EXHIBIT 62

UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

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

REBUTTAL EXPERT WITNESS REPORT

OF

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Introduction

This rebuttal report will address: 1) the draft manuscript[s] of the unpublished Agricultural Health Study (AHS) dated February 6, 2013 (Exhibit 19A to the deposition of Dr. Aaron Earl Blair taken March 20, 2017) and March 15, 2013 (Exhibit 19B to the deposition of Dr. Aaron Earl Blair taken March 20, 2017); 2) epidemiology issues raised by Defendant's experts Dr. Lorelei A. Mucci, Dr. Jennifer S. Rider and Dr. William Fleming; 3) the North American Pooled Project ("NAPP") study.

The Draft Manuscripts of the Unpublished AHS

The draft manuscripts of the unpublished AHS provide analyses of 333 NHL cases within the AHS cohort (DeRoos 2005) that followed individuals from through December 2008 for cancer incidence. The draft manuscripts also purport to give new exposure data collected in the second phase interview of the AHS between 1998 and 2004, together with the original data collected at enrollment of the cohort between 1993 and 1997.

The main problem with these draft AHS manuscripts are the authors' attempts to impute and 'guestimate' exposure for glyphosate or glyphosate-based formulations ("GBFs", including Roundup®). The problems arise because there has been a dramatic increase in the use of and exposure to glyphosate or GBFs in the mid-1990s (Aspelin and Grube 2016; Grube et al 2016; Coupe and Capel 2015; Thelin and Stone 2016; Service. USDoANAS 2016; Benbrook 2015). The authors failed to address this major issue in their draft manuscripts of unpublished AHS data. While under some, limited circumstances it is an acceptable epidemiological approach to impute or 'guestimate' certain unavailable data, one must be extremely careful when imputing/guestimating a critical piece of data, such as exposure or outcome of interest. In the case of the draft AHS manuscripts, the guestimation was conducted to answer the question as to whether or not the cases and controls were even exposed to the products being studied. In the instance of the draft AHS manuscripts, the imputation/guestimation failed, in part, because the draft manuscripts could not accurately account for the major change in the use of GBFs, including Roundup®. The validity of the results of such an imputation/guestimation become extremely questionable because when applied, the study authors need to assume glyphosate/GBF use was based on historical use, and do

not apply the increased use for any person who did not report their pesticide use, i.e. the non-responders. Consequently, such imputation/guestimation is unable to fully contemplate major changes in the professional agricultural environment as seen with the use of glyphosate/GBFs. Further, this change was not captured in the original reporting by AHS participants and generates a unique problem for glyphosate/GBFs compared with all other pesticide exposure assessments performed in this prospective study. After registration in the U.S. in 1974, glyphosate/GBFs were mainly used to kill weeds before planting of crops or spraying for weed control in pastures and non-crop areas, with 6 - 8 million pounds applied by U.S. farmers and ranchers in 1987 [Grube 2016]. The dramatic change in glyphosate/GBF use began in 1996, the first year genetically engineered, glyphosate -tolerant crops were planted commercially in the U.S. Specifically, in 1996, Monsanto first introduced genetically engineered, glyphosate resistant soybeans (Roundup® Ready) to the commercial market, followed by cotton and canola in 1997, corn in 1998, and alfalfa and sugar beets in 2005. Prior to the introduction of genetically modified seeds, glyphosate/GBFs accounted for only 3.8% of the total volume of herbicide active ingredients applied in agriculture, while this changed to 180–185 million pounds by 2007 [EPA reports; Coupe 2015]. This substantial increase established glyphosate/GBFs as 53.5% of total agricultural herbicide use in 2009 according to USGS [Thelin and Stone 2016]; annual farm-sector glyphosate/GBF usage further increased to approximately 240 million pounds in 2014 [based on average annual crop use reported by the NASS; Service. USDoANAS 2016, Benbrook 2015. The original AHS enrollment (Dec 1993-Dec 1997) preceded this tremendous increase in agricultural use of glyphosate/GBFs. Thus, this increase in use was never captured for members of the AHS cohort who did not respond to follow-up interviews in phase 2 (1999-2003) or phase 3 (2005-2010) of the AHS, as set forth below.

