Attachment 3: Forty six studies by overseas scholars found that glyphosate or glyphosate formulated herbicides cause cell toxicity, DNA damage, teratogenic, mutagenic, and reproductive toxicity, along with miscarriage!

**Summary of Main Points**

1) **Kale, P.G. et al, (1995):** Roundup and others total nine herbicides and pesticides were tested for their mutagenicity using the Drosophila sex-linked recessive lethal mutation assay. These are Ambush, Treflan, Blazer, Roundup, 2,4-D Amine, Crossbow, Galecron, Pramitol, and Pondmaster. All chemicals induced significant numbers of mutations in at least one of the cell types tested.

2) **Yousef MI et al. (1995):** Two sublethal doses of Carbofuran (carbamate insecticide) and Glyphosate (organophosphorus herbicide) treatment resulted in a decline in body weight, libido, ejaculate volume, sperm concentration, semen initial fructose and semen osmolality. This was accompanied with increases in the abnormal and dead sperm and semen methylene blue reduction time. The hazardous effect of these pesticides on semen quality continued during the recovery period, and was dose-dependent. These effects on sperm quality may be due to the direct cytotoxic effects of these pesticides on spermatogenesis and/or indirectly via hypothalam-pituitary-testis axis which control the reproductive efficiency.

3) **Savitz, D.A. et al (1997):** A variety of chemicals (atrazine, glyphosate, organophosphates, 4-[2,4-dichlorophenoxy] butyric acid, and insecticides)... Based on these data, despite limitations in exposure assessment, the authors encourage continued evaluation of male exposures, particularly in relation to miscarriage and preterm delivery.
4) Claudia Bolognesi et al. (1997): In this study, the formulated commercial product, Roundup, and its active agent, glyphosate, were tested in the same battery of assays for the induction of DNA damage and chromosomal effects in vivo and in vitro. ... A DNA-damaging activity as DNA single-strand breaks and 8-OHdG and a significant increase in chromosomal alterations were observed with both substances in vivo and in vitro. A weak increment of the genotoxic activity was evident using the technical formulation.

5) Lioi, M.B. et al. (1998): The pesticides glyphosate, vinclozolin, and atrazine have been studied ... In our experimental conditions, each chemical compound tested produced a dose-related increase in the percent of aberrant cells and an increase of SCE/cell.

6) Peluso M. (1968): Roundup is able to induce a dose-dependent formation of DNA adducts in the kidneys and liver of mice. The levels of Roundup-related DNA adducts observed in mouse kidneys and liver at the highest dose of herbicide tested (600 mg/kg) were 3.0 +/- 0.1 (SE) and 1.7 +/- 0.1 (SE) adducts/10(8) nucleotides, respectively. The Roundup DNA adducts were not related to the active ingredient, the isopropylammonium salt of glyphosate, but to another, unknown component of the herbicide mixture. Additional experiments are needed to identify the chemical specie(s) of Roundup mixture involved in DNA adduct formation.

7) Peggy J. Perkins at el. (2000): Comparative toxicity of Roundup and its surfactant polyoxyethyleneamine (POEA) to *Xenopus laevis*, and comparing Roundup formulation and the Rodeo formulation (with no surfactant POEA), using Frog Embryo Teratogenesis Assay—*Xenopus* (FETAX). A comparison of LC50 concentrations
indicated that the Roundup formulation of glyphosate was 700 times as toxic as the Rodeo formulation. Even though a limited number of tests were performed to evaluate the effects of the surfactant POEA on *X. laevis*, each test showed a lower LC50 value for POEA alone than for either Roundup or Rodeo. More studies are needed, but it seems likely that the surfactant itself is responsible for the greater toxicity displayed by the Roundup formulation of glyphosate. It seems less likely that the greater toxicity is due to enhanced uptake of glyphosate by the embryos.

8) Walsh, L.P. et al. (2000): In conclusion, Roundup disrupted steroidogenesis in Leydig cells through a post-transcriptional reduction in StAR protein expression. The use of StAR as an end point in studies concerning endocrine disruption merits further consideration. Although Roundup decreased steroidogenesis, the active ingredient of this herbicide, glyphosate, did not alter steroid production, indicating that at least one other component of the formulation is required to disrupt steroidogenesis. Because the formulation of Roundup is proprietary, further studies are needed to identify the components in Roundup and their ability to disrupt steroidogenesis.

