Response to Questions From the Domestic Policy Subcommittee of the House Oversight and Government Reform Committee

With Regard to Herbicide-Resistant Weeds Following Testimony Delivered Before the Subcommittee on September 30, 2010

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What does your research reveal about when Monsanto should have known and reacted to development of Roundup-resistant weeds?

Prior to the confirmation of the first glyphosate-resistant weed population in 1996, weed scientists had collected abundant evidence showing that resistant weeds were likely to evolve with frequent use of glyphosate. For instance, Duncan & Weller (1987) conducted experiments on five biotypes of field bindweed that had been shown by DeGennaro & Duncan (1984) to have substantial variability in their tolerance to glyphosate. They concluded from their experiments that: "These results further suggest that glyphosate tolerance in a field bindweed population could be enhanced by selection pressure in the form of ***repeated glyphosate applications***."[1] Boerboom et al (1990) similarly found a three-fold range of glyphosate tolerance in specimens of the weed birdsfoot trefoil.[2] As with field bindweed, repeated glyphosate applications would kill off the more susceptible types, leaving the more tolerant to propagate, potentially leading to a resistant population quite rapidly.[3]

In 1996, the eminent weed scientist Dr. Jonathan Gressel reviewed some of the evidence pointing to the likelihood that glyphosate-resistant weeds would emerge, and rebuked Monsanto scientists for giving the false impression that glyphosate was "invincible" to

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[1] DeGennaro, F.P. & S.C. Weller (1984). "Differential susceptibility of field bindweed (*Convolvulus arvensis*) biotypes to glyphosate," Weed Science 32: 472-476; Duncan, C.N. & S.C. Weller (1987). "Heritability of glyphosate susceptibility among biotypes of field bindweed," The Journal of Heredity 78: 257-60.

[2] Boerboom, C.M. et al (1990). "Mechanism of glyphosate tolerance in birdsfoot trefoil," Weed Science 38: 463-467.

[3] Although "tolerance" and "resistance" to herbicides are formally distinct, in practice the terms are often used interchangeably by weed scientists. In common usage, tolerance denotes a weed that withstands lower doses of an herbicide, while resistant weeds survive higher doses. "Resistant" rather than "tolerant" is the term preferred by scientists for crops intentionally manipulated to withstand application of an herbicide.

resistance.[4] Dr. Gressel first presented the following excerpt of a paper written by Monsanto scientists Steven Padgette and colleagues for a symposium in Spain.[5]

> "Evolution 'of weed resistance to glyphosate appears to be an unlikely event, based on the ***lack of weeds or crops that are inherently tolerant to glyphosate*** and the long history of extensive use of the herbicide resulting in no resistant weeds. ***Unique properties*** of glyphosate such as its mode of action, chemical structure, limited metabolism in plants, and lack of residual activity in soil indicates that ***the herbicide exerts low selection pressure*** on weed populations' (Padgette et al 1995)." (emphasis added)

As noted above, at least two weeds had been shown in peer-reviewed studies in the 1980s and 1990 to have precisely the "inherent tolerance to glyphosate" of which Monsanto's Padgette and colleagues profess ignorance in 1995. Many other weeds, such as morningglories, yellow nutsedge, field horsetail, prairie cupgrass, wild buckwheat, and dayflower species have long been recognized as glyphosate-tolerant.[6] The statement that glyphosate "exerts low selection pressure on weed populations" is grossly misleading, in that it considers only certain chemical properties of glyphosate, and ignores the much more important factor of how glyphosate is used. The frequency, intensity and timing of glyphosate use with Roundup Ready crops generate tremendous selection pressure for evolution of resistant weeds, whatever "unique properties" the glyphosate molecule may or may not possess.

Dr. Gressel's commentary on the quote presented above makes it clear that Monsanto scientists were not innocently wrong, but rather guilty of intentional misrepresentation. Speaking directly to Padgette and colleagues' 1995 paper that contains the statement quoted above, Gressel said:

> "The impression of invincibility from resistance was enhanced by ***not citing*** the growing literature on the known inter-, and especially intra-specific, genetic variability in quantitative levels of glyphosate resistance. ***This literature was known to the various authors,*** yet must have been considered irrelevant. ***In turn, this led to dismissing the need to set resistance management strategies in motion, and the ensuing appearance of a glyphosate-resistant population*** in the management system and the weed where it was most likely to occur." (emphasis added).

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[4] Gressel, J. (1996). "Fewer constraints than proclaimed to the evolution of glyphosate-resistant weeds," Resistant Pest Management Newsletter, Vol. 8, No. 2 (Winter 1996), pp. 20-23. http://whalonlab.msu.edu/Newsletter/pdf/8_2.pdf.

[5] Padgette, S.R., X. Delannay, L. Bradshaw, B. Wells & G. Kishore (1995). "Development of glyphosate-tolerant crops and perspectives on the potential for weed resistance to glyphosate," in: International Symposium on Weed and Crop Resistance to Herbicides, Cordoba, Spain. Abstract 92.

[6] Boerboom, C. & M.D. Owen (2006). "Facts about Glyphosate-Resistant Weeds," The Glyphosate, Weeds and Crop Series, Purdue University Extension, Dec. 2006. http://www.extension.purdue.edu/extmedia/GWC/GWC-1.pdf.

Dr. Gressel goes on to discuss the ample evidence for likely resistance that Monsanto scientists had conveniently ignored. For instance, he cites and discusses eight published scientific articles that present ***eight different mechanisms*** by which weeds might evolve resistance to glyphosate. The lead author of one of these papers was Monsanto scientist Stephen Padgette. We discuss Dr. Gressel's reference to a glyphosate-resistant weed population below.

Why would Monsanto scientists misrepresent this important issue of glyphosate's potential to foster glyphosate-resistant weeds? (And they did this not only in Padgette et al (1995), but in a flurry of papers presenting essentially the same distorted view, for instance: Bradshaw et al (1995), Padgette et al (1996) and Bradshaw et al (1997)[7]). The answer is clear. In the mid 1990s when these papers appeared, Monsanto was in the midst of launching the company's first Roundup Ready (RR) crop, RR soybeans, which were first planted commercially in 1996. While Monsanto's microscopic focus on the supposedly "unique properties" of the glyphosate molecule had some success in quelling resistance concerns (e.g. see Jasienuik 1996),[8] most weed scientists were not fooled. Dr. Gressel and many others were convinced that Roundup Ready crop systems would likely do what two decades of glyphosate use had thus far largely failed to do: foster rapid evolution of GR weeds.

As early as 1990, public interest scientists published a strong critique of the herbicide-resistant (HR) crop paradigm entitled *Biotechnology's Bitter Harvest*, which highlighted the high potential for HR weed evolution presented by HR crop systems, among other risks, such as increased use of toxic herbicides.[9] In 1992, Dr. Rebecca Goldburg (co-author of *Biotechnology's Bitter Harvest*) published a peer-reviewed paper in the journal *Weed Technology*, which made similar points. Interestingly, Dr. Goldburg conceded that HR crops resistant to newer and safer herbicides (e.g. glyphosate vs. older, more toxic herbicides like 2,4-D) might offer some short-term benefits in terms of displacing more toxic herbicides, but cautioned that: "resistant weeds already limit use of some of the newer chemicals, and the availability of crops that tolerate the newer herbicides could further encourage the evolution of resistant weeds…"[10]

Weed scientist Dr. Brian Sindel (1996) made the same point in an article discussing the first glyphosate-resistant weed population (discussed further below), quoting his colleague Dr. Roger Cousens of Latrobe University to the effect that herbicide-resistant crops that rely entirely on herbicides for weed control are "in danger of crashing down around our

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[7] See Gressel (1996), op. cit., for references.

8 Jasieniuk, M. (1996). "Constraints on the evolution of glyphosate resistance in weeds," Resistant Pest Management Newsletter, Vol. 7, No. 2 (Winter 1995), pp. 25-26. http://whalonlab.msu.edu/Newsletter/pdf/7_2.pdf.

[9] Goldburg, R., J. Rissler, H. Shand, C. Hassebrook (1990). *Biotechnology's Bitter Harvest: Herbicide-Tolerant Crops and the Threat to Sustainable Agriculture,* Biotechnology Working Group, March 1990.

[10] Goldburg, R. (1992). "Environmental concerns with the development of herbicide-tolerant plants," Weed Technology 6: 647-652.

ears" due to weeds developing resistance to herbicides.[11] Dr. Sindel also explained why glyphosate had thus far fostered so little weed resistance. Used as a "pre-sowing, knockdown herbicide" (Australian terminology for pre-emergence burndown use) with conventional crops, any resistant weeds would likely be killed off by tillage or subsequent use of other herbicides. Such would not be the case with Roundup Ready crops, where glyphosate would likely be the only weed control tool applied. Dr. Sindel concluded by stating that "glyphosate must be retained as an effective herbicide. Integrated weed management, a combination of weed control techniques, is promoted to avoid the further emergence of herbicide resistance."[12]

In 1997, Dr. Ian Heap, who has long managed an online database that registers the occurrence of herbicide-resistant weed populations worldwide, also warned of the need for resistance management with Roundup Ready crops:

> "The recently developed glyphosate-resistant crops ***will need to be used in rotation with conventional cultivars, and in conjunction with non-chemical weed control*** and other herbicides if the selection of glyphosate-resistant weeds is to be avoided." (emphasis added).[13]

Finally, we cite a prescient 1992 article by EPA scientist Dr. Diana Horne in the journal *Weed Technology* entitled "EPA's response to resistance management and herbicide-tolerant crop issues."[14] In 1992, U.S. regulation of genetically engineered (GE) crops was still in the planning stages, and EPA's role had not yet been fixed. While it was clear that EPA would regulate insecticide-producing insect-resistant GE crops by virtue of its traditional role as pesticide regulator, "EPA's role in the regulation of herbicide-tolerant (HTC) varieties is more oblique. EPA has no direct authority over the plant, as herbicide tolerance does not include production of pesticidal compounds. But, EPA will regulate new herbicide uses."

