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Glyphosate effects on diseases of plants

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ABSTRACT

Glyphosate, N-(phosphonomethyl)glycine, is the most extensively used herbicide in the history of agriculture. Weed management programs in glyphosate resistant (GR) field crops have provided highly effective weed control, simplified management decisions, and given cleaner harvested products. However, this relatively simple, broad-spectrum, systemic herbicide can have extensive unintended effects on nutrient efficiency and disease severity, thereby threatening its agricultural sustainability. A significant increase in disease severity associated with the wide spread application of the glyphosate herbicide can be the result of direct glyphosate-induced weakening of plant defenses and increased pathogen population and virulence. Indirect effects of glyphosate on disease predisposition result from immobilization of specific micronutrients involved in disease resistance, reduced growth and vigor of the plant from accumulation of glyphosate in meristematic root, shoot, and reproductive tissues, altered physiological efficiency, or modification of the soil microflora affecting the availability of nutrients involved in physiological disease resistance. Strategies to ameliorate the predisposing effects of glyphosate on disease include judicious selection of herbicide application rates, micronutrient amendment, glyphosate detoxification in meristematic tissues and soil, changes in cultural practices to enhance micronutrient availability for plant uptake, and biological amendment with glyphosate-resistant microbes for nitrogen fixation and nutrient availability. Given that recommended doses of glyphosate are often many times higher than needed to control weeds, we believe the most prudent method to reduce the detrimental effects of glyphosate on GR crops will be to use this herbicide in as small a dose as practically needed. Such a frugal approach will not only curtail disease predisposition of GR crops, but will also benefit the grower and the environment.

1. Introduction

Changes in agricultural practices such as crop rotation, crop sequence, tillage, and fertility that affect the soil microflora or nutrient availability generally result in changes in disease expression (Datnoff et al., 2007; Englehard, 1989; Huber and Graham, 1999). This is commonly observed for soilborne diseases where only limited innate resistance is available in commercial cultivars so that cultural controls become important management practices to minimize the impact of these diseases. Threatening to make things worse in this regard is the introduction of herbicide-resistant crops (canola, corn, cotton, soybeans, alfalfa, etc.) that are now grown extensively throughout the world. This new trend in agriculture has increased the usage and intensity of specific herbicides while limiting genetic diversity in the specific crops that have been genetically modified.

Herbicides are known to increase specific plant diseases (Altman and Campbell, 1977; Hornby et al., 1998; Mekwatanakarn and Sivasithamparam, 1987), and several are reported to influence micronutrient availability (Evans et al., 2007; Huber et al., 2004, 2005). Micronutrients are the activators or inhibitors of many critical physiological functions. Thus, a deficiency or change in availability of these regulatory elements can greatly affect plant growth and resistance to diseases and pests (Datnoff et al., 2007). The virulence mechanism of some pathogens such as *Gaeumannomyces*, *Magnaporthe*, *Phymatotrichum*, *Corynespora*, and *Streptomyces* involves Mn oxidation at the infection site to compromise the plant's resistance mechanisms involving the shikimate pathway (Thompson and Huber, 2007). Isolates of these pathogens that cannot oxidize physiologically available Mn^{2+} to the non-available Mn^{4+} are avirulent and not able to cause significant tissue damage (Roseman et al., 1991). Production of the Mn oxidizing enzyme(s) occurs soon after spore germination and during epiphytic growth (Cheng, 2005; Schulze et al., 1995; Thompson et al., 2005). Environmental conditions that reduce the availability of micronutrients for plant uptake also predispose plants to disease (Huber and McCay-Buis, 1993; Huber and Graham, 1999; Thompson and Huber, 2007).

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Table 1
Some diseases increased in glyphosate weed control programs.

