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Attorneys for Plaintiff
DEWAYNE JOHNSON

SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF SAN FRANCISCO

DEWAYNE JOHNSON,

Plaintiff,

v.

MONSANTO COMPANY

Defendants.

Case No. CGC-16-550128

DECLARATION OF CURTIS G. HOKE IN SUPPORT OF PLAINTIFF'S OPPOSITION TO MONSANTO'S MOTION *IN LIMINE* NO. 26 TO EXCLUDE EVIDENCE OR ARGUMENT THAT MONSANTO DECEIVED THE EPA

Trial Judge: TBD

Trial Date: June 18, 2018
Time: 9:30 a.m.
Department: TBD

DECLARATION OF CURTIS G. HOKE

I, Curtis Hoke, declare and state as follows:

1. I am an attorney at law admitted to practice before all of the courts in the state of California. I am an attorney at The Miller Firm, LLC, attorneys of record for Plaintiff Dewayne Johnson. I am over eighteen years of age and am fully competent to make this Declaration in support of Plaintiff's Opposition to Monsanto's Motion *in Limine* No. 26 to Exclude Evidence or Argument that Monsanto Deceived the EPA. Except as otherwise expressly stated below, I have personal knowledge of the facts stated in this declaration, and if called to testify, I could and would competently testify to the matters stated herein.

2. Attached hereto as **Exhibit A** is a true and correct copy of portions of the Expert Report Regarding the Regulatory Review of Glyphosate by John R. Fowle III, Ph. D., DABT.

3. Attached hereto as **Exhibit B** is a true and correct copy of a document entitled Evaluation of the potential genotoxicity of Glyphosate, Glyphosate mixtures and component surfactants by James M. Parry.

4. Attached hereto as **Exhibit C** is a true and correct copy of MONGLY03734971.

5. Attached hereto as **Exhibit D** is a true and correct copy of relevant portions of the deposition transcript of Donna Farmer, Ph.D., taken on January 11, 2017.

6. Attached hereto as **Exhibit E** is a true and correct copy of relevant portions of the deposition transcript of John R. Fowle, III, Ph.D., taken on February 22, 2018.

7. Attached hereto as **Exhibit F** is a true and correct copy of MONGLY03738295 – MONGLY03738296.

I declare under penalty of perjury under the laws of the State of California that the foregoing is true and correct.

Executed on June 7, 2018 in Orange, Virginia.

By: _____
Curtis G. Hoke,
Declarant

EXHIBIT A

Expert Report
Regarding the Regulatory Review of Glyphosate
John R. Fowle III, Ph.D., DABT
Principal, Science to Inform, LLC

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The SAP is chartered under the Federal Advisory Committee Act (FACA) in accordance with the provisions of 5 U.S.C. App.2. The FIFRA SAP is a statutory advisory committee created on November 28, 1975 pursuant to section 25(d) of the FIFRA, as amended by Public Laws 94-140, 95-396, 96-539, 98-201, and 100-532. Section 104 of the Food Quality Protection Act of 1996 (Public Law 104-170) establishes a Science Review Board consisting of sixty scientists who shall be available to the Scientific Advisory Panel on an ad hoc basis to assist in reviews conducted by the Panel.

The purpose of the FIFRA SAP is to provide comments, evaluations, and recommendations on pesticides, and pesticide-related issues, as to the impact on health and the environment of regulatory actions. The FIFRA SAP and FQPA SRB neither make nor recommend policy decisions. Rather, they provide recommendations and advice about OPP's analyses, reports and operating guidelines to improve the effectiveness and quality of scientific analyses made by EPA. The SAP meets on about 8-10 topics per year in public meetings to provide advice on the science used to inform decisions. To ensure timeliness, minutes and recommendations are published within 90 days of each meeting.

XI. Communications Between EPA and Stakeholders, Including Regulated Entities

I am aware that certain EPA employees, or former employees, are being accused of collusion with Monsanto. My experiences with, and observations of, these people are in direct contrast to these allegations as I know them to be of high integrity. Based on my observations during my time at EPA, they followed and complied with all relevant

processes, rules and regulations including those dealing with ethics and conflicts of interest.

Further, I know that allegations have been made by some that Monsanto employees inappropriately met with EPA and that they inappropriately affected glyphosate registration decisions. Based on my experience in the pesticides program, I never experienced, or witnessed, any inappropriate communication between any registrant, including Monsanto, and EPA. Meetings with registrants are a standard part of the EPA process, expected, and not inherently inappropriate. In fact, FIFRA calls for them.

Periodic meetings, and communications between EPA and the public, including registrants, during the registration process was intended by Congress and is a long-standing part of the EPA "fishbowl" approach to open, transparent government. It is a normal and necessary process. In fact it is specified for the registration of pesticides under 40 CFR §§155.27, 155.30 "Meetings and communications" that "EPA personnel may, upon their own initiative or upon request of any interested person or party, meet or communicate with persons or parties outside of government concerning a Registration Standard under development." It is further stated that "the purpose of such meetings is to receive and consider information, exchange views, explore factual and substantive positions, discuss regulatory options or for any other purpose deemed appropriate by the Agency in its deliberations concerning development of a Registration Standard."

EXHIBIT B

Evaluation of the potential genotoxicity of Glyphosate,

Glyphosate mixtures and component surfactants

James M. Parry

Centre for Molecular Genetics and Toxicology

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University of Wales Swansea

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Introduction

The available data concerning the potential genotoxic activity of glyphosate, glyphosate mixtures and surfactants have been evaluated and the results of the evaluation are presented in Tables 1 to 14. Each of the tables reviews the data for the three groups of chemicals grouped according to the type of test system used to assess potential genotoxicity, the effect produce and reference to the appropriate data set.

Conclusions

Evaluation of the genotoxicity of Glyphosate

I. Bacterial mutagenicity (Table 1)

Two comprehensive studies (Scantox 10.9.91-A, Li and Long 1988) provide no evidence of mutagenic activity for glyphosate in *Salmonella typhimurium*.

One study of differential DNA repair in the *Bacillus subtilis* rec assay gave negative results.

I conclude that there was no evidence that glyphosate is genotoxic in bacteria.

II. *In vitro* cytogenetic assays (Table 3)

(a) Chromosomal aberrations

Two studies in human and bovine lymphocytes report positive results over dose ranges up to 170μM following exposure for 72 hrs in the absence of S9 mix (Lioi *et al* 1998a, 1998b).

One negative study in human lymphocytes over a dose range of up to 562μg/ml in both the presence and absence of S9 mix and at sampling times of up to 48 hrs (Notox 141918).

Note: the Lioi *et al* studies present a combined data set of experiments from 3 separate donors.

One negative study in *Allium cepa* root tips has been reported.

(b) Sister chromatid exchange

Two studies report positive results in human and bovine lymphocytes over dose ranges of up to 170μM following exposure for 72 hrs in the absence of S9 mix (Lioi *et al* 1998a, 1998b).

Evaluation. There is published evidence that glyphosate shows clastogenic activity following 72 hrs exposure of both bovine and human lymphocytes (Lioi *et al* 1998a, 1998b).

In my view there is a need to repeat the studies of Lioi *et al* to a comprehensive protocol to clarify the potential clastogenic activity of glyphosate.

III. Point mutation in cultured mammalian cells (Table 5)

Negative results are reported in both the Tk assay using mouse lymphoma cells (up to 5000μg/ml) and the HGPRT assay using Chinese hamster cells (up to 22500 μg/ml) in both the presence and absence of S9 mix (Scantox 10.9.91-B, Li and Long 1988).

There is no evidence that glyphosate is a point mutagen in cultured mammalian cells.