Dr. Horne went on to discuss the widespread occurrence of weeds resistant to other herbicides, and EPA's "strong interest in promoting the development and broader use of integrated pest management (IPM) technologies" to forestall evolution of resistant insects and weeds and reduce use of herbicides overall and their adverse environmental impacts. In a passage that could not have escaped Monsanto, she posed the following question:

> "Would it be appropriate, for example, for the Agency to require that transgenic plants (both of the pesticidal, as well as the herbicide-tolerant varieties), be used ***only within the context of a resistance management program***?" (emphasis added)

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[11] Sindel, B. (1996). "Glyphosate resistance discovered in annual ryegrass," Resistant Pest Management Newsletter, Vol. 8, No. 2 (Winter 1996), pp. 23-24.
[12] Ibid.
[13] Heap, I. (1997). "Occurrence of herbicide-resistant weeds worldwide," Pesticide Science 51: 235-243.
[14] Horne, D. (1992). "EPA's response to resistance management and herbicide-tolerant crop issues," Weed Technology 6: 657-661.

Unfortunately, Dr. Horne's paper was prescient only in its discernment of the weed resistance threat posed by HR crop systems. While EPA went on to institute mandatory resistance management for insect-resistant GE crops, its halting efforts to establish even weak voluntary weed resistance management plans for glyphosate-resistant and other HR crops foundered on opposition from HR crop developers and growers.[15]

Why were Monsanto scientists virtually alone in denying the threat of glyphosate-resistant weeds? The answer seems clear. Any resistance management plan with a chance to be effective would have to limit selection pressure by imposing restrictions on the use of glyphosate and/or Roundup Ready crops. This is consistent with Dr. Heap's statement above that RR crops would need to be rotated to conventional cultivars to avert further weed resistance. We note that EPA's successful insect resistance management (IRM) plans for insect-resistant crops involve the requirement that growers plant substantial refugia of non-Bt corn and cotton alongside their Bt crop plantings.[16] In short, resistance management would have meant a perhaps substantial crimp in Monsanto's profits via reduced sales of glyphosate and RR crop seed. Another important factor is that farmers often respond to lower-level glyphosate-resistance in weeds by "increasing the magnitude and frequency of glyphosate applications"[17] – a counterproductive, but for Monsanto profitable, response. This helps explain why Monsanto has always recommended using the "full rate" of glyphosate as its keystone "weed resistance prevention" strategy, despite rebukes from weed scientists that use of alternatives to glyphosate is the proper response.[18]

Still, didn't Monsanto understand that it had a longer-term financial interest in preventing the evolution of glyphosate-resistant weeds so as to prolong the useful life of its Roundup Ready technology? Dr. Gressel in fact appeals to Monsanto with this "enlightened self-interest" argument in the conclusion of his piece, cited above. CFS believes that such appeals are based on a misunderstanding of the market forces guiding biotechnology-pesticide firms such as Monsanto.

First, consider that the pesticide industry has long familiarity with weed resistance, which has been evolving since the 1970s. Second, that the pesticide treadmill phenomenon whereby a frequently used herbicide fosters resistance, leading to supplementation or replacement with a new "mode of action" (different type of herbicide), has been a major driver in the pesticide industry's development and sale of new herbicides. Finally, consider

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[15] Jones, Jim (2010). Testimony before the Domestic Policy Subcommittee, House Oversight and Government Reform Committee, Sept. 30, 2010. Mr. Jones is the EPA's Deputy Assistant Administrator for Chemical Safety and Pollution Prevention. http://oversight.house.gov/images/stories/Hearings/pdfs/20100930Jones.pdf.
[16] Jones (2010), op. cit.
[17] NRC (2010). *The Impact of Genetically Engineered Crops on Farm Sustainability in the United States,* National Research Council, National Academy of Sciences, 2010 (pr-publication copy), p. 2-15.
[18] Hartzler, B. (2004). Weed Science, Iowa State University, December 17, 2004. http://www.weeds.iastate.edu/mgmt/2004/twoforone.shtml; Hartzler, B. et al (2004). "Preserving the value of glyphosate," Iowa State University, Feb. 20, 2004, a joint statement by 12 leading Midwestern weed scientists. http://www.weeds.iastate.edu/mgmt/2004/preserving.shtml.

that the most profitable period for sale of patented HR seeds and their associated herbicides is limited to the 20-year terms of the associated patents.

Glyphosate went "off-patent" in the year 2000. Despite competition from cheaper generic versions of glyphosate, Monsanto continued to sell large quantities of its Roundup formulations of glyphosate after the year 2000 by tying the use of Roundup to its patented Roundup Ready seeds.[19] The major patent on Roundup Ready soybeans (the largest acreage RR crop) expires in 2014.[20] For a variety of reasons, Monsanto has been relatively unsuccessful in selling farmers on its second generation RR2 soybeans: some object to their high price; others that they do not provide the promised yield boost; and still others find the value of the technology eroded by glyphosate-resistant weeds, which require use of expensive, supplemental herbicides anyway.[21] For many, it is a combination of these factors – more expensive seed plus the expense of additional herbicides to combat GR weeds. When Roundup Ready 1 soybeans go off-patent in 2014, cheap generic versions will presumably become available; and farmers will likely have the legal right to save and replant them, offering further potential savings. Finally, other firms are poised to introduce their own glyphosate-resistant crops, posing a competitive challenge to the company.[22] In short, Monsanto could be facing the imminent loss of its lucrative Roundup Ready soybean franchise, followed by loss of market share in Roundup Ready corn and cotton when their associated patents expire.

What would persuade farmers to continue buying Monsanto soybeans, corn and cotton? One strong enticement would be the ability to control glyphosate-resistant weeds. Indeed, Monsanto has developed and is awaiting USDA approval of soybean varieties resistant to the broad-spectrum herbicide dicamba, which will be "stacked" with resistance to glyphosate as well.[23] These dual HR soybeans are being offered as a tool to help manage glyphosate-resistant weeds. Triple-stack versions of corn and cotton – which combine resistance to dicamba, glyphosate and a third herbicide, glufosinate – are not far behind.[24]

Finally, consider the market potential for these dual and triple-stack HR crops, which will certainly be more expensive than their Roundup Ready-only predecessors. Clearly, those farmers with GR and other HR weed-infested fields would be the most likely market, since

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[19] Barboza, D. (2001). "The Power of Roundup; A Weed Killer Is a Block For Monsanto To Build On," New York Times, August 2, 2001. http://www.nytimes.com/2001/08/02/business/the-power-of-roundup-a-weed-killer-is-a-block-for-monsanto-to-build-on.html.

[20] Pollack, A. (2009). "As patent ends, a seed's use will survive," New York Times, December 18, 2009. http://www.nytimes.com/2009/12/18/business/18seed.html.

[21] Agrimoney (2010). "Monsanto faces revenue risk if seed drive misfires," Agrimoney, August 16,2010. http://www.agrimoney.com/news/monsanto-faces-revenue-risk-if-seed-drive-misfires--2111.html; Bennett, D. (2009). "Conventional soybeans draw interest," Delta Farm Press, April 3, 2009, http://deltafarmpress.com/soybeans/conventional-soybeans-0403/.

[22] See recent entries for glyphosate-tolerant crops – Stine Seed, Bayer CropScience and Pioneer – at http://www.aphis.usda.gov/biotechnology/not_reg.html.

[23] Monsanto (2010a). "Monsanto completes key regulatory submission for soybeans withy dicamba herbicide tolerance trait," News Release, July 13, 2010. http://monsanto.mediaroom.com/index.php?s=43&item=863

[24] Monsanto (2010b). "Monsanto Announces Record 11 Project Advancements in Annual Research and Development Pipeline Update," News Release, Jan 6, 2010.

those without resistant weeds would have little incentive to purchase pricier multiple HR crops if cheaper Roundup Ready-only varieties do the job. Glyphosate-resistant weeds are currently estimated to infest 6% of the 173 million acres planted to soybeans, corn and cotton in the U.S., or 10.4 million acres.[25] This represents roughly four-fold greater acreage than in late 2007, when CFS collated figures from the same definitive data source on resistant weeds and found the GR weed-infested acreage totaled just 2.4 million acres. Though no one can say with certainty how rapidly GR weeds will emerge in the future, Syngenta's weed resistance manager, Chuck Foresman, estimates that 38 million acres – or one of every four row crop acres – will be infested with GR weeds in the U.S. by the year 2013.[26] This 38 million acres of GR weed-infested fields would represent a substantially greater market opportunity for the sale of Monsanto's dual and triple-resistant HR crops than the current 10 million acres. Clearly, glyphosate-resistant weed evolution opens up substantial new marketing opportunities for Monsanto. In contrast, serious stewardship measures to slow or stop GR weed evolution works against the company's financial interest.

