Weeds, Herbicides and Plant Disease Management

Babak Pakdaman Sardrood and Ebrahim Mohammadi Goltapeh

Abstract Plants growing unwantedly in an agro-ecosystem are defined as weeds. Weeds modify crop plant growth, development and yield, not only through their competition for light, water, nutrients, but also through the establishment of ecological niches suitable for the growth and development of plant pathogens and pests, and the role they play as the host of hazardous organisms. Furthermore, some weeds produce allelopathic compounds that directly affect the growth and development of agricultural crops and even lead to their death. Such toxic compounds may be found in the leaf, flower, fruit, root, rhizome, and seed of the producing plants. To control weeds, various mechanical, agricultural, biological and chemical approaches are applied. Chemical control with herbicides is considered as the most easiest and attractive method applied in the control of weeds. Recently, compounds of biological origin have been introduced against weeds, of which mycoherbicides, of fungal origin, are the most famous group. Herbicides can impact the growth, development, reproduction, distribution, and survival of plant pathogens in several ways. Therefore, their rational use is considered as a vital part in integrated plant disease management programs.

Keywords Herbicide · Plant disease · Integrated plant disease management
Weed · Control

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1 Introduction

The plants unwantedly growing in fields and gardens are called weeds. Weeds as a part of agro-ecosystems play various roles in the fluctuation of plant disease risk and are important from several different view points. While most of weeds may help in disease risk diminishment, however, there may be some weeds that considerably affect crop plant vigor and its potential for growth and development throughout its germination of seed till the end of its life in various direct and indirect manners, and necessitate weed control programs. The easiest strategy to control weeds is the application of herbicides that if applied in a rational and aware manner, can help in disease risk decrease. Therefore, any action against weeds shall be taken with enough knowledge of their direct and indirect impacts on weed-weed, weed-crop, weed-pathogen, and weed-vector, weed-beneficial organisms, pathogen-biological control agent interactions, and it is very clear that such a high level of disease management in a stable agricultural system necessitates detailed information of agro-ecosystem ecology as well as more innovative work on the development of new generations of herbicides that are environmentally non-hazarduos and toxicologically safe for crop plant.

The effects of herbicides on environment was the subject of the book "Herbicides and Environment" edited by Kortekamp (2011), and the impact of herbicides on soil biology and function has been reviewed by Rose et al. (2016). The application of herbicides requires more precise information not on groups of microorganisms, but on relevant species in agriculture and plant pathology, and their inter-relationships with other important species (such as weeds, host plant, vectors, beneficial microorganisms, biological control agents, and pathogens). Here, we intend to imply to the importance of the weeds in the increase of disease risk, and to the capacity of different herbicides of various classes for the applications in integrated disease management programs. The necessity of such a review was severely sensed because of the increased crises with the environmental pollutions and health risks related to the residues from the applied agrochemicals left on/in the food stuff ready for human as well as livestock consumption, and mounting list of pesticide resistance development in hazardous organisms, as well as the current global trend towards the establishment of stable agricultural systems. There are increasing reports of the involvement of cellular membrane Adenosine triphosphate-Binding Cassette transporters (ABC-transporters) in the development and appearance of resistant adapted microorganisms (Del Sorbo et al. 2000). Herbicides of different molecular topology and structure, and of antimicrobial activity can play a considerable role in pesticide resistance management, especially when they are applied in combination with biological control agents.

The widespread use of herbicides in agriculture fields raises serious and often controversial issues related to ambient soil pollution and changes in biological balance. Herbicides act as a "stress factor", and change soil microbiological balance. In a study on the changes of soil microbiology after herbicidal treatment, soil samples were collected from a cambic chernozem at depth ranges of 0–20 cm from

