Exhibit 9

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	

Supplemental Report of Dr. Beate Ritz, M.D., Ph.D.

Pursuant to PTO No. 34 and In Support of General Causation

On Behalf of Plaintiffs

In this report, I will present my expert opinion on non-Hodgkin's lymphoma ("NHL") and glyphosate in the context of the recently published Agricultural Health Study, "*Glyphosate Use and Cancer Incidence in the Agricultural Health Study*, Journal of the National Cancer Institute 2017) ("AHS"). This article was published after my earlier expert reports and my deposition in this case, during which I was questioned extensively about the draft manuscript of the now published AHS. As more fully explained below, the publication of the AHS has not changed my opinions. None of the AHS publications, starting with De Roos in 2005, have shown a statistical association between glyphosate and NHL. This is because of the major methodological flaws in the study with respect to assessing this association as more fully explained below. In fact, these flaws were also predicted by several commentators, who were more generally concerned that exposure misclassification would produce false results.

First, the new AHS article presents results for glyphosate that are very similar to those from an unpublished 2013 draft manuscript that was previously provided to me and discussed in my earlier reports and at my deposition. Most pesticide results presented in the 2013 draft paper were published by AHS authors in 2014 (Alavanja 2014). However, the 2014 manuscript did not report analyses and results for glyphosate. The latest publication by Andreotti et al. (2017) in contrast focuses solely on glyphosate use in the AHS and reports on several cancers including NHL and its subtypes. This new paper (Andreotti, 2017) updates the earlier AHS publication by DeRoos (2005) that I discussed in my previous report; i.e. the 2017 paper presents results for NHL with an attempt to increase the length of follow-up to include cancer outcomes until 2012 for North Carolina and until 2013 for Iowa. The publication employs follow-up questionnaire data collected between 1999-2005 from cohort members to update the original exposure information obtained at enrollment in 1993-1997. The publication of this AHS paper, however, does not change my opinion about the epidemiologic evidence and the role of glyphosate in causing NHL, because it does not provide new data or new conclusions beyond what was already presented in the 2013 AHS draft I reviewed. Specifically, this paper shows no association between glyphosate use and NHL. While the publication presents some additional analyses such as exposure lagging and subtype analyses, it does not address or remedy the major flaw of the AHS in terms of glyphosate exposure assessment. Thus, the published paper is no more informative or useful than the unpublished draft from 2013—a draft I previously reviewed and which was the subject of much discussion during my first deposition.

In my previous reports as well as during my deposition, I described the major methodological flaws of the AHS with respect to glyphosate exposure assessment that undermine the weight of the AHS null findings for glyphosate. Here, I will concentrate on major exposure assessment issues while highlighting aspects that have not received enough attention, especially exposure misclassification introduced by data obtained in the 1999-2005 interviews, which are essential for my critique of this publication.

The AHS cohort study started enrollment in 1993 and was designed to follow licensed restricted use pesticide applicators in two states – North Carolina and Iowa. It prospectively collects health outcome data – such as from cancer registries - and attempts to update exposure information in subsequent interviews with cohort members. At time of recruitment, applicators attended pesticide-licensing exams in each state and, if they agreed to participate, were asked to report past pesticide use via a questionnaire. Pesticide licensing exam takers were thought to be primed to think about pesticide applications, and expected, on the spot, to be able to provide details about their pesticide use history. It is important to note that applicators were asked to recall details of their past use of specific pesticides potentially going back 3-4 decades while being present at the licensing exam facility. Hence, this exposure assessment relied entirely on instantaneous recall for the 22 pesticides –including glyphosate – for which the applicators were asked to report detailed use. Notably, cohort members were not able to review records of past pesticide purchases, ask family members or co-workers to help recall specific use periods and agents, or take time to retrieve necessary information – a practice commonly encouraged in case control studies that assess pesticide health effects.

It is well known that faulty recall of past exposures leads to measurement error. In a cohort study, this error contributes to ‘non-differential’ exposure misclassification; i.e. it is as likely for those who remain healthy and those who later develop a disease to make mistakes and not recall and report exposures correctly. For example, some applicators, who used a lot of different pesticides over decades, might get confused about which ones they used, when they used them, and for how long. Some may not recall less frequently used pesticides, occasionally used pesticides, or pesticides used at low volume. Recently used and large volume pesticides might get recalled more often and with better accuracy. Older or less educated participants might have worse recall than younger or educated participants, etc.