IV. In vivo chromosome studies in rodents. (Table 6)

a) Rat bone marrow cytogenetics assay

There is one negative study reported in the bone marrow of rats exposed to 1000mg/kg bw (Li and Long 1988),

b) Mouse bone marrow micronucleus assay.

There are two negative studies at concentrations of up to 5000mg/kg bw available for evaluation (Rank *et al* 1993, Scantox 12.9.91) However, in neither study is there substantive evidence of bone marrow toxicity.

There is one positive study at 300mg/kg with multiple dosing, sampled at 24hrs (Bolognesi *et al* 1997). However, this study only involved the use of 4 animals per dose point however bone marrow toxicity was observed.

Evaluation. There are conflicitng results concerning the bone marrow activity of glyphosate which can only be resolved by repeating the Bolognesi *et al* (1997) study.

V Dominant Lethal Study (Table 10)

There is one negative dominant lethal assay involving exposure of male mice of concentration up to 200mg/kg bw (RD 300, SRRS L1147)

Evaluation. There is no evidence that glyphosate is capable of inducing dominant lethal mutations in mouse male germ cells.

VI Miscellaneous Endpoints (Table 8)

a) **G6PD activity**

Two studies demonstrate increases in G6PD activity (as a marker of a pro-oxidant state) in human and bovine lymphocytes at concentrations of up to 170μM (Lioi *et al* 1998a, 1998b). G6 PD activity was reduced in presence of an antioxidant.

Note : no genetic endpoint was measured in these studies.

b) **Induction of 8-OHdG**

One study demonstrates the production of 8-OHdG (as a marker of oxidative damage) in the liver of mice exposed to glyphosate (Bolognesi *et al* 1997)

c) **Induction of DNA damage measured by alkaline elution**

One study demonstrates the production of single strand breaks in liver and kidney of mice following exposure to 300mg/kg bw of glyphosate (Bolognesi *et al* 1997).

d) **Induction of DNA adducts measured by ^{32}P post - labelling**

One study reports no increase in adducts in the liver and kidneys of mice following exposure to 130 and 270mg/kg of glyphosate (Peluso *et al* 1998)

e) **Hepatocyte DNA repair assay**

One limited study (low concentrations used) reported negative results for its ability of glyphosate to induce repairable DNA assay using rat hepatocytes (Li and Long 1988).

Evaluation. These studies provide some evidence that glyphosate may be capable of inducing oxidative damage under both *in vitro* and *in vivo* conditions

Evaluation of the genotoxicity of Glyphosate mixtures

Bacterial mutagenicity (Table 2)

1) The limited published study (Rank *et al* 1993) showed single dose point increases in mutagenicity of a Glyphosate mixtures in *Salmonella* strains TA98 and TA100.

Four comprehensive studies with glyphosate mixtures of concentration of 31% to 72% (MSL – 11731, MSL – 11729, MSL – 11730, BioAgri G.1.1.050/96) provide no evidence of mutagenic activity in *Salmonella typhimurium.*

Evaluation. In view of the extensive negative data in studies performed to comprehensive protocols I conclude that Glyphosate mixtures are not mutagenic to *Salmonella typhimurium.*.

11) **In vitro cytogenetics (Table 4)**

a) **Chromosomal aberrations**

There are no available studies involving the analysis of the induction of chromosome aberrations in cultured mammalian cells.

There is one published study in *Allium cepa* root tips reporting positive results (described as being indicative of spindle disturbances) at concentrations greater then 720 µg/ml (Rank *et al* 1993).

b) **Sister chromatid exchange**

There are two studies reporting positive results in human lymphocytes at concentrations from 100µg/ml to 2500µg/ml (Bolognesi *et al* 1997, Vigfusson and Vysa 1980).

Evaluation. The *in vitro* cytogenetic data for glyphosate mixtures are inadequate for evaluation.

IV **_In vivo_ mouse bone marrow micronucleus assay (Table 7)**

There are 5 studies in mouse bone marrow which report negative results for micronucleus induction for various mixtures of glyphosate at concentrations of up to 3400mg/kg bw (Rank *et al* 1993, BioAgri C.1.2-60/96, MSL – 11771, MSL7173, MSL – 1172). However, most of the studies provide only limited evidence of bone marrow toxicity.

There is one positive study of a Roundup mixtures at 450mg/kg bw with multiple dosing and sampled at 24 hrs (Bolognesi *et al* 1997). Bone marrow toxicity was reported in this study but only 3 animals were used per dose point.

Evaluation. Conflicting results concerning the bone marrow activity of glyphosate mixtures can only be resolved by repeating the Bolognisi *et al* (1997) study.

V **Drosophila sex linked recessive lethal mutation assays (Table 11)**

One study provides limited evidence that following larval feeding both Roundup and Pondmaster mixtures produced some positive results in spermatocyte broods (Kale *et al* 1995)

Evaluation. Some limited evidence that Glyphosate mixtures are capable of inducing sex linked recessive mutations in the male germ cells of *Drosophila melanogaster*.

VI **Miscellaneous Endpoints (Table9)**

(a) **Induction of 8-OHdG**

One study demonstrates the production of 8-OHdG (as a marker of oxidative damage) in the liver and kidneys of mice exposed to Roundup mixture (Bolognesi *et al* 1997).

(b) **Induction of DNA damage measured by alkaline elution**

One study demonstrates the production of single strand breaks in the liver and kidney of mice exposed to 300mg/kg bw of Roundup mixture (Bolognesi *et al* 1997)

c) **Induction of DNA adducts measured by ^{32}P post labelling**

One study reports an increase in adducts in the liver and kidneys of mice following exposure to 400, 500 and 600mg/kg bw of Roundup Mixtures (Bolognesi *et al* 1997)

d) **COMET assay**

One study demonstrates the induction of chromosome damage as measures in the COMET assay following exposure of tadpoles to Roundup at concentrations above 27mg/litre (Clements *et al* 1997)

Evaluation. These studies provide some evidence that Roundup mixture produces DNA lesions *in vivo*, probably due to the production of oxidative damage.

Evaluation of the genotoxicity of Surfactants

I) **Bacterial Mutagenicity (Table 12)**

Three comprehensive studies failed to demonstrate any mutagenic activity for the surfactants in bacterial assays (MSL – 10625, MSL – 1538, Hoecht 92.0487).

II) ***In vitro* chromosome aberration assay (Table 13)**

One study failed to demonstrate any significant increase in chromosome aberrations after exposure to Dodigen 4022 at concentrations of up to 6000µg/ml (Hoecht 92.1025).

However, a number of non-significant changes in various parameters were reported. This study should be repeated.

III) **Mouse bone marrow micronucleus assay (Table 14)**

One limited experiment (ML-89-463) produced negative results in mouse bone marrow with MON 0818 at 100mg/kg bw.

Evaluation. The only adequate studies with the surfactants are those involving bacterial mutagenicity assays. There was no evidence that the various surfactants are bacterial mutagens.

Overall Conclusions

1) It is clear from the data provided that with the exception of one limited study (Rank *et al* 1993) there is an extensive range of studies which demonstrate that glyphosate and glyphosate are **not** genotoxic in bacteria.

2) There is published *in vitro* evidence that glyphosate is clastogenic and capable of inducing sister chromatid exchange in both human and bovine lymphocytes (Lioi *et al* 1998a, 1998b).

3) *In vitro* cytogenetic data on glyphosate mixtures are inadequate for evaluation.

4) There are two studies (Scantox 10.9.91, Li and Long 1988) which demonstrate that glyphosate is not a point mutagen in cultured mammalian cells.

5) This is a published study indicating that glyphosate was not clastogenic in rat bone marrow (Li and Long 1988). There are two studies which indicate that glyphosate was not capable of inducing micronuclei in mouse bone marrow (Rank *et al* 1993, Scantox 12.9.99). However, in neither study was there substantive evidence of bone marrow toxicity.