It will perhaps be objected that this is a cynical interpretation of Monsanto's motives. Not at all. CFS is intimately familiar with Monsanto's long-standing voluntary stewardship efforts with Roundup Ready crops, whose ostensible purpose is indeed to slow the emergence of GR weeds. While it is beyond the scope of these comments to elaborate, we have done so elsewhere, demonstrating that some of Monsanto's supposed resistance management recommendations are not only ineffectual, but exacerbate the problem by supporting continual planting of Roundup Ready crops every year.[27] However, the bottom line of rapidly expanding GR weed populations speaks more than any analysis to the inefficacy of Monsanto's recommendations. Of course, having such programs in place is good public relations. And it must be said that biotech-friendly USDA regularly touts such voluntary, Monsanto-sponsored measures as an excuse not to take regulatory action, ignoring their failure.[28] Recall, however, that the EPA's Jim Jones has testified that biotech companies successfully foiled weak attempts by EPA and USDA to introduce voluntary weed resistance management programs under their auspices in 2001. As virtually the sole provider of genetically engineered HR crops at that time, the objectors must have included Monsanto.

Yet in fairness, it should be stated that Monsanto is not alone in anticipating considerable profits from the GR weed epidemic. As recently reported in the Wall Street Journal, pesticide-biotechnology companies are investing hundreds of millions of dollars in new HR crops as a temporary hi-tech "fix" to glyphosate-resistant weeds. Dow Agrosciences

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[25] USDA APHIS (2010). "Draft environmental assessment of supplemental request for partial deregulation of sugar beet genetically engineered to be tolerant to the herbicide glyphosate," USDA Animal and Plant Health Inspection Service, October 2010, p. 93.
[26] Syngenta (2009). "Leading the fight against glyphosate resistance," http://www.syngentaebiz.com/DotNetEBiz/ImageLIbrary/WR%203%20Leading%20the%20Fight.pdf.
[27] CFS (2010). CFS Science Comments on USDA APHIS's draft environmental assessment for partial deregulation of Roundup Ready sugar beets," Dec. 6, 2010. http://www.centerforfoodsafety.org/wp-content/uploads/2010/12/RRSB-Partial-Dereg-EA-Science-Comments-BF.pdf.
[28] USDA APHIS (2010), op. cit.

scientist Jim Jachetta stated that these new HR crops represent "a very significant opportunity" and "a new era" for chemical companies.[29] Mr. Jachetta was probably thinking in particular of Dow's new corn and soybeans varieties that resist high doses of 2,4-D, a close chemical cousin of dicamba that formed part of the Vietnam War defoliant Agent Orange. Dow took the opportunity of press attention to the GR weed epidemic to issue a press release touting its 2,4-D-resistant crops as a fix to GR weeds.[30]

When should Monsanto have known and reacted to the development of Roundup-resistant weeds? The short answer is, no later than the introduction of the first Roundup Ready crop in 1996. As documented above, there was widespread concern in the weed science community that Roundup Ready systems would foster GR weeds, and Monsanto scientists not only ignored the evidence, but in several publications intentionally gave the false impression that resistant weeds would not emerge, so as to avoid resistance management regulations that the EPA was seriously considering, and that would have limited the company's profits.

However, there is also solid evidence that Monsanto scientists denied the existence of the first confirmed GR weed population, rigid ryegrass in Australia, in a peer-reviewed scientific publication. This is the GR weed population referred to by Dr. Gressel (in the above-cited article), who stated that Australian researchers had confirmed to him its existence in discussions at a weed science conference in June of 1996. The Australian press had reported the resistant ryegrass even before that. In another paper (also cited above) appearing in the same issue of the same journal as Dr. Gressel's, Dr. Brian Sindel stated that: "Researchers at the Centre for Conservation Farming at Charles Stuart University at Wagga Wagga ***confirmed*** that the ryegrass was resistant to glyphosate," and that Monsanto Australia's Bill Blowes was working with the University to determine the cause of the resistance. Nevertheless, Monsanto scientists said not a word about this GR weed in a 1997 paper that appeared in the journal *Weed Technology*,[31] and in fact repeatedly denied the existence of any "verified" GR weed population in the world, despite the confirmation cited above.[32] Though the editors received the original paper in April of 1995, they note that a revised version was received on July 17, 1996 – at least weeks and probably months after University researchers had confirmed the resistance.

This historical footnote, however revealing it may be as to Monsanto's (lack of) corporate character, is of minor importance now. Much more significant is the company's ***continuing*** obfuscation of the glyphosate-resistant weed issue, even today, as it strives to introduce new Roundup Ready crops (such as alfalfa and sugar beets) free from the regulation that is urgently

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[29] As quoted in: Kilman, S. (2010). "Superweed outbreak triggers arms race," Wall Street Journal, June 4, 2010.

[30] Kaskey, J (2010). "Dow plans new trait to combat Roundup-resistant weeds," Bloomberg, May 05, 2010, http://www.businessweek.com/news/2010-05-05/dow-plans-new-trait-to-combat-roundup-resistant-weeds-update2-.html.

[31] Bradshaw, L. et al (1997). "Perspectives on glyphosate resistance," Weed Technology 11: 189-198.

[32] In one passage that reveals they know of the resistant population, Bradshaw and colleagues tellingly state that "evidence of weeds evolving resistance to this herbicide [glyphosate] under field situations has not been verified," citing two papers from 1993 and 1994. Elsewhere in the paper, they state: "no verified reports of a glyphosate-resistant plants have arisen following an extensive history of broad-scale glyphosate applications in the field." Yet as noted by Dr. Gressel, the population had been confirmed as resistant by no later than June of 1996.

needed to prevent further epidemic spread of weed resistance. Monsanto's position today is that planting a GR crop every year in the same field is consistent with forestalling GR weed evolution, provided only that it is not the same GR crop every year. This position – uncritically adopted by USDA[33] – stands in direct contradiction to the consensus view of every legitimate member of the weed science community, as expressed in a recent National Research Council report, which stated explicitly that the value of crop rotation to forestall glyphosate-resistant weeds is undermined when the crops in the rotation are glyphosate-resistant.[34]

Thus, the question of when should Monsanto have known and reacted to the development of Roundup-resistant weeds is perhaps wrongly put, as it implies that the company has in fact reacted in an effective manner to glyphosate-resistant weeds. The truth, however, is that Monsanto continues to employ its considerable expertise not to forestall GR weeds, but rather to obfuscate the issue. This in turn serves the interests of avoiding any serious resistance management, selling as many Roundup Ready seeds and as much Roundup as possible, and generating (via GR weeds) market demand for its successor herbicide-resistant crops.

Can you elaborate on why multiple-resistant crops are not, as some claim, a solution to the resistant weed epidemic?

Agrichemical-biotechnology companies have invested hundreds of millions of dollars in the development of crops resistant to high rates of older, more toxic herbicides as the supposed "solution" to glyphosate-resistant weeds.[35] In most cases, such crops are resistant to multiple herbicides, often including glyphosate.

Prominent examples include corn, soybeans and cotton resistant to 2,4-D, developed by Dow Agrosciences; and soybeans, corn and cotton resistant to dicamba, developed by Monsanto. Dow's 2,4-D resistant corn also resists the "fop" class of ACCase inhibiting herbicides,[36] and will be offered with resistance to glyphosate and/or glufosinate as well for triple or "quad-stack" resistance to three or four major classes of herbicide. Dow's soybeans will be resistant to glufosinate and glyphosate as well as 2,4-D, for "triple-stack" resistance to three herbicide families.[37] Monsanto's dicamba-resistant soybeans will also be resistant to glyphosate, while the company has triple-stack versions of corn and cotton in the works that resist dicamba, glufosinate and glyphosate.[38] There are many other

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[33] CFS (2010), op. cit.
[34] NRC (2010), op. cit., pp. 2-19, 2-20. See CFS (2010) for further support.
[35] Kilman, S. (2010). "Superweed outbreak triggers arms race," Wall Street Journal, June 4, 2010.
[36] Wright, T.R. et al (2010). "Robust crop resistance to broadleaf and grass herbicides provided by aryloxyalkanoate dioxygenase transgenes," PNAS 107: 20240-45.
[37] See corresponding entries at USDA's list of genetically engineered crops pending nonregulated status, at http://www.aphis.usda.gov/biotechnology/not_reg.html. For Dow's plans to "stack" their 2,4-D-resistant crops with glyphosate resistance, see: Kaskey, J (2010). "Dow plans new trait to combat Roundup-resistant weeds," Bloomberg, May 05, 2010, http://www.businessweek.com/news/2010-05-05/dow-plans-new-trait-to-combat-roundup-resistant-weeds-update2-.html.
[38] Monsanto (2010a). "Monsanto completes key regulatory submission for soybeans withy dicamba herbicide tolerance trait," News Release, July 13, 2010. http://monsanto.mediaroom.com/index.php?s=43&item=863.

examples from other companies. In fact, fully eleven HR crops are awaiting deregulation (approval for commercial cultivation) by USDA. These include 2,4-D resistant corn and soybeans and dicamba-resistant soybeans.[39]

The agrichemical-biotechnology industry intends for these crops to be sprayed with either premixed herbicide cocktails containing some or all the herbicides to which the crop is resistant, or with one or more of them sequentially, on an as-needed basis.[40]

The rationale behind these multiple HR crop systems is simple. Weeds resistant to one herbicide mode of action will be killed by the other(s). Unfortunately, such a simple-minded approach to weed control will offer at best short-term relief to growers, and even then only at the cost of sharply increased use of more toxic herbicides, with associated adverse impacts on the environment and public health. In the medium to longer-term, Nature will evolve clever responses to the chemical onslaught accompanying multiple-HR crop systems in the form of multiple herbicide-resistant weeds. Real solutions to resistant weeds, as opposed to temporary fixes, will have to involve a renewed commitment to integrated approaches that prioritize non-chemical means of weed control.[41]

Weed resistance is an evolutionary phenomenon. Frequent, repeated use of an herbicide selects for the preferential survival of those initially rare individuals with the genetic predisposition to survive its application. Over time, the resistant individuals propagate and gradually supplant susceptible weeds, resulting in a resistant weed population. The rate of evolution is critically dependent on the "selection pressure." The more frequently an herbicide is used, the more rapidly a resistant weed population will evolve.