9) Daruich, J. et al. (2001): We determined the effects of these compounds on the levels and activities of the P450scc enzyme (which converts cholesterol to pregnenolone) and the 3beta-hydroxysteroid dehydrogenase (3beta-HSD) enzyme (which converts pregnenolone to progesterone). Of the pesticides screened, only the pesticide Roundup inhibited dibutyryl [(Bu)(2)cAMP-stimulated progesterone production in MA-10 cells without causing cellular toxicity. Roundup inhibited steroidogenesis by disrupting StAR protein expression, further demonstrating the susceptibility of StAR to environmental pollutants.

10) T E Arbuckle et al. (2001): For late abortions, preconception
exposure to glyphosate (OR = 1.7; 95% CI, 1.0-2.9), ... was associated with elevated risks. Postconception exposures were generally associated with late spontaneous abortions.

11) Marc J et al. (2002): Roundup delayed the activation of CDK1/cyclin B in vivo. Roundup inhibited also the global protein synthetic rate without preventing the accumulation of cyclin B. In summary, Roundup affects cell cycle regulation by delaying activation of the CDK1/cyclin B complex, by synergic effect of glyphosate and formulation products. Considering the universality among species of the CDK1/cyclin B regulator, our results question the safety of glyphosate and Roundup on human health.

12) Dallegrave, E. et al. (2003): The aim of this study was to assess the teratogenicity of the herbicide glyphosate-Roundup (as commercialized in Brazil) to Wistar rats. Dams were treated orally with water or 500, 750 or 1000 mg/kg glyphosate from day 6 to 15 of pregnancy. Results showed a 50%, mortality rate for dams treated with 1000 mg/kg glyphosate. Skeletal alterations were observed in 15.4, 33.1, 42.0 and 57.3% of fetuses from the control, 500, 750 and 1000 mg/kg glyphosate groups, respectively. We may conclude that glyphosate-Roundup is toxic to the dams and induces developmental retardation of the fetal skeleton.

13) Lajmanovich RC et al. (2003): Larval maldevelopment (craniofacial and mouth deformities, eye abnormalities and bent curved tails) occurred in all tests and increased with time and GLY-F concentration.

14) Cox, C. (2004): Glyphosate has been shown to have carcinogenic effects. Three recent studies found a link between
glyphosate exposure and non-Hodgkin's lymphoma (a type of cancer).

15) Marc, J. et al. (2004): Several glyphosate-based pesticides from different manufacturers were assayed in comparison with Roundup 3plus for their ability to interfere with the cell cycle regulation. All the tested products, Amega, Cargly, Cosmic, and Roundup Biovert induced cell cycle dysfunction.

16) Marc, J et al. (2004): We conclude that formulated glyphosate's effect on the cell cycle is exerted at the level of the DNA-response checkpoint of S phase. The resulting inhibition of CDK1/cyclin B Tyr 15 dephosphorylation leads to prevention of the G2/M transition and cell cycle progression.

17) Benedettia, A.L. et al. (2004): The object of this study was to analyze the hepatic effects of the herbicide Glyphosate-Biocarb (as commercialized in Brazil) in Wistar rats. ... We may conclude that Glyphosate-Biocarb may induce hepatic histological changes as well as AST and ALT leaking from liver to serum in experimental models.

18) John F Acquavella et al. (2004): This study by Monsanto reported: Sixty percent of farmers had detectable levels of glyphosate in their urine on the day of application. The geometric mean (GM) concentration was 3 ppb, the maximum value was 233 ppb, and the highest estimated systemic dose was 0.004 mg/kg. For spouses, 4% had detectable levels in their urine on the day of application. Their maximum value was 3 ppb. For children, 12% had detectable glyphosate in their urine on the day of application, with a maximum concentration of 29 ppb.

19) Marc, J. et al. (2005): The surfactant polyoxyethylene amine
(POEA), the major component of commercial Roundup, was found to be highly toxic to the embryos when tested alone and therefore could contribute to the inhibition of hatching.

20) Lajmanovich, R.C. et al. (2005): Larval maldevelopment (craniofacial and mouth deformities, eye abnormalities and bent curved tails) occurred in all tests and increased with time and GLY-F concentration. ... Malformation were minimal at 3.07 mg/L exposed for one day, whereas greater that 90% were malformed at a GLY-F level of 7.5 mg/L. The current test confirmed the malformation effects of GLY-F on tadpoles.