   There is one published study which suggests that glyphosate may be capable of inducing micronuclei in mouse bone marrow when delivered by multiple dosing (Bolognesi *et al* 1997).

6) Five studies report negative results for micronucleus induction in the bone marrow of mice following exposure to glyphosate mixtures. However, these studies provide only limited evidence of bone marrow toxicity. None of the studies were performed to a protocol equivalent to that of Bolognesi *et al* (1997) which gave positive results with glyphosate.

7) There is one dominant lethal study which failed to demonstrate any capacity to induce genotoxicity in mouse male germ cells (RD300, SRRS L1147). However, it should be noted that this is a relatively insensitive methodology.

8) No dominant lethal assay results are available for glyphosate mixtures.

9) No sex-linked recessive lethal assay in *Drosophila* results are available for glyphosate.

10) Following larval feeding, Roundup and Pondmaster mixtures containing glyphosate produced some positive results in spermatocyte broods (Kale *et al* 1995).

11) Glyphosate induced G6PD activity in both bovine and human lymphocytes (Lioi *et al* 1998a, 1998b) and the production of 8-OHdG in mouse liver (Bolognesi *et al* 1997). Both observations indicate that glyphosate may be capable of inducing a pro-oxidant state leading to the formation of the oxidative damage lesion 8-OHdG.

12) A Roundup mixture containing glyphosate was shown to produce 8-OHdG in both the liver and kidneys of mice (Bolognesi *et al* 1997). These observations indicate the Roundup mixture is capable of inducing oxidative damage *in vivo*.

13) Glyphosate failed to induce repairable DNA damage in a limited *in vitro* study in rat hepatocytes (Li and Long 1988).

14) Glyphosate induced single strand breaks *in vivo* in the liver and kidneys of mice (Bolognesi *et al* 1997).

15) Roundup mixture produced single strand breaks *in vivo* in the liver and kidneys of mice (Bolognesi *et al* 1997).

16) Glyphosate mixture but not Glyphosate produced an increase in uncharacterised DNA adducts *in vivo* in the liver and kidneys of mice (Peluso *et al* 1998).

The overall genotoxicity profiles of glyphosate and glyphosate mixtures are illustrated in Figures 1 and 2 respectively.

17) None of the surfactants demonstrated any mutagenic activity in bacteria.

18) There are no adequate data to evaluate the *in vitro* clastogenic activity of surfactants.

19) One limited bone marrow micronucleus assay failed to detect any micronucleus inducing activity with the surfactant MON0818.

Specific evaluation of the genotoxicity of glyphosate

On the basis of the study of Lioi *et al* (1998a and 1998b) I conclude that glyphosate is a potential clastogenic *in vitro*. The study of Bolognesi *et al* (1997) indicates that this clastogenic activity **may** be reproduced *in vivo* in somatic cells. However, the dominant lethal assay (of limited sensitivity) indicates that this genotoxic activity is not reproduced in germ cells. The work of Bolognesi *et al* (1997) and Lioi *et al* (1998a and 1998b) suggests that the genotoxicity observed may be derived from the generation of oxidative damage in the presence of glyphosate.

Specific evaluation of genotoxicity of glyphosate mixtures

In view of the absence of adequate data no evaluation of the clastogenic potential *in vitro* of glyphosate mixtures is possible. In the absence of a micronucleus study to the protocol of that used by Bolognesi *et al* (1997) no adequate assessment of the potential activity of glyphosate mixtures in bone marrow is possible. The available studies do not provide any evidence of genotoxicity in rodent bone marrow. There is some evidence from *Drosophila* to suggest that glyphosate mixtures may have some germ cell activity.

The studies of Bolognesi *et al* (1997) suggests that glyphosate mixtures may be capable of inducing oxidative damage *in vivo*.

Specific evaluation of surfactants

None of the surfactants were capable of inducing mutations in bacteria. No adequate data available to evaluate the *in vitro* or *in vivo* clastogenicity of the surfactants.

Publications utilized in the assessment of the genotoxic activity of glyphosate and glyphosate formulations.

Lioi *et al* (1998a). Genotoxicity and oxidative stress induced by pesticide exposure in bovine lymphocyte cultures *in vitro*. *Mutation Research* **403**, 13-20.

Lioi *et al* (1998b). Cytogenetic damage and the induction of pro-oxidant state in human lymphocytes exposed *in vitro* to glyphosate, vinclazolin, atrazine and DPX-E9636. *Environ. Molec. Mutagenesis* **32**, 39-46.

Rank *et al* (1993). Genotoxicity testing of the herbicide Roundup and its active ingredient glyphosate isopropylamine using the mouse bone marrow micronucleus test, *Salmonella* mutagenicity test and *Allium* anaphase-telophase test. *Mutation Research* **300**, 29-30.

Bolognesi *et al* (1997). Genotoxic activity of glyphosate and its technical formulation Roundup. *J. Agric. Food Chem.* **45**, 1957-1962.

Kale *et al* (1995). Mutagenicity testing of nine herbicides and pesticides currently used in agriculture. *Environ. Molec. Mutagenesis* **25**, 148-153.

Vigfusson and Vyse (1980). The effect of the pesticides, Dexon, Captan and Roundup on sister chromatid exchange in human lymphocytes *in vitro*. *Mutation Research* **79**, 53-57.

Clements *et al* (1997). Genotoxicity of select herbicides in *Ranacates beiana* tadpoles using the alkaline single-cell gel DNA electrophoresis (COMET) assay. *Environ. Molec. Mutagenesis* **29**, 277-288.

Peluso *et al* (1998). ^{32}P-postlabelling detection of DNA adducts in mice treated with the herbicide Roundup. *Environ. Mol. Mutagenesis* **31**, 55-59.

Li and Long (1988). An evaluation of the genotoxic potential of glyphosate. *Fundamental and Applied Toxicology* **10**, 537-546.

Reports utilized in the assessment of the genotoxic activity of glyphosate and glyphosate formulations

1. BioAgri G.1.2-60, Micronucleus study with Glifos.
2. BioAgri G.1.1-050/96, Ames/Salmonella assay of Glifos.
3. Hoecht 92.0487, Bacterial mutagenicity assay of Dodigen 4022.
4. Hoechst 92.1024, Chromosome aberration assay of Dodigen 4022 in V79 cells.
5. ML-89-463, Mouse micronucleus assay of MON 0818
6. MSL-1538, Ames/Salmonella assay of MON 8080
7. MSL-10625, Ames/Salmonella assay with surfactant MON 0818.
8. MSL-11729, Ames/Salmonella assay with Roundup MON 2139.
9. MSL-11730, Ames/Salmonella assay of Rodeo.
10. MSL-11731, Ames/Salmonella assay of Direct of MON 14445.
11. MSL-11771, Mouse micronucleus test with Roundup.
12. MSL-11772, Mouse micronucleus study of Rodeo.
13. Notox 141918, Chromosome aberration study of Glyfosaat *in vitro* in human lymphocytes.
14. MSL-11773, Mouse micronucleus study of Direct.
15. RD 300 SRRSL1147, Dominant Lethal Study of glyphosate in mice.
16. Scantox, 12.9.91 Micronucleus test with glyphosate.
17. Scantox, 10.9.91-B, *In vitro* mammalian cell gene mutation test.

