approach is based on p-values, which he has selectively reported and used to highlight those findings that are statistically significant, without applying any commonly recommended methods to account for the hundreds of individual tumor types evaluated across the 12 experiments. Dr. Portier has further employed other flawed strategies, including the use of historical controls and the "pooling" of subsets of the data to generate additional p-values, which he has computed using inconsistent and arbitrary standards. Dr. Portier's "pooled" analyses are deeply defective, lacking any accounting for study heterogeneity or differences in observed effects as recommended by Dr. Portier's own cited sources. His simple aggregating of data – as though data from disparate studies arose from the same experiment – is completely inappropriate and unsupported by any credible statistical text or manual

regarding methods for analyzing data from multiple sources. Dr. Portier's analytic strategy seriously violates our own profession's "Statement on p-Values: Context, Process, and Purpose" (*The American Statistician*), referenced in Section III, which notes in part: "*P-values and related analyses should not be reported selectively. Conducting multiple analyses of the data and reporting only those with certain p-values (typically those passing a significance threshold) renders the reported p-values essentially uninterpretable. Cherry-picking promising findings, also known by such terms as data dredging, significance chasing, significance questing, selective inference, and 'p-hacking,' leads to a spurious excess of statistically significant results…and should be vigorously avoided.*"

July 31, 2017

Christopher D. Corcoran — Date

APPENDIX A – RESULTS FOR RAT FEEDING STUDIES

TABLE A.1 – Lankas Rat Results, by Tumor Type and Adjusted for Multiple Tests.

MALES	
TUMOR SITE AND TYPE	p
pituitary adenoma	0.394
pituitary carcinoma	0.785
brain glioma	0.703
heart sarcoma	0.253
lung met undiff sarcoma	0.250
lung cell carcoma	0.514
lung lymphoma	0.750
lung met ost sarcoma	0.750
lung met mixed tumor	0.500
liver cell sarcoma	0.440
liver lymphoma	0.626
liver met undiff sarcoma	0.750
liver neo nodule	0.474
liver hep carcinoma	0.061
mes lymph angioma	0.547
mes lymph lymphoma	0.623
mes lymph cell sarcoma	0.454
pancreas islet cell adenoma	0.509
pancreas islet cell carcinoma	0.251
pancreas acinar cell adenoma	0.251
pancreas lymphoma	0.749
pancreas cell sarcoma	0.644
salivary cell sarcoma	0.250
med lymph fibrosarcoma	0.241
med lymph cell sarcoma	0.593
spleen angiosarcoma	0.750
spleen lymphoma	0.626
spleen cell sarcoma	0.201
stomach cell sarcoma	0.250
jejunum cell sarcoma	0.255
kidney adenoma	0.813
kidney lymphoma	0.750
kidney cell sarcoma	0.735
kidney lipoma	0.735
testis cell tumor	0.009
prostate cell sarcoma	0.251
bladder papilloma	0.494

FEMALES	
TUMOR SITE AND TYPE	p
pituitary adenoma	0.938
pituitary carcinoma	0.084
brain carcinoma	0.189
brain lymphoma	0.251
brain glioma	0.251
spinal cord	0.250
heart lymphoma	0.250
heart sarcoma	0.750
trachea fibrosarcoma	0.751
esophagus fibrosarcoma	0.636
lung cell carcoma	0.282
lung lymphoma	0.317
lung mamm adenocarcinoma	0.253
lung adrenal carcinoma	0.253
lung met fibrosarcoma	0.753
liver cell sarcoma	0.490
liver lymphoma	0.062
liver met fibrosarcoma	0.750
liver hep carcinoma	0.156
liver neo nodule	0.732
mes lymph lymphoma	0.267
mes lymph cell sarcoma	0.070
pancreas islet cell adenoma	0.874
pancreas islet cell carcinoma	0.292
salivary fibrosarcoma	0.250
thymus lymphoma	0.224
thymus thymoma	0.266
med lymph fibrosarcoma	0.744
med lymph cell sarcoma	0.094
med lymph lymphoma	0.058
spleen lymphoma	0.062
spleen sarcoma	0.062
stomach lymphoma	0.250
stomach cell sarcoma	0.750
stomach fibrosarcoma	0.750
jejunum leiomyosarcoma	0.500
ileum cell sarcoma	0.249

thyroid ccell adenoma	0.738
thyroid carcinoma	0.253
thyroid foll adenoma	0.123
parathyroid adenoma	0.743
adrenal cell sarcoma	0.250
adrenal chromocytoma	0.158
adrenal cort adenoma	0.844
adrenal lymphoma	0.750
skin cell tumor	0.251
skin adenoma	0.251
muscle cell sarcoma	0.747
harderian lymphoma	0.759
marrow lymphoma	0.646
marrow sarcoma	0.597

colon cell sarcoma	0.244
kidney lymphoma	0.250
kidney ret cell sarcoma	0.108
kidney trans cell sarcoma	0.250
bladder trans cell carcinoma	0.232
ovary gran cell tumor	0.657
ovary theca cell tumor	0.234
uterus cell carcinoma	0.247
uterus endo sarcoma	0.247
uterus adenoma	0.197
uterus polyp	0.605
uterus ret cell sarcoma	0.809
thyroid ccell adenoma	0.671
thyroid ccell carcinoma	0.003
thyroid foll adenoma	0.964
thyroid fbirosarcoma	0.244
parathyroid adenoma	0.240
adrenal cell sarcoma	0.109
adrenal chromocytoma	0.351
adrenal cortical adenoma	0.850
adrenal cortical carcinoma	0.386
adrenal lymphoma	0.246
mammary adenoma	0.497
mammary fibroadenoma	0.804
mammary adenocarcinoma	0.457
mammary ret cell sarcoma	0.746
eye fibrosarcoma	0.242
harderian lymphoma	0.240
harderian fibrosarcoma	0.240
marrow lymphoma	0.188
marrow sarcoma	0.062

1

2

1 **TABLE A.2 – Stout Rat Results, by Tumor Type and Adjusted for Multiple Tests.**

MALES	
TUMOR SITE AND TYPE	p
adrenal adenoma	0.063
adrenal chromocytoma B	0.248
adrenal chromocytoma M	0.585
adrenal ganglione	0.504
brain astrocytoma	0.297
bone sarcoma	0.245
cervical astrocytoma	0.496
cervical glioma	0.747
duodenum carcinoma	0.749
eyes sarcoma	0.250
kidney lipoma	0.938
kidney liposarcoma	0.500
kidney mesenchymal	0.500
kidney adenoma	0.751
liver adenoma	0.016
liver carcinoma	0.610
liver sarcoma	0.313
liver neoplasm	0.500
mammary gland adenoma	0.282
mammary gland carcinoma	0.717
mammary gland canthoma	0.243
lymph node gioma	0.250
nose adenoma	0.245
pancreas adenoma	0.147
pancreas carcinoma	0.752
pituitary distalis	0.665
pituitary intermedia	0.251
prostate carcinoma	0.750
parathyroid adenoma	0.243
skin canthoma	0.077
skin carcinoma	0.546
skin adenocarcinoma	0.752
skin cytoma	0.500
skin zymbal's gland adenoma	0.498
skin basal cell	0.248
skin papilloma	0.735
skin sebaceous gland adenoma	0.312
skin fibroma	0.752
sp cord thoracic cytoma	0.500
testies interstitial	0.297

FEMALES	
TUMOR SITE AND TYPE	p
adrenal adenoma	0.664
adrenal chromocytoma b	0.268
adrenal chromocytoma m	0.250
adrenal carcinoma	0.015
brain cell tumor	0.500
cecum sarcoma	0.507
kidney lipoma	0.500
kidney carcinoma	0.500
kidney hemangioma	0.750
liver adenoma	0.922
liver carcinoma	0.167
liver sarcoma	0.500
liver giosarcoma	0.500
liver cholangioma	0.750
lung adenoma	0.750
mammary gland adenoma	0.252
mammary gland carcinoma	0.770
mammary gland carcinosarcoma	0.438
nose carcinoma	0.500
ovary granulosa	0.684
ovary theca	0.749
pancreas adenoma	0.962
pituitary adenoma	0.996
pituitary carcinoma	0.434
parathyroid adenoma	0.859
skin carcinoma	0.248
skin zymbal's cell adenoma	0.500
skin basal cell	0.748
skin clitoral gland adenoma	0.748
spleen lymphoma	0.250
spleen hemangioma	0.250
spleen sarcoma	0.750
thyroid adenoma	0.050
thyroid carcinoma	0.500
thyroid cystadenoma	0.438
thyroid foll cell carcinoma	0.250
thymus lymphoma	0.937
urinary papilloma	0.500
uterus polyp	0.355
uterus hamartoma	0.498

thyroid adenoma	0.067
thyroid c cell carcinoma	0.441
thyroid cystadenoma	0.407
thyroid follicular cell carcinoma	0.254
thymus lymphoma	0.479

uterus sarcoma	0.498
uterus adenoma	0.749
uterus leiomyoma	0.749
uterus fibroma	0.749

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1 **TABLE A.3(i) – Atkinson Male Rat Results, by Tumor Type and Adjusted for Multiple Tests.**

MALES	
TUMOR SITE AND TYPE	p
adrenals cortical adenoma	0.909
adrenals uni phaeochromocytoma (M)	0.517
adrenals uni phaeochromocytoma (B)	0.134
adrenals bi phaeochromocytoma (B)	0.517
brain granular cell tumor	0.307
brain glioma	0.685
kidneys tubular adenoma	0.800
kidneys urothelial carcinoma	0.400
liver carcinoma	0.681
liver adenoma	0.322
lungs squamous cell carcinoma	0.403
lungs alveolar/bronchiolar adenoma	0.763
mammary glands fibroadenoma	0.303
mammary glands carcinoma	0.548
mesenteric lymph nodes haemangioma	0.819
pancreas exocrine adenoma	0.945
pancreas islet adenoma	0.973
parathyroids adenoma	0.699
pituitary carcinoma	0.750
pituitary adenoma	0.981
prostate carcinoma	0.307
prostate adenoma	0.307
salivary glands parotid fibroma	0.796
skin trichoepithelioma	0.331
skin basal cell tumor	0.697
skin zymbal's carcinoma	0.697
skin squamous-cell carcinoma	0.303
skin sarcoma	0.690
skin schwannoma	0.545
skin papilloma	0.303
skin fibrosarcoma	0.296
skin fibroma	0.489
skin dermal fibroma	0.561
skin lipoma	0.725
skin epithelioma	0.047
testes uni interstitial-cell adenoma	0.976
testes bi interstitial-cell adenoma	0.303
testes interstitial-cell adenoma	0.303
thymus thymoma	0.684
thyroids follicular carcinoma	0.310

FEMALES	
TUMOR SITE AND TYPE	p
adrenals cortical carcinoma	0.269
adrenals uni phaeochromocytoma (B)	0.975
adrenals bi phaeochromocytoma (B)	0.736
brain glioma	0.586
duodenum carcinoma	0.263
kidneys mesenchymal tumor	0.798
liver adenoma	0.235
lungs alveolar/bronchiolar carcinoma	0.400
lungs sarcoma	0.800
mammary glands fibroadenoma	0.334
mammary glands met carcinoma	0.267
mammary glands carcinoma	0.259
mammary glands adenoma	0.450
ovaries granulosa cell tumor	0.425
pancreas exocrine carcinoma	0.732
pancreas islet adenoma	0.733
parathyroids adenoma	0.448
pituitary carcinoma	0.384
pituitary adenoma	0.525
salivary glands mandibular fibroma	0.395
skin basal cell tumor	0.428
skin sebaceous carcinoma	0.733
skin zymbal's carcinoma	0.744
skin squamous-cell carcinoma	0.583
skin sarcoma	0.733
skin fibroma	0.505
skin lipoma	0.070
skin epithelioma	0.733
thyroids uni c-cell adenoma	0.108
thyroids bi c-cell adenoma	0.927
uterus stromal sarcoma	0.265
uterus met endometrial carcinoma	0.584
uterus endometrial carcinoma	0.461
uterus endometrial adenoma	0.735
uterus polyp	0.367

thyroids follicular adenoma	0.067
thyroids uni c-cell carcinoma	0.310
thyroids bi met c-cell carcinoma	0.310
thyroids uni c-cell adenoma	0.400
thyroids bi c-cell adenoma	0.310
thyroids uni adenoma	0.310

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TABLE A.4 – Brammer Rat Results, by Tumor Type and Adjusted for Multiple Tests.