21) Beuret CJ et al. (2005): The present study has investigated the effects that 1% glyphosate oral exposure has on lipoperoxidation and antioxidant enzyme systems in the maternal serum and liver of pregnant rats and their term fetuses at 21 days of gestation. The results suggest that excessive lipid peroxidation induced with glyphosate ingestion leads to an overload of maternal and fetal antioxidant defense systems.

22) Richard S et al. (2005): We show that glyphosate is toxic to human placental JEG3 cells within 18 hr with concentrations lower than those found with agricultural use, and this effect increases with concentration and time or in the presence of Roundup adjuvants. Surprisingly, Roundup is always more toxic than its active ingredient. The glyphosate-based herbicide disrupts aromatase activity and mRNA levels and interacts with the active site of the purified enzyme, but the effects of glyphosate are facilitated by the Roundup formulation in microsomes or in cell culture. We conclude that endocrine and toxic effects of Roundup, not just glyphosate, can be observed in mammals. We suggest that the presence of Roundup adjuvants enhances glyphosate bioavailability and/or bioaccumulation.
23) Sparling DW et al. (2006): Hatching success at the highest concentration was significantly lower (73%) than in other treatments (80-100%). ... Genetic damage, as measured by flow cytometry, increased with treatment concentration except for the highest dose. ... There also is a risk that the health of turtle embryos may be affected in ways not measured in the present study.

24) Oliveira AG et al. (2007): The exposure to the herbicide resulted in alterations in the structure of the testis and epididymal region as well as in the serum levels of testosterone and estradiol, with changes in the expression of androgen receptors restricted to the testis. The harmful effects were more conspicuous in the proximal efferent ductules and epididymal ducts, suggesting higher sensitivity of these segments among the male genital organs. The effects were mostly dose dependent, indicating that this herbicide may cause disorder in the morphophysiology of the male genital system of animals.

25) César Paz-y-Miño et al. (2007): We analyzed the consequences of aerial spraying with glyphosate added to a surfactant solution in the northern part of Ecuador. ... The results showed a higher degree of DNA damage in the exposed group (comet length = 35.5 µm) compared to the control group (comet length = 25.94 µm). These results suggest that in the formulation used during aerial spraying glyphosate had a genotoxic effect on the exposed individuals.

26) Bellé R et al. (2007): New insights in cancer biology lead to two fundamental concepts about the very first origin of cancerogenesis. Cancers result from dysfunction of DNA-damaged checkpoints and cancers appear as a result of normal stem cell (NCS) transformation into a cancer stem cell (CSC). The second aspect suggests a new definition of
"cancer", since CSC can be detected well before any clinical evidence. Since early development starts from the zygote, which is a primary stem cell, sea urchin early development allows analysis of the early steps of the cancerization process. In the field of toxicology and incidence on human health, the sea urchin experimental model allows assessment of cancer risk from single or combined molecules long before any epidemiologic evidence is available. Sea urchin embryos were used to test the worldwide used pesticide Roundup that contains glyphosate as the active herbicide agent; it was shown to activate the DNA-damage checkpoint of the first cell cycle of development.

27) Dallegrave E et al. (2007): The results showed that glyphosate-Roundup did not induce maternal toxicity but induced adverse reproductive effects on male offspring rats: a decrease in sperm number per epididymis tail and in daily sperm production during adulthood, an increase in the percentage of abnormal sperms and a dose-related decrease in the serum testosterone level at puberty, and signs of individual spermatid degeneration during both periods. There was only a vaginal canal-opening delay in the exposed female offspring. These findings suggest that in utero and lactational exposure to glyphosate-Roundup may induce significant adverse effects on the reproductive system of male Wistar rats at puberty and during adulthood.

28) McComb et al. (2007): In in vitro tests found that glyphosate acts in the mitochondria of the rat liver cells as an oxidative phosphorylation decoupling agent.

29) Soso AB et al. (2007): The results indicate that the presence of glyphosate in water was deleterious to Rhamdia quelen reproduction, altering steroid profiles and egg viability.
30) Hokanson R et al. (2007): Among the chemicals most commonly used, both commercially and in the home, is the herbicide glyphosate. Although glyphosate is commonly considered to be relatively non-toxic, we utilized in vitro DNA microarray analysis of this chemical to evaluate its capacity to alter the expression of a variety of genes in human cells. We selected a group of genes, determined by DNA microarray analysis to be dysregulated, and used quantitative real-time PCR to corroborate their altered states of expression. We discussed the reported function of those genes, with emphasis on altered physiological states that are capable of initiating adverse health effects that might be anticipated if gene expression were significantly altered in either adults or embryos exposed in utero.