Figure 1

Profile of genotoxicity of Glyphosate

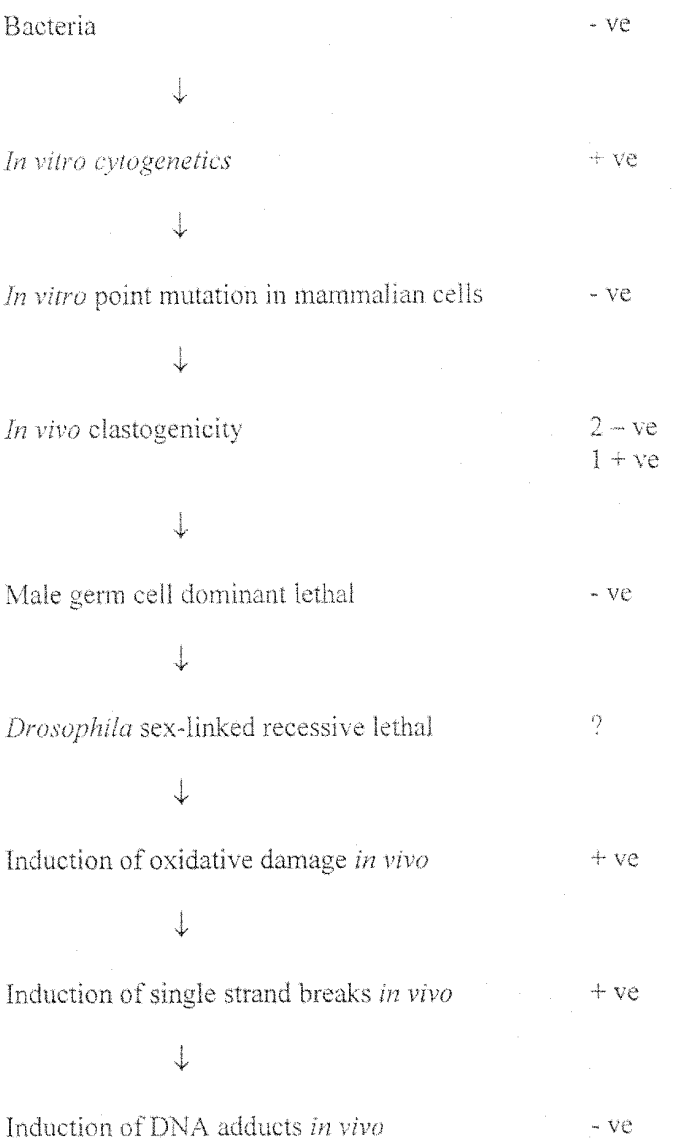

Figure 2

Profile of Genotoxicity of Glyphosate Mixtures

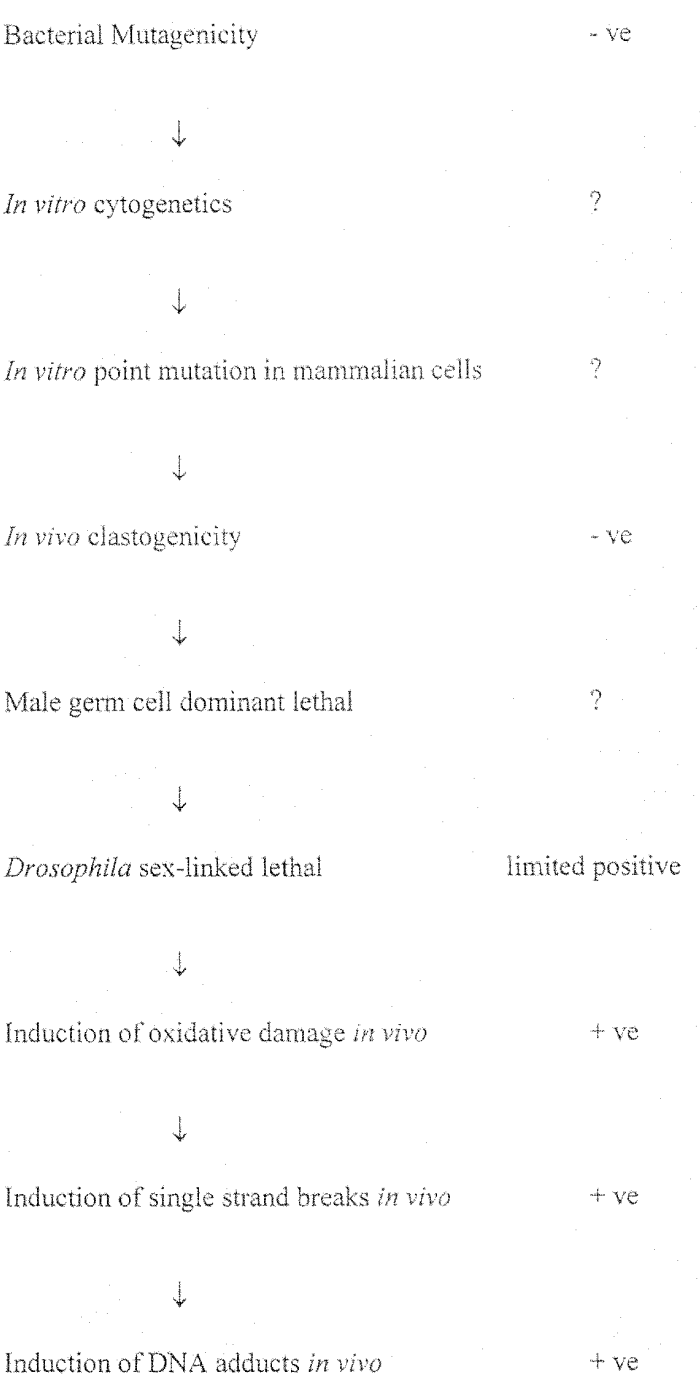

Table 1

Glyphosate

Endpoint	Effect	Cell type	Reference
Glyphosate (206-Jak-25-1) Point Mutation Induction in Ames test	Negative 310 to 2500µg/plate + S9 mix 160 to 2500µg/plate – S9 mix	Salmonella TA 98 TA 100 TA 1535 TA 1537	Scantox 10.9.91-A
Glyphosate Differential sensitivity rec assay	Negative 20 to 2000µg/test disc	*Bacillus subtilis*	Li and Long (1988)
Point mutation induction in Ames test	Negative 10 to 5000µg/plate + and – S9	**Salmonella** TA 98 TA 100 TA 1535 TA 1537 TA 1538 *E. coli* WP2 hcr	Li and Long (1988)

Table 2

Glyphosate Mixtures

Endpoint	Effect	Cell type	Reference
Roundup Point Mutation Induction in Ames Test	Positive minus S9 mix at 360µg/plate	TA 98	Rank *et al* 1993
	Positive in presence of S9 mix at 720µg/plate	TA 100	Rank *et al* 1993
	Note: Single point increases No evidence of dose response		
Direct Mixture (72%)			
Point mutation induction in Ames test	Negative 15 to 1500µg/plate + S9 5 to 500µg/plate –S9	TA 98 TA 100 TA 1535 TA 1537	MSL-11731
Roundup (31%) Point mutation induction in Ames test	Negative 15 to 1500µg/plate + S9 5 to 500µg/plate – S9	TA 98 TA 100 TA 1535 TA 1537	MSL-11729
Roundup Mixtures			
Rodeo (40%) Point Mutation in Ames test	Negative 50 to 5000µg/plate + and – S9 mix	TA 98 TA 100 TA 1535 TA 1537	MSL-11730
Glifos (41%) Point Mutation in Ames test	Negative 1 to 5000µg/plate + and – S9 mix	TA 97a TA 98 TA 100 TA 1535	BioAgri G.1.1-050/96