MALES	
TUMOR SITE AND TYPE	p
adrenal phaeochromoytoma b	0.806
adrenal adenoma	0.313
adrenal phaeochromocytoma m	0.313
brain astrocytoma	0.438
brain meningioma	0.313
brain ependymoma	0.250
epididymis mesothelioma b	0.316
epididymis mesothelioma m	0.502
heart schwannoma	0.750
kidney haemangioma	0.250
kidney mesenchymal tumour	0.250
lacrimal gland neurofibrosarcoma	0.750
liver adenoma	0.008
liver liposarcoma	0.250
lung adenocarcinoma	0.500
lymph node-m haemangioma	0.687
lymph node-m haemangiosarcoma	0.814
nasal cavity fibrosarcoma	0.250
nasal cavity papilloma	0.250
nasal cavity ameloblastoma	0.500
pancreas exocrine adenoma	0.095
pancreas exocrine adenocarcioma	0.500
pancreas islet cell adenoma	0.576
parathyroid gland adenoma	0.500
pharynx carcinoma	0.753
pituitary gland adenoma pars distalis	0.386
pituitary gland adenoma pars intermedia	0.387
salivary gland neurofibrosarcoma	0.254
skin papilloma	0.247
skin basal cell tumour	0.387
skin basal cell carcinoma	0.247
skin pilomatrixoma	0.430
skin xeratoacanthoma	0.387
skin adenoma	0.496
skin trichofolliculoma	0.498
skin sarcoma	0.749
spleen not otherwise specified sarcoma	0.500
spleen not otherwise specified sarcoma	0.500
testis leydig cell tumor	0.791
testis mesothelioma b	0.502

FEMALES	
TUMOR SITE AND TYPE	p
adrenal ganglioneuroma	0.498
adrenal phaeochromocytoma	0.890
brain astrocytoma	0.202
brain meningioma	0.250
brain pineal gland tumour	0.250
cervix stromal cell polyp	0.250
cervix adenocarcinoma	0.438
cervix sarcoma	0.062
cervix haemangiosarcoma	0.250
duodenum adenocarcinoma	0.506
duodenum leiomyoma	0.506
harderian gland anaplastic sarcoma	0.502
ileum leiomyosarcoma	0.519
kidney liposarcoma	0.250
liver adenoma	0.250
lymph node-m haemangioma	0.762
lymph node-m haemangiosarcoma	0.432
mammary gland adenocarcinoma	0.264
mammary gland adenoma	0.894
mammary gland cystadenoma	0.519
mammary gland fibroadenoma	0.377
nasal cavity papilloma	0.187
nasal cavity adenoma	0.500
pancreas adenocarcinoma	0.252
pancreas islet cell adenoma	0.252
pituitary gland adenoma pars distalis	0.280
salivary gland adenoma	0.751
skin squamous carcinoma	0.313
skin basal cell tumour	0.250
skin pilomatrixoma	0.438
spleen haemangiosarcoma	0.502
stomach squamous papilloma	0.251
thymus thymoma b	0.629
thymus thymoma m	0.626
thymus not otherwise specified sarcoma	0.252
thyroid gland follicular cell adenoma	0.833
thyroid gland parafollicular cell adenoma	0.499
thyroid gland parafollicular cell carcinoma	0.252
uterus stromal cell polyp	0.950
uterus adenocarcinoma	0.816

testis mesothelioma m	0.502
thymus benign thymoma	0.112
thyroid gland follicular cell adenoma	0.072
thyroid gland parafollicular cell adenoma b	0.882
thyroid gland parafollicular cell adenoma m	0.502
voluntary muscle haemangioma	0.251

uterus leiomyoma	0.438
uterus carcinoma	0.297
uterus haemangiosarcoma	0.625
uterus haemangioma	0.250

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1 **TABLE A.5 – Suresh Rat Results, by Tumor Type and Adjusted for Multiple Tests.**

MALES	
TUMOR SITE AND TYPE	p
salivary gland duct palpinoma	0.691
stomach adenocarcinoma	0.307
stomach papilloma-forestomach	0.503
pancreas islet cell adenoma	0.742
pancreas carcinoma	0.509
pancreas sarcoma	0.308
pancreas lymphosarcoma	0.698
liver cholangiocarcinoma	0.263
liver hepatocellular adenoma	0.391
liver carcinoma	0.418
liver b.d. adenoma	0.937
liver histiocytic sarcoma	0.624
liver tumour emboli	0.370
liver fibrosarcoma	0.495
liver lymphosarcoma	0.747
liver benign b.d. adenoma	0.253
lungs histiocytic sarcoma	0.433
lungs cholangiocarcinoma	0.503
lungs adenocarcinoma	0.296
lungs hepatocellular carcinoma	0.322
lungs squamous cell carcinoma	0.704
lungs giant cell tumour	0.296
heart histiocytic sarcoma	0.445
spleen cholangiocarcinoma	0.515
mesentric lymph nodes sarcoma	0.695
mediastinal lymph node sarcoma - metastatic	0.634
mediastinal lymph node cholangiocarcinoma	0.494
mediastinal lymph node hepatocellular carcinoma	0.494
mediastinal lymph node giant cell tumour	0.306
mediastinal lymph node sarcoma	0.306
mandibular lymph node lymphoma	0.087
kidneys carcinoma	0.503
kidneys histiosarcoma	0.299
testes leydig cell tumor	0.182
testes seminoma	0.296
epididymes sarcoma	0.250
brain squamous cell carcinoma	0.309
thyroids c cell adenoma	0.595
pituitary adenocarcinoma	0.503
pituitary adenoma	0.376

FEMALES	
TUMOR SITE AND TYPE	p
stomach papilloma-forestomach	0.355
pancreas islet cell adenoma	0.355
pancreas cholangio-carcinoma	0.638
pancreas histiocytic sarcoma	0.638
liver cholangiocarcinoma	0.746
liver adenoma	0.922
liver carcinoma	0.869
liver b.d. adenoma	0.503
liver histiocytic sarcoma	0.711
lungs bronchio alveolar adenoma	0.434
lungs histiocytic sarcoma-metastatic	0.667
lungs adenoma	0.633
lungs fibroma	0.480
lungs round cell sarcoma	0.333
lungs histiocytic sarcoma	0.633
trachea sarcoma	0.472
heart histiocytic sarcoma	0.594
heart round cell sarcoma	0.350
mediastinal lymph node histiocytic sarcoma-m	0.650
mediastinal lymph node cholangiocarcinoma	0.650
mediastinal lymph node histiocytic sarcoma	0.482
kidney lymphosarcoma	0.352
urinary bladder carcinoma	0.350
uterus adenoma	0.289
uterus adenocarcinoma	0.643
uterus carcinoma	0.514
uterus leiomyosarcoma	0.289
uterus adenoma papillary	0.514
uterus hemangioma	0.289
thyroids c cell adenoma	0.537
pituitary adenocarcinoma	0.684
pituitary adenoma	0.967
adrenals cortical cell adenoma	0.400
adrenals pheochromocytoma	0.133
thymus thymoma	0.755
mammary gland adenoma	0.538
mammary gland adenocarcinoma	0.982
tumour/mass histiocytic sarcoma	0.658
tumour/mass cholangiocarcinoma	0.635
tumour/mass fibroma	0.635

adrenals cortical cell adenoma	0.922
adrenals pheochromocytoma	0.066
adrenals m. pheochromocytoma	0.213
tumour/mass squamous cell carcinoma	0.301
tumour/mass histiocytic sarcoma	0.123
tumour/mass cholangiocarcinoma	0.659
tumour/mass giant cell tumour	0.123
tumour/mass fibroma	0.315
bone sarcoma	0.694
sternum sarcoma	0.690

tumour/mass undifferentiated sarcoma	0.635

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1 **TABLE A.6 – Enemoto Rat P-Values, by Tumor Type and Adjusted for Multiple Tests.**

MALES	
TUMOR SITE AND TYPE	p
heart schwannoma	0.297
hematopoietic and lymphatic myelogenic leukemia	0.750
hematopoietic and lymphatic malignant lymphoma	0.813
hematopoietic and lymphatic cell leukemia	0.813
spleen histiocytic sarcoma	0.500
lung adenoma	0.146
lung squamous cell carcinoma	0.250
lung adenocarcinoma	0.250
stomach leiomyosarcoma	0.250
small intestine leiomyoma	0.250
small intestine adenocarcinoma	0.250
small intestine malignant schwannoma	0.500
liver adenoma	0.250
liver carcinoma	0.323
pancreas acinar cell adenoma	0.120
pancreas islet cell adenoma	0.846
pancreas islet cell carcinoma	0.250
kidney adenoma	0.004
kidney lipoma	0.250
testis cell tumor	0.576
coagulating gland adenoma	0.250
pituitary anterior adenoma	0.132
pituitary adenoma (intermediate part)	0.500
pituitary (mass not in section)	0.250
thyroid follicullar adenoma	0.947
thyroid c-cell adenoma	0.623
thyroid follicullar adenocarcinoma	0.750
thyroid c-cell carcinoma	0.514
adrenal cortical adenoma	0.620
adrenal pheochromromocytoma	0.892
adrenal cortical adenocarcinoma	0.250
cerebrum glioma	0.392
cerebrum malignant reticulosis	0.750
cerebellum cell tumor	0.250
bone (femur) osteochondroma	0.250
bone (other) osteosarcoma	0.250
eye shwannoma	0.750
skin papilloma	0.946
skin keratoacanthoma	0.029
skin trichoepithelioma	0.500