Importantly, the second phase of the AHS was plagued by low response: i.e. it generated no more than a 64% response rate among AHS cohort members who were private applicators contacted in 1998-2004 (or a 36% non-response). This is an extremely low response rate when usage increased this much and this fast (furthermore, concerning future glyphosate/GBF analyses in AHS, only 46%, less than half, of all private applicators responded to the third phase 2005-2010 interviews). Thus, one-third

of all cohort subjects never reported their actual exposures or changes in exposures after enrolment interviews were conducted, even though use of glyphosate/GBFs started to change dramatically.

The AHS researchers knew that such a large non-response rate would raise questions about the validity of certain results of their study, so they were forced to come up with a method to address this problem. Otherwise, these studies would be questioned by peer reviewers and unlikely to be published. The AHS researchers attempted to address the loss of active participants with a method called 'imputation' to avoid having large amounts of missing exposure data –for those who did not respond – or generating selection bias (cohort studies may be affected by selection bias due to 'differential' loss to follow-up among the exposed or unexposed cases and controls) (Heltsche, et al. 2012). The method the authors used was a "data driven imputations of exposures"; or, in other words, a 'guestimation' of what exposures would have been in those who did not respond and report. This procedure assumes that it is sufficient to use the data in hand to predict/guestimate all future exposure in AHS participants who did not respond; i.e. that the past and current exposures and characteristics of the participants who responded to multiple interviews over time would accurately predict the use of those who did not respond. For glyphosate/GBFs with a use pattern change as dramatic as described above, it is a flawed approach to predict who would or would not start using Roundup® Ready crops after baseline, and likewise to predict the use of glyphosate/GBFs. This is because this imputation method assumes that those who did not respond had similar pesticide use and exposure pattern as those who did respond whether or not they developed NHL (this is called the 'missing at random assumption'). This assumption - if wrong - may cause enough exposure misclassification (undifferential with regard to disease status) for a large proportion of AHS participants to bias effect estimates towards the null of not finding any associations. An alternative to imputation for non-responders is to restrict the analyses to include only data from those cohort members who actually responded. However, this can cause strong selection bias if the response to the follow-up questionnaires depends on participant characteristics and health status. This is not an issue for assessing effects for exposures measured at enrollment on cancer when outcomes are being obtained through linkage with registries (i.e. cases are almost always found), but it is an issue for assessing effects of time varying

exposures especially when there are considerable changes in exposure that may affect future cancer occurrence. It has been stated in published AHS studies that response to follow-up interviews depended on education and age and on some farming practices including personal pesticide use and a number of health conditions (see for example Rinsky, et al. 2017). Methods have been developed to address selection bias and the most recent paper by Rinsky et al. 2017 for the AHS group addresses the need for bias correction in the AHS and shows how to implement such methods to assess and correct this bias in a quantitative manner. This paper concludes that as long as exposure and disease are not strongly associated with response during follow-up (i.e. to respond to interviews) resulting bias would be small. However, for bias to be assessed and bias correction to work, one needs accurate data for exposure as well as variables identified as predictors of response and disease status. Given that glyphosate/GBF exposure patterns changed dramatically after enrollment and that updated exposure information was only available for responders, this method does not work for glyphosate/GBF exposure in the AHS (in fact the authors state that "farming activities after enrollment may be strongly associated with response to later interviews"). Possibly severe selection bias in estimating these time varying glyphosate/GBF exposures cannot be avoided or corrected in the described way and will continue to affect future glyphosate/GBF exposure and NHL association studies in the AHS.

Another important issue relates to the outcome assessment, i.e. the diagnosis of NHL: how to address the influence of the recent ICD re-classification of NHL subtypes on the AHS results. The issue of disease classifications becomes apparent when we examine the Alavanja 2014 paper supplement that shows major changes by redistributing NHL according to subtypes and newly adding more than 100 cases of NHL cancers from multiple myeloma and chronic lymphocytic leukemia. Most importantly, these changes in outcome classification also affect the pesticide exposure distributions among NHL cases. For example, in the draft manuscript of the unpublished 2013 AHS study, 173 NHL cases were considered unexposed to DDT (in dose-response analyses) while only 152 NHL cases in the published 2014 manuscript are considered unexposed to DDT. But, DDT exposures were assessed with the same method and same data in both manuscripts; the change between the two papers was the disease classification used. Importantly, this resulted in increased risk estimates for