Table 3

Glyphosate

Endpoint	Effect	Cell type	Reference
Glyphosate-N-(phosphonomethyl) glycine Chromosome aberrations	Positive 5 to 51μM 72 hrs exposure in absence of S9 mix	Human lymphocytes	Lioi *et al* 1998(a)
Sister chromosome exchange	Positive 5 to 51μM 72 hrs exposure in absence of S9 mix	Human lymphocytes	Lioi *et al* 1998(a)
Chromosome aberrations	Positive 17 to 170μM 72 hrs exposure in absence of S9 mix	Bovine lymphocytes	Lioi *et al* 1998(b)
Sister chromosome exchange	Positive 17 to 170μM 72 hrs exposure in absence of S9 mix	Bovine lymphocytes	Lioi *et al* 1998(b)
Note: Lioi *et al* studies indicate data derived from 3 donors combined.			
Glyfosaat Chromosome aberrations	Negative 33 to 237μg/ml –S9 14hrs 56 to 333μg/ml –S9 48hrs 33 to 562μg/ml +S9 24hrs 100 to 562μg/ml +S9 48 hrs	Human lymphocytes	Notox 141918
Note: Reduction in mitotic index in absence of +S9 mix and at 24 hrs in presence of S9 mix.			
Glyphosate isopropylamine salt Cytogenetic changes	Negative	*Allium cepa* root tips	Rank *et al* (1993)

Table 4

Glyphosate Mixture

Endpoint	Effect	Cell type	Reference
Roundup Sister chromatid exchange	Positive at 100µg/ml 72 hrs exposures	Human lymphocytes	Bolognesi *et al* (1997)
Cytogenetic changes	Positive response at concentrations greater than 720µg/litre Characterised as spindle disturbance	*Allium cepa* root tip	Rank *et al* (1993)
Sister chromatid exchange	Small positive increase at 250 and 2500µg/ml	Human lymphocytes	Vigfusson and Vyse (1980)

Table 5

Glyphosate

Endpoint	Effect	Cell type	Reference
Glyphosate (206-Jak-25-1) Tk mutation induction in mammalian cells	Negative 0.65, 1.3, 2.5, 5.0mg/ml –S9 mix 0.52, 1.0, 2.1, 4.2mg/ml +S9 mix	Mouse lymphoma L5178Y	Scantox 10.9.91-B
Glyphosate HGPRT Mutation induction in mammalian cells	Negative 5 to 22.5mg/ml + and – S9 mix	Chinese hamster	Li and Long (1988)

Table 6

Glyphosate

Endpoint	Effect	Cell type	Reference
Glyphosate isopropylamine salt Micronucleus induction	Negative up to 200mg/kg by i.p. injection Note: only 1 dose point gave reduction in PCE/NCE ratio	Mouse bone marrow	Rank *et al* (1993)
Glyphosate (analar grade) Micronucleus induction	Positive response at 300mg/kg at 24hrs Multiple dosing i.p. injection 4 animals analysed Reduction in PCE/NCE ratio	Mouse bone marrow	Bolognesi *et al* (1997)
Glyphosate (206-Jak-25-1) Micronucleus induction	Negative 5000mg/kg at 24, 48, 72hrs No evidence of bone marrow toxicity	Mouse bone marrow	Scantox 12.9.91
Glyphosate Chromosomal aberrations	Negative 1gm/kg sampled at 6, 12, 24hrs	Rat bone marrow	Li and Long (1988)

Table 7

Glyphosate Mixtures

Endpoint	Effect	Cell type	Reference
Roundup (41%) Micronucleus induction	Negative up to 200mg/kg only sampled at 48hrs	Mouse bone marrow	Rank *et al* (1993)
Roundup Micronucleus induction	Positive response at 450mg/kg Multiple dose 3 animals sampled reduction in PCE/NCE ratio	Mouse bone marrow	Bolognesi *et al* (1997)
Glifos (41%) Micronucleus induction	Negative 68, 137, 206mg/kg i.p. delivered 2 x at 24hr intervals Note: Inadequate study	Mouse bone marrow	BioAgri G.1.2-60/96
Roundup 31% Micronucleus induction	Negative 140, 280, 555mg/kg i.p. injection sampled at 24, 48, 72hrs Note: Limited evidence of bone marrow toxicity One male 268 showed increase in micronuclei	Mouse bone marrow	MSL-11771
Direct (72%) Micronucleus induction	Negative 91, 183, 365mg/kg by i.p. sampled at 24, 48, 72hrs Note: Limited evidence of bone marrow toxicity one female 186 183mg/kg at 48hrs showed an increase	Mouse bone marrow	MSL-11773
Rodeo (40%) Micronucleus induction	Negative 850, 1700, 3400mg/kg by i.p. sampled at 24, 48, 72hrs	Mouse bone marrow	MSL-11772

Table 8

Miscellaneous Endpoints

Glyphosate, N- (phosphonomethyl)glycine

Endpoint	Effect	Cell type	Reference
G6PD activity	Increase in activities 5 to 51μM	Human lymphocytes	Lioi *et al* 1998(a)

Note, increase in G6PD activity reduced by presence of antioxidant N-acetyl cysteine, but not eliminated.

G6PD activity	Increase in activity 17 to 170μM	Bovine Lymphocytes	Lioi *et al* 1998(b)

Note, increase in G6PD activity reduced by presence of antioxidant N-acetyl cysteine, but not eliminated

Glyphosate (Analar Grade)

Induction of 8-OHdG	Increase in 8--OHdG in liver	Mice *In vivo*	Bolognesi *et al* (1997)
Induction of DNA damage measured by alkaline elution	Increase in single-strand breaks in liver and kidney at 4 hrs following 300mg/kg	Mice *In vivo*	Bolognesi *et al* (1997)

Glyphosate isopropylammonium salt.

Induction of DNA adducts measured by ^{32}P post-labelling	Negative no increase in adducts in liver and kidney at 130 and 270mg/kg	Mice *In vivo*	Peluso *et al* (1998)

Table 8 continued

Glyphosate

Hepatocyte DNA repair assay	Negative 12.5ng to 125µg/ml	Rat Hepatocytes	Li and Long (1988)

Note Very low concentrations used, study adds very little value to the analysis of the potential genotoxicity of Glyphosate.

Table 9

Miscellaneous Endpoints

Glyphosate Mixtures

Endpoint	Effect	Cell type	Reference
Roundup (41%) Mon 35050			
Induction of 8-0HdG	Increase in 8–0HdG in Liver and Kidney	Mice *In vivo*	Bolognesi *et al* (1997)
Induction of DNA damage measured by alkaline elution	increase in single-strand breaks in Liver and Kidney at 4hrs following 300mg/kg	Mice *In vivo*	Bolognesi *et al* (1997)
Induction of DNA adducts measured by ^{32}Ppost-labelling Note. Adducts were not characterised	increase in adducts in liver and kidney at 400, 500 and 600mg/kg	Mice *In vivo*	Peluso *et al* (1998)
Roundup			
COMET assay	Positive results observed at concentrations above 27mg/ litre	Tadpoles of *Rana catosbeiana*	Clements *et al* 1997

Table 10

Glyphosate

Endpoint	Effect	Cell type	Reference
Dominant Lethal Study	Negative Small reduction in viable foetuses in week 1 at 800mg/kg, week 3 at 2000mg/kg Increase in late reabsorptions at week 8 at 200mg/kg	Mouse male gametes exposed Effect measured in embryos	RD300 SRRS L1147

Table 11

Glyphosate Mixtures

Endpoint	Effect	Cell type	Reference
Roundup Sex linked recessive lethal mutations	Positive result in Spermatocyte broods At 1µg/ml.	*Drosophila melanogaster* Larval exposure	Kale *et al* (1995)
Pondmaster Sex linked recessive lethal mutations	Positive result in spermatocyte broods at 0.1µg/ml	*Drosophila melanogaster* larval exposure	Kale *et al* (1995)