FEMALES	
TUMOR SITE AND TYPE	p
heart schwannoma	0.250
hematopoietic/lymphatic lymphoma	0.259
small intestine leiomyoma	0.250
large intestine histioctytoma	0.750
liver hepatocellular adenoma	0.813
pancreas islet cell adenoma	0.812
pancreas islet cell carcinoma	0.500
kidney lipoma	0.500
kidney trans cell carcinoma	0.750
bladder papilloma	0.500
ovary granulosa cell tumor	0.750
ovary luteoma	0.250
uterus stromal polyp	0.656
uterus grnaular cell tumor	0.750
uterus adenocarcinoma	0.750
uterus schwannoma	0.250
uterus (mass not in section)	0.750
vagina polyp	0.750
vagina leiomysarcoma	0.250
pituitary anterior adenoma	0.819
pituitary anterior adenocarcinoma	0.750
thyroid follicular adenoma	0.688
thyroid c-cell adenoma	0.908
adrenal cortical adenoma	0.500
adrenal ganglioneuroma	0.500
adrenal pheochromocytoma	0.500
cerebrum meningioma	0.500
cerebrum reticulosis	0.813
bone (vertebra) chordoma	0.750
skin papilloma	0.500
skin keratoacanthoma	0.250
skin fibroma	0.400
skin lipoma	0.932
skin (mass not in section)	0.580
mammary gland adenoma	0.813
mammary gland fibroadenoma	0.106
mammary gland adenocarcinoma	0.595

skin sabaceous gland adenoma	0.500
skin basal cell adenoma	0.015
skin fibroma	0.262
skin lipoma	0.873
skin squamous cell carcinoma	0.187
skin basal cell carcinoma	0.250
skin fibrosacoma	0.500
skin liposarcoma	0.750
skin hemangiosarcoma	0.250
skin hemangiopericytoma	0.250
skin osteosarcoma	0.313
skin schwannoma	0.250
skin histiocytic sarcoma	0.250

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1 **TABLE A.7 – Wood Rat Results, by Tumor Type and Adjusted for Multiple Tests.**

MALES	
TUMOR SITE AND TYPE	p
adrenal cortical adenoma	0.813
adrenal cortical carcinoma	0.750
adrenal phaeochromocytoma b	0.062
adrenal phaeochromocytoma m	0.805
bone osteoma	0.250
brain/spinal cord astrocytoma	0.250
brain/spinal cord granular cell tumour b	0.813
brain/spinal cord granular cell tumour m	0.250
intestinal tract leiomyoma	0.250
intestinal trace leiomsarcoma	0.250
epididymis mesothelioma b	0.750
epididymis mesothelioma m	0.751
heart schwannoma	0.500
kidney lipoma	0.250
kidney tubular carcinoma	0.750
liver hepatocellular adenoma	0.418
liver hepatocellular carcinoma	0.750
lymph node angioma	0.357
lymph node angiosarcoma	0.945
nasal cavities adenoma	0.938
pancreas islet cell adenoma	0.827
parathyroid adenoma	0.750
pituitary adenoma	0.045
pituitary adenocarcinoma	0.750
skin - subcutaneous fibroma	0.595
skin - subcutaneous fibrosarcoma	0.903
skin - subcutaneous histiocytic sarcoma	0.500
skin - subcutaneous lipoma	0.250
skin - leiomyosarcoma	0.250
skin - cutaneous basal cell tumor	0.750
skin- cutaneous carcinoma	0.675
skin - cutaneous keratoacanthoma	0.030
skin - cutaneous adenoma	0.500
skin - cutaneous adenocarcinoma	0.500
skin - cutaneous trichoepithelioma	0.250
skin - cutaneous papilloma	0.250
skin - cutaneous s.s. carcinoma	0.500
spleen angioma	0.250
spleen angiosarcoma	0.750
stomach papilloma	0.370

FEMALES	
TUMOR SITE AND TYPE	p
adrenal cortical adenoma	0.813
adrenal ganglioneuroma	0.250
brain/spinal cord oligodendroglioma	0.750
brain/spinal cord ependymoma	0.813
heart schwannoma	0.938
kidney clear cell carcinoma	0.250
liver adenoma	0.392
liver carcinoma	0.250
liver cholangioma	0.750
lymph node angioma	0.748
mammary gland fibroadenoma	0.824
mammary gland adenoma	0.062
mammary gland adenocarcinoma	0.042
ovary granulosa cell tumour	0.928
ovary granulosa-theca cell tumour	0.943
ovary sarcoma	0.750
pancreas adenocarcinoma	0.250
pharynx papilloma	0.250
pituitary adenoma	0.014
pituitary adenocarcinoma	0.500
skin - subcutaneous fibroma	0.250
skin - subcutaneous lipoma	0.313
skin - subcutaneous angioma	0.062
skin - cutaneous basal cell tumour	0.750
skin - cutaneous carcinoma	0.250
skin - cutaneous papilloma	0.500
stomach papilloma	0.500
thymus thymoma b	0.765
thymus carcinoma	0.250
thyroid follicular adenoma	0.372
thyroid follicular adenocarcinoma	0.813
thyroid parafollicular adenoma	0.997
thyroid parafollicular adenocarcinoma	0.500
tongue granular cell tumor	0.250
uterus polyp	0.221
uterus adenocarcinoma	0.602
uterus sarcoma	0.438
uterus leiomyoma	0.514
uterus angiosarcoma	0.500
lymhoid/haemopoietic lymphoma	0.830

testis interstitial cell tumour	0.778
thymus thymoma b	0.187
thymus thymoma m	0.313
thyroid adenoma	0.066
thyroid adenocarcinoma	0.250
thyroid parafollicular adenoma	0.823
thyroid parafollicular adenocarcinoma	0.938
thyroid hibernoma	0.250
urinary bladder papilloma	0.500
abdominal adenocarcinoma	0.500
abdominal carcinoma	0.250
lymphoid/haemopoietic lymphoma	0.500

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APPENDIX B – RESULTS FOR MOUSE FEEDING STUDIES

TABLE B.1 –Knezevich Mouse P-Values, by Tumor Type and Adjusted for Multiple Tests.

MALES	
TUMOR SITE AND TYPE	p
brain lymphoblastic lymphosarcoma w/leuk	0.251
heart lymphoblastic lymphosarcoma w/leuk	0.337
lung adenoma	0.294
lung adenocarcinoma	0.906
lung lymphoblastic lymphosarcoma w/leuk	0.772
lung lymphoblastic lymphosarcoma	0.505
liver adenocarcinoma	0.717
liver adenoma	0.251
liver carcinoma	0.062
liver sarcoma	0.503
liver liposarcoma	0.189
liver composite lymphosarcoma	0.754
liver lymphoblastic lymphosarcoma	0.539
mesenteric sarcoma	0.492
mesenteric lymphosarcoma	0.624
mesenteric lymphoblastic lymphosarcoma (S)	0.827
mesenteric lymphoblastic lymphosarcoma (M)	0.061
mesenteric lymphoblastic lymphosarcoma w/leuk	0.492
mediastinal sarcoma	0.489
mediastinal lymphosarcoma	0.631
mediastinal lymphoblastic lymphosarcoma w/leuk (S)	0.373
mediastinal lymphoblastic lymphosarcoma w/leuk (M)	0.463
salivary glands lymphoblastic lymphosarcoma w/leuk	0.628
spleen hemangioendothelioma	0.250
spleen sarcoma	0.505
spleen composite lymphosarcoma	0.631
spleen lymphoblastic lymphosarcoma w/leuk (S)	0.827
spleen lymphoblastic lymphosarcoma w/leuk (M)	0.442
stomach lymphoblastic lymphosarcoma w/leuk	0.746
pancreas sarcoma	0.508
pancreas lymphoblastic lymphosarcoma w/leuk	0.256
ileum composite lymphosarcoma	0.733
ileum lymphoblastic lymphosarcoma w/leuk	0.733
cecum lymphoblastic lymphosarcoma w/leuk	0.753
colon composite lymphosarcoma	0.755
kidney adenoma (using EPA reeval)	0.442
kidney carcinoma (using EPA reeval)	0.063
kidney sarcoma	0.505

FEMALES	
TUMOR SITE AND TYPE	p
brain lymphoblastic lymphosarcoma w/leuk	0.251
heart lymphoblastic lymphosarcoma w/leuk	0.433
lung adenoma	0.999
lung adenocarcinoma	0.183
lung granulosa cell tumor	0.500
lung leiomyosarcoma	0.500
lung liposarcoma	0.753
lung composite lymphosarcoma	0.442
lung lymphoblastic lymphosarcoma w/leuk	0.717
lung lymphoblastic lymphosarcoma	0.253
liver adenocarcinoma	0.828
liver adenoma	0.497
liver hemangioendothelioma (M)	0.249
liver leiomyosarcoma	0.497
liver granulocytic leukemia	0.875
liver hemangioendothelioma (S)	0.437
liver composite lymphosarcoma	0.064
liver lymphoblastic lymphosarcoma w/leuk	0.787
liver lymphoblastic lymphosarcoma	0.061
mesenteric leiomyosarcoma	0.495
mesenteric granulocytic leukemia	0.495
mesenteric adenocarcinoma	0.747
mesenteric composite lymphosarcoma	0.141
mesenteric lymphoblastic lymphosarcoma w/leuk (M)	0.522
mesenteric lymphoblastic lymphosarcoma w/leuk (S)	0.782
mesenteric composite lymphosarcoma	0.141
mesenteric lymphoblastic lymphosarcoma (M)	0.060
mesenteric lymphoblastic lymphosarcoma (S)	0.247
mesenteric hemangioendothelioma	0.247
mediastinal leiomyosarcoma	0.489
mediastinal granulocytic leukemia	0.489
mediastinal liposarcoma	0.761
mediastina composite lymphosarcoma	0.266
mediastinal lymphoblastic lymphosarcoma w/leuk (S)	0.717
mediastinal lymphoblastic lymphosarcoma w/leuk (M)	0.760
mediastinal lymphoblastic lymphosarc (M)	0.489
mediastinal lymphoblastic lymphosarc (S)	0.267
salivary glands leiomyosarcoma	0.239

kidney composite lymphosarcoma	0.753
kidney lymphoblastic lymphosarcoma w/leuk	0.463
testes cell tumor	0.649
testes lymphoblastic lymphosarcoma w/leuk (S)	0.508
testes lymphoblastic lymphosarcoma w/leuk (M)	0.254
epididymides leiomysarcoma	0.317
bladder histiocyticsarcoma	0.500
bladder lymphoblastic lymphosarcoma w/leuk	0.810
renal gland adenoma	0.574
renal gland lymphoblastic lymphosarcoma w/leuk (U)	0.503
renal gland lymphoblastic lymphosarcoma w/leuk (B)	0.246
skin/ears fibrosarcoma	0.245
skin/ears liposarcoma	0.245
skin/ears composite lymphosarcoma	0.745
skin/ears lymphoblastic lymphosarcoma w/leuk	0.245
eyes lymphoblastic lymphosarcoma w/leuk	0.643
harderian gland adenoma	0.750
harderian gland liposarcoma	0.255
marrow lymphoblastic lymphosarcoma w/leuk	0.566