Weeds have evolved many different mechanisms for surviving the application of herbicides. The best studied are so-called "target–site" alterations in the enzyme whose activity is normally blocked by the herbicide.[42] Disablement of the enzyme, which performs some critical function in the plant, results in the death of the normal weed. The target-site alteration makes the enzyme immune to the herbicide, conferring resistance on the weed. If the herbicide is regarded as a key and the target enzyme as a lock, the normal susceptible weed is killed when the key fits and opens the lock; the resistant weed has evolved an altered lock that the herbicidal key no longer opens. Target-site alterations normally confer resistance only to herbicides (one to many) that have the same "mode of

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Monsanto (2010b). "Monsanto Announces Record 11 Project Advancements in Annual Research and Development Pipeline Update," News Release, Jan 6, 2010. http://monsanto.mediaroom.com/index.php?s=43&item=788.

[39] See USDA's list of GE crops pending nonregulated status at http://www.aphis.usda.gov/biotechnology/not_reg.html, last updated August 20, 2010.

[40] Green et al (2007). "New multiple-herbicide crop resistance and formulation technology to augment the utility of glyphosate," Pest Management Science 64(4): 332-9.

[41] PSU (2010). "Suppressing Weeds Using Cover Crops in Pennyslvania," Pennsylvania State University, College of Agricultural Sciences, Agricultural Research and Cooperative Extension, 2010.

[42] For a recent review, see: Powles, S.B. & Q. Yu (2010). "Evolution in Action: Plants Resistant to Herbicides," *Annu. Rev. Plant Biol.,* 61: 8.1–8.31.

action."[43] Each "mode of action" (corresponding to a family or class of herbicides) represents a key that opens a particular lock.[44] Use of another herbicide with a different mode of action is usually effective in killing these types of weed.

A weed may also become resistant by evolving the ability to generate many-fold more copies of the target enzyme than are normally produced. In this case, the usual dose of the herbicide is only able to shut down a certain small proportion of the much more numerous enzyme molecules, while the others continue to function, allowing the thereby resistant weed to survive.[45] In terms of the key-lock analogy, the herbicide still fits the lock, but there are not enough herbicidal keys to open the more numerous locks.

Weeds may also evolve the ability to prevent or minimize internal movement of the herbicide, once absorbed by the plant, to the tissues (e.g. roots) it must reach to exert its killing effect, a mechanism known as reduced translocation. Still another mechanism involves reduced absorption of the herbicide, for instance via leaves with a thicker or tougher cuticle.[46] In these cases, the herbicide is unable to reach the lock (or not in sufficient quantities) to open it and so kill the weed.

In all of these cases, switching to an herbicide with a different mode of action will often provide control, at least for a time, though as discussed further below there are complications.

Another different class of resistance mechanisms is called "metabolic degradation" or "enhanced metabolism." Weeds with this form of resistance have the ability to degrade or metabolize the herbicide into a form that is not toxic to the plant. Interestingly, this mechanism often utilizes the plant's natural repertoire of detoxification enzymes, and involves several classes of enzyme that are quite similar to those present in the livers and other tissues of mammals, where they perform a similar detoxification function. Weeds that evolve resistance via metabolic degradation often have the ability to detoxify herbicides from several different families with different modes of action, making them particularly difficult to control. Powles and Yu (2010), in the paper already cited, note that the P450 class of detoxification enzymes represents "a very threatening resistance mechanism, because P450 enzymes can simultaneously metabolize herbicides of different modes of action, potentially including never-used herbicides."

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[43] For weeds resistant to different modes of action, see links under "Herbicide site of action" at http://www.weedscience.org/In.asp. Note that weeds highlighted in red with "Multiple – 2, 3, 4 or more MOAs" indicate multiple herbicide resistant weed populations that withstand herbicides from the specified number of herbicide families (MOAs = modes of action).

[44] The reality is more complicated. Each herbicide family, corresponding to a distinct mode of action, is actually comprised of several to dozens of active ingredients with slightly differing versions of the same basic key which all open the same lock. Resistant weeds may have resistance to all or in some cases only some members of the herbicide family. In terms of our analogy, the lock may be altered such that none of the keys in a particular family opens it, or in such a way that some keys do and others do not fit it.

[45] A population of the most damaging glyphosate-resistant weed, Palmer amaranth, recently evolved this mode of resistance. See: Gaines, T.A. et al (2010). "Gene amplification confers glyphosate resistance in Amaranthus palmeri," PNAS 107: 1029-34.

[46] For a recent review, see: Powles & Yu (2010), op. cit.

It is extremely important to observe that in most cases of weed resistance, the mechanism involved remains unknown. It requires extremely sophisticated molecular analysis as well and lengthy greenhouse testing to ascertain the mechanism of resistance in any particular case. And weed scientists are rapidly discovering that some weed populations possess two or more mechanisms of resistance to a single herbicide family, each lending only limited resistance, but together offering higher and more threatening levels.[47] Nature's evolution of weed resistance to herbicides takes quite ingenious turns,[48] and has far outpaced our technical capacities to ascertain the causes. This becomes clearer when one considers the vast numbers of resistant weeds in the world today.

According to the latest counts, over 400,000 fields in the world are infested with 348 herbicide-resistant biotypes of 194 different weed species.[49] A biotype is a particular weed species-herbicide family combination. The number of resistant biotypes exceeds the number of resistant species because a particular weed species can have various populations resistant to different herbicide families. Thus, separate waterhemp populations with resistance to glyphosate alone, or to ALS inhibitors alone, represent two distinct herbicide-resistant biotypes of a single weed species. The considerable excess of biotypes to species indicates that a number of weed species have different populations that are resistant to different herbicide modes of action.

The U.S. is by far the world leader in herbicide-resistant weeds, with 132 confirmed resistant biotypes infesting roughly 30 million acres.[50] Second place belongs to Australia, with just 54 resistant biotypes.[51] The most extensive populations of resistant weeds in the U.S. involve three major herbicide modes of action: resistance to photosystem II inhibitor family herbicides (chiefly the triazine class), which emerged chiefly in the 1970s; resistance to ALS inhibitor family herbicides, which evolved mainly in the 1980s and early 1990s when these herbicides were most heavily used; and resistance to glyphosate, which has evolved in dramatic fashion over just the past decade.[52]

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[47] Dinellli, G. et al (2006). "Physiological and molecular insight on the mechanisms of resistance to glyphosate in *Conyza Canadensis* (L.) Cronq. Biotypes," Pesticide Biochemistry and Physiology 86: 30-41.
[48] Gressel, J. & A.A. Levy (2006). "Agriculture: The selector of improbable mutations," PNAS 103: 12215-16.
[49] See http://www.weedscience.org/In.asp.
[50] Based on Center for Food Safety's compilation of data on herbicide-resistant weeds in the U.S. from the International Survey of Herbicide-Resistant Weeds (ISHRW), at www.weedscience.org, on November 30, 2010. 30 million acres is near the upper-bound estimate of 32.3 million acres, which is closer to reality than the lower bound estimate of 9.4 million acres. One indication of this is that a recent point estimate for acreage infested by glyphosate-resistant weeds alone, made by Dr. Ian Heap, who manages the ISHRW website, is 10.4 milllion acres, exceeding the lower-bound estimate for acreage infested by ***all*** herbicide-resistant weeds.
[51] http://www.weedscience.org/summary/CountrySummary.asp.
[52] Benbrook, C. (2009). Impacts of Genetically Engineered Crops on Pesticide Use in the United States: The First Thirteen Years," The Organic Center, November 2009, pp. 12-13 and Figure 2.4. Note that acreage infested with glyphosate-resistant weeds as well as ALS inhibitor-resistant weeds has increased greatly since February of 2009, which is when the figures upon which the figure is based were compiled from ISHRW.

There are two basic pathways for weeds to evolve multiple herbicide resistance. In one pathway, weed populations ***accumulate*** resistance mechanisms, one by one, to different families of herbicides over years, while the other pathway (enhanced metabolism) involves resistance to several families of herbicides all at once.

The one-by-one pathway is made possible by the fact that weed populations, once they evolve resistance to a particular type of herbicide, often retain that resistance trait indefinitely. This is not necessarily the case, but it is often so. Weed scientists once assumed that an herbicide-resistant weed population would gradually disappear if farmers stopped applying the pertinent herbicide. This notion was based on the theoretical idea that in the absence of herbicide use, weeds ***without*** the resistance trait would always be more vigorous – grow faster and bigger, produce more seed and pollen – than resistant weeds. Thus, the latter would thrive only when the herbicide was used, but would be "outcompeted" by normal weeds in its absence. The theoretical underpinning of this idea is that the resistance trait imposes a "metabolic cost" or "fitness cost." That is, the resistant weed expends energy and resources to generate the resistance mechanism, and consequently has less to devote to growth and reproduction. According to this theory, the resistant weed, though of course favored when the herbicide is used, is less vigorous and fecund when not the herbicide is not applied.