Table 12

Surfactant

Endpoint	Effect	Cell type	Reference
Surfactant MON 0818			
Point Mutation induction in Ames test	Negatives 1 to 100µg/plate +S9 0.3 to 30µg/plate –S9	**Salmonella** TA 98 TA 100 TA 1535 TA 1537	MSL – 10625
Surfactant MON 8080			
Point Mutation induction in Ames test	Negatives 0.003 t0 3µl /plates + ad – S9 mix	**Salmonella** TA 98 TA 100 TA1535 TA 1537	MSL – 1538
Surfactant Dodigan 4022			
Point Mutation Induction in Ames test	Negatives 4 to 10,000 µg/plats in both presence and absence at S9 Mix	**Salmonella** TA 98 TA 100 TA 1535 TA 1537 TA 1538 *E. coli* WP2uvrA	Hoecht 92.0487

Table 13

Surfactant Dodigen 4022

Endpoint	Effect	Cell type	Reference
In vitro chromosome aberrations	Complex set of results – None significant Concentration range 600 to 6000µg/ml sampled at 7, 18 and 28hrs **Mitotic index minus S9** decreased at 7hrs increased at 18hrs decreased at 28hrs **Mitotic index plus S9** decreased at 7hrs increased at 18hrs no change at 28hrs **Polyploidy minus S9** decreased at 7hrs decreased at 18hrs increased at 28hrs **Polyploidy plus S9** decreased at 7hrs decreased at 18hrs increased at 28hrs **Aberrations minus S9** increased at 7hrs no change at 18hrs increased at 28hrs **Aberrations plus S9** increased at 7hrs no change at 18hrs increased at 28hrs	Chinese hamster V79	Hoecht 92.1024

Note: Experiments are difficult to interpret and should have been repeated.

Table 14

Surfactant MON 0818

Endpoint	Effect	Cell type	Reference
Micronucleus induction Note – limited experiment	Negatives 100mg/kg by I.p. sampled at 24 and 48 hrs No evidence of animal or bone marrow toxiety	Mouse Bone marrow	ML-89-463

Key Issues concerning the potential genotoxicity of glyphosate, glyphosate formulations and surfactants; recommendations for future work.

James M. Parry

Centre for Molecular Genetics and Toxicology
School of Biological Sciences
University of Wales Swansea
Swansea SA2 8PP, UK

Key Questions

1. Is glyphosate an *in vitro* clastogen? Can the positive studies of Lioi *et al* (1998a, 1998b) be reproduced?
2. Is glyphosate an *in vivo* clastogen? Can the positive studies of Bolognesi *et al* (1997) be reproduced?
3. If glyphosate is an *in vitro* and *in vivo* clastogen, what is its mechanism of action and does the mechanism lead to other types of genotoxic activity *in vivo* such as point mutation induction?
4. Does glyphosate produce oxidative damage?
5. Can we explain the reported genotoxic effects of glyphosate on the basis of the induction of oxidative damage?
6. If glyphosate is an *in vivo* genotoxin is its mechanism of action thresholded? Under what conditions of exposure are the antioxidant defences of the cell overwhelmed?
7. Are there differences in the genotoxic activities of glyphosate and glyphosate formulations?
8. Do any of the surfactants contribute to the reported genotoxicity of glyphosate formulations?

Deficiencies in the Data Set

1. No adequate *in vitro* clastogenicity data available for glyphosate formulations.

2. No bone marrow micronucleus study of glyphosate available using multiple dosing and adequate animal numbers.

3. No studies available demonstrating the effects of anti-oxidants upon the induction of genotoxic endpoints by glyphosate.

4. No adequate *in vitro* or *in vivo* clastogenicity data for surfactants used in glyphosate formulations.

Actions Recommended

a) Provide comprehensive *in vitro* cytogenetic data on glyphosate formulations.

b) On the assumption that the reported *in vitro* positive clastogenic data for glyphosate is due to oxidative damage determine the influence of antioxidants. Evaluate the clastogenic activity of glyphosate in the presence and absence of a variety of antioxidant activities. Such a study should also incorporate glyphosate formulations to clarify the validity of reports of differences in activity. I recommend that both a) and b) should be undertaken using the *in vitro* micronucleus assay in human lymphocytes. The *in vitro* micronucleus assay would provide a more cost-effective method for evaluating a large number of experimental variables.

c) Evaluate the induction of oxidative damage *in vivo* and determine the influence of the antioxidant status of the animals. Determine the exposure concentrations of glyphosate which overwhelm the antioxidant status of tissues.

d) Perform an *in vivo* bone marrow micronucleus assay with multiple dosing with adequate numbers of animals to determine whether the work of Bolognesi *et al* (1997) can be reproduced.

e) I am making no recommendation to repeat any of the sister chromatid exchange studies. Chromosomal aberration data will always take priority over SCE data so I

see no point in repeating SCE studies as they involve an endpoint which is poorly defined and doesn't lead to genetic changes.

f) In view of the increasing appreciation of the value of the COMET assay as marker of tissue-specific damage I recommend the consideration of its use in any *in vivo* studies performed. The COMET assay would provide the ability to determine whether damage is produced in a wide range of tissues following glyphosate exposure. Such studies would also indicate whether the COMET positive results for glyphosate formulations in tadpoles (Clements *et al* 1997) are reproduced in mammals. In view of the data on oxidative damage (Bolognesi *et al* 1997) I would recommend COMET assays in the liver and kidney of mice if the oxidative data are confirmed as indicated under c).

g) I do not recommend any transgenic point mutation assay at this time. There is no available evidence that glyphosate is a point mutagen and the relatively low sensitivity of the transgenic assay means that negative results would have little value in the assessment of the hazard and risk of glyphosate exposures.

h) I do not recommend any studies of DNA adduct induction at this time. Such a study would only be of value if the adducts formed were characterised which would require major efforts. If the adducts reported by Peluso *et al* (1998) are the result of oxidative damage they are likely to be of the same type as those produced in the absence of glyphosate exposure by background oxidative damage.

i) Provide comprehensive *in vitro* data on the surfactants.

My overall view is that if the reported genotoxicity of glyphosate and glyphosate formulations can be shown to be due to the production of oxidative damage then a case could be made that any genetic damage would be thresholded. Such genetic damage would only be biologically relevant under conditions of compromised antioxidant status. If such an

oxidative damage mechanism is proved then it may be necessary to consider the possibility of susceptible groups within the human population.

If the genotoxic activity of glyphosate and its formulations is confirmed it would be advisable to determine whether there are exposed individuals and groups within the human population. If such individuals can be identified then the extent of exposure should be determined and their lymphocytes analysed for the presence of chromosome aberrations. In such populations micronucleus studies would probably only be of value in aspleenic individuals.

Comments on Parry Evaluation of
Glyphosate and Glyphosate Formulation Potential Genotoxicity.
Larry Kier
September 18, 1999

There is no summary evaluation in the initial section and no overall conclusions are presented on the genotoxicity of glyphosate or glyphosate formulations.

Although the summary says most studies (i.e. unpublished reports) were conducted according to OECD guidelines, this is clearly not the case for several published studies cited but this is not mentioned in the evaluation.

The depth of analysis of the studies is rather superficial. The analysis of the unpublished reports appears to be much more thorough than analysis of the published reports.

Ames tests--There are numerous published and unpublished negative Ames studies with glyphosate that contradict the reported positive findings of Rank et al. The evaluation doesn't go into any depth on the quality of the Rank et al. data in comparison with the other reports. (e.g., reproducibility or testing at equivalent doses).

Micronucleus--There is no analysis of the possible significance of differences in protocol between Bolognesi et al. and the other negative studies. In particular, what are the implications of multiple dosing (actually 2 doses) compared with a single dose. How many instances of clear positive/negative differences exist for these two protocols?

There is no conclusion about what the data say about glyphosate. The published studies are presented as some evidence of genotoxicity and the reports are presented as giving no evidence.