As mentioned above, such recall error can be reduced through improved data collection efforts such as home visits to farmers by occupational professionals or trained researchers who review purchasing records and other materials with the study subject as has been done in a French case control study of 800 farmers (Elbaz 2009). There, 224 Parkinson Disease (PD) cases were matched to 557 controls free of PD affiliated with the same health insurance. "Pesticide exposure was assessed using a 2-phase procedure, including a case-by-case expert evaluation." Professional users were interviewed at home by an occupational health professional to reconstruct the history of pesticide use. Participants were asked to list each farm where they had worked (start/end years). They were asked to describe each farm in terms of land size, crops (size), and animal breeding (number), and whether they had personally sprayed pesticides for each of them. For each crop/animal for which pesticides were used, detailed information was obtained: pesticides, frequency (days/year), duration (hours/year), spraying method (portable device, tractor), and start/end years. The point was to "use as many sources of information as possible, interviewers visited the farm, discussed technical issues, looked for old pesticide containers and packages, and reviewed bills and farming calendars. Farm interviews usually lasted half a day or more."

It is a great disadvantage of large cohort studies, such as the AHS, that such in-depth person-by-person exposure assessment is too burdensome and expensive; in fact, cohorts generally collect lower quality exposure data on a much larger number of study subjects, substituting quantity for quality of data. The error generated in cohorts, and especially the AHS, is considered 'non-differential' such that there is no systematic difference between the error in reporting for those who later become cases (diseased) and those who remain healthy (controls) – this is 'by design' in a cohort since at enrolment no-one has the disease of interest such that remembering would be influenced by disease status. Thus, exposure information ascertained in cohorts can be considered 'free of recall bias' since it is not 'differential' (differential referring to the issue that diseased individuals recall exposures differently from non-diseased controls because they may have an incentive to recall exposures and explain their disease). On the other hand, it is well known that non-differential exposure misclassification is 1) stronger in situations when exposure assessment quality is low; and 2) it is the type of exposure error that most often biases results towards the null (no effect); i.e. studies with lower quality exposure assessment (including large cohort studies) are more likely to find no association even if one truly exists.

This is known as drowning a true signal in noise, meaning that the more inaccurate the exposure assessment is, the greater the noise, and the stronger any true signal (effect) has to be in order to be noticed above the noise that is generated by measurement error. The first AHS by DeRoos was most likely affected by non-differential exposure measurement error. Interestingly, Monsanto scientists criticized the AHS design and especially the exposure assessment it employed in 1997, mirroring my concerns described above (see MONGLY00885870):[1]

> "*The exposure assessment in the AHS will be inaccurate. Exposure assessment will be based on historical usage as reported by the farmer or applicator on the study questionnaire(s). There are two problems with this approach: 1. usage does not necessarily mean exposure (work practices/ equipment/ environmental conditions determine exposure to a large degree); 2. recall can be faulty or biased, especially when historical usage information is collected. Attempts at verification over a 3 year period have found less than 70% agreement between purchasing records and reported usage.*
>
> *Inaccurate exposure classification can produce spurious results. The conventional thinking in epidemiology is that exposure misclassification will most often obscure exposure disease relationships."*

Monsanto commissioned a report by a Harvard expert group that came to similar conclusions: "*Important limitations of the AHS include ….…limited understanding of the reliability and validity of self-reporting of chemical use, an insufficient program of biological monitoring to validate the exposure surrogates employed in the AHS questionnaires*." Gray et al. (2000) (page 48). Importantly, they also concluded that "*Misclassification will reduce the power of the study to detect any genuine cause-effect relationships and will also reduce the validity of findings. Reductions in power are a serious issue because they will undermine the ability of government and industry to regulate harmful exposures and to reassure farmers with "negative" results."* (page 58). Finally, the paper published by Alavanja in 2014 received reviewers' comments that also refer to this problem, they state: "*...misclassification of exposures can occur and can have a sizable impact on estimates of relative risk, which in a prospective cohort design would tend to produce false negative results*." (page 9).