As it turns out, this theory fits reality in some cases, but not in others. While some resistant weeds are indeed less "fit" in the absence of the pertinent herbicide's use, others are as just as fit or even more vigorous than their herbicide-susceptible brethren. As with mechanisms of resistance, weed scientists simply have not determined the fitness of the great majority of herbicide-resistant biotypes. Based on what little is known, however, we can make the following cautious generalizations about resistance to the three major modes of action presented above.

In general, weeds resistant to triazines tend to be less fit.[53] Some weeds resistant to ALS inhibitors exhibit lesser fitness, but others appear to have equivalent or even greater fitness than susceptible weeds.[54] Since glyphosate-resistant biotypes have emerged rapidly over just the past decade, in most cases their fitness has not been tested, and remains unknown. Given the importance of glyphosate in world agriculture, and the rapid emergence of glyphosate-resistant (GR) biotypes, elucidation of the fitness of GR weeds should be a top research priority.[55] Below, we discuss recent research that addresses this question.

The fitness of a resistant weed population helps determine how well it thrives in situations where farmers stop using the pertinent herbicide. Where fitness costs obtain, the resistant weed population will subside. Where there is no fitness cost, or indeed the resistant weed

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[53] Gronwald, J.W. (1994). "Resistance to photosystem II inhibiting herbicides," in: Powles, S.B. & J.A.M. Holtum, eds., *Herbicide Resistance in Plants: Biology and Biochemistry,* Ann Arbor, MI, Lewis, 1994.
[54] Tranel, P.J. & T.R. Wright (2002). "Resistance of weeds to ALS-inhibiting herbicides: what have we learned?" Weed Science 50: 700-712. Further examples are discussed below.
[55] Vila-Aiub, M.M. et al (2009). "Fitness costs associated with evolved herbicide resistance alleles in plants," New Phytologist 184: 751-767.

is more vigorous, ending use of the pertinent herbicide will do nothing to reduce resistant populations. In these cases, resistant weed populations may well persist indefinitely or perhaps even increase in scope even when the herbicide is not used.

Another important factor is the herbicide regime used by farmers. While we often think simplistically of farmers switching to a new mode of action when afflicted with weeds resistant to a particular herbicide, the reality is more complex. Often, herbicide A to which one or several weeds have evolved resistance will still be effective in controlling other troublesome weed species. In these cases, a common response of farmers is to supplement herbicide A with herbicide B rather than stop using A altogether. Thus, weed populations that have evolved resistance to A will continue to be exposed to it, and will continue to have an advantage over their susceptible brethren. Even if there is a fitness cost to herbicide A resistance, weeds resistant to it will continue to be favored.

The hope, of course, is that herbicide B will save the day by killing off weeds resistant to A. This forms the basis of the agrichemical-biotechnology industry's strategy of introducing multiple-herbicide resistant crops. And this will sometimes be an effective strategy. However, here too the reality is more complex. In those cases where the population of weeds resistant to A is small, the supplementation (or switching) strategy has a greater chance of success. However, this strategy is more likely to fail with larger resistant weed populations, for the following reason.

The larger the population of weeds resistant to herbicide A, the more likely that there exists among them individual weeds that have the rare genetic predisposition that confers resistance to herbicide B. Suppose that a small population of herbicide A-resistant weeds numbers 1,000, while a large population has 1 million individual plants. If on average only one in a million weeds are resistant, it is unlikely that the small population harbors one, while quite likely that the larger one does. It's essentially a numbers game, equivalent to tickets in a lottery. The small weed population is equivalent to buying just a few lottery tickets, while a large population corresponds to buying most of the tickets. The likelihood that the A-resistant population has a "winning ticket" (an individual with resistance to B as well as A) increases with its size. Winning the lottery, of course, is precisely what one wants to avoid in this case.[56]

What this means is that when a farmer either switches from herbicide A to herbicide B, or supplements A with B, he may well select for weeds that have resistance to ***both*** herbicides. This is the pathway by which weed populations accumulate resistance, one by one, to different herbicide modes of action.

This is the theory, and of course theory (as we have seen above with fitness) can be wrong. What do the facts on the ground tell us? One fact is that multiple herbicide-resistant weed populations are on the rise in the U.S., and have increased sharply over just the past three years. This is depicted in the table below, which is based on data compiled by Center for

[56] This lottery analogy is borrowed (and adapted) from Iowa State University weed scientist Bob Hartzler. See http://www.weeds.iastate.edu/mgmt/2004/twoforone.shtml.

Food Safety on resistant weeds from the best available source, the International Survey of Herbicide-Resistant Weeds (ISHRW).[57] The ISHRW is an online database that records populations of herbicide-resistant weeds, and is supported by agrichemical-biotechnology companies and academic weed scientists. Note that both the number of sites and acreage infested figures are given in ranges due to the difficulty of estimating the precise geographic extent of resistant weed populations. While most weed biotypes in the U.S. and the world today still have confirmed resistance to just one mode of action, the table below demonstrates a disturbing trend to proliferation of multiple herbicide-resistant (MHR) weed populations.

Data on Multiple Herbicide-Resistant Weeds in the U.S Over the Past Three Years

Date Compiled	No. of Species	No. of Reports	No. of States	Sites (min.)	Sites (max.)	Acreage (min.)	Acreage (max.)
11/21/07	11	20	12	679	1,459	25,829	245,755
11/30/10	14	32	15	1,016	3,078	127,799	1,258,605
% increase	27%	60%	25%	50%	111%	395%	412%

As the table shows, the number of reports of MHR weeds has increased by 60%, from 20 to 32, since just November 2007. More concerning is the increase in the aggregate number of sites and acreage infested by these MHR weed populations. The number of sites infested has increased by half to more than double over the past three years, while the acreage infested has increased by a still more troubling 400%.

Two populations of MHR weeds that have emerged since November 2007 are resistant to glyphosate and paraquat. However, the most prevalent MHR weeds resist applications of ALS inhibitors and/or glyphosate. ALS inhibitor-resistant weeds emerged primarily in the 1980s and early 1990s following the introduction of herbicides with this mode of action in 1982. The fact that many weeds resistant to this mode of action have no loss of fitness (and in some cases have enhanced fitness) means that their populations have tended to persist or increase even as farmers made a large scale switch from reliance on them to use of glyphosate in tandem with the adoption of glyphosate-resistant Roundup Ready crops beginning in 1996. Many populations of ALS inhibitor-resistant weeds are also extremely large, infesting from hundreds of thousands to millions of acres. Two populations (in Missouri and Illinois) infest anywhere from 2 to 5 million acres each.

Over the past 14 years, glyphosate has largely displaced ALS inhibitors on the three crops – soybeans, cotton, and to a lesser extent corn – where Roundup Ready varieties have become predominant. These are also the three crops that receive the bulk of herbicides applied in U.S. agriculture as a whole. Consequently, it is no surprise that the majority of weeds evolving resistance over the past decade have become resistant to glyphosate. As with ALS inhibitors, glyphosate-resistant weed populations are often large, with several infesting hundreds of thousands to millions of acres.

As noted above, there has been a sharp rise in reports of weeds resistant to both ALS inhibitors and glyphosate. In November 2007, ISHRW recorded just 3 reports of two

[57] www.weedscience.org.

species of weed infesting at most 10,600 acres that were dual resistant to glyphosate and ALS inhibitors. By November 2010, just three years later, there were 7 reports of five species of weeds with dual resistance to these two modes of action, and they infested hundreds of thousands to as much as 1 million acres. This represents from 10-fold to 100-fold more infested acreage. The most extensive populations of these weeds (waterhemp in Missouri) also resist a third mode of action, PPO inhibitors,[58] that are otherwise being relied upon by growers to combat resistance to glyphosate and ALS inhibitors. Waterhemp has a demonstrated ability to evolve resistance to two, three or more herbicide modes of action, and is for that and other reasons particularly feared. [59] University of Illinois weed scientists recently sounded the alarm about multiple herbicide-resistant waterhemp (*Amaranthus tuberculatus*) in their state and in Missouri:

> "Herbicide resistance in *A. tuberculatus* appears to be on the threshold of becoming an unmanageable problem in soybean."[60]

Noting that glufosinate is one of the few remaining options for control of late season waterhemp, they fear its loss to resistance as well:

> "Furthermore, on the basis of *A. tuberculatus's* history, there is no reason to expect it will not evolve resistance to glufosinate if this herbicide is widely used. If this happens, and no new soybean postemergence herbicides are commercialized, ***soybean production may not be practical in many Midwestern fields***." (emphasis added)

The emergence of dual resistance to glyphosate and ALS inhibitors fits the model of one-by-one accumulation of resistances presented above. Weeds initially evolved ALS inhibitor resistance in the 1980s and 1990s. Because many of these populations have no apparent loss of fitness, they have persisted into this decade; because they tend to be large, there existed among them weeds that had the rare genetic predisposition to survive glyphosate application. Massive use of glyphosate with Roundup Ready crops beginning in 1996 then fostered evolution of the dual-resistant biotypes.

To make matters still worse, a recent study of the most prevalent glyphosate-resistant weed species, horseweed, suggests that it has fitness equal to or greater than glyphosate-susceptible horseweed (at least in California), and that the glyphosate-resistant populations appear to be expanding whether or not glyphosate is applied to them.