31) Mañas F et al. (2009): AMPA is the major environmental breakdown product of glyphosate. The purpose of this study is to evaluate the in vitro genotoxicity of AMPA using the Comet assay in Hep-2 cells after 4h of incubation and the chromosome aberration (CA) test in human lymphocytes after 48h of exposition. Potential in vivo genotoxicity was evaluated through the micronucleus test in mice. In the Comet assay, the level of DNA damage in exposed cells at 2.5-7.5mM showed a significant increase compared with the control group. In human lymphocytes we found statistically significant clastogenic effect AMPA at 1.8mM compared with the control group. In vivo, the micronucleus test rendered significant statistical increases at 200-400mg/kg. AMPA was genotoxic in the three performed tests.

32) Nora Benachour and Gilles-Eric Séralini (2009): We have evaluated the toxicity of four glyphosate (G)-based herbicides in Roundup (R) formulations, from $10^5$ times dilutions, on three different human cell types. This dilution level is far below agricultural recommendations and corresponds to low levels of residues in food or
feed. The formulations have been compared to G alone and with its main metabolite AMPA or with one known adjuvant of R formulations, POEA. HUVEC primary neonate umbilical cord vein cells have been tested with 293 embryonic kidney and JEG3 placental cell lines. All R formulations cause total cell death within 24 h, through an inhibition of the mitochondrial succinate dehydrogenase activity, and necrosis, by release of cytosolic adenylate kinase measuring membrane damage. They also induce apoptosis via activation of enzymatic caspases 3/7 activity. This is confirmed by characteristic DNA fragmentation, nuclear shrinkage (pyknosis), and nuclear fragmentation (karyorrhexis), which is demonstrated by DAPI in apoptotic round cells. G provokes only apoptosis, and HUVEC are 100 times more sensitive overall at this level. The deleterious effects are not proportional to G concentrations but rather depend on the nature of the adjuvants. AMPA and POEA separately and synergistically damage cell membranes like R but at different concentrations. Their mixtures are generally even more harmful with G. In conclusion, the R adjuvants like POEA change human cell permeability and amplify toxicity induced already by G, through apoptosis and necrosis. The real threshold of G toxicity must take into account the presence of adjuvants but also G metabolism and time-amplified effects or bioaccumulation. This should be discussed when analyzing the in vivo toxic actions of R. This work clearly confirms that the adjuvants in Roundup formulations are not inert. Moreover, the proprietary mixtures available on the market could cause cell damage and even death around residual levels to be expected, especially in food and feed derived from R formulation-treated crops.

33) Mariana, A. et al. (2009): We studied the effect of chronic pesticide exposure in rats injected i.p. for 5 weeks with doses between 1/50 and 1/250 LD50 of dimethoate, glyphosate and zineb, either alone or in combination. All tested agrochemicals increased the oxidative stress
status in the plasma, liver, and testes, and also modified hormonal parameters involved in reproductive function. The increase in oxidative stress and damage biomarker levels, as well as the alteration of the antioxidant defence system decreased testosterone, FSH and LH levels in the plasma of pesticide-treated rats.

34) Mañas F et al. (2009): In the present study glyphosate was genotoxic in the comet assay in Hep-2 cells and in the MNT test at 400 mg/kg in mice. ... The results showed an increase in these enzyme activities. According to the obtained results we cannot discard the oxidative stress as a potential genotoxicity mechanism. Due to the fact that the results were not conclusive we believe it is necessary to carry on researching the possible connection between oxidative stress an genetic damage.

35) Prasad S et al. (2009): Glyphosate treatment significantly increases CAs (chromosomal aberrations) and MN (micronuclei) induction at both treatments and time compared with the vehicle control (P < .05). The cytotoxic effects of glyphosate were also evident, as observed by significant decrease in mitotic index (MI). The present results indicate that glyphosate is clastogenic and cytotoxic to mouse bone marrow.