[1]Dr. Acquavella was employed by Monsanto as an epidemiologist at the time of this quote. He is currently a consultant for Monsanto Company.

While these recall-related exposure assessment issues are generic for any cohort study that relies on self-reports of past exposures, the AHS exposure assessment for glyphosate or GBFs has a much more compounded misclassification problem due to the time-varying character of the exposure and - as described in my previous reports - the dramatic change in glyphosate or GBFs use in the mid-1990s that completely overlaps with the initial AHS exposure assessment at enrollment between 1993-1997 (Aspelin and Grube 2016; Grube et al 2016; Coupe and Capel 2015; Thelin and Stone 2016; Service. USDoANAS 2016; Benbrook 2015).

This also explains that the problems for GBFs in the AHS are different from those for any other time-varying pesticide exposure that did not have this pattern of change. Specifically, the initial exposure assessment was compromised because the answers to the questions of whether, how much, and in which decade glyphosate had been used by applicators was heavily influenced by the timing of when subjects entered the cohort, that is, whether the enrollment happened before (1993-95) or after (1996-97) the introduction of glyphosate resistant crops. This will have influence on the measures of frequency and intensity of use reported at baseline enrollment because GMO adopters would be expected to report heavy use if asked in 1996-97, but not if asked in 1993-95 prior to GMO introduction. In other words, the study would put an applicator into the 'no/low intensity use' group if he applied glyphosate only occasionally or not at all before adopting GMOs/glyphosate use in 1995 as long as he was enrolled and asked to report his use early i.e. in the period 1993-95. The exact same individual would be put into a 'high intensity use' group if asked to report the same use in 1996 or 1997 after he adopted GMOs. It is therefore likely that many high intensity glyphosate users were incorrectly grouped in the no or low intensity use groups.

After the initial enrollment that determined the exposure status of each participant (ever/never or low/high intensity exposure to a specific pesticide), the cohort members are followed for the occurrence of events such as a cancer diagnosis. In the DeRoos (2005) analyses, cancer data were collected for everyone from enrollment until the end of 2001. In her analysis, DeRoos then compared NHL rates in those with glyphosate exposure (by low/high intensity) with the rates among those having reported no/low exposure at enrollment. Due to the dramatic changes in glyphosate use in 1995, in this cohort it is possible that the 'high use' group may include: 1) an applicator who used glyphosate frequently/intensely throughout the 1970s or 80s and reported this at any time during enrollment; or 2) an applicator who never or sparingly used

glyphosate before 1995 but switched to high intensity use on GMO crops and reported this high use at enrollment in 1996 or 1997. On the other hand, the 'low/no' use group may consist of applicators that also switched to heavy use on GMO crops in 1996 or 1997 but enrolled in the AHS prior to 1996 as well as applicators who truly used glyphosate infrequently or not at all. Thus, the AHS may classify true users as non-users or combine in the exposed group very different periods of exposure (early users and those who switched to heavy use after 1995). This is a classic example of exposure misclassification due to changes in exposure over time that are not captured by the study's exposure assessment. Again, since this is a cohort study, the exposure misclassification happens prior to event (disease) and thus is non-differential. This aspect (exposure changing in such a dramatic manner during the initial enrollment) is unique to glyphosate in this cohort and greatly compounds the non-differential exposure misclassification that occurs due to faulty memories in reporting past pesticide use I described above. The combined impact of these two sources of non-differential exposure misclassification can strongly bias results towards the null i.e. not finding a true association.

Importantly, the dramatic change in glyphosate use impacts studies with longer follow-up such as the AHS publication in addition to the initial exposure assessment and the DeRoos (2005) results. These problems are compounded by the exposures updated in the telephone assisted interviews in 1999-2005. This is because there are two additional problems introduced by the updated exposure assessment that further increase non-differential exposure misclassification during the follow-up period and cause misclassification of participants at baseline and during follow-up. One reason – as I explained previously (See Ritz Expert and Rebuttal Reports) - is the tremendous loss to follow-up (almost 40% of AHS participants did not respond to follow-up calls in 1999-2005) that prevented AHS researchers from updating the glyphosate exposure measure in these subjects with actual data and the problems related to guessing (imputing) exposure data for these participants. The second problem arises because the AHS asked only about the use of specific pesticides during the last year of farming prior to the phase 2 interview thus leaving potentially 9-10 years of actual pesticide use between the first questionnaire (1993) and the phase 2 interview (2003) unreported. That is, the interview did not ask participants to fill in the gap between enrollment (1993-1997) and follow-up (1999-2005), but "*at follow-up applicators reported the number of days each pesticide was used in the most recent year farmed*" (AHS 2017), which generally was the past year prior to interview. In the

very time period when exposures to glyphosate dramatically changed, reporting only one year of use leaves us to guess when and if glyphosate use changed in the interim and generates another source of additional non-differential exposure misclassification.