Plant	Disease	Pathogen	References
Apple	Canker	*Botryosphaeria dothidea*	Rosenberger and Fargione (2004)
Banana	Panama disease	*Fusarium oxysporum* f. sp. *cubense*	Harper (2007)
Barley	Root rot	*Magnaporthe grisea*	Smiley et al. (1992)
Bean	Anthracnose	*Colletotrichum lindemuthianum*	Johal and Rahe (1984, 1988, 1990)
Bean	Damping off, root rot	*Pythium* spp.	Johal and Rahe (1984)
Bean	Root rot	*Fusarium solani* f. sp. *phaseoli*	Harper (2007)
Bean	Hypocotyl rot	*Phytophthora megasperma*	Keen et al. (1982)
Canola	Crown rot	*Fusarium* spp.	Harper (2007)
Canola	Wilt	*Fusarium oxysporum*	Harper (2007), Large and McLaren (2002)
Citrus	Citrus variegated chlorosis	*Xylella fastidiosa*	Yamada (2006)
Citrus	Crown rot	*Phytophthora* spp.	Yamada (2006)
Cotton	Damping off	*Pythium* spp.	Harper (2007)
Cotton	Bunchy top	Manganese deficiency	Harper (2007)
Cotton	Wilt	*F. oxysporum* f. sp. *vasinfectum*	Harper (2007)
Grape	Black goo	*Phaeomoniella chlamydospora*	Harper (2007)
Melon	Root rot	*Monosporascus cannonbalus*	
Soybeans	Root rot	*Corynespora cassiicola*	Huber et al. (2005)
Soybeans	Target spot	*Corynespora cassiicola*	Huber et al. (2005)
Soybeans	Sudden Death Syndrome	*Fusarium solani* f. sp. *glycines*	Keen et al. (1982)
Soybeans	Root rot	*Phytophthora megasperma*	Keen et al. (1982)
Soybeans	Cyst nematode	*Heterodera glycines*	Geisler et al. (2002), Kremer et al. (2000)
Soybeans	White mold	*Sclerotinia sclerotiorum*	Harper (2007)
Sugar beet	Yellows	*Fusarium oxysporum* f. sp. *betae*	Larson et al. (2006)
Sugar beet	Root rot	*Rhizoctonia solani*	Larson et al. (2006)
Sugarcane	Decline	*Marasmius* spp.	Huber (unpublished)
Tomato	Crown root rot	Fusarium	Bramhall and Higgins (1988)
Tomato	Wilt	*Fusarium oxysporum* f. sp. *pisi*	Harper (2007)
Various	Canker	*Phytophthora* spp.	Harper (2007)
Weeds	Biocontrol	*Myrothecium verrucaria*	Boyette et al. (2006)
Wheat	Bare patch	*Rhizoctonia solani*	Harper (2007)
Wheat	Glume blotch	*Septoria* spp.	Harper (2007)
Wheat	Root rot	*Fusarium* spp.	Fernandez et al. (2005, 2007), Harper (2007)
Wheat	Head scab	*Fusarium graminearum*	Fernandez et al. (2005)
Wheat	Take-all	*Gaeumannomyces graminis*	Hornby et al. (1998)

The herbicide glyphosate, N-(phosphonomethyl)glycine, is a strong systemic metal chelator and was initially patented for that purpose (Bromilow et al., 1993). Its herbicidal action is by chelating with Mn, a cofactor for the 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase enzyme in the shikimate pathway, to inhibit this metabolic pathway of plants and many microorganisms (Cerdeira and Duke, 2006; Grossbard and Atkinson, 1985; Jaworski, 1972). Many cations chelate with glyphosate, thus reducing its herbicidal efficacy (Bernards et al., 2005; Hickman et al., 2002). Plants with a compromised shikimate metabolism are predisposed to various plant pathogens (Johal and Rahe, 1988; Rahe et al., 1990), and glyphosate is patented as a synergist for mycoherbicides to enhance the virulence and pathogenicity of organisms used for biological weed control (Boyette et al., 2006; Duke and Cerdeira, 2005). The synergistic activity of glyphosate weed control in predisposing plants to infectious organisms has been observed for many diseases (Table 1), and the extensive use of glyphosate in agriculture is a significant factor in the increased severity or "reemergence" of diseases once considered efficiently managed.

The extensive adoption of Roundup Ready® crops such as soybeans, canola, cotton, and corn has intensified the application of glyphosate in these production systems. The applied glyphosate is readily translocated to roots and released throughout the rhizosphere in root exudates of Roundup Ready® plants as well as glyphosate-sensitive plants (Bromilow et al., 1993; Grossbard and Atkinson, 1985). The toxic microbial effects of glyphosate are cumulative with continued use so that Mn deficiency is now observed in areas that were previously considered Mn sufficient because of reduced populations of Mn-reducing soil organisms (Huber, unpublished). The presence of the glyphosate-resistance gene in corn and soybeans also reduces Mn uptake and physiological efficiency (Dodds et al., 2002a,b,c; Gordon, 2006; Reichenberger, 2007). Along with glyphosate-induced Mn deficiency, there has been a gradual recognition of increased disease severity (Harper, 2007; Larson et al., 2006). A few examples are presented to illustrate this relationship.

2. Some diseases increased by glyphosate

2.1. Corynespora root rot of soybean

The damage from Corynespora root rot, previously considered minor, may become economically damaging in Roundup Ready® soybeans since application of glyphosate to Roundup Ready® soybeans greatly increases severity of this disease (Fig. 1). This fungal root rot is more severe when glyphosate is applied to soybeans under weedy conditions even though the weeds may not be hosts for *Corynespora cassiicola*. The weeds serve to translocate and release more glyphosate into the rhizosphere environment to reduce the population of Mn-reducing organisms and increase Mn-oxidizing organisms. This change in soil biology limits manganese availability for plant uptake and active defense reactions, and acts synergistically with *Corynespora* to increase disease (Huber et al., 2005).

2.2. Take-all of cereal crops

The most comprehensive understanding of the interaction of micronutrients influenced by glyphosate and disease is with the take-all disease of cereals. Increased take-all of cereals after a pre-plant "burn-down" use of glyphosate has been recognized for over 15 years (Hornby et al., 1998). Take-all is also increased when glyphosate is applied to Roundup Ready® soybeans the preceding year compared with the use of a non-glyphosate herbicide (Fig. 2). All of the conditions known to affect Mn availability are inversely related to the severity of take-all (and other diseases, Table 2) so that