salivary lymphoblastic lymphosarcoma w/leuk	0.485
spleen hemangioendothelioma (M)	0.370
spleen hemangioma	0.250
spleen granulocytic leukemia	0.877
spleen adenocarcinoma	0.745
spleen hemangioendothelioma (S)	0.250
spleen composite lymphosarcoma (S)	0.580
spleen lymphoblastic lymphosarcoma w/leuk (S)	0.824
spleen lymphoblastic lymphosarcoma w/leuk (M)	0.438
spleen composite lymphosarcoma (M)	0.016
spleen lymphoblastic lymphosarcoma (M)	0.250
spleen lymphoblastic lymphosarcoma (S)	0.250
stomach leiomyosarcoma	0.254
stomach adenocarcinoma	0.254
duodenum composite lymphosarcoma	0.770
pancreas granulocytic leukemia	0.508
pancreas composite lymphosarcoma	0.638
pancreas lymphoblastic lymphosarcoma w/leuk	0.746
jejunum composite lymphosarcoma	0.761
ileum composite lymphosarcoma	0.758
cecum composite lymphosarcoma	0.766
colon composite lymphosarcoma	0.743
colon lymphoblastic lymphosarcoma w/leuk	0.743
kidney leiomyosarcoma	0.500
kidney granulocytic leukemia	0.500
kidney composite lymphosarcoma	0.395
kidney lymphoblastic lymphosarcoma w/leuk	0.597
kidney lymphoblastic lymphosarcoma	0.250
bladder granulocytic leukemia	0.519
bladder composite lymphosarcoma	0.822
bladder lymphoblastic lymphosarcoma w/leuk	0.838
ovaries luteoma	0.246
ovaries teratoma	0.508
ovaries cell tumor	0.508
ovaries leiomyosarcoma	0.508
ovaries adenocarcinoma	0.754
ovaries lymphoblastic lymphosarcoma w/leuk (U)	0.508
ovaries lymphoblastic lymphosarcoma w/leuk (B)	0.641
ovaries composite lymphosarcoma	0.246
uterus leiomyoma	0.619
uterus leiomyosarcoma	0.385
uterus sarcoma	0.505
uterus hemangioma	0.505

uterus adenocarcinoma	0.939
uterus hemangioendothelioma	0.255
uterus lymphoblastic lymphosarcoma w/leuk	0.821
thyroid adenoma	0.271
skin/ears fibrosarcoma	0.516
skin/ears liposarcoma	0.759
skin/ears rhabdomyosarcoma	0.759
skin/ears lymphoblastic lymphosarcoma w/leuk	0.592
mammary adenocarcinoma	0.842
mammary lymphoblastic lymphosarcoma w/leuk	0.250
muscle liposarcoma	0.749
muscle lymphoblastic lymphosarcoma w/leuk	0.623
harderian gland adenoma	0.830
harderian lymphoblastic lymphosarcoma w/leuk	0.250
marrow lymphoblastic lymphosarcoma w/leuk	0.385
marrow lymphoblastic lymphosarcoma	0.065
marrow composite lymphosarcoma	0.257

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TABLE B.2 – Atkinson Mice Results, by Tumor Type and Adjusted for Multiple Tests.

MALES	
TUMOR SITE AND TYPE	p
adrenals phaeochromocytoma (M)	0.486
adrenals carcinoma	0.486
adrenals phaeochromocytoma (B)	0.338
adrenals adenoma	0.648
adrenals subcap adenoma	0.716
brain meningioma	0.347
kidneys carcinoma	0.813
kidneys adenoma	0.813
liver carcinoma	0.450
liver adenoma	0.583
liver (assoc) adenoma	0.077
lungs carcinoma	0.456
lungs adenoma	0.339
lungs (assoc) adenoma	0.217
pancreas adenoma	0.340
pituitary intermediate adenoma	0.326
prostate sarcoma	0.350
skin carcinoma	0.655
skin sarcoma	0.641
skin papilloma	0.345
skin lipoma	0.345
spinal cord ganglioneuroma	0.655
stomach carcinoma	0.340
testes adenoma	0.520
vascular haemangiosarcoma (using IARC)	0.004

FEMALES	
TUMOR SITE AND TYPE	p
adrenals carcinoma	0.500
adrenals subcap adenoma	0.750
liver carcinoma	0.750
liver adenoma	0.642
lungs carcinoma	0.105
lungs adenoma	0.358
lungs (assoc) adenoma	0.072
lungs secondary tumor	0.201
lymphoreticular sarcoma	0.575
lymphoreticular lymphoma	0.475
mammary glands carcinoma	0.845
mammary glands adenocarcinoma	0.250
ovaries granulosa cell tumor	0.750
ovaries luteal cell tumor	0.250
ovaries adenoma	0.062
pancreas adenoma	0.500
pituitary anterior adenoma	0.155
pituitary intermediate adenoma	0.250
skin carcinoma	0.187
skin sarcoma	0.392
skin papilloma	0.750
spleen sarcoma	0.250
thyroids adenoma	0.250
uterus sarcoma	0.299
uterus stromal tumor	0.250
uterus polyps	0.433
uterus leiomyoma	0.108

TABLE B.3 – Wood Mice Results, by Tumor Type and Adjusted for Multiple Tests.

MALES	
TUMOR SITE AND TYPE	p
adrenal adenoma	0.172
adrenal carcinoma	0.754
bone marrow lipoma	0.750
brain sarcoma	0.251
brain oligodendroglioma	0.754
harderian adenoma	0.502
kidney haemangiosarcoma	0.250
liver adenoma	0.335
liver carcinoma	0.921
liver haemangiosarcoma	0.615
lung adenoma	0.926
lung adenocarcinoma	0.030
seminal adenoma	0.938
seminal leiomyosarcoma	0.250
skin fibrosarcoma	0.542
spleen haemangioma	0.750
testis cell tumor	0.938
abdominal mesothelioma	0.250
abdominal sarcoma	0.250
lymphoid/haemopoietic myeloid leukaemia	0.500
lymphoid/haemopoietic lymphoma	0.007

FEMALES	
TUMOR SITE AND TYPE	p
bone osteoma	0.750
bone marrow sarcoma	0.250
brain oligodendroglioma	0.750
harderian adenoma	0.155
harderian adenocarcinoma	0.938
intestinal adenoma	0.750
liver carcinoma	0.500
liver haemangioma	0.500
liver haemangiosarcoma	0.250
lung adenoma	0.637
lung adenocarcinoma	0.591
mammary adenocarcinoma	0.391
mammary carcinoma	0.500
mesenteric sarcoma	0.534
ovary luteoma	0.514
ovary haemangioma	0.250
ovary cell tumor	0.250
ovary cystadenoma	0.062
ovary sarcoma	0.500
pancreas adenocarcinoma	0.750
pituitary adenoma	0.108
skin haemangiosarcoma	0.500
spleen haemangiosarcoma	0.438
thymus sarcoma	0.250
uterus polyp	0.170
uterus haemangioma	0.500
uterus leiomyoma	0.250
uterus carcinoma	0.750
uterus sarcoma	0.719
uterus leiomyosarcoma	0.750
abdominal lipoma	0.750
lymphoid/haemopoietic myeloid leukaemia	0.250
lymphoid/haemopoietic lymphoma	0.353
lymphoid/haemopoietic sarcoma	0.482

TABLE B.4 – Sugimoto Mice Results, by Tumor Type and Adjusted for Multiple Tests.

MALES	
TUMOR SITE AND TYPE	p
hematopoietic & lymphatic system lymphoma	0.016
lymph nodes lymphoma	0.500
spleen sarcoma	0.750
lung adenoma	0.512
lung adenocarcinoma	0.148
intestine adenoma	0.500
intestine adenocarcinoma	0.250
liver adenoma	0.984
liver hemangioma	0.750
liver sarcoma	0.750
liver carcinoma	0.391
kidney adenoma	0.062
urinary bladder papilloma	0.751
testis cell tumor	0.500
testis hemangioma	0.750
thyroid adenoma	0.751
adrenal b cell tumor	0.500
cerebrum lipoma	0.500
ilarderian gland adenoma	0.515
skin papilloma	0.813
skin hemangiosarcoma	0.062
skin leiomysarcoma	0.187
skin osteosarcoma	0.250

FEMALES	
TUMOR SITE AND TYPE	p
hematopoietic & lymphatic system leukemia	0.250
hematopoietic & lymphatic system lymphoma	0.307
thymus lymphoma	0.250
spleen hemangioma	0.250
spleen hemangiosarcoma	0.250
spleen sarcoma	0.250
lung adenoma	0.800
lung adenocarcinoma	0.597
small intestine adenoma	0.250
liver adenoma	0.735
liver hemangioma	0.250
urinary bladder leiomyoma	0.187
ovary hemangioma	0.250
uterus polyp	0.751
uterus hemangioma	0.062
uterus leiomyoma	0.370
uterus sarcoma	0.500
uterus leiomyosarcoma	0.624
pituitary adenoma	0.500
thyroid adenoma	0.751
adrenal a cell tumor	0.595
adrenal pheochromocytoma	0.751
bone osteoma	0.250
harderian gland adenoma	0.040
skin papilloma	0.750
skin lipoma	0.626
skin carcinoma	0.500
skin liposarcoma	0.250
skin hemangiosarcoma	0.250
mammary gland adenoma	0.500
mammary gland adenocarcinoma	0.814
thoracic cavity osteosarcoma	0.400
abdominal cavity hemangioma	0.257
abdominal cavity osteosarcoma	0.257

TABLE B.5 – Kumar Mice Results, by Tumor Type and Adjusted for Multiple Tests.

MALES	
TUMOR SITE AND TYPE	p
cecum adenoma	0.648
liver hemangiosarcoma	0.327
liver adenoma	0.846
liver carcinoma	0.249
lungs squamous cell carcinoma	0.500
lungs broncheo-alveolar adenoma	0.463
lungs broncheo-alveolar carcinoma	0.347
mesenteric hemangioma	0.431
mesenteric hemangiosarcoma	0.245
kidneys adenoma	0.090
kidneys hibernoma	0.671
testes tumor	0.345
epididymes leiomyoma	0.503
skin carcinoma	0.791
tumor/mass hemangioma	0.304
bone osteoma	0.582
lymphoreticular sarcoma	0.624
lymphoreticular lymphoma	0.064
lymphoreticular leukemia	0.744

FEMALES	
TUMOR SITE AND TYPE	p
stomach sarcoma	0.515
pancreas sarcoma	0.515
liver sarcoma	0.769
liver adenoma	0.515
lungs endometrial stromal sarcoma	0.365
lungs broncheo-alveolar adenoma	0.165
lungs broncheo-alveolar carcinoma	0.750
mesenteric hemangioma	0.016
mesenteric sarcoma	0.500
kidneys sarcoma	0.511
bladder sarcoma	0.368
ovaries hemangioma	0.304
ovaries sarcoma	0.735
ovaries tumor	0.304
ovaries luteoma	0.304
uterus leiomyosarcoma	0.311
uterus sarcoma	0.793
uterus leiomyoma	0.311
pituitary adenoma	0.372
adrenals sarcoma	0.511
adrenals adenoma	0.363
adrenals pheochromocytoma	0.363
skin carcinoma	0.588
thymus lymphoma	0.629
mammary adenocarcinoma	0.598
tumor/mass hemangiosarcoma	0.562
femur osteoma	0.297
lymph node sarcoma (M)	0.189
lymph node sarcoma (I)	0.189
hemolymphoreticular sarcoma	0.199
hemolymphoreticular lymphoma	0.070
hemolymphoreticular leukemia	0.602

TABLE B.6 – Kumar Mice Results Using Data from Weber Reanalysis, by Tumor Type and Adjusted for Multiple Tests.