There is mixing of glyphosate and formulations in the analysis.

What's the significance of one animal showing an increase in micronuclei noted for micronucleus studies of Roundup and Direct? Apparently the conclusion is that these studies are negative, but if that is the case why mention single animal results. Are these considered significant?

There appears to be no evaluation of the significance of different endpoints--e.g. comet in tadpoles, oxidative damage, in vivo vs. in vitro, etc. These are all apparently considered as equivalent in this evaluation.

It's not clear how these data and reports lead to a concern about stability of glyphosate formulations.

WRATTEN, STEPHEN J [FND/1000]

To: MARTENS, MARK A [FND/5045]; FARMER, DONNA R [FND/1000]
Cc: KIER, LARRY D [NCP/1000]; HEYDENS, WILLIAM F [FND/1000]; GRAHAM, WILLIAM [FND/5040]
Subject: Comments on Parry write-up

Mark and Donna

I was somewhat disappointed in the Parry report, not particularly from his conclusions but just the way they're presented. The style and rather casual lack of completeness and preciseness would make it hard to circulate this around to anyone as supporting information. Has he ever worked with industry before on this sort of project?

I will mail the marked-up paper back to you, but some other general comments need to be made:

1. It is odd that the one study by BioAgri is discussed right on the first page in rather extensive detail but none of the others are. I understand that he didn't like this one, but it is still strange to read this way.

2. The whole report could benefit from a couple of introductory paragraphs about what he was asked to do and what he received as far as reports. Did he have all the Monsanto reports as well as the literature articles? Was he asked to compare these, evaluate the methods, explain the differences, identify any faults, or what?

3. Some where the report needs to identify the full citations of each report evaluated and give the full Literature references for the public documents. Also the test material should be clearly identified, ideally by both MON number and brand name if needed, but at least to say which are glyphosate and which are formulations - this is done, sort of, but not as clearly as I'd like. Separate tables would be good.

4. He has an odd way of starting all conclusions with a negative - ie., points 2, 3, and 4 on page 3. Couldn't the sentence structure be modified to be less awkward? When he says "no data were provided..." time and again, it makes it sound as though he was suspicious that there were data but he didn't get them. I know this is not the intent, but it could be cleaned up.

5. Table 1 seems to state repeatedly that "there was no evidence of xxx mutagenicity". It would be more powerful if it said "there was convincing evidence that glyphosate does not act as a xxx mutagen". "no evidence of" is a very weak way of stating a conclusion.

6. He says very little about the literature reports. So little that one almost forgets about them. Can he not provide some critique about their quality and methodology as compared to the Monsanto reports? Are they included in or excluded from the statement in the first paragraph sentence "these studies were performed to a high standard and to OECD recommended guidelines"? In the section entitled "Assessment of the published..." on p. 2, I am hard-pressed to find any assessment. It is almost merely a listing of what everyone already knew from casually reading the abstract.

7. In his conclusions (p. 2), do the "studies evaluated" (line 2) include the literature reports or not? IN other words, is he saying that none of the studies (Monsanto plus literature) had evidence of glyphsoate genotoxic potential, or is he limiting this conclusion to the Monsanto studies?

8. Of course we know there were no data of the type listed in points 2, 3, and 4 on p. 3. We didn't need him to tell us that. The key point is whether the conclusions of Bolognesi, and Rank can be discounted on the basis of the strength and number of studies at hand, or whether their experiments need to be repeated independently to credibly refute the findings. Of course we knew that the latter would be the most convincing approach, but we need him to make any arguments that can be made on the data we have.

Overall, I guess we have his recommendation of studies that could be used to strengthen the database on p. 4. , but that is about it. I do not see that he has stuck his neck out on anything at all controversial, and therefore, there is little value in the write-up as written that could be useful. Hope it didn't cost much. Perhaps this is too harsh, and I don't know what your proposal to him was, but I guess I would expect more than this of a Professor.

Steve

EXHIBIT C

Message

From: HEYDENS, WILLIAM F [FND/1000] [/O=MONSANTO/OU=NA-1000-01/CN=RECIPIENTS/CN=230737]
Sent: 9/16/1999 6:18:36 PM
To: [redacted] [FND/5045] [/O=MONSANTO/OU=EA-5040-01/CN=RECIPIENTS/CN=21606]; 'KIER, LARRY D [NCP/1000]' [/O=MONSANTO/OU=GLB-STL/CN=LEGACY ADDRESSES/CN=33322]; 'FARMER, DONNA R [FND/1000]' [/O=MONSANTO/OU=GLB-STL/CN=LEGACY ADDRESSES/CN=180070]
CC: 'HEYDENS, WILLIAM F [FND/1000]' [/O=MONSANTO/OU=GLB-STL/CN=LEGACY ADDRESSES/CN=230737]
Subject: RE: Parry report

[redacted] All,

I have read the report and agree with the comments - there are various things that can be done to improve the report.

However, let's step back and look at what we are really trying to achieve here. We want to find/develop someone who is comfortable with the genetox profile of glyphosate/Roundup and who can be influential with regulators and Scientific Outreach operations when genetox. issues arise. My read is that Parry is not currently such a person, and it would take quite some time and $$$/studies to get him there. We simply aren't going to do the studies Parry suggests. [redacted] do you think Parry can become a strong advocate without doing this work Parry? If not, we should ***seriously*** start looking for one or more other individuals to work with. Even if we think we can eventually bring Parry around closer to where we need him, we should be currently looking for a second/back-up genetox. supporter. We have not made much progress and are currently very vulnerable in this area. We have time to fix that, but only if we make this a high priority now.

Bill

-----Original Message-----
From: [redacted] [FND/5045]
Sent: Thursday, September 16, 1999 2:02 AM
To: KIER, LARRY D [NCP/1000]; FARMER, DONNA R [FND/1000]
Cc: HEYDENS, WILLIAM F [FND/1000]
Subject: Parry report
Importance: High

Larry and Donna,

I would like to get some feedback to Jim Parry on his report. I sent you my comments but didn't get a reaction. Can I get your opinions and then have a discussion on the action to take?

Regards, [redacted]

EXHIBIT D

UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF CALIFORNIA

IN RE: ROUNDUP PRODUCTS LIABILITY LITIGATION	)	MDL No. 2741
THIS DOCUMENT RELATES TO ALL CASES	)	Case No. 16-md-02741-VC

WEDNESDAY, JANUARY 11, 2017

CONFIDENTIAL - SUBJECT TO PROTECTIVE ORDER

- - -

Videotaped deposition of Donna Farmer, Ph.D., Volume I, held at the offices of HUSCH BLACKWELL, L.L.C., 190 Carondelet Plaza, Suite 600, St. Louis, Missouri, commencing at 9:04 a.m., on the above date, before Carrie A. Campbell, Registered Diplomate Reporter, Certified Realtime Reporter, Illinois, California & Texas Certified Shorthand Reporter, Missouri & Kansas Certified Court Reporter.

- - -

GOLKOW TECHNOLOGIES, INC.
877.370.3377 ph | 917.591.5672 fax
deps@golkow.com

it?

MR. JOHNSTON: Same objection.

THE WITNESS: Again, that was the same study where they injected the formulated product directly into the abdomens of the animals. There was direct damage to the organs and to the animal, and the results are secondary to cytotoxicity.

QUESTIONS BY MR. MILLER:

Q. He tells us on -- he tells Monsanto in this report at 4266 -- I'm just about done with this report.

But at 4266, Dr. Parry tells us that there is -- this is in F. "In view of the increasing appreciation of the value of COMET assay as a marker of tissue-specific damage, I recommend the consideration of its use in any in vivo studies performed."

Do you see that?

MR. JOHNSTON: Objection. Foundation.