DDT and a statistically significant trend by lifetime years of exposure not seen in the draft manuscript of the unpublished 2013 AHS (according to the supplemental table of the published manuscript, a significant trend would not be seen when using the old ICD classification even though additional years of follow-up added cases (old ICD classification p-trend=0.32; new ICD classification p trend=0.02). This proves that the results presented in the draft manuscript of the unpublished AHS are not a good substitute for glyphosate/GBF exposures related effect estimates with additional follow-up. Furthermore, it contradicts the statement made by Dr. Mucci in her expert report that the draft manuscript of the unpublished AHS results from 2013 are good enough to be included in a meta-analysis; i.e. that: "One minor weakness is that the updated analysis on glyphosate and other herbicides has not been published to date, although the findings on insecticides, fungicides, and fumigants were published" and "concern [about including the results form an unpublished study] is minimized since the methodology is the same as those studies that have undergone peer review." (page 35, Mucci). Thus, the results and conclusions from the draft manuscript of the unpublished 2013 AHS cannot be considered fit for inclusion into a meta-analysis nor are they of the same quality as peer-reviewed and published manuscripts that are included in meta-analysis.

Other reasons for the draft manuscripts of the unpublished 2013 AHS results for NHL overall, or NHL subtypes with glyphosate/GBF exposures may also relate to the very high and almost ubiquitous exposure to glyphosate/GBFs in this cohort. Effects for ubiquitous exposures are difficult or even impossible to estimate since, in order to see effects, we rely on exposure contrasts (i.e. we need both exposed and unexposed subjects; or low and high exposures). In other words, when everyone smokes heavily, we cannot estimate the effect of smoking on lung cancer; or, if the exposure contrast is too small, it is impossible to estimate an incremental increase in risk for the exposure, *i.e.* we need enough of a difference in exposure to see a difference in effect.

Also, the high frequency of co-exposures in those listed as unexposed to glyphosate/GBFs might be yet another problem if these co-exposure chemicals indeed cause NHL. As the 2005 DeRoos paper shows, exposures to potentially carcinogenic pesticides 2,4 D, alachlor and atrazine were very high among both glyphosate/GBF exposed and unexposed AHS participants at baseline. If these chemicals indeed cause NHL, we would expect them to increase the baseline rate of NHL in the glyphosate/GBF

unexposed such that an incremental increase due to glyphosate/GBF exposure would require a much larger sample size to be estimable. This is because we are estimating relative increases in risk of cancer. Now, assume we are interested in estimating the risk of lung cancer from smoking and find in our population among non-smokers 4 lung cancers/100,000 and in smokers 20/100,000; we can use these rates to estimate a (20/4=) 5 fold risk increase for lung cancers due to smoking in this population. Now imagine that we examine smoking in an occupational cohort of miners and that radon exposure adds 10 extra cases of lung cancer per 100,000 miners i.e. no matter whether they smoke. Thus, we would see in non-smoking miners a rate of (10+4 =) 14/100,000 lung cancers (the reference group) to which we compare the rate in smokers of (10+20=) 30/100,000 and estimate a (30/14=) 2.14-fold increase in risk for smoking and lung cancer in miners, i.e. a relative risk much smaller than we estimated in non-miners (5 fold). Statistically, I need less power to be able to estimate a larger relative risk increase than a smaller one i.e. a 5-fold compared with a 2.14 fold risk increase.

Finally, as is the case for most farmer focused studies, the AHS has to address multiple pesticide exposure scenarios and decide whether it is appropriate to adjust for 'proxies' i.e. co-exposures that are not risk factors for the outcome but related to the exposure of interest. This generates the necessity to distinguish between true confounding co-exposures (pesticides that truly cause NHL and are also associated with glyphosate exposures) and co-exposures that solely act as 'proxy measures' for glyphosate/GBFs but do not cause NHL. For the latter, one should not adjust since this would lead to over-adjustment and introduce major bias. There is no analytical or statistical fix for this problem.