MALES		FEMALES	
TUMOR SITE AND TYPE	p	TUMOR SITE AND TYPE	p
cecum adenoma	0.750	stomach sarcoma	0.500
liver adenoma	0.846	pancreas sarcoma	0.500
liver carcinoma	0.155	liver sarcoma	0.688
lungs squamous cell carcinoma	0.500	liver adenoma	0.395
lungs broncheo-alveolar adenoma	0.438	lungs endometrial stromal sarcoma	0.250
lungs broncheo-alveolar carcinoma	0.250	lungs broncheo-alveolar adenoma	0.069
kidneys adenoma	0.250	lungs broncheo-alveolar carcinoma	0.750
kidneys hibernoma	0.750	mesenteric sarcoma	0.500
testes tumor	0.237	kidneys sarcoma	0.500
epididymes leiomyoma	0.500	bladder sarcoma	0.250
skin carcinoma	0.813	ovaries sarcoma	0.495
lymphoreticular sarcoma	0.534	ovaries tumor	0.250
lymphoreticular lymphoma	0.141	uterus leiomyosarcoma	0.250
lymphoreticular leukemia	0.830	uterus sarcoma	0.704
femur osteoma	0.750	uterus leiomyoma	0.830
hemangioma	0.261	pituitary adenoma	0.250
hemangiosarcoma	0.438	adrenals sarcoma	0.500
		adrenals adenoma	0.250
		adrenals pheochromocytoma	0.250
		skin carcinoma	0.500
		mammary adenocarcinoma	0.438
		femur osteoma	0.113
		hemolymphoreticular sarcoma	0.199
		hemolymphoreticular lymphoma	0.085
		hemolymphoreticular leukemia	0.602
		hemangioma	0.014
		hemangiosarcoma	0.750

APPENDIX C – MULTIPLE TESTING ADJUSTMENTS

TABLE C.1 – Summary of findings with individual p-values < 0.05 for exact one-sided trend tests for increasing tumor incidence with increased dose, computed across 1,016 total tumor types, with multiple testing adjustment for the false discovery rate.

Study	Rodent/Strain/Sex	Tumor Type	Exact Trend P-Value	P-Value Adjusted for False Discovery Rate
Lankas	Rat/SD/Male	Testis Cell Tumor	0.009	0.473
	Rat/SD/Female	Thyroid Cell Carcinoma	0.003	0.175
Stout	Rat/SD/Male	Liver Adenoma	0.016	0.703
	Rat/SD/Female	Adrenal Carcinoma	0.015	0.662
Atkinson	Rat/SD/Male	Skin Epithelioma	0.047	0.801
Brammer	Rat/Wistar/Male	Liver Adenoma	0.008	0.370
Enemoto	Rat/SD/Male	Kidney Adenoma	0.004	0.189
	Rat/SD/Male	Skin Keratoacanthoma	0.029	0.510
	Rat/SD/Male	Skin Basal Cell Adenoma	0.015	0.395
Wood	Rat/Wistar/Male	Pituitary Adenoma	0.045	0.684
	Rat/Wistar/Male	Skin Cutaneous Keratoacanthoma	0.030	0.684
	Rat/Wistar/Female	Mammary Gland Adenocarcinoma	0.042	0.616
	Rat/Wistar/Female	Pituitary Adenoma	0.014	0.557
Knezevich	Mouse/CD-1/Female	Spleen Composite Lymphosarcoma (M)	0.016	0.858
Atkinson	Mouse/CD-1/Male	Vascular Haemangiosarcoma	0.004	0.089
Wood	Mouse/CD-1/Male	Lung Adenocarcinoma	0.030	0.312
	Mouse/CD-1/Male	Lymphoid/Haemopoietic Lymphoma	0.007	0.139
Sugimoto	Mouse/CD-1/Male	Hematopoietic & Lymphatic System Lymphoma	0.016	0.373
	Mouse/CD-1/Female	Harderian Gland Adenoma	0.040	0.554
Kumar	Mouse/Swiss/Female	Mesenteric Hemangioma	0.016	0.468

APPENDIX D – MULTIPLE TESTING ADJUSTMENTS

TABLE D.1 – Summary of findings with individual p-values < 0.05 for exact one-sided trend tests for decreasing tumor incidence with increased dose, computed across 1,016 total tumor types, with multiple testing adjustment for the false discovery rate.

Study	Rodent/Strain/Sex	Tumor Type	Exact Trend P-Value	P-Value Adjusted for False Discovery Rate
Lankas	Rat/SD/Female	Thyroid Follicular Adenoma	0.036	0.956
Stout	Rat/SD/Female	Pancreas Adenoma	0.038	0.693
	Rat/SD/Female	Pituitary Adenoma	0.004	0.166
Atkinson	Rat/SD/Male	Pancreas Islet Adenoma	0.027	0.410
	Rat/SD/Male	Pituitary Adenoma	0.019	0.410
	Rat/SD/Male	Testes Uni Interstitial-Cell Adenoma	0.024	0.410
	Rat/SD/Female	Adrenals Uni Phaeochromocytoma (B)	0.025	0.781
Brammer	Rat/Wistar/Female	Uterus Stromal Cell Polyp	0.050	0.805
Suresh	Rat/Wistar	Pituitary Adenoma	0.033	0.671
	Rat/Wistar	Mammary Gland Adenocarcinoma	0.018	0.671
Wood	Rat/Wistar/Female	Thyroid Parafollicular Adenoma	0.003	0.120
Knezevich	Mouse/CD-1/Female	Lung Adenoma	0.001	0.056
Sugimoto	Mouse/CD-1/Male	Liver Adenoma	0.016	0.378

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STAT 5100, Linear Regression and Time Series, 3 courses.
STAT 5120, Categorical Data Analysis, 6 courses.
STAT 5810, Topics in Statistics, 4 courses.
STAT 5820, Topics in Statistics, 1 course.
STAT 5820, 6910, Topics in Statistics, 1 course.
STAT 5970, Seminar, 3 courses.
STAT 6250, Graduate Internship/Co-op, 1 course.
STAT 6550, Statistical Computing, 1 course.
STAT 6810, Topics in Statistics (Topic), 1 course.
STAT 6820, Topics in Statistics (Topic), 1 course.
STAT 6910, Seminar in Statistics, 14 courses.
STAT 6950, Directed Reading and Conference, 1 course.
STAT 6990, Continuing Graduate Advisement, 2 courses.
STAT 7810, Topics in Statistics (Topic), 1 course.
STAT 7990, Continuing Graduate Advisement, 1 course.

Directed Student Learning

Dissertation Committee Chair, "Network Meta-Analysis," Mathematics & Statistics. (September 1, 2014 - Present).
Advised: Brinley Zabriskie

Master's Committee Chair, "Statistical Strategies for Public Database Access and Analysis," Mathematics & Statistics. (September 1, 2014 - Present).
Advised: Christina Stevens

Dissertation Committee Chair, Mathematics & Statistics. (August 2013 - Present).
Advised: Divya Nair

Dissertation Committee Chair, Mathematics & Statistics. (August 2013 - Present).
Advised: Sarah Schwartz

Master's Committee Chair, Mathematics & Statistics. (August 2013 - Present).
Advised: Michael Steelman

Master's Committee Chair, Mathematics & Statistics. (August 2012 - Present).
Advised: Jenny Clements

Dissertation Committee Member, Nutrition, Dietetics and Food Sciences. (August 2010 - Present).
Advised: Meo La

Dissertation Committee Chair, "Computational methods for family-based association tests." (August 2008 - May 2012).
Advised: William Welbourn

Master's Committee Chair, "Serum cytokine levels and risk of dementia." (2011).
Advised: Austin Bowles

Master's Committee Chair, "TBD." (2011).
Advised: Elizabeth Giles

Master's Committee Chair, "Patterns of stressful life events and Alzheimer's disease risk." (2011).
Advised: Megan Platt

Supervised Research/URF, "Effectiveness of surgical strategies for hysterectomy," Biology. (2010).
Advised: Erica Huelsmann

Master's Committee Chair, "Comparing methods for family-based association tests." (2009).
Advised: Abbie Lundgreen

Master's Committee Chair, "Heritability of cognitive change." (2009).
Advised: Colette Childs

Dissertation Committee Chair, "Small-sample inference for correlated categorical data." (2008).
Advised: Larry Cook

Master's Committee Chair, "Heritability of cognitive traits using complex pedigrees and sibships." (2008).
Advised: Cassidy Allen

Master's Committee Chair, "Multivariate analysis of longitudinal neuropsychological measures in the Cache County Memory Study." (2006).
Advised: Sarah Schwartz

Master's Committee Chair, "Computational efficiency of exact family-based association tests." (2006).
Advised: Yanwei Ouyan

Supervised Research/URF, "Nutritional risk factors for cognitive decline among the elderly," Biology. (2006).
Advised: Angela Dunn

Dissertation Committee Chair, "Exact family based association tests." (2004).
Advised: Kady Schneiter

Supervised Research/URF, "Cognitive decline and antioxidant, Vitamin C, and Vitamin E intake among the elderly," Biology. (2003 - 2004).
Advised: Leila King

Supervised Research/URF, "Haplotypes of candidate genes as predictors of hip fracture in the elderly," Biology. (2003 - 2004).
Advised: Sara Anderson

Master's Committee Chair, "Use of classification methods for dementia screening." (2003).
Advised: Leslie Toone

Supervised Research/URF, "Using patient characteristics of the demented to classify dementia type," Mathematics & Statistics. (2003).
Advised: Kimberly Peterson

Supervised Research/URF, "Persistence of behavioral disturbances among the demented," Mathematics & Statistics. (2002 - 2003).
Advised: Craig Huber

Master's Committee Chair, "Correcting for left truncation bias when evaluating survival among the elderly with dementia." (2002).
Advised: Jennifer Harrick

Supervised Research/URF, "General advising for submitting abstract regarding NIH internship project to CUR Posters on the Hill," Mathematics & Statistics. (2002).
Advised: Randy Johnson

Supervised Research/URF, "Genetic factors in shortening time-to-onset of Alzheimer's disease," Mathematics & Statistics. (2002).
Advised: Sunni Mumford

Master's Committee Chair, "Operating characteristics of exact methods for corelated categorical data." (2001).
Advised: Shea Watrin

RESEARCH & OTHER CREATIVE ACTIVITIES

Published Intellectual Contributions

Book Chapters

Book, Chapter in Scholarly Book (Published)
Corcoran, C. D., Senchaudhuri, P., Mehta, C., Patel, N. (2010). Exact Methods for Categorical Data Analysis. In BS Everitt, CR Palmer (Ed.), *Encyclopaedic Companion to Medical Statistics*. London: Hodder Arnold.

Book, Chapter in Non-Scholarly Book (Published)

Corcoran, C. D. (2009). Analysis of Correlated Data. *StatXact Version 8.0 User Manual* (pp. 895-935).

Book, Chapter in Scholarly Book (Published)
Cutler, A., Corcoran, C. D., Toone, L. (2005). Bagging. *Encyclopedia of Statistics in Behavioral Science*. New York: Wiley & Sons.