> "In a survey conducted in 2006 and 2007, the majority of horseweed plants sampled in the southern SJV [San Joaquin Valley] were GR [glyphosate-resistant], regardless of nearby cropping systems (Hanson et al. 2009), suggesting the possibility that increased fitness may have contributed to the very rapid expansion in the range of

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[58] See http://www.weedscience.org/Case/Case.asp?ResistID=5269.
[59] Tranel,P.J. (2010). "Introducing QuadStack waterhemp," Agronomy Day 2010, University of Illinois Extension.
[60] Tranel, P.J. et al (2010). "Herbicide resistances in Amaranthus tuberculatus: a call for new options," Journal of Agricultural and Food Chemistry, DOI: 10.1021/jf103797n.

> the GR biotype. … Observations of vigorous and productive GR horseweed, regardless of whether it is growing in treated or untreated areas, suggests that the GR horseweed in California may be more competitive than the glyphosate-susceptible (GS) biotype in addition to being resistant to the most commonly used herbicide in orchards, vineyards, and adjacent noncrop areas (Shrestha, personal observation)."[61]

Still more troubling are the results of recent research on horseweed populations in Indiana and Ohio variously resistant to glyphosate alone, to ALS inhibitors alone, or to both classes of herbicides. The authors of this study reported that all three types of resistant horseweed displayed equal fitness to susceptible horseweed, as measured by "growth and seed production potential." They further warn that these populations are likely to persist and even increase in range with continued use of glyphosate and ALS inhibitors – and would be unlikely to "disappear" even if the growers were to stop using them. This latter possibility is unlikely, given the fact that these two modes of action are very commonly used to control many different weed species beyond horseweed in their region.

> "…. we conclude that horseweed populations composed of biotypes with single resistance to glyphosate and ALS-inhibiting herbicides, or multiple resistance to glyphosate + ALS-inhibiting herbicides have similar growth and seed production potential. Furthermore, the variation within these herbicide-resistant populations following exposure to herbicides would suggest that repeated applications will only increase the ability of these populations to compete and reproduce following repeated applications of the same herbicide or combination of herbicides. … To control these herbicide-resistant horseweed populations, and to offset the evolution of more herbicide-resistant weeds, multiple integrated weed management practices need to be implemented with the idea that resistant biotypes will not just disappear after growers stop the application of these herbicide modes of action."[62]

Authors from the same team have also done several studies showing the clear potential for horseweed to evolve resistance to 2,4-D.[63] They note that:

> "Multiple-resistant and cross-resistant horseweed populations have evolved to various combinations of the previous herbicide modes of action in Israel, Michigan, and Ohio (Heap 2009), providing evidence for the plasticity of this weed."[64]

Importantly, their studies of potential 2,4-D resistance in horseweed have been driven by concern over the advisability of relying on some of the new herbicide-resistant crops, such

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[61]Shrestha, A. et al (2010). "Growth, Phenology, and Intraspecific Competition between Glyphosate-Resistant and Glyphosate-Susceptible Horseweeds (*Conyza canadensis*) in the San Joaquin Valley of California," Weed Science 58: 147-153.

[62] Davis, V.M. et al (2009). "Growth and Seed Production of Horseweed (*Conyza canadensis*) Populations Resistant to Glyphosate, ALS-Inhibiting, and Multiple (Glyphosate + ALS-Inhibiting) Herbicides," Weed Science 57: 494-504.

[63] Kruger, G.R. et al (2008). "Response and Survival of Rosette-Stage Horseweed (*Conyza canadensis*) after Exposure to 2,4-D," Weed Science 56: 748-752.

[64] Kruger, G.R. et al (2010). "Growth and Seed Production of Horseweed (*Conyza canadensis*) Populations after Exposure to Postemergence 2,4-D," Weed Science 58: 413-419.

as the 2,4-D and dicamba-resistant varieties mentioned above. Note that 2,4-D and dicamba are both "growth regulator" type herbicides:

> "With the impending commercialization of 2,4-D- and dicamba-resistant crops, it appears that additional options for control of glyphosate-resistant annual broadleaf weeds will be available. However, growth regulator herbicide-resistant technologies ***may not provide long-term solutions*** if resistant or tolerant populations currently exist or if populations become resistant under selection pressure from overreliance on growth regulators for broadleaf weed management."[65]

The implications of these various studies and data are clear. Weeds – including some of the most agronomically damaging and costly species like horseweed and waterhemp – have demonstrated the ability to evolve resistance to single modes of action as well as multiple herbicides. The single-resistant and in some cases dual-resistant weeds often suffer no "fitness cost," and thus their populations are likely to persist indefinitely, rather than conveniently "disappear" if farmers were to stop using them. The persistence of single- and multiple herbicide-resistant weed populations means that switching to, or supplementation with, new modes of action like 2,4-D and dicamba – in association with crops engineered with resistance to them – may backfire. While short-term relief is possible, these new 2,4-D and dicamba-resistant crops "may not provide long-term solutions…" if growers rely excessively on them. Rather, the introduction of multiple-herbicide resistant crops is quite likely to foster increasingly costly and damaging populations of weeds resistant to ever more herbicides.

The all-at-once pathway of herbicide-resistance is also concerning. As noted above, metabolic degradation mechanisms employing the plant's natural detoxification systems can evolve to confer resistance to multiple herbicides at one time – and potentially even to herbicides that have never before been used. At present, this mechanism of weed resistance has been observed mostly in grass-type weeds in Europe and Australia. Powles and Yu (2010) report 11 weed species that have the P450-mediated herbicide degradation mechanism alluded to above. Of these species, populations of blackgrass (*Alopecurus myosuroides*) and rigid ryegrass (*Lolium rigidum*) are among the worst, with resistance to multiple herbicides from three and four different herbicide families, respectively.[66] There have thus far been few reports of weeds with this mechanism of resistance in the U.S.,[67] though further investigations may reveal others.

The rapid increase in the number of weed populations resistant to glyphosate and to multiple herbicides as well as the acreage they infest poses serious problems for U.S. agriculture. Agronomists are wary of the agrichemical-biotechnology industry's preferred response to this problem – introduction of new crops resistant to older, more toxic herbicides, often in stacked versions conferring resistance to multiple herbicides. While

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[65] Ibid.

[66] Powles, S.B. & Q. Yu (2010). "Evolution in Action: Plants Resistant to Herbicides," *Annu. Rev. Plant Biol.,* 61: 8.1–8.31, Table 4.

[67] Park, K.W. et al (2004). "Absorption, translocation, and metabolism of propoxycarbazone-sodium in ALS-inhibitor resistant *Bromus tectorum* biotypes," Pesticide Biochemistry and Physiology 79: 18-24.

new technologies may provide some short-term relief, it will come only at the cost of increased herbicidal pollution of the environment, harm to human health, and greatly increased weed control costs for farmers. In the medium to longer term, Nature is likely to win this chemical arms resistance race between crops and weeds.

Do you know of any specific health threats presented by any of the herbicide resistant crop systems under development?

As noted above, two of the leading herbicide-resistant crop systems involve resistance to 2,4-D and dicamba. According to Pennsylvania State University weed scientist Dr. Dave Mortensen, widespread deployment of these crop systems would likely lead to a substantial increase in the use of these herbicides in U.S. agriculture. In testimony before this Subcommittee on July 28, 2010, Dr. Mortensen estimated that herbicide use on soybeans would increase by 70% within three years of introduction of 2,4-D and dicamba-resistant soybeans, assuming rapid adoption,[68] an increase of roughly 55 million lbs.[69]

Increased use of these herbicides, especially at that magnitude, would have adverse impacts on the environment, public health, and in particular the health of farmers.

The toxicity of 2,4-D (dichlorophenoxyacetic acid) has been exhaustively reviewed in a petition by public interest scientists to EPA requesting that the herbicide's registration be cancelled.[70] Ingestion or inhalation of 2,4-D has adverse effects on the nervous system – loss of coordination, limb stiffness, stupor, coma. A growing body of evidence points to 2,4-D as a carcinogen. Studies in the U.S., Italy, Canada, and several other countries link 2,4-D exposure to non-Hodgkin's lymphoma, a cancer of the immune system. Studies of farm workers exposed to 2,4-D revealed higher than normal rates of birth defects in their children. 2,4-D is also a mutagen and an endocrine disruptor, and can be contaminated during the production process with the even more toxic compound dioxin, which is highly carcinogenic, weakens the immune system, decreases fertility, and causes birth defects.[71] 2,4-D is banned in Norway.

Dicamba is a chlorinated benzoic acid herbicide similar in structure and mode of action to 2,4-D, and is used in both agriculture (e.g. corn, wheat) and on lawns.[72] In 1992, the

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[68] Mortensen, D. (2010). Accessible at: http://oversight.house.gov/index.php?option=com_content&view=article&id=921%3A07-28-2010-domestic-policy-qare-superweeds-an-outgrowth-of-usda-biotech-policy-part-iq&catid=18%3Asubcommittee-on-regulatory-affairs&Itemid=1.

[69] Mercer, D. (2010). "Roundup resistant weeds pose environmental threat," Associated Press, June 21, 2010. http://www.usatoday.com/tech/science/environment/2010-06-21-roundup-weeds_N.htm.

[70] Comments to EPA on its 2,4-D Risk Assessment, Docket ID No OPP-2004-0167, submitted by a coalition of public health groups, including Natural Resources Defense Council and Beyond Pesticides, August 23, 2004.

[71] Beyond Pesticides (2004). 2,4-D: chemicalWATCH Fact Sheet, updated July 2004, Beyond Pesticides. http://www.beyondpesticides.org/pesticides/factsheets/2,4-D.pdf.

[72] Cox, C. (1994). "Dicamba Factsheet," *Journal of Pesticide Reform* 14(1): 30-35.