Loss to follow-up impacts exposure estimates used in the AHS (2017) paper because the AHS researchers decided to impute (guess) exposures between enrollment and follow-up for those who did not respond in Phase 2. They used a sophisticated imputation model (Heltsche 2012) that bases its exposure guesses for non-responders on what is known about exposure levels for responders at both times (enrollment and at follow-up) and what is known about non-responders at enrollment (Heltshe: "*a data-driven multiple imputation for the 20,968 applicators who did not complete the Phase 2 questionnaire was employed*"). It is important to realize that this data driven approach makes speculative assumptions that may be wrong. For example, it assumes one can use what we know about responders and non-responders at enrollment to guess exposures after enrollment for non-responders. Rinsky et al (2017), for example, modeled potential bias from non-response in the AHS and found that non-response depended on many lifestyle factors as well as on pesticide use i.e. "*those indicating personal pesticide use or raising animals responded more frequently than their counterparts*" (page 398). But, since this approach relies on exposure data at enrollment that is already misclassified - because of inaccurate recall and change of glyphosate use mid-enrollment in 1995 - these guesses for non-responders are likely to propagate the non-differential exposure errors further making the imputed and updated exposure estimates less valid and reliable than the original ones.

In support of my arguments above, the Harvard group stated in the year 2000; "*Periodic follow-up surveys are necessary to determine how exposures and disease states change as the cohort ages, thereby maintaining the prospective character of the study. If low response rates occur with the follow-up questionnaires, the potential for bias will increase, partly from misclassification of subjects (and personyears) with regard to chemical exposure*." (page 52). Similarly, the 1997 report by John Acquavella (Monsanto employed epidemiologist) concludes with this cautionary note: "*We also have to keep in mind that even the most sophisticated statistical analysis can't correct for other aspects of the study that are less than optimum (e.g. exposure misclassification).*" A 2016 Exponent report commissioned by Monsanto and CropLifeAmerica, a pesticide industry group, listed various flaws in the AHS, including that "*only 44% of enrolled pesticide applicators completed the detailed take-home questionnaire*

shortly after enrollment, and participation in follow-up questionnaires was also highly incomplete". Furthermore, the editorial by Dr E. Ward in JNCI 2017 commenting on the AHS 2017 paper acknowledges exposure assessment problems when stating "*given the nature of pesticide use in agriculture, applicators may only be exposed to specific pesticides for short periods of time each year*" and "*the intermittent nature and limited range of exposure may limit the ability of studies in these populations to detect cancer hazards.*"

Thus, overall and in summary, there is non-differential exposure misclassification from several sources that impact the AHS findings:

- simple recall error of past pesticide use history at enrollment
- dramatic increase in glyphosate use and exposure mid-enrollment
- exclusively referring to the most recent year of pesticide exposure in follow-up interviews leaving a considerable gap in ascertaining true use for a period when use changed dramatically
- having to impute exposures for a large proportion (40%) of non-responders with misclassified data at both time points

These errors compound each other in the AHS and strongly reduce the study's ability to detect actual cause-effect relationships. This greatly reduces the validity and reliability of the AHS findings. These flaws in exposure assessment leading to non-differential misclassification of exposure are described in standard epidemiological methodology books (Rothman et al Modern Epidemiology), and are generally well accepted in the epidemiological community - as also stated by Monsanto's own epidemiologist, Dr. Acquavella (see above). It is my opinion that this compounded exposure misclassification, from both the baseline and follow-up exposure assessment for glyphosate in the AHS, explains why the AHS found no association for NHL - in stark contrast to all the other epidemiological studies.