Book, Chapter in Scholarly Book (Published)
Corcoran, C. D., Ryan, L. M. (2002). Exact Dose-Response Inference. In M Aerts, H Geys, G Molenberghs, and LM Ryan (Ed.), *Topics in Modelling of Clustered Data* (pp. 195-206). New York: Chapman and Hall.

Book, Chapter in Scholarly Book (Published)
Corcoran, C. D. (2002). Trend tests for binary data. In AH El-Shaarawi and WW Piegorsch (Ed.), *Encyclopedia of Environments* (vol. 4, pp. 2260-2264). Chichester: John Wiley & Sons.

Book, Chapter in Non-Scholarly Book (Published)
Corcoran, C. D., Kannappan, A. R., Senchaudhuri, P., Coull, B. (1999). *Egret User Manual*. Cytel Software Corporation.

Refereed Journal Articles

Journal Article, Professional Journal (Accepted)
Rattinger, G. B., Fauth, E. B., Behrens, S., Sanders, C., Schwartz, S., Norton, M. C., Corcoran, C. D., Mullins, C. D., Lyketsos, C. G., Tschanz, J. T. (in press). Closer caregiver and care recipient relationships predict lower informal costs of dementia care. *Alzheimer's & Dementia*.

Journal Article, Professional Journal (Accepted)
Matyi, J. A., Tschanz, J. T., Rattinger, G. B., Sanders, C., Vernon, E. K., Corcoran, C. D., Kauwe, J. S., Buhusi, M. C. (in press). Sex differences in risk for Alzheimer's Disease related to neurotrophin gene polymorphisms: the Cache County Memory Study. *Journal of Gerontology: Biological Sciences*.

Journal Article, Professional Journal (Published)
Sanders, C., Behrens, S., Schwartz, S., Wengreen, H., Corcoran, C. D., Lyketos, C. G., Tschanz, J. T. (2016). Nutritional status is associated with faster cognitive decline and worse functional impairment in the progression of dementia: The Cache County Dementia Progression Study. *Journal of Alzheimer's Disease*.

Journal Article, Professional Journal (Published)
Rattinger, G. G., Fauth, E. B., Behrens, S., Sanders, C., Schwartz, S., Norton, M. C., Corcoran, C. D., Mullins, C. D., Lyketos, C. G., Tschanz, J. T. (2016). Closer caregiver and care-recipient relationships predict lower informal costs of dementia care: The Cache County Dementia Progression Study. *Alzheimer's & Dementia, 12*(8), 917-924.

Journal Article, Professional Journal (Published)
Hippen, A. A., Ebbert, M. t., Norton, M. C., Tschanz, J. T., Munger, R. G., Corcoran, C. D., Kauwe, J. S. (2016). Presenilin E318G variant and Alzheiemr's disease risk: The Cache County Study. *BMC Genomics, 17*(Suppl 3), 438.

Journal Article, Professional Journal (Published)
Rattinger, G. G., Schwartz, S., Mullins, C. D., Corcoran, C. D., Zuckerman, I. H., Sanders, C., Norton, M. C., Fauth, E. B., Leoutsakos, J. M. S., Lyketsos, C. G., Tschanz, J. T. (2015). Dementia severity and the longitudinal costs of informal care in the Cache County population. *Alzheimer's & Dementia, 11*, 946-954.

Journal Article, Academic Journal (Published)
Snyder, C. M., Fauth, E. B., Wanzek, J., Piercy, K. W., Norton, M. C., Corcoran, C. D., Rabins, P. V., Lyketsos, C. G., Tschanz, J. T. (2015). Dementia caregivers' coping strategies and their relationship to health and well-being: The Cache County Study. *Aging & Mental Health, 19*(5), 390-399.

Journal Article, Academic Journal (Published)
Wang, L., Naj, A. C., Graham, R. R., Crane, P. K., Kunkle, B. W., Chrucaga, C., Murcia, J. D., Cannon-Albright, L., Baldwin, C. T., Zetterberg, H., Blennow, K., Kukull, W. A., Faber, K. M., Schupf, N., Norton, M. C., Tschanz, J. T., Munger, R. G., Corcoran, C. D., et al., Yu, L. (2015). Rarity of the Alzheimer Disease-Protective APP A673T Variant in the United States. *JAMA Neurology, 72*(2), 209-216. http://www.ncbi.nlm.nih.gov/pubmed/25531812

Journal Article, Academic Journal (Published)
Lythgoe, C., Perkes, A., Peterson, M., Schmutz, C., Leary, M., Ebbert, E. T. W., Ridge, P. G., M., J., Munger, R. G., Corcoran, C. D., Kauwe, J. S. K. (2015). Population-based analysis of cholesteryl ester transfer protein identifies association between I405V and cognitive decline: the Cache County Study. *Neurobiology of Aging, 36*(547), e1-3.

Journal Article, Academic Journal (Published)
Rattinger, G. B., Schwartz, S., Mullins, C. D., Corcoran, C. D., Zuckerman, I. H., Sanders, C., Norton, M. C., Fauth, E. B., Leoutsakos, J. M. S., Lyketsos, C. G., Tschanz, J. T. (2015). Dementia severity and the longitudinal costs of informal care in the Cache County population. *Alzheimer's & Dementia, 11*(8), 946-954.

Journal Article, Professional Journal (Published)
Greene, D., Tschanz, J. T., Smith, K. R., Østbye, T., Corcoran, C. D., Welsh-Bohmer, K. A., Norton, M. C. (2014). Impact of offspring death on cognitive health in late life: The Cache County Study. *American Journal of Geriatric Psychiatry, 22*(11), 1307-15.

Journal Article, Professional Journal (Published)
Chuang, Y.-f., Breitner, J. C., Chiu, Y. L., Khachaturian, A., Hayden, K., Corcoran, C. D., Tschanz, J. T., Norton, M. C., Munger, R. G., Welsh-Bohmer, K. A., Zandi, P., For the Cache County Investigators (2014). Use of diuretics is associated with reduced risk of Alzheimer's disease: The Cache County Study. *Neurobiology of Aging, 35*(11), 2429-35.

Journal Article, Professional Journal (Published)
Gilbert, M., Snyder, C., Corcoran, C. D., Norton, M. C., Lyketsos, C. G., Tschanz, J. T. (2014). The association of traumatic brain injury with rate of progression of cognitive and functional impairment in a population-based cohort of Alzheimer's disease: The Cache County Dementia Progression Study. *International Psychogeriatrics, 26*(10), 1593-1601.

Journal Article, Professional Journal (Published)
Snyder, C. M., Fauth, E., Wanzek, J., Piercy, K. W., Norton, M. C., Corcoran, C. D., Rabins, P. V., Lyketsos, C. G., Tschanz, J. T. (2014). Dementia caregivers' coping strategies and their relationship to health and well-being: The Cache County Study. *Aging Mental Health, 5*, 1-10.

Journal Article, Public or Trade Journal (Published)
Ridge, P. G., Maxwell, T. J., Foutz, S. J., Bailey, M. H., Corcoran, C. D., Tschanz, J. T., Norton, M. C., Munger, R. G., O'Brien, E., Kerber, R. A., Cawthon, R. M., Kauwe, J. S. (2014). Mitochondrial genomic variation associated with higher mitochondrial copy number: The Cache County Study on Memory Health and Aging. *BMC Bioinformatics, 15*(7), S6.

Journal Article, Public or Trade Journal (Published)
Sharp, A. R., Ridge, P. G., Bailey, M. H., Boehme, K. L., Norton, M. C., Tschanz, J. T., Munger, R. G., Corcoran, C. D., Kauwe, J. S., Alzheimer's Disease Neuroimaging Initiative (2014).

Population substructure in Cache County, Utah: The Cache County study. *BMC Bioinformatics, 15*(7), S8.

Journal Article, Professional Journal (Published)
Ebbert, M. T., Ridge, P. G., Wilson, A. R., Sharp, A. R., Bailey, M., Norton, M. C., Tschanz, J. T., Munger, R. G., Corcoran, C. D., Kauwe, J. S. (2014). Population-based analysis of Alzheimer's disease risk alleles implicates genetic interactions. *Biological Psychiatry, 75*(9), 732-737.

Journal Article, Professional Journal (Published)
Peterson, D., Munger, C., Crowley, J., Corcoran, C. D., Cruchaga, C., Goate, A. M., Norton, M. C., Green, R. C., Munger, R. G., Breitner, J. C., Welsh-Bohmer, K. A., Lyketsos, C. G., Tschanz, J. T., Kauwe, J. S. (2014). Variants in PPP3R1 and MAPT are associated with more rapid functional decline in Alzheimer's disease: The Cache County Dementia Progression Study. *Alzheimer's and Dementia, 10*(3), 366-371.

Journal Article, Professional Journal (Published)
Steinberg, M., Hess, K., Corcoran, C. D., Mielke, M. M., Norton, M. C., Breitner, J., Green, R., Leoutsakos, J.-M., Welsh-Bohmer, K., Lyketsos, C., Tschanz, J. T. (2014). Vascular risk factors and neuropsychiatric symptoms in Alzheiemr's disease: The Cache County Study. *International Journal of Geriatric Psychiatry, 29*(2), 153-159.

Journal Article, Professional Journal (Published)
Cruchaga, C., Karch, C. M., Jin, S. C., Benitez, B. A., Cai, Y., Guerreiro, R., Harari, O., Norton, J., Budde, J., Bertelsen, S., Jeng, A. T., Cooper, B., Skorupa, T., Carrell, D., Levitch, D., Hsu, S., Choi, J., Ryten, M., UK Brain Expression Consortium, Hardy, J., Ryten, M., Trabzuni, D., Weale, M. E., Ramasamy, A., Smith, C., Sassi, C., Bras, J., Gibbs, J. R., Hernandez, D. G., Lupton, M. K., Powell, J., Forabosco, P., Ridge, P. G., Corcoran, C. D., Tschanz, J. T., Norton, M. C., Munger, R. G., Schmutz, C., Leary, M., Demirci, F. Y., Bamne, M. N., Lopez, O. L., Ganguli, M., Medway, C., Turton, J., Lord, J., Braae, A., Barber, I., Brown, K., Alzheimer's Research UK Consortium, Passmore, P., Craig, D., Johnston, J., McGuinness, B., Todd, S., Heun, T., Kölsch, H., Kehoe, P. G., Hooper, N. M., Vardy, E. R., Mann, D. M., Pickering-Brown, S., Brown, K., Kalsheker, K., Lowe, J., Morgan, K., David Smith, A., Wilcock, G., Warden, D., Holmes, C., Pastor, P., Lorenzo-Betancor, O., Brkanac, Z., Scott, E., Topol, E., Morgan, K., Rogaeva, E., Singleton, A. B., Hardy, J., Kamboh, M. I., St. George-Hyslop, P., Cairns, N., Morris, J. C., Kauwe, J. S., Goate, A. M. (2014). Rare coding variants in the phospholipase D3 gene confer risk for Alzheimer's disease. *Nature, 505*(7484), 550-554.

Journal Article, Professional Journal (Published)
Gonzalez Murcia, J. D., Schmutz, C., Munger, C., Perkes, A., Gustin, A., Peterson, M., Ebbert, M. T. W., Norton, M. C., Tschanz, J. T., Munger, R. G., Corcoran, C. D., Kauwe, J. S. K. (2013). Assessment of TREM2 rs75932628 association with Alzheimer's disease in a population-based sample: The Cache County Study. *Neurobiology of Aging, 34*(12), 2889:e11-e13.