National Cancer Institute (NCI) found that farmers exposed to dicamba were twice as likely to contract non-Hodgkin's lymphoma.[73] A subsequent NCI study reported associations between dicamba exposure and higher incidence of lung and colon cancer in pesticide applicators.[74] Researchers have also found a 20% percent inhibition of the nervous system enzyme acetylcholinesterase in a group of certified pesticide applicators whose only common pesticide used was dicamba.[75] Exposure to organophosphate insecticide residues in food has recently been linked to increased rates of attention-deficit/hyperactivity disorder in children, and the presumed mechanism is inhibition of acetylcholinesterase, an enzyme essential for normal brain development.[76] Dicamba is moderately persistent in soil and water, and is frequently found contaminating ground water supplies.[77] Pregnant mice that ingested drinking water spiked with low doses of a commercial herbicide product containing dicamba, 2,4-D and mecoprop had reduced litter size, suggesting that this herbicide mixture may have developmental toxicity.[78] A study of the frequency of sister chromatid exchanges (SCEs) and cell-cycle progression assays revealed that high doses of dicamba can damage DNA, leading the study authors to warn that dicamba is a "potentially hazardous compound to humans."[79]

Dicamba is also highly volatile, and under the right conditions (hot days, no rainfall) can revolatilize after application and drift to damage neighboring crops or plants bordering fields.[80] This drift can cause significant economic damage to other farmers,[81] and may also destroy habitat for pollinators and other beneficial insects.[82] The greatly increased use of dicamba to be expected with dicamba-resistant crops will likely exacerbate these adverse impacts. South Africa completely prohibited use of dicamba in some districts, and banned aerial application in others.[83]

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[73] Cantor, K.P. (1992). "Pesticides and other agricultural risk factors for non-Hodgkin's lymphoma among men in Iowa and Minnesota," *Cancer Res.* 52: 2447-2455.
[74] Samanic, C. et al (2006). "Cancer Incidence among Pesticide Applicators Exposed to Dicamba in the Agricultural Health Study," *Environmental Health Perspectives* 114: 1521-1526.
[75] Potter, WT, et al. (1993). "Radiometric assay of red cell and plasma cholinesterase in pesticide appliers from Minnesota." *Toxicology and Applied Pharmacology* 119: 150-155.
76 Bourchard, M. F. et al (2010). "Attention-Deficit/Hyperactivity Disorder and Urinary Metabolites of Organophosphate Pesticides," Pediatrics 2010; 125:e1270–e1277.
[77] Thurman, E.M. et al (2003). "Regional Water-Quality Analysis of 2,4-D and Dicamba in River Water Using Gas Chromatography-Isotope Dilution Mass Spectrometry," *International Journal of Environmental Analytical Chemistry* 79: 185-198.
[78] Cavieres, M.F., J. Jaeger & W. Porter (2002). "Developmental Toxicity of a Commercial Herbicide Mixture in Mice: I. Effects on Embryo Implantation and Litter Size," *Environmental Health Perspectives* 110: 1081-1085.
[79] Gonzalez, N.V. et al (2006). "Genotoxicity analysis of the phenoxy herbicide dicamba in mammalian cells in vitro," *Toxicology in Vitro* 20: 1481-87.
[80]Hartzler, B. (2004). "Dicamba Volatility," Iowa State University posting, July 24, 2001, http://www.weeds.iastate.edu/mgmt/2001/dicambavolatility.htm.
[81] See testimony of Steve Smith, Director of Agriculture for Indiana-based tomato processor Red Gold, at http://oversight.house.gov/images/stories/Hearings/pdfs/20100930Smith.pdf.
[82] Mercer, D. (2010), op. cit.
[83] "Banned and restricted substances in the republic of South Africa." April 22, 2008. Accessed online July 19, 2010. http://www.nda.agric.za/act36/Banned%20and%20restricted.htm.

2,4-D-resistant crops may pose a new food safety risk beyond the risks attributable to the increased use of 2,4-D to be expected with its adoption. First, one must understand that monocot plants (cereal crops like corn) have a natural tolerance to low levels of 2,4-D, facilitating the use of this pesticide on major field crops like wheat and corn. Numerous studies have examined precisely how 2,4-D is metabolized by non-genetically engineered plants so as to render it non-toxic to the plant. Genetically engineered 2,4-D-resistant plants incorporate a bacteria-derived gene that metabolizes 2,4-D in a different way, transforming it into 2,4-dichlorophenol (2,4-DCP). 2,4-DCP is not produced, or only in very small amounts, when naturally tolerant plants metabolize 2,4-D.

2,4-DCP is a chlorophenol compound that is individually listed by EPA in its toxics release inventory of toxic chemicals.[84] The European Union also lists 2,4-DCP as a hazardous substance. Animals dosed with high levels of chlorophenols in their food or drinking water experienced adverse liver and immune system effects, and did not gain as much weight as control animals. Some studies have shown increased risk of cancer, as well as acne and liver damage, among workers in pesticide plants that make chlorophenols, though it is not clear whether the effects were due to chlorophenols or other chemicals.[85]

2,4-DCP is used as a raw material in the manufacture of 2,4-D.[86] In a material safety data sheet for 2,4-DCP,[87] Dow notes that exposure of just 1% of a worker's body (an area the size of the palm of a hand) to molten 2,4-DCP may cause death. Dow's industrial hygiene guideline for 2,4-DCP is 1 part per million, skin. Dow reports that animal testing has revealed that 2,4-DCP has adverse effects on blood forming organs (bone marrow & spleen), kidney and liver; that 2,4-DCP may be contaminated by the more toxic 2,4,6-trichlorophenol (known to the State of California to cause cancer); and that this contaminant (present at a level of 0.1% in current samples) may explain the inconclusive results in carcinogenicity tests on animals. Dow further notes that in-vitro genetic toxicity (mutagenicity) studies with 2,4-DCP were negative in some cases and positive in other cases, and that it found no relevant information with respect to possible reproductive effects from 2,4-DCP exposure. Dow found that 2,4-DCP is moderately toxic to aquatic organisms on an acute basis (LC50 or EC50 between 1 and 10 mg/L in most sensitive species tested).

French scientists conducted experiments to determine whether the 2,4-DCP generated by transgenic, 2,4-D-resistant plants after spraying with 2,4-D would be broken down into less toxic compounds. They found that the basic structure of the 2,4-DCP molecule remained intact. The French team concluded that 2,4-D-resistant plants sprayed with 2,4-D "may not

84 EPA (1999). Emergency Planning and Community Right-to-Know Section 313: List of Toxic Chemicals within the Chlorophenols Category, Environmental Protection Agency, June 1999 (Technical Update November 2005).

85 USDHHS (1999). "Toxicological Profile for Chlorophenols," Agency for Toxic Substances and Disease Registry, Public Health Service, US Dept of Health and Human Services, July 1999.

86 http://nzic.org.nz/ChemProcesses/production/1J.pdf.

87 Dow (2006). 2,4-Dichlorophenol Material Safety Data Sheet, Product Code: 20636, MSDS Number: 000715. Dow AgroSciences LLC, Effective Date: 7-Sept-06.

be acceptable for human consumption."[88] They further point to the potential for 2,4-DCP residues in foods derived from 2,4-D resistant plants to be transformed *in vivo* into more highly chlorinated compounds that have greater toxicity.[89]

BASF is awaiting USDA deregulation of genetically engineered, imidazolinone-resistant soybeans (BPS-CV127-9).[90] Imazethapyr, one of the most widely used of the imidazolinone class of herbicides (a form of heterocyclic aromatic amine), has been associated with increased risk of bladder and colon cancers in farmers who use this herbicide.[91]

Could you elaborate on the external costs imposed on growers and the environment caused by the cultivation of herbicide-resistant crops?

As explained by Steve Smith in testimony before this Subcommittee on September 30, 2010, herbicide-resistant crops make it possible to apply large quantities of herbicides much later in the growing season than is possible otherwise. This facilitation of postemergence herbicide use means that neighboring growers will be vulnerable to crop injury from herbicide drift to a much greater extent than they were before HR crops were introduced. Costs incurred from crop injury by growers whose crops are not resistant to the pertinent herbicide are difficult to estimate, but could be substantial, especially in the case of a volatile herbicide like dicamba.

In order to defend their crops from herbicide drift damage, some and perhaps very many growers will purchase seed that is herbicide-resistant for defensive purposes, not because they want to make use of the trait and associated herbicide for weed control. In fact, this has already happened with Roundup Ready technology, and is happening now with Clearfield.

According to Arkansas weed consultant Ford Baldwin:

> "A lot of growers planted Roundup Ready corn in the beginning out of self defense. I looked at enough glyphosate drift on conventional corn to understand why. Most growers initially used conventional [i.e. non-glyphosate] herbicides in the Roundup Ready corn. Over time though the progression was to glyphosate-based programs and we lost a lot of the benefit of what could have been a great resistance management tool."[92]

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[88] Laurent, F. et al (2006). "Metabolism of [14C]-2,4-dichlorophenol in edible plants," Pest Management Science 62: 558-564.
[89] Wittsiepe, J. et al (2000). "Myeloperoxidase-catalyzed formation of PCDD/F from chlorophenols," Chemosphere 40: 963-968.
90 See petition 09-015-01p at http://www.aphis.usda.gov/biotechnology/not_reg.html.
[91] Koutros, S. et al (2009). "Heterocyclic aromatic amine pesticide use and human cancer risk: Results from the U.S. Agricultural Health Study," Int. J. Cancer 124: 1206-1212.
[92] Baldwin, F.L. (2010). "Herbicide drift damaging rice," Delta Farm Press, June 7, 2010. http://deltafarmpress.com/rice/herbicide-drift-damaging-rice-0607/.