36) Robin Mesnage et al. (2009): In January 2009, a farming couple contacted us because two of their three children were born with congenital malformations. One had a somatotropic deficiency, an imperforate anus and a small atrial septal defect at birth. Another was suffering from hypospadias, had a micropenis, a total deficiency of growth hormone and presented also an imperforate anus. All these disorders are rarely encountered in the same person or family. Yet, in some cases these symptoms with others have been grouped under the
Stratton-Parker syndrome, whose etiology remains unknown. They noticeably overlap our cases. Only males are affected up to date and all cases occurred sporadically, some authors have therefore proposed an X-linked recessive inheritance. Due to the absence of known familial antecedents, and lack of genetic origins evidenced to date, the hypothesis of an environmental origin can be explored. In particular, many pesticides were used by this family around pregnancies. The father sprayed, without protection, more than 1.3 tons of pesticides per year including 300 liters of glyphosate based herbicides. Among them are well-known endocrine disruptors such as carbendazim, 2,4-Dichlorophenoxyacetic acid, glyphosate, ioxynil, linuron, trifluralin and vinclozolin. The whole family had close contact with the father, consumes products of their garden and can be exposed through the consumption of pigs and poultry fed with the farm harvest.

37) Gasnier C et al. (2009): Glyphosate-based herbicides are the most widely used across the world; they are commercialized in different formulations. Their residues are frequent pollutants in the environment. In addition, these herbicides are spread on most eaten transgenic plants, modified to tolerate high levels of these compounds in their cells. Up to 400 ppm of their residues are accepted in some feed. We exposed human liver HepG2 cells, a well-known model to study xenobiotic toxicity, to four different formulations and to glyphosate, which is usually tested alone in chronic in vivo regulatory studies. We measured cytotoxicity with three assays (Alamar Blue, MTT, ToxiLight), plus genotoxicity (comet assay), anti-estrogenic (on ERalpha, ERbeta) and anti-androgenic effects (on AR) using gene reporter tests. We also checked androgen to estrogen conversion by aromatase activity and mRNA. All parameters were disrupted at sub-agricultural doses with all formulations within 24h. These effects were more dependent on the formulation than on the glyphosate concentration. First, we observed a human cell endocrine
disruption from 0.5 ppm on the androgen receptor in MDA-MB453-kb2 cells for the most active formulation (R400), then from 2 ppm the transcriptional activities on both estrogen receptors were also inhibited on HepG2. Aromatase transcription and activity were disrupted from 10 ppm. Cytotoxic effects started at 10 ppm with Alamar Blue assay (the most sensitive), and DNA damages at 5 ppm. A real cell impact of glyphosate-based herbicides residues in food, feed or in the environment has thus to be considered, and their classifications as carcinogens/mutagens/reprotoxics is discussed.

38) Romano RM et al. (2010): These results suggest that commercial formulation of glyphosate is a potent endocrine disruptor in vivo, causing disturbances in the reproductive development of rats when the exposure was performed during the puberty period.

39) Jayawardene, U.A et al. (2010): Glyphosate recorded the highest percentage of malformation (69%) compared to other pesticides in 1.00 ppm concentration. Malformations observed were mainly in the spine, such as hunched back (kyphosis) and curvature (scoliosis), while edema and skin ulcers were also observed.

40) Paganelli, A.et al. (2010): Xenopus laevis embryos were incubated with 1/5000 dilutions of a commercial GBH. The treated embryos were highly abnormal with marked alterations in cephalic and neural crest development and shortening of the anterior–posterior (A-P) axis. Alterations on neural crest markers were later correlated with deformities in the cranial cartilages at tadpole stages. Embryos injected with pure glyphosate showed very similar phenotypes. Moreover, GBH produced similar effects in chicken embryos, showing a gradual loss of rhombomere domains, reduction of the optic vesicles, and microcephaly. This suggests that glyphosate itself was responsible for the phenotypes.
observed, rather than a surfactant or other component of the commercial formulation. A reporter gene assay revealed that GBH treatment increased endogenous retinoic acid (RA) activity in Xenopus embryos and cotreatment with a RA antagonist rescued the teratogenic effects of the GBH. Therefore, we conclude that the phenotypes produced by GBH are mainly a consequence of the increase of endogenous retinoid activity.

41) Rick A. Relyea (2012): Even more striking was the discovery that Roundup induced morphological changes in the tadpoles. In wood frog and leopard frog tadpoles, Roundup induced relatively deeper tails in the same direction and of the same magnitude as the adaptive changes induced by dragonfly cues. To my knowledge, this is the first study to show that a pesticide can induce morphological changes in a vertebrate. Moreover, the data suggest that the herbicide might be activating the tadpoles' developmental pathways used for antipredator responses. Collectively, these discoveries suggest that the world's most widely applied herbicide may have much further-reaching effects on nontarget species than previous considered.