Journal Article, Professional Journal (Published)
Wengreen, H., Munger, R. G., Nelson, C., Corcoran, C. D., Tschanz, J. T., Norton, M. C., Welsh-Bohmer, K. A. (2013). Prospective Study of DASH- and Mediterranean-style Dietary Patterns and Age-related Cognitive Change. *98*(5), 1263-71.

Journal Article, Academic Journal (Published)
Wengreen, H., Munger, R. G., Cutler, A., Quach, A., Bowles, A., Corcoran, C. D., Tschanz, J. T., Norton, M. C., Welsh-Bohmer, K. (2013). Prospective study of dietary approaches to stop hypertension- and mediterranean-style dietary patterns and age-related cognitive change:

The Cache County Study on Memory, Health and Aging. *American Journal of Clinical Nutrition, 98*(5), 1263-1271.

Journal Article, Professional Journal (Published)
Piercy, K. W., Fauth, E. B., Norton, M. C., Pfister, R., Corcoran, C. D., Rabins, P. V., Lyketsos, C., Tschanz, J. T. (2013). Predictors of dementia caregiver depressive symptoms in a population: The Cache County Dementia Progression Study. *Journal of Gerontology: Psychological Sciences, 68*(6), 921-926.

Journal Article, Professional Journal (Published)
Norton, M. C., Clark, C., Fauth, E. B., Piercy, K. W., Pfister, R., Green, R. C., Corcoran, C. D., Rabins, P. V., Lyketsos, C. G., Tschanz, J. T. (2013). Caregiver personality predicts rate of cognitive decline in a community sample of persons with Alzheimer's Disease. The Dementia Progression Study. *International Psychogeriatrics, 25*(10), 1629-1637.

Journal Article, Academic Journal (Published)
Norton, M. C., Clark, C., Fauth, E. B., Piercy, K. W., Pfister, R., Green, R. C., Corcoran, C. D., Rabins, P. V., Lyketsos, C. G., Tschanz, J. T. (2013). Caregiver personality predicts rate of cognitive decline in a community sample of persons with Alzheimer's disease: The Cache County Dementia Progression Study. *International Psychogeriatrics, 25*, 1629-1637.

Journal Article, Professional Journal (Published)
Tschanz, J., Pfister, R., Steffens, D., Corcoran, C. D., Smith, K., Østbye, T., Schwartz, S., Welsh-Bohmer, K., Norton, M. C. (2013). Stressful events in late-life: Effects on cognitive decline: The Cache County Study. *International Journal of Geriatric Psychiatry, 28*, 821-830.

Journal Article, Academic Journal (Published)
Greene, D., Tschanz, J. T., Smith, K. R., Ostbye, T., Corcoran, C. D., Welsh-Bohmer, K. A., Norton, M. C. (2013). Impact of Offspring Death on Cognitive Health in Late Life: The Cache County Study. *The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry*.

Journal Article, Academic Journal (Published)
Tschanz, J. T., Pfister, R., Wanzek, J., Corcoran, C. D., Smith, K., Tschanz, B. T., Steffens, D. C., Østbye, T., Welsh-Bohmer, K. A., Norton, M. C. (2013). Stressful life events and cognitive decline in late life: moderation by education and age. The Cache County Study. *International journal of geriatric psychiatry, 28*(8), 821-30.

Journal Article, Academic Journal (Published)
Fauth, E. B., Schwartz, S., Tschanz, J. T., Ostbye, T., Corcoran, C. D., Norton, M. C. (2013). Baseline disability in activities of daily living predicts dementia risk even after controlling for global cognitive ability and depressive symptoms. *International Journal of Geriatric Psychiatry, 28*(6), 597-606.

Journal Article, Professional Journal (Published)
Peterson, D., Crowley, J., Munger, C., Corcoran, C. D., Cruchaga, C., Goate, A., Norton, M. C., Green, R., Munger, R. G., Breitner, J. C., Welsh-Bohmer, K., Lyketsos, C., Kauwe, J. S. (2013). Variants in PPP3R1 and MAPT are associated with more rapid functional decline in Alzheimer's disease: The Cache County Dementia Progression Study. *Alzheimer's and Dementia*.

Journal Article, Academic Journal (Published)
Rabins, P. V., Schwartz, S., Black, B. S., Corcoran, C. D., Fauth, E. B., Mielke, M., Christensen, J., Lyketsos, C., Tschanz, J. T. (2013). Predictors of progression to severe Alzheimer Disease in an incidence sample. *Alzheimer's and Dementia, 9*(2), 204-207.

Journal Article, Professional Journal (Published)
Piercy, K. W., Corcoran, C. D., Fauth, E. B., Norton, M. C., Rabins, P. V., Tschanz, B. T., Deberard, M. S., Snyder, C., Smith, C., Lee, L., Lyketsos, C. G. (2013). Caregiver coping strategies predict rate of cognitive and functional decline in dementia: The Cache County Dementia Progression Study. *American Journal of Geriatric Psychiatry, 21*(1), 57-66.

Journal Article, Academic Journal (Published)
Tschanz, J. T., Piercy, K. W., Corcoran, C. D., Fauth, E. B., Norton, M. C., Rabins, P. V., Tschanz, B. T., Deberard, M. S., Snyder, C., Smith, C., Lee, L., Lyketsos, C. G. (2013). Caregiver coping strategies predict cognitive and functional decline in dementia: The Cache County Dementia Progression Study. *The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry, 21*(1), 57-66.

Journal Article, Professional Journal (Published)
Ridge, P., Maxwell, T. J., Corcoran, C. D., Norton, M. C., Tschanz, J. T., O'Brien, E., Kerber, R. A., Cawthon, R. M., Munger, R. G., Kauwe, J. S. (2012). Mitochondrial genomic analysis of late onset Alzheimer's disease reveals protective haplogroups H6A1A/H6A1B. *PLoS, 7*(9), e45134.

Journal Article, Professional Journal (Published)
Leoutsakos, J. M., Han, D., Mielke, M., Forrester, S. N., Tschanz, J. T., Corcoran, C. D., Green, R., Norton, M. C., Welsh-Bohmer, K., Lyketsos, C. (2012). Effects of General Medical Health on Alzheimer Progression: the Cache County Dementia Progression Study. *International Journal of Psychogeriatrics, 24*(10), 1561-70.

Journal Article, Academic Journal (Published)
Leoutsakos, J. M., Han, D., Mielke, M. M., Forrester, S. N., Tschanz, J. T., Corcoran, C. D., Green, R. C., Norton, M. C., Welsh-Bohmer, K. A., Lyketsos, C. G. (2012). Effects of general medical health on Alzheimer's progression: the Cache County Dementia Progression Study. *International psychogeriatrics / IPA, 24*(10), 1561-70.

Journal Article, Academic Journal (Published)
Shao, H., Breitner, J. C., Whitmer, R., Wang, J., Hayden, K., Wengreen, H., Corcoran, C. D., Tschanz, J. T., Norton, M. C., Munger, R. G., Welsh-Bohmer, K., Zandi, P. (2012). Hormone Therapy and AD Dementia: New Findings from the Cache County Study. *Neurology, 79*(18), 1846-1852.

Journal Article, Professional Journal (Published)
Ridge, P. G., Maxwell, T. J., Corcoran, C. D., Norton, M. C., Tschanz, J. T., Kerber, R. A., Cawthon, R. M., Munger, R. G., Kauwe, J. S. (2012). Mitochondrial genomic analysis of late onset Alzheimer's disease reveals protective haplogroups H6a1a/ H6a1b. The Cache County Study on Memory in Aging. *PLoS ONE*.

Journal Article, Academic Journal (Published)
Ridge, P., Maxwell, T., Corcoran, C. D., Norton, M. C., Tschanz, J. T., Obrien, E., Kerber, R., Cawthon, R., Munger, R. G., Kauwe, J. (2012). Mitochondrial genomic analysis of late onset Alzheimer's disease reveals protective haplogroups H6A1A/H6A1B: the Cache County Study on Memory in Aging. *PLoS One, 7*(9), e45134.

Journal Article, Professional Journal (Published)
Mielke, M. M., Leoutsakos, J. M., Corcoran, C. D., Green, R. C., Norton, M. C., Welsh-Bohmer, K. A., Tschanz, J. T., Lyketsos, C. G. (2012). Effects of FDA approved medications for Alzheimer’s disease on dementia progression. *Alzheimer's and Dementia, 8*(3), 180-187.

Journal Article, Professional Journal (Published)

Rosenberg, P. B., Mielke, M. M., Leoutsakos, J. S., Lyketsos, C. G., Rabins, P. V., Zandi, P. P., Breitner, J. C., Norton, M. C., Welsh-Bohmer, K. A., Zuckerman, I. H., Rattinger, G. B., Green, R. C., Corcoran, C. D., Tschanz, J. T. (2012). The association of psychotropic medication use with the cognitive, functional and neuropsychiatric trajectory of Alzheimer's disease.

Journal Article, Academic Journal (Published)
Fauth, E. B., Hess, K., Piercy, K. W., Norton, M. C., Corcoran, C. D., Rabins, P., Lyketsos, C., Tschanz, J. T. (2012). Caregivers' relationship closeness with the person with dementia predicts both positive and negative outcomes for caregivers' physical health and psychological well-being. *Aging & mental health, 16*(6), 699-711.

Journal Article, Professional Journal (Published)
Treiber, K., Carlson, M., Corcoran, C. D., Norton, M. C., Piercy, K. W., Deberard, M. S., Stein, D. M., Foley, B. E., Welsh-Bohmer, K. A., Breitner, J. C. S., Lyketsos, C. G. (2011). Cognitive Stimulation and Cognitive and Functional Decline in Alzheimer's Disease: The Cache County Dementia Progression Study. *The Journals of Gerontology, Series B: Psychological Sciences and Social Sciences, 66*(4), 416-425.

Journal Article, Professional Journal (Published)
Norton, M. C., Smith, K. R., Ostbye, T., Tschanz, J. T., Corcoran, C. D., Schwartz, S., Skoog, I., Steffens, D. C., Welsh-Bohmer, K. A., Breitner, J. C. S. (2011). Early parental death and remarriage of widowed parents as risk factors for Alzheimer's disease. *American Journal of Geriatric Psychiatry, 19*, 814-824.

Journal Article, Professional Journal (Published)
Tschanz, J. T., Corcoran, C. D., Schwartz, S., Treiber, K., Green, R. C., Norton, M. C., Mielke, M. M., Piercy, K. W., Steinberg, M., Rabins, P. V., Leoutsakos, J., Welsh-Bohmer, K. A., Breitner, J. C. S., Lyketsos, C. G. (2011). Progression of cognitive, functional and neuropsychiatric symptom domains in a population cohort with Alzheimer's Dementia: The Cache County 5 dementia progression study. *American Journal of Geriatric Psychiatry, 19*, 532-542.

Journal Article, Professional Journal (Published)
Wengreen, H., Neilson, C., Munger, R. G., Corcoran, C. D. (2011). Prospective study of ready-to-eat breakfast cereal consumption and cognitive decline among elderly men and women. *Journal of Nutrition, Health and Aging, 15*(3), 202-207.