THE WITNESS: I see that's what he says.

QUESTIONS BY MR. MILLER:

Q. And Monsanto never performed a COMET assay on any of its in vivo studies?

A. We have a difference of opinion of the value of the COMET study. There are other studies that are -- the COMET study, you can actually get positive effects if you take blood from people who have been on a treadmill for 30 minutes. So, again, you have to look at the study and what it provides.

And this, again, comes back to talking about the oxidative damage with Bolognesi. And again, remember, he is talking about doing an assay where -- in talking about looking at the liver and the kidneys where we actually went and did the studies in the whole animals that we shared with you about the Heydens report.

Q. The answer is Monsanto never did COMET assays, true?

A. No, we would not do COMET assays. We do not see it as a really valuable assay.

Q. And this expert who you asked

EXHIBIT E

SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF SAN FRANCISCO

DEWAYNE JOHNSON,
Plaintiff,
v.
MONSANTO COMPANY, et al.,
Defendants.

No. CGC-16-550128

Judge: Hon. Curtis E.A. Karnow

Dept. 304

C O N F I D E N T I A L

DEPOSITION OF JOHN R. FOWLE, III, Ph.D.

Washington, D.C.

February 22, 2018

GOLKOW LITIGATION SERVICES

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or report?

MR. COPLE: Objection, asked and answered.

A. It was not a published article or report, but EPA had access to these documents, had experts who could evaluate that. And so, on that basis, basically, is what I made my -- my -- my statement, my conclusion.

Q. I'm not finding -- if it's not a publication, how could it fall under the number 3 that refers only to published articles?

A. You're right.

MR. COPLE: Objection, asked and answered.

A. You're right. You're right. I don't know whether it would fall under that. I really don't.

Q. Okay.

A. But -- yeah. Okay. But -- yeah.

Q. Okay. So you don't know if it needed to be -- you can't say that it didn't need to be provided to the EPA, fair?

A. I can't say for -- for -- for certain whether it needed to be or not --

Q. And you --

A. -- but I can say that what's contained in that report was available to EPA.

Q. Dr. Parry, a leading mutagenicity expert's

not a toxicological --

A. No.

Q. Okay. So do you agree that this provision does not exempt Monsanto from an obligation to provide the TNO study to the EPA?

MR. COPLE: Objection, asked and answered.

A. That's my reading of that, yes.

Q. All right. You can put that exhibit down.

Does the EPA or has it at any time in your review of the record prevented Monsanto from performing testing above and beyond the minimum testing requirements of the EPA?

MR. COPLE: Objection, lacks foundation.

A. To my knowledge, no.

Q. Okay. Has the EPA prevented Monsanto in any way from performing any of the studies that Dr. Parry suggested Monsanto perform?

MR. COPLE: Objection, asked and answered.

A. No. No, to my knowledge, no.

Q. A Monsanto -- well, would you agree with me that Monsanto has not conducted subchronic, chronic or teratogenicity studies with their formulated products?

MR. COPLE: Objection, lacks foundation.

A. I believe that EPA waived that requirement

EXHIBIT F

Message

From: HEYDENS, WILLIAM F [redacted] [/O=MONSANTO/OU=NA-1000-01/CN=RECIPIENTS/CN=230737]
Sent: 4/2/2002 12:45:18 PM
To: HEALY, CHARLES E [redacted] [/O=MONSANTO/OU=NA-1000-01/CN=RECIPIENTS/CN=297008]
Subject: RE: TNO dermal penetration studies: new issues and topics for the conf call of Tuesday, 2 April (8 A.M STL time)

Chuck,

Thanks. I would like to sit in but will probably not do so due to time considerations. My primary concern is with the glyphosate in terms of the potential for this work to blow Roundup risk evaluations (getting a much higher dermal penetration than we've ever seen before.

Bill

-----Original Message-----
From: HEALY, CHARLES E [redacted]
Sent: Monday, April 01, 2002 3:42 PM
To: HEYDENS, WILLIAM F [redacted]
Subject: FW: TNO dermal penetration studies: new issues and topics for the conf call of Tuesday, 2 April (8 A.M STL time)

Bill,

As a follow-up to [redacted] message below, we are having a conference call with Brussels Tuesday morning (Apr 2) at 8 a.m. in A2215N. I don't know if you want to sit in or even have time to or just would like an update afterwards. In any case, just wanted to make you aware of the call. Joel, Donna, Abby, [redacted] Ruth, Steve, and I will all be on the call with [redacted] and [redacted]

Chuck

-----Original Message-----
From: [redacted]
Sent: Friday, March 29, 2002 9:29 AM
To: HEALY, CHARLES E [redacted]
Subject: RE: TNO dermal penetration studies: new issues and topics for the conf call of Tuesday, 2 April (8 A.M STL time)

Chuck,

I will send you a copy of [redacted] fax. Could you circulate it to Abby, Joel,Donna and Steve Wratten please. Did Cam find us a room for Tuesday (8:10:30)?

Thanks,
[redacted]

-----Original Message-----
From: [redacted]
Sent: Friday, March 29, 2002 7:58 AM
To: [redacted]; LI, ABBY A [redacted]; HEALY, CHARLES E [redacted]; [redacted]; KRONENBERG, JOEL M [redacted]
Cc: HEYDENS, WILLIAM F [redacted]; FARMER, DONNA R [redacted]; MCKENNA, RUTH M [redacted]
Subject: TNO dermal penetration studies: new issues and topics for the conf call of Tuesday, 2 April (8 A.M STL time)
Importance: High

Dear all,

As today we received preliminary surprising results on *in vitro* dermal penetration of propachlor and glyphosate through rat skin, it is imperative that we work closely together and communicate well on the conduct, the practical difficulties and the results associated with these studies. [redacted] could you please circulate the fax I will send to you this morning ?

Please find herewith a summary of issues (with data) we have to discuss during our conf call of next Tuesday:

Propachlor:
- Our attempt to demonstrate that the dermal penetration of propachlor through human skin is lower than with rat

skin failed. Indeed,
* concentrate formulation: % penetration with human skin = % penetration with *rat skin*
* spray dilution: % penetration *human skin* > % dermal penetration *rat skin* ($p<0.05$) (see attached table)
- Microautoradiographies clearly show stores of propachlor in the epidermis of human skin

Triallate:
- The UK's PSD wants to take into consideration the stores of triallate in skin tissues for the derivation of the dermal penetration factor (% dermal penetration + stores = 0.22% + 26.9% = 27.1%) (see attached table)
- Our attempt to show that triallate is stored in the stratum corneum by micro-autoradiography failed due to experimental problems

Glyphosate:
- The EU rapporteur for glyphosate used a dermal penetration factor of 3% based on several published *in vitro/in vivo* dermal penetration studies
- We launched human and rat *in vitro* dermal penetration studies with MON 35012 with and without surfactant
- Preliminary results with rat skin are not acceptable (see fax); due to very bad reproductibility that TNO cannot explain, they proposed to repeat the study in parallel with the human skin study. However, we can already conclude that:
* for the concentrate MON 35012, the % *in vitro* dermal penetration of glyphosate through rat skin is between 5 and 10%
* for the spray dilution of MON 35012, the % *in vitro* dermal penetration of glyphosate through rat skin will be around 2%
* The dermal penetration of glyphosate itself in the absence of surfactant is lower than 1.5%.

In the light of these results, should we continue to place *in vitro* dermal penetration studies at TNO ?
Propachlor: can we use rat dermal penetration results for the risk assessment ?
Triallate:
- pharmacokinetics modelling
- tape-stripping
Glyphosate:
- Reproductibility by repeating the study ?
- Results with human skin ?

<< File: TNO_Dermal_Penetration.doc >>

Regards, [redacted]