Differences Between the Draft Manuscripts of the Unpublished AHS Data and the Peer-Reviewed NAPP Study

There are other problems with the draft manuscripts of the unpublished AHS data which tend to be typical of a non-peer reviewed unpublished study and clearly show why we as both academics and epidemiologists do not normally rely upon such non-peer reviewed unpublished information. As an example, if one looks at page 25 of the February 6, 2013 draft manuscripts of the unpublished AHS, the authors note in

footnote two: "Numbers do not sum to totals (333 cases, 714,770 person-years) due to missing data," with similar comments about "missing data" on page 27. The missing data references continue in the draft manuscript dated March 15, 2013 – see e.g. pages 30 and 45. Furthermore, the comments of certain "unknown" authors are equally telling as to the problems with this draft manuscript of the unpublished AHS. See e.g. page 19 of the March 15, 2013 draft manuscript: "Although this is a large prospective study, there are limitations...need to add a paragraph of exposure assessment. Discuss the information on our exposure scale in relation to the monitoring work. Discuss the likely magnitude of misclassification and its likely impact on the estimates of RR."

For the above-stated reasons, it is not appropriate from an epidemiologically perspective to rely on the data contained in the two draft manuscripts of the unpublished AHS which I have reviewed, or on its conclusions. Furthermore, as I was an external advisor for the AHS for more than a decade, I certainly would have pointed out the above-mentioned significant problems if this data had gotten closer to publication. My reliance on the NAPP report is appropriate because the data contained in the NAPP study has been presented at meetings, both in poster and published abstract form, and thus HAS been peer-reviewed, making reliance on the NAPP appropriate.

Statistical Power and Meta or Pooled Analyses

I would like to briefly comment on the issue of statistical power, since both defense experts Drs. Rider and Mucci misrepresented a major issue when discussing this point or the epidemiology studies in their reports. While the reports are correct in pointing out that statistical power of a study does not only depend on the number of cases and controls but – in addition – on exposure prevalence, they failed to acknowledge or describe a basic fact i.e. that statistical power does not increase linearly with exposure prevalence. Rather the highest power is generally achieved at a 50:50 split of exposed and unexposed – this is why most clinical treatment trials employ this type of treatment allocation. In other words, we cannot estimate effects at the extremes of the exposure distribution i.e. with everyone either exposed or unexposed we cannot study an exposure. As an example: we cannot estimate the effect of smoking on lung cancer in a population in which everyone smokes heavily – in such a population one might have to conclude that lung cancer is a genetic disorder i.e. the only difference

between cases and controls is their genetic/biologic susceptibility to smoke. Thus, the ability to estimate effects in a population with either very low or very high exposure is restricted in terms of statistical power; i.e. it requires more and more subjects to be enrolled in such studies to estimate an effect for the exposure. The latter is the case in the AHS study, rather than becoming the 'statistically most powerful study' nearly universal exposure to glyphosate/GBFs will make it impossible to estimate some of its effects.

In terms of meta-analysis and pooled analysis, Dr. Rider, in her expert report, stated that "Given the potential threats to internal validity in the case-control studies, a meta-analysis that attempts to summarize all of the published data could be misleading. In addition, the published meta-analyses of glyphosate and NHL do not include the unpublished data from the AHS or the findings from the NAPP, which plaintiffs' experts agree should be incorporated. These studies would effectively reduce the summary effect estimate in the meta-analyses and render that point estimate no longer statistically significant [this refers to the Delzell and Chang meta-analysis]." (page 4, Rider). First, the internal validity issues Dr. Rider attributes to population-based case control studies are questionable, because: a) recall bias has not been shown to affect pesticide studies, and is unlikely to affect one specific agent only in studies that assess multiple pesticides; b) similarly, the issue of confounding control as raised by both defense experts is clearly out of step with the current thinking in epidemiology. This methodology, used by both Drs. Rider and Mucci, is not the methodology that is currently accepted by epidemiologists, especially those who study and analyze complex exposures. For example, multiple exposures have to be cautiously addressed in terms of what is or isn't a risk factor for the outcome or should be considered a confounder. We have to consider prior knowledge, and just claiming that something is a confounder is not enough. Rather, the question would be how strong a confounder we would need to change the results we observe and in what direction this change would be [not all confounding changes the estimates away from the null]; and what variables would qualify as confounders (most of the adjustments for a number of moderately strong risk factors including previous cancer history - in McDuffie et al. – did not change the effect estimates for the pesticides by much [for example: for dicamba basic adjustment for age and province resulted in an OR of 1.92 (1.39–2.66) while additional adjustment for all

