CENTER FOR FOOD SAFETY

May 2015 FACT SHEET

GLYPHOSATE AND CANCER RISK: FREQUENTLY ASKED QUESTIONS

WHY IS THERE CONCERN ABOUT GLYPHOSATE AND CANCER? The World Health Organization's (WHO's) cancer authorities – the International Agency for Research on Cancer (IARC) – recently determined that glyphosate is "probably carcinogenic to humans" (Group 2A). Glyphosate is the most heavily used pesticide in the world thanks to widespread planting of Monsanto's Roundup Ready crops, which are genetically engineered to survive spraying with it. Use and exposure will increase still more if glyphosate-resistant turfgrasses currently being developed for lawns, playing fields and golf courses are introduced.

WHERE DO EPA AND WHO'S IARC STAND ON GLYPHOSATE'S CARCINOGENICITY? In 1985, EPA classified glyphosate as a possible carcinogen based on experiments showing tumors in glyphosate-treated rodents. Input from Monsanto led to a dubious reinterpretation of these studies by EPA, and reclassification of glyphosate as non-carcinogenic in 1991.[1] IARC has thus far published only a brief summary of its glyphosate assessment, which is based on multiple lines of evidence: kidney, pancreatic and other tumors in glyphosate-treated test animals; epidemiology studies showing higher rates of cancer in glyphosate-using farmers; and research showing that glyphosate damages DNA and chromosomes, one mechanism by which cancer is induced.[2] IARC's full assessment is due out in 2016.

WHOSE ASSESSMENT IS MORE RELIABLE: IARC OR EPA? IARC is the world's leading authority on cancer. Its glyphosate determination was made by unanimous decision of 17 qualified scientists led by Dr. Aaron Blair, a distinguished epidemiologist recently retired from the U.S. National Cancer Institute.[3] IARC's assessment is up-to-date, analyzing all the relevant available research, while EPA's last comprehensive assessment of glyphosate occurred in 1993. IARC considered a broad range of evidence, including human epidemiology and other peer-reviewed studies, while EPA did not assess epidemiology and relied almost entirely on unpublished industry studies.[4] IARC is an independent agency whose sole mission is human health. While EPA is charged with protecting human health as well, it is also subject to considerable pressure from pesticide companies whose products it regulates. EPA is currently re-assessing glyphosate, and has said it will consider IARC's findings.

NATIONAL OFFICE: 660 Pennsylvania Avenue, SE, Suite 302, Washington, DC 20003
NORTHWEST OFFICE: 917 SW Oak Street, Suite 300 Portland, OR 97205
CALIFORNIA OFFICE: 303 Sacramento St., 2nd Floor, San Francisco, CA 94111
HAWAI'I OFFICE: 1132 Bishop, Suite 2107, Honolulu, HI 96813

HOW DOES GLYPHOSATE COMPARE TO OTHER AGENTS THAT CAUSE OR MAY CAUSE CANCER?

The evidence implicating glyphosate as a human carcinogen is not as strong as that for smoking or asbestos (IARC Group 1, "carcinogenic"), but stronger than that for DDT, parathion (both insecticides) or infection with type 2 HIV virus (Group 2B, "possibly carcinogenic").[5]

BUT DOESN'T IARC CONSIDER SUNLIGHT AND ALCOHOL TO BE CARCINOGENIC?

Although IARC primarily assesses chemicals, it also evaluates the carcinogenic potential of other "agents,"[6] which has unfortunately been used by some in a misguided attempt to cast doubt on its glyphosate determination. In fact, IARC's classifications of UV radiation and alcohol as "carcinogenic" are well-supported by science. Dermatologists regard excessive exposure to UV radiation (a component of sunlight) as the most important preventable cause of skin cancer.[7] According to the American Cancer Society, "alcohol is a known cause of cancers" of eight different organs.[8] The point is not that sunshine or drinking a few beers will kill you, but that you can reduce your risk of cancer by avoiding frequent sunburns and cutting back on heavy drinking. One important distinction here is that you can choose to wear sunscreen or drink less, but for most of us it is difficult to reduce our exposure to chemicals like glyphosate.

IS IARC'S ASSESSMENT RELEVANT TO ACTUAL HUMAN RISK OF CANCER?

A formal risk assessment evaluates both the inherent toxicity of a substance (called hazard) and our exposure to it. While a toxic substance is always hazardous, the risk it poses depends upon the circumstances of exposure.[9] While IARC does not directly evaluate exposure (it is a hazard assessment), it does consider the results of qualified epidemiological studies, which evaluate risk from actual exposure under real-world conditions. Three epidemiology studies of farmers show a link between glyphosate and non-Hodgkin's lymphoma (NHL), an immune system cancer.[10] Another finds a "suggestive association" between glyphosate and a related immune system cancer, multiple myeloma (but not NHL), and recommends follow-up given the herbicide's widespread use.[11] Because there is typically a time lag of decades between exposure to a carcinogen and elevated cancer rates, and glyphosate use has skyrocketed over the past 10-15 years, the full effects of glyphosate's rising use remain to be discovered.