Journal Article, Academic Journal (Published)
Norton, M. C., Smith, K. R., Østbye, T., Tschanz, J. T., Schwartz, S., Corcoran, C. D., Breitner, J. C., Steffens, D. C., Skoog, I., Rabins, P. V., Welsh-Bohmer, K. A. (2011). Early parental death and remarriage of widowed parents as risk factors for Alzheimer disease: the Cache County study. *The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry, 19*(9), 814-24.

Journal Article, Academic Journal (Published)
Tschanz, J. T., Corcoran, C. D., Schwartz, S., Treiber, K., Green, R. C., Norton, M. C., Mielke, M. M., Piercy, K. W., Steinberg, M., Rabins, P. V., Leoutsakos, J. M., Welsh-Bohmer, K. A., Breitner, J. C., Lyketsos, C. G. (2011). Progression of cognitive, functional, and neuropsychiatric symptom domains in a population cohort with Alzheimer dementia: the Cache County Dementia Progression study. *The American journal of geriatric psychiatry : official journal of the American Association for Geriatric Psychiatry, 19*(6), 532-42.

Journal Article, Academic Journal (Published)
Mielke, M. M., Leoutsakos, J. M., Tschanz, J. T., Green, R. C., Tripodis, Y., Corcoran, C. D., Norton, M. C., Lyketsos, C. G. (2011). Interaction between vascular factors and the APOE ε4

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Milman, L., Faroqi-Shah, Y., Corcoran, C. D., Clinical Aphasiology Conference, "Normative data for the WAB-R: A comparison of monolingual English speakers, Asian Indian-English bilinguals, and Spanish-English bilinguals.," St. Simons Island, GA. (May 29, 2014).

Corcoran, C. D. (Invited Lecture), International Webinar, "Exact Nonparamatric Inference for Correlated Categorical Data," Cytel Software Corporation. (April 7, 2014).

Corcoran, C. D., Annual Meeting of the Utah Chapter of the American Statistical Association, "The Perils of P-Values: A Case Study in Statistical Genetics," American Statistical Association, Utah Chapter, Salt Lake City, UT. (March 25, 2014).

Corcoran, C. D. (Presenter & Author), Boston University Department of Biostatistics Seminar, "Permutation-Based Tests and Rare Variants in Genetic Association Studies," Department of Biostatics, Boston University, Boston University, Boston, MA. (March 20, 2014).

Corcoran, C. D. (Presenter & Author), Brigham Young University Department of Statistics Seminar, "Doctoral Research Programs in Statistics at Utah State University," Brigham Young University, Brigham Young University, Provo, UT. (February 2014).

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Corcoran, C. D. (Presenter & Author), Food and Drug Administration Workshop, "StatXact Training Course," Food and Drug Administration, Chevy Chase, MD. (September 19, 2013).

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Klein, E., Tschanz, J., Corcoran, C. D. (Author Only), Norton, M., Welsh-Bohmer, K., Breitner, J., Zandi, P., Lyketsos, C., Society of Epidemiological Research, "Estimating Survival Duration from Memory Symptom Onset: A Comparison of Methods. The Cache County Study," Salt Lake City, UT. (June 2004).

Norton, M., Skoog, I., Toone, L., Tschanz, J., Corcoran, C. D. (Author Only), Zandi, P., Hart, A., Breitner, J., Welsh-Bohmer, K., Steffens, D., Society of Epidemiological Research, "Improving Assessment of Incidence of First-Onset Geriatric Depression in Population-Based Studies," Salt Lake City, UT. (June 2004).

Lensegrav-Benson, T., Lisota, R., Tschanz, J., Masters, K., Norton, M., Carlson, M., Corcoran, C. D. (Author Only), Lyketsos, C., Heath, E., Leslie, C., Munger, R. G., Ostybe, T., Welsh-Bohmer, K., Annual Meeting of the Western Psychological Association, "Physical Activity is Associated with Better Cognitive Performance," Phoenix, AZ. (April 2004).

Corcoran, C. D. (Invited Lecture), Schneiter, K., Laird, N., Invited Seminar, University of Colorado Health Sciences Center, "Implementing an exact family based association test in the presence of two alleles," Denver, CO. (April 2004).

Lisota, R., Steffens, D., Toone, L., Tschanz, J. T., Norton, M., Corcoran, C. D. (Author Only), Welsh-Bohmer, K. A., Breitner, J. S., Annual AAGP Meeting, "Vascular Risk Factors Predict Chronicity of Depression in the Elderly," Baltimore, MD. (February 2004).

Schneiter, K., Corcoran, C. D., The Western North American Region of The International Biometric Soceity, "An Exact Approach to Family Based Association Tests Using a Network Algorithm.," The International Biometric Society, Golden, CO. (2003).

Corcoran, C. D. (Invited Lecture), Invited Seminar, Brigham Young University, "A network algorithm for exact family based association tests," Provo, UT. (September 2003).

Corcoran, C. D. (Author Only), Schneiter, K., Spring Meeting of the Western North America Region of the International Biometrics Society, "A Network Algorithm for Exact-Based Association Tests," Denver, CO. (June 2003).

Corcoran, C. D. (Presenter & Author), Senchaudhuri, P., Spring Meeting, Western North America Region, "Exact Dose-Response Estimation for Clustered Binary Data," International Biometrics Society, Denver, CO. (June 2003).

Huber, C., Steinberg, M., Tschanz, J., Corcoran, C. D. (Author Only), Posters on the Hill, "A Longitudinal Model for Behavioral Disturbances among the Elderly with Dementia: The Cache County Memory Study," Washington D.C. (April 2003).

Norton, M. C., Steffens, D. C., Skoog, I., Corcoran, C. D. (Author Only), Welsh-Bohmer, K. A., Breitner, J. S., American Association for Geriatric Psychiatry, "Prior Minor Depression Is More Predictive of Future Episodes of Depression in the Elderly than Gender, Age, or APOE status. The Cache County Study," Honolulu, HI. (March 2003).

Tschanz, J., Welsh-Bohmer, K., Norton, M., Corcoran, C. D. (Author Only), Breitner, J., International Neuropsychological Society 31st Annual Meeting, "Progression to Dementia in Diverse Types of Mild Cognitive Impairments of Aging," Honolulu, HI. (February 2003).

Corcoran, C. D. (Presenter & Author), Senchaudhuri, P., Mehta, C., Joint Statistical Meeting, "Order-restricted inference for several binomials," NYC, NY. (August 2002).

Norton, M., Tschanz, J., Corcoran, C. D., Mumford, S., Welsh-Bohmer, K., Breitner, J., International Conference on Alzheimer's Disease and Related Disorders, "Apolipoprotein E4 interacts with mild cognitive deficit to shorten time to dementia onset," Stockholm, Sweden. (July 2002).

Tschanz, J., Norton, M., Corcoran, C. D., LaCaille, R., Welsh-Bohmer, K., Breitner, J., International Conference on Alzheimer's Disease and Related Disorders, "Cognitive screening and self-perception of memory problems predict mild cognitive impairment and dementia," Stockholm, Sweden. (July 2002).

Corcoran, C. D. (Author Only), International Conference on Alzheimer's Disease and Related Disorders, "Differential impact of genetic and demographic variables on clinical course of dementia and Alzheimer's disease," Stockholm, Sweden. (July 2002).

Hayden, K., Khachaturian, A., Breitner, J., Tschanz, J., Corcoran, C. D. (Author Only), Norton, M., International Conference on Alzheimer's Disease and Related Disorders, "Evaluation of performance of a two-stage screen for incident dementia," Stockholm, Sweden. (July 2002).

Wengreen, H. J., Munger, R. G., West, N., Cutler, D., Corcoran, C. D., Zhang, J., Sassano, N. E., International Conference on Nutrition and Aging, "Protein Intake and Risk of Osteoporotic Hip Fracture in Elderly Utah Residents," Paris, France. (July 2001).

Corcoran, C. D. (Presenter & Author), WHO Meeting for the Prevention of Craniofacial Anomalies, "Deisgn consideration for dose-response studies.," Park City, UT. (May 2001).

West, N., Tschanz, J., Welsh-Bohmer, K., Corcoran, C. D., Wyse, B., Weight, C., Breitner, J., Annual Meeting of the International Neuropsychological Society, "Genetic and nongenetic risk factors for cognitive decline in the normal elderly," Chicago, IL. (February 2001).

Contracts, Grants and Sponsored Research

Contract

Kauwe (Brigham Young University), Keone (Principal), Munger, Ronald G. (Supporting), Corcoran, Christopher D (Supporting), "Alzheimer’s disease candidate gene genotyping: The Cache County Study," Sponsored by USTAR, State, $42,000.00. (February 1, 2011 - May 30, 2011).

Grant

Tschanz, Joann T (Principal), Corcoran, Christopher D (Supporting), Munger, Ronald G. (Supporting), Lefevre, Michael (Supporting), "Epidemiology of Alzheimer’s Disease resilience and risk pedigrees," Sponsored by NIH, Federal, $1,067,869.00. (September 1, 2016 - August 31, 2021).

Corcoran, Christopher D (Supporting), Stevens, John R. (Supporting), "miRNA and colorectal cancer: Associations with tumor phenotype and survival," Sponsored by National Institutes of Health, Federal, $1,250,000.00. (July 2012 - June 2017).

Corcoran, Christopher D (Supporting), "Pleiotropic and interaction effects on Alzheimer’s disease risk and progression," Sponsored by National Institutes of Health, Federal, $1,250,000.00. (July 2012 - June 2017).

Corcoran, Christopher D (Supporting), "Prenatal and Neonatal Biologic Markers for Autism," Federal, $576,008.00. (July 2010 - June 2015).

Intellectual Contributions in Submission

Refereed Journal Articles

Milman, L., Faroqi-Shah, Y., Corcoran, C. D., Damele, D. Interpreting MMSE scores in highly proficient bilingual Asian Indian-English and Spanish-English speakers: Demographic adjustments, item analyses, and supplemental measures.

SERVICE

General Service

Department

Chairperson, Graduate Committee, August 2012 - Present.

Undergraduate Statistics Advisor, 1999 - Present.

Committee Member, Undergraduate Curriculum Committee, 2003 - 2005.

Committee Member, Graduate Committee, 2002 - 2003.

Committee Member, Undergraduate Committee, 2001 - 2002.

Other

Committee Chair, Computing Committee, 2005 - 2009.

Professional/Public

Officer, Secretary, American Statistical Association, Utah Chapter. 2002 - 2006.

Member, Sunrise Elementary School Community Council. 2002 - 2006.

Committee Member, Cache School District Building Task Force. 2003 - 2004.

Program Organizer, Bioinformatics Working Group. 2002 - 2003.

Contribuing Author of User Manuals. 1999 - 2003.

Program Organizer, Statistics Brown Bag Seminar Series. 2000 - 2001.

Utah State University

Committee Member, Promotion and Tenure Central Committee, September 2014 - Present.

Committee Member, Utah State University Faculty Senate, 2007 - Present.

Committee Chair, Utah State University Faculty Senate Committee on Committees, 2008 - 2009.