Growers who bought Roundup Ready corn "out of self defense" paid a substantial premium (technology fee) for a trait they did not want. This is an external cost imposed by the Roundup Ready crop system, as it is used in the real world. Mr. Baldwin's article, however, focuses on an analogous situation with another herbicide-resistant crop, Clearfield rice. Clearfield is a non-GE type HR crop, resistant to the imidazolinone class of herbicides of the ALS inhibitor family. Newpath is BASF's formulation of imazethapyr.

> "My university counterparts have received more Newpath drift calls than normal as well. At present, four out of every five requests to come to a field involve some problem with Newpath on conventional rice. Most involve drift, but there have also been several cases of miscommunication between neighbors, and also between farmers and applicators on whether a particular field was Clearfield or conventional rice.
>
> These situations are never good. They have led to more talk of "defensive" planting of Clearfield rice. While it is easy for the good doctor to sit at his desk and say that is a bad idea, I have looked at several fields this year where I must admit I couldn't blame the farmer for his thinking."

Baldwin is clearly sympathetic to the crop injury and losses incurred by growers of conventional corn (due to Roundup drift from Roundup Ready fields) and conventional rice (due to Newpath drift from Clearfield rice fields). Yet he is no enemy of either technology. On the contrary, he regards them as useful tools for farmers, but tools that are having unfortunate and costly impacts on those who choose not to use them.

But the real thrust of the article has to do with the difficulty of using these herbicide-resistant crop systems in a ***sustainable*** manner, which is exacerbated by the drift issue. Growers initially bought Roundup Ready corn for defensive reasons: "Over time though the progression was to glyphosate-based programs ***and we lost a lot of the benefit of what could have been a great resistance management tool.***" What is the "great resistance management tool" that was lost? First, it was growing conventional corn with "conventional" [non-glyphosate] herbicides. That is, growers who planted Roundup Ready soybeans or cotton and then rotated to conventional corn were practicing "resistance management" by not using glyphosate for at least one year in their rotations. When they began switching to Roundup Ready corn for defensive reasons, they continued at first to use non-glyphosate herbicides with it, retaining the resistance management benefit. However, eventually they switched over to glyphosate with RR corn, increasing selection pressure for glyphosate-resistant weeds.

While Baldwin does not elaborate, it was probably economics that drove this decision. When a farmer pays a hefty technology fee for an RR traited seed, it makes economic sense to make use of it through using inexpensive glyphosate, rather than mostly more expensive "conventional" herbicides. If they hadn't been forced for "defensive" reasons to buy more expensive Roundup Ready corn, they probably would have continued planting cheaper conventional corn, which entails using conventional herbicides, and provides a resistance-managing "break" from continual glyphosate use.

Baldwin sees the same thing happening with Clearfield rice.

> "Most weed scientists I know feel we are growing more Clearfield rice now than is sustainable over time – unless we get a breakthrough in new technology. As we continue to increase the acres, most likely we are shortening the life of the technology. … If you plant every acre to Clearfield and continue to pound it with Newpath and Beyond, resistant barnyardgrass will be the most likely end result."

When adoption of these two HR crops – RR corn and Newpath rice – reached a certain tipping point, the crop-damaging drift that is a consistent feature of these HR technologies forced many other growers to unwillingly adopt them. This led to massive overreliance on the HR crop-associated herbicides, loss of the resistance management benefits provided by retaining a conventional crop in the rotation, and a spate of new herbicide-resistant weeds. The resistant weeds drive the demand for "new technology" in the form of a new herbicide or new herbicide-resistant crop – spurring yet another turn in the vicious spiral of increasing herbicide use and weed resistance. It's hard to imagine a more unsustainable technology than herbicide-resistant crop systems, at least as they are used in the real world, in the absence of regulation.

The only way to get off this pesticide treadmill is through integrated weed management that prioritizes non-chemical weed control measures. Unfortunately, mainstream American agriculture has been so thoroughly fixated on the chemical-only approach that most farmers, extension agents, and weed scientists have no clue where to begin. The silver lining in the HR weed epidemic may perhaps be that it is opening minds like that of Dr. Stanley Culpepper, weed scientist at the University of Georgia.

Culpepper is in the midst of a glyphosate-resistant pigweed epidemic that is rapidly making cotton-growing a near-impossible task in Georgia. In 2009, half of Georgia's one million acres of cotton had to be weeded by hand to remove this GR weed, at a cost of $11 million. Growers who until recently spent $25 per acre on weed control are now forced to spend $60 to $100 per acre. According to Culpepper: "We're talking survival, at least economically speaking, in some areas, because some growers aren't going to survive this."[93]

While Culpepper does not advocate giving up herbicides, he understands that the old approach of relying upon them exclusively is doomed to fail. Culpepper now recommends deep tillage to bury the resistant pigweed seed so that it will not sprout, which can reduce seed germination by up to 50%. He also recommends the planting of heavy cover crops like rye to provide a thick mat between crop rows that likewise reduces weed seed germination by as much as 50%. Together, the two techniques reduce the emergence of resistant pigweed that actually emerges, by up to 80%. The much reduced populations of weeds (resistant or not) that do emerge can then be managed with much lesser quantities of herbicides.

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[93] As quoted in: Haire, B. (2010). "Pigweed threatens Georgia cotton industry," Southeast Farm Press, July 6, 2010. http://southeastfarmpress.com/pigweed-threatens-georgia-cotton-industry.

While Dr. Culpepper appears to be a recent convert to the virtues of cover cropping and other non-chemical modes of weed control, other scientists have been working to improve and encourage adoption of such practices for many years, mostly without recognition and with far too little support from our pesticide-friendly U.S. Dept. of Agriculture. Dr. Adam Davis recently published a study showing the effectiveness of the "cover crop roller-crimper" for use in no-till soybean cultivation.[94] The roller-crimper is a heavy, flanged cylinder that is attached to a tractor and rolled over a cover crop like rye in the spring to kill it. The killed cover crop forms a heavy mat into which soybeans can be drilled, and which physically suppresses weed emergence, as discussed above. Some cover crops also exude allelopathic compounds into the soil that also inhibit the emergence of weeds.

Dr. Matt Liebman at Iowa State University has shown the great benefits to farmers from adopting more complex rotations involving three or more crops (including a winter cover crop or alfalfa), rather than the standard corn-soybean rotation.[95] In addition to decreasing use of (and expenditures on) synthetic nitrogen fertilizers by half to three-fourths, the more complex three- and four-year rotations reduced herbicide use by 76% and 82%, respectively, with weed suppression equivalent to the herbicide-intensive, conventional corn/soybean rotation, and yields that were equal or higher. These "low-external input" (LEI) systems were also more profitable than the conventional rotation, especially when considered in the absence of subsidies. Our perverted subsidy system, however, reduces the differences between the systems, and act as an impediment to adoption of such beneficial systems by American growers. A perhaps even more important factor, however, is the paucity of support for truly sustainable weed management systems such as this on the part of the U.S. Dept. of Agriculture, which like the major agrichemical-biotechnology firms is fixated on chemical-only approaches to weed control and farming in general.

We conclude by citing a very recent paper by Illinois agronomists, who are at ground zero of an extremely threatening outbreak of multiple herbicide-resistant waterhemp (*Amaranthus tuberculatus*). Patrick Tranel and colleagues have recently surveyed fields in Illinois and Missouri, and found a startlingly high proportion of waterhemp populations to be resistant to various combinations of two, three or even four different types of herbicide.[96] Waterhemp is regarded as one of the most threatening weeds to soybean and to a lesser extent corn cultivation in the Midwest, particularly in Illinois and Missouri. Waterhemp populations with individuals resistant to only one herbicide mode of action are practically a thing of the past. The majority of populations now contain multiple-herbicide resistant plants. Tranel and colleagues state that:

> "Herbicide resistance in *A. tuberculatus* appears to be on the threshold of becoming an unmanageable problem in soybean."

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[94] Davis, A.S. (2010). "Cover-Crop Roller–Crimper Contributes to Weed Management in No-Till Soybean," Weed Science 58: 300-309.

[95] Liebman, M. et al (2008). "Agronomic and Economic Performance Characteristics of Conventional and Low-External-Input Cropping Systems in the Central Corn Belt," Agronomy Journal 100: 600-610.

[96] Tranel, P.J. (2010). "Herbicide resistances in Amaranthus tuberculatus: A call for new options," Journal of Agricultural and Food Chemistry, DOI:10.1021/jf103797n.

They further warn that these weed populations will likely evolve resistance to glufosinate, one of the few postemergence herbicidal options available to growers afflicted with these multiple herbicide-resistant populations. This would occur with widespread deployment of glufosinate-resistant, LibertyLink soybeans, which at present are very little grown. If this happens, they warn, and no new soybean postemergence herbicides are commercialized:

> "Soybean production may not be practical in many Midwest U.S. fields."

The inability to economically cultivate the second most widely grown crop in America, a mainstay of Midwestern agriculture, would represent a huge cost imposed by unregulated use of HR crop systems on American farmers and U.S. agriculture as a whole. Clearly, USDA and land grant university agronomists must begin devoting serious attention to the sorts of sustainable, integrated weed control practices described above, which make non-chemical approaches a priority, and deemphasize the use of herbicides.