IS THE GENERAL PUBLIC AT RISK FROM GLYPHOSATE?

Because of glyphosate's extremely intensive use (300 million lbs./year, more than four times that of the second-leading pesticide, atrazine), it is regularly found in food (e.g. bread), the air, rainfall and surface waters.[12] Glyphosate is found at similar frequencies and levels in the urine of farm and non-farm family members, including children, suggesting similar levels of exposure.[13] Glyphosate has also been detected in human blood.[14] EPA's maximal "safe" level of glyphosate exposure is six times higher than Europe's,[15] and 17.5-fold higher than the level EPA itself set in the early 1980s.[16] EPA's latest high-end estimate of infant exposure to glyphosate exceeds the level it regarded as safe in the 1980s;[17] and is five times higher than the maximum level suggested by independent scientists.[18]

ARE THERE OTHER PROBLEMS WITH GLYPHOSATE ASSESSMENTS?

EPA's assessments of glyphosate share the weaknesses of all the Agency's pesticide regulation. Most testing has involved only the active ingredient glyphosate, even though formulations (e.g. Roundup) used in the real world are often more toxic due to the presence of additional, often undisclosed, ingredients.[19] While we are all exposed to multiple pesticides in our food, water and air, EPA does not consider the additive or synergistic effects of exposure to glyphosate together with other pesticides. Finally, EPA's practice of basing its decisions almost entirely on studies conducted or commissioned by the pesticide registrant introduces serious conflicts of interest,[20] and excludes pertinent evidence from peer-reviewed studies by independent scientists.[21]

DO OTHER HERBICIDES POSE CANCER RISKS?

Massive use of glyphosate with Roundup Ready crops has generated an epidemic of glyphosate-resistant weeds. In response, pesticide companies are poised to introduce a host of "next-generation" GE crops resistant to herbicides such as 2,4-D and dicamba as well as glyphosate. These new GE crops will trigger an unprecedented and increasingly toxic spiral of weed resistance and herbicide use in American agriculture, for instance, a several-fold rise in 2,4-D and dicamba applications, with no countervailing reduction in glyphosate.[22] Exposure to 2,4-D and dicamba is linked to non-Hodgkin's lymphoma (NHL), the same cancer with which glyphosate has been associated.[23] Herbicide exposure in general is also linked to increased rates of Parkinson's disease.[24]

ENDNOTES

[1] EPA (1991). Memorandum on Second Peer Review of Glyphosate, U.S. Environmental Protection Agency, 10/30/91.

[2] Guyton KZ et al. (2015). Carcinogenicity of tetrachlorvinphos, parathion, malathion, diazinon, and glyphosate. Lancet Oncology (March 20th), doi:10.1016/S1470-2045(15)70134-8.

[3] Pollack A (2015). Weed killer, long cleared, is doubted. New York Times 3/27/15; and Gillam C (2015). Scientist defends WHO group report linking herbicide to cancer. Reuters 3/26/15.

[4] EPA (1993). Glyphosate Reregistration Eligibility Decision. U.S. Environmental Protection Agency, Sept. 1993, App. C.

[5] See IARC List of classifications at http://monographs.iarc.fr/ENG/Classification/index.php.

[6] IARC also assesses biological organisms (e.g. viral infections), behavioral practices (e.g. tobacco smoking), occupational exposure (e.g. as firefighter), physical agents (e.g. surgical implants), and foods or components of food (e.g. coffee and caffeine), collectively referred to as "agents." See IARC Monongraphs on the Evaluation of Carcinogenic Risks to Humans, Preamble, at http://monographs.iarc.fr/ENG/Preamble/CurrentPreamble.pdf.

[7] American Academy of Dermatology: Melanoma FAQs. https://www.aad.org/media-resources/stats-and-facts/conditions/melanoma-faqs.

[8] American Cancer Society: Alcohol Use and Cancer. The organs are the mouth, throat, larynx, esophagus, liver, colon, rectum and breast, see: http://www.cancer.org/cancer/cancercauses/dietandphysicalactivity/alcohol-use-and-cancer.

[9] Key factors include the timing and level of exposure. Children and fetuses are generally more susceptible to harm than adults; and while greater exposure is generally thought to mean greater risk, lower levels of hormone-disrupting chemicals sometimes cause more harm than higher levels (Vandenberg LN et al. 2012. Hormones and endocrine-disrupting chemicals: low-dose effects and nonmonotonic dose responses. Endocrine Reviews 33(3): 378-455).