I have not arrived at these opinions recently and have previously discussed these exposure assessment issues in my epidemiologic teaching at UCLA. I used the AHS as an example of problems that can arise in cohort studies. As seen in slides I created for my student courses from 2012, page 5, I list the disadvantages of the cohort design. The disadvantages of the cohort design include:

1) Large numbers of subjects required (thus, low feasibility to study rare diseases);

2) Relatively expensive to conduct;

3) Potentially a very long duration for follow-up is necessary;

4) Exposures may change, making findings irrelevant unless the exposure assessment is adapted;

5) Maintaining follow-up may be difficult.

Points 4 and 5 are specifically relevant for the AHS, which I discuss as an example in the slides. In my slides, I show how much active loss to follow-up the AHS experienced, which is listed along with other disadvantages of a cohort study. I teach the AHS as an example to my students, because I can then explain to them the need to distinguish between active and passive follow-up. I further point out to my students that even though we have almost perfect 'passive follow-up' through cancer registries in the AHS, this will not suffice to give adequate follow-up in terms of changing exposures. Rather, active follow-up is needed to constantly update time varying (changing) exposures in order to avoid massive exposure misclassification. Because of the lack of active follow-up in the AHS, there is indeed massive non-differential exposure misclassification as I discussed above. (For example, the Harvard Nurses Cohorts have been followed for 3 to 4 decades and actively re-contacted their subjects every 2 years in order to update time-varying exposures; a very high participation rate of more than 90% in each follow-up cycle of questionnaires gives this particular cohort a very high rate of compliance with active follow-up as contrasted with the very low rate of passive follow-up in the AHS- i.e. 64% in the first cycle and less than 50% in the second cycle).

Finally, it would be inappropriate to include this recent AHS publication in any meta-analysis, since meta-analyses generally proceed after pre-selecting studies of overall similar high quality. It is not uncommon to exclude certain studies deemed not to be of adequate quality in some respect. Often meta-analyses provide separate estimates for case control and cohort studies or studies different in terms of exposure or outcome assessment ,since a summary estimate for studies that are truly dissimilar is not scientifically useful. Rather, we gain more scientific insight by considering what contributes to the heterogeneity of study results – which is generally assessed with both formal statistical as well as qualitative tools (see: Blair et al, 1995: "*In particular, meta-analysis can identify heterogeneity in effects among multiple studies, and, where appropriate, provide summary statistics that portray the relationship between environmental exposures and health effects*." These 1995 guidelines for meta-analyses also recommended: "*Study design: Under ideal circumstances, studies of different design should be*

included. The contribution of study design to heterogeneity in the effect estimates should be analyzed, and separate meta-analyses should be conducted by study design when effect estimates systematically vary by design." AND "*As part of a heterogeneity analysis, it is important to evaluate the variation of exposure between and within studies. Studies with very different exposure levels or definitions of exposure may be inappropriate for combined analyses. In such cases separate analysis may be appropriate.*") It is more important to generate explanations for differences in estimated effects than to produce summary estimates across studies that are clearly heterogeneous. In this case, including the AHS in a meta-analysis is methodologically unsound because its data output is unreliable on multiple levels, as identified above and in my previous reports and testimony.

Overall, this 2017 AHS publication perpetuates the fundamental design weaknesses I have already pointed out in the unpublished AHS draft manuscript. Simply put, it does not provide useful new information on whether or not glyphosate causes cancers or NHL because of the strong exposure misclassification, an error that cannot be remedied. I hold all the above opinions to a reasonable degree of scientific certainty. Furthermore, as previously stated in my earlier expert and rebuttal reports, 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.

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

Date: December 18, 2017

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

Exhibit 19-17 to the Deposition of Dr. Beate Ritz, taken September 18, 2017.

Exhibit 53 to Plaintiffs' Opposition to Monsanto Company's *Daubert* and Summary Judgment Motion, ECF No. 647, filed October 27, 2017. (MONGLY02314040)

Exhibit 75 to Plaintiffs' Opposition to Monsanto Company's *Daubert* and Summary Judgment Motion, ECF No. 647, filed October 27, 2017.

Exhibit 1 to Plaintiffs' Reply Memorandum in Support of Their Opposition to Monsanto's *Daubert* and Summary Judgment Motion, ECF No. 793, filed November 20, 2017. (MONGLY00885870)