42) Fabio Leonardo Meza-Joya et al. (2012): Glyphosate formulation at application rates above 5.4 µg a.e./cm² (in vivo) and concentrations above 95 µg a.e./mL (in vitro) showed clear evidence of cytotoxicity. In vivo and in vitro exposure of *E. johnstonei* erythrocytes to the glyphosate formulation induced DNA breaks in a dose-dependent manner with statistically significant values (*P* < 0.05) at all doses tested. DNA damage initially increased with the duration of exposure and then decreased, suggesting that DNA repair events were occurring during in vivo and in vitro exposures.

43) Koller VJ et al. (2012): Glyphosate (G) is the largest selling herbicide worldwide; the most common formulations (Roundup, R)
contain polyoxyethyleneamine as main surfactant. Recent findings indicate that G exposure may cause DNA damage and cancer in humans. R induced acute cytotoxic effects at concentrations >40 mg/l after 20 min, which were due to membrane damage and impairment of mitochondrial functions. Both G and R induced DNA migration in single-cell gel electrophoresis assays at doses >20 mg/l. Furthermore, an increase of nuclear aberrations that reflect DNA damage was observed. The frequencies of micronuclei and nuclear buds were elevated after 20-min exposure to 10–20 mg/l, while nucleoplasmatic bridges were only enhanced by R at the highest dose (20 mg/l). R was under all conditions more active than its active principle (G). Comparisons with results of earlier studies with lymphocytes and cells from internal organs indicate that epithelial cells are more susceptible to the cytotoxic and DNA-damaging properties of the herbicide and its formulation. Since we found genotoxic effects after short exposure to concentrations that correspond to a 450-fold dilution of spraying used in agriculture, our findings indicate that inhalation may cause DNA damage in exposed individuals.

44) Vandenberg LN et al. (2012): For decades, studies of endocrine-disrupting chemicals (EDCs) have challenged traditional concepts in toxicology, in particular the dogma of “the dose makes the poison,” because EDCs can have effects at low doses that are not predicted by effects at higher doses. Here, we review two major concepts in EDC studies: low dose and nonmonotonicity. ... We conclude that when nonmonotonic dose-response curves occur, the effects of low doses cannot be predicted by the effects observed at high doses. Thus, fundamental changes in chemical testing and safety determination are needed to protect human health.

45) Benedetti D et al. (2013): Soybean cultivation is widespread in
the State of Rio Grande do Sul (RS, Brazil), especially in the city of Espumoso. Soybean workers in this region are increasingly exposed to a wide combination of chemical agents present in formulations of fungicides, herbicides, and insecticides. ... Comet assay and BMCyt (micronuclei and nuclear buds) data revealed DNA damage in soybean workers. Cell death was also observed (condensed chromatin, karyorhectic, and karyolitic cells). Inhibition of non-specific choline esterase (BchE) was not observed in the workers. The trace element contents of buccal samples were analyzed by Particle-Induced X-ray Emission (PIXE). Higher concentrations of Mg, Al, Si, P, S, and Cl were observed in cells from workers. No associations with use of personal protective equipment, gender, or mode of application of pesticides were observed. Our findings indicate the advisability of monitoring genetic toxicity in soybean farm workers exposed to pesticides.

46) Thongprakaisang S et al. (2013): Glyphosate is an active ingredient of the most widely used herbicide and it is believed to be less toxic than other pesticides. However, several recent studies showed its potential adverse health effects to humans as it may be an endocrine disruptor. This study focuses on the effects of pure glyphosate on estrogen receptors (ERs) mediated transcriptional activity and their expressions. Glyphosate exerted proliferative effects only in human hormone-dependent breast cancer, T47D cells, but not in hormone-independent breast cancer, MDA-MB231 cells, at $10^{-12}$ to $10^{-6}$ M in estrogen withdrawal condition. The proliferative concentrations of glyphosate that induced the activation of estrogen response element (ERE) transcription activity were 5-13 fold of control in T47D-KBluc cells and this activation was inhibited by an estrogen antagonist, ICI 182780, indicating that the estrogenic activity of glyphosate was mediated via ERs. Furthermore, glyphosate also altered both ERα and β expression. These results indicated that low and environmentally relevant
concentrations of glyphosate possessed estrogenic activity. Glyphosate-based herbicides are widely used for soybean cultivation, and our results also found that there was an additive estrogenic effect between glyphosate and genistein, a phytoestrogen in soybeans. However, these additive effects of glyphosate contamination in soybeans need further animal study.


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