[10] Guyton et al., 2015.

[11] De Roos AJ et al. (2005). Cancer incidence among glyphosate-exposed pesticide applicators in the Agricultural Health Study. Environmental Health Perspectives 113(1): 49-54.

[12] For 2012 agricultural use of glyphosate (>280 million lbs.) and atrazine (70 million lbs.) in the U.S., see charts below maps at pertinent links on http://water.usgs.gov/nawqa/pnsp/usage/maps/compound_listing.php?year=2012&hilo=L. For non-farm uses of glyphosate (18-23 million lbs./year), see: EPA (2011). Pesticide Industry Sales and Usage: 2006 and 2007 Market Estimates, EPA, Feb. 2011, Tables 3.7 & 3.8. For glyphosate in food, see FoEE (2013). Human contamination by glyphosate. Friends of the Earth Europe, June 2013. For lack of testing in U.S., see: Gillam C (2015). Regulators may recommend testing food for glyphosate residues, Reuters, 4/20/15. For glyphosate in air, rain and surface water, see Chang F-C et al. (2011). Occurrence and fate of the herbicide glyphosate and its degradate aminomethylphosphonic acid in the atmosphere. Environ Toxicol Chem 30(3): 548-555; and Coupe RH et al. (2011). Fate and transport of glyphosate and aminomethylphosphonic acid in surface waters of agricultural basins. Pest Manag Sci 68(1): 16-30.

[13] Curwin BD et al. (2007a). Urinary pesticide concentrations among children, mothers and fathers living in farm and non-farm households in Iowa. Ann. Occup. Hyg. 51(1): 53-65; and Curwin BD et al. (2007b). Pesticide dose estimates for children of Iowa farmers and non-farmers. Environmental Research 105: 307-315. For Europe, see FoEE (2013). Human contamination by glyphosate. Friends of the Earth Europe, June 2013.

[14] Aris A, Leblanc S. Maternal and fetal exposure to pesticides associated to genetically modified foods in Eastern Townships of Quebec, Canada. Reprod Toxicol. 2011: 31(4): 528-533.

[15] "Acceptable daily intake" (ADI) or the equivalent "chronic population adjusted dose" (cPAD), expressed as milligrams glyphosate per kilogram body weight per day: 0.3 in Europe vs. 1.75 mg/kg/day in the U.S. For Europe, see http://ec.europa.eu/food/plant/protection/evaluation/existactive/list1_glyphosate_en.pdf, Appendix II; for US, see EPA (2006). Glyphosate human health risk assessment for proposed use on Indian mulberry and amend use on pea. EPA, 9/29/06, p. 21.

[16] For EPA's setting of the glyphosate ADI at 0.1 mg/kg/day in the early 1980s (vs. 1.75 today), see EPA (1983). Glyphosate (Roundup) on wheat. March 3, 1983.

[17] See EPA (2006) in footnote 14, Table 6.1.2, maximum infant exposure = 0.127562 mg/kg/day, 28% higher than the 1980's ADI of 0.1 mg/kg/day (see EPA 1983 in last footnote).

[18] Antoniou M et al. (2012). Teratogenic effects of glyphosate-based herbicides: divergence of regulatory decisions from scientific evidence. J Environ Anal Toxicol S4:006. doi:10.4172/2161-0525.S4-006, suggesting an ADI of 0.025 mg/kg/day based on teratogenic rather than carcinogenic effects.

[19] See references at: http://earthopensource.org/gmomythsandtruths/sample-page/4-health-hazards-roundup-glyphosate/4-2-myth-strict-regulations-ensure-exposed-safe-levels-roundup/

[20] Boone MD et al. (2014). Pesticide regulation amid the influence of industry. BioScience 64: 917-922.

[21] Myers JP et al. (2009). Why public health agencies cannot depend on good laboratory practices as a criterion for selecting data: the case of bisphenol A. Environmental Health Perspectives 117(3): 309-315.

[22] Mortensen DA et al. (2012). Navigating a critical juncture for sustainable weed management. Bioscience 62(1): 75-85.

[23] Schinasi L, Leon ME (2014). Non-Hodgkin lymphoma and occupational exposure to agricultural pesticide chemical groups and active ingredients: a systematic review and meta-analysis. Int. J Environ. Res. Public Health 11: 4449-4527. McDuffie HH et al (2001). Non-Hodgkin's lymphoma and specific pesticide exposures in men: cross-Canada study of pesticides and health. Cancer Epidemiology, Biomarkers & Prevention 10: 1155-1163.

[24] For instance, see: Brighina L et al. (2008). Alpha-synuclein, pesticides and Parkison disease. Neurology 70: 1461-1469.