other risk factor for NHL including history of cancer resulted in an OR of 1.88 (1.32–2.68); for Mecoprop basic adjustment for age and province resulted in an OR of 2.23 (1.38–3.07) while additional adjustment for all risk factor for NHL including history of cancer resulted in an OR of 2.33 (1.58–3.44) – i.e. minimal changes in both directions towards and away from the null); c) selection bias is not a concern in properly conducted population-based studies. Furthermore, this issue has been addressed adequately in the Canadian studies. Even more importantly, the AHS has the potential for severe selection and exposure misclassification biases due to the necessity of active follow-up for exposure assessment and time varying exposures, an issue which has not been addressed in the reports of Dr. Rider or Dr. Mucci. Dr. Rider contradicts herself and Dr. Mucci when stating that the data summary (meta-analysis) should include the unpublished studies (AHS and NAPP) since the AHS is a cohort study with a methodology in design and analysis very different from the case control studies and hence should be considered on its own merits; while the NAPP study summarizes previous data that, if included in the meta-analysis without excluding the primary studies; such an estimate would "double-up" on those studies. Importantly, the statement that "Any limitations of both the study design and statistical analysis of included studies carry forward through the results of the meta-analysis" (page 18, Rider) is only partially correct i.e. this statement assumes that each study has exactly the same bias and moreover that all are biasing the results in the exact same direction - which is highly unlikely in practice.

Fleming Report

As the President Elect of the International Society for Environmental Epidemiology, a sub-discipline of Epidemiology that specifically concentrates among its members those with expertise in examining a wide range of spatial and temporal patterns in exposures and disease, I object strongly to the naïve use of both temporal cancer rates and spatial cancer patterns in Dr. Fleming's report in order to draw conclusions about NHL causes specifically whether or not glyphosate/GBF exposures cause NHL. Our discipline uses maps and graphs extensive because they are very important tools for the purpose of visualizing data i.e. to show general patterns of disease or exposure rates over time and/or space. However, the first thing I teach my

students in environmental epidemiology is that using these tools to claim that a very specific exposure (pesticide) does or does not cause a chronic disease is highly unscientific and unnecessarily invalidates the good use of these tools. For example, the pretty graphs and maps shown by Dr. Fleming cannot tell us anything about the influence of the AIDS epidemic over the years on NHL rates or about other time varying influences. Specifically, if glyphosate/GBFs are not the only agents capable of causing NHL – which defense experts seems to agree to since they are worried about confounding risk factors - and we accept that for example DDT and lindane – pesticides widely used in the 1950 to 70th – may also cause NHL, how could any of these graphs/ maps depict the influence of complex waxing and waning causal exposures over time, some of them increasing and some decreasing and therefore influencing rates in different directions? The spatial map by Fleming includes all races and both sexes, thus, it seems that he assumes that NHL rates in men and women or immigrant Hispanic laborers in central California can be easily compared with all San Francisco inhabitants including white males and that factors such the AIDS epidemic can be ignored; i.e. that we can simply compare age adjusted rates from San Francisco populations to those in central California populations and deduce whether or not glyphosate/GBF alone is the single agent causing NHL. Again, this is not only scientifically untenable but simply wrong.

Conclusion

I hold the above opinions to a reasonable degree of scientific certainty. Furthermore, as previously stated in my earlier expert report, I hold the opinion, to a reasonable degree of scientific certainty that glyphosate and GBFs including Roundup, cause non-Hodgkin's lymphoma. I reserve my right to supplement or amend this report as additional materials become available.

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Beate Ritz, M.D., Ph.D.

Date: August 18, 2017

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Defendant's Expert Report of Dr. William Fleming

Defendant's Expert Report Dr. Lorelei A. Mucci,

Defendant's Expert Report of Dr. Jennifer S. Rider

Exhibits 19A and 19B to Deposition of Dr. Aaron Earl Blair, taken March 20, 2017.