the experimental field. Herbicides used were clorsulfuron (Dacsulfuron® with doses ranging from 0.005 to 0.035 mg g^{-1} soil) and MCPB-Na also known as sodium 4-(4-chloro-2-methylphenoxy)butanoate (Butoxone®, with doses ranging from 0.5 to 3.5 MCPB-Na of 400 g L^{-1} g^{-1} soil). Potato Glucose Agar (PGA) was used for the isolation and identification of fungal colonies. Determination of mold colonies was performed up to genus level using identification keys. The following types of molds were identified in the control sample: *Penicillium* sp., *Humicola* sp., *Actinomucor* sp., *Aspergillus* sp., *Fusarium* sp. and *Mucor* sp. In the experimental variants treated with Dacsulfuron®, the presence of additional genera was noticed: *Trichoderma*, *Cladosporium*, *Chaetomium*, *Botrytis* and *Curvularia*. By comparison with the control samples, in the variants treated with Butaxone®, additional presence of the genus *Rhizomucor* was found. Some types were found in the soil samples treated with increasing doses of Dacsulfuron® and Butaxone®, others reduced their number or disappeared as the result of their sensitivity to the toxic action of xenobiotics. These xenobiotic substances disturbed soil biology and microbiology, and negatively influenced the quality and quantity of soil fungi communities. Dacsulfuronu® had lower toxicity on soil fungi communities than Butoxone® (Craciun et al. 2013). This was due to the large number of genera that occur in experimental variants treated with Dacsulfuron®. This information was confirmed by analysis of the diversity expressed by the Shannon-Weaver index (Shannon and Weaver 1949) which indicated a value of 0.84 for dacsufuron and 0.63 for Butaxone®.

Praveena et al. (2007) reported that herbicides could either negatively affect the growth and sporulation of the fungus *Fusarium pallidoroseum*, known as a potential biocontrol agent of water hyacinth [*Eichhornia crassipes* (Mart.) Solms], or had no significant effect on the fungus. They evaluated the in vitro effect of different herbicides including paraquat, pretilachlor, glyphosate, 2,4-D, and 2,4-D/anilofos mixture on *Fusarium pallidoroseum* colonies. Three treatments of the tested herbicides (pretilachlor, 2,4-D and 2,4-D/anilofos mixture) inhibited fungal growth and sporulation completely, whereas paraquat and glyphosate exhibited a relatively low level of inhibitory. Additionally, when lower concentration of the herbicides was used, paraquat, glyphosate, 2,4-D and 2,4-D/anilofos mixture could support the fungal growth (Praveena et al. 2007). These findings highlight the importance of herbicide dosage applied in order to suppress the weed‘s development. Highly concentrated herbicides not only damage the environment, but also might reduce the biocontrol effect of fungal agents.

2 The Effect of Weeds on Plant Diseases

Weeds as unwanted components of an agroecosystem interact with cultivated plants, therefore, the control of weeds is considered as an inseparatable part of the management of plant pathogens (Wisler and Norris 2005). Weeds can influence on

plant diseases through their direct and indirect effects on the components of disease triangle (host plant, pathogen, and biotic as well as abiotic environmental factors):

2.1 Direct Effect of Weeds on Plant Physiology

Weeds generally exert their hazardous effects on crop plant growth through competition for the limited water supplies, limited rates of available nutrients, space and light. Fast growing weeds can germinate and emerge earlier and reduce crop plant germination and growth through the interception of sun beams and use of water supplies. For example, the seeds of the weeds like *Datura stramonium*, and *Abutilon theophrasti* (Fig. 1) germinate synchronous to soybean seed germination, grow fast and form their well-developed canopy over soybean seedlings, the result is clear: soybean seedlings will not be able to catch light required for photosynthesis, and will consequently be etiolated and ultimately die (Merrill and Lembi 2009). In sugarcane fields, the increased growth and development of weed grasses leads to the decreased and weak growth and development of sugarcane, so that the weakened plants are difficult to discriminate from those suffering from ratoon stunting disease.

Fig. 1 Velvetleaf (*Abutilon theophrasti*), an allelopathic malvaceaous weed with velvet-like heart-shaped leaves, yellow flowers and capsular button-like fruits (*Source* http://nyc.books.plantsofsuburbia.com/) (Color figure online)

Additionally, some of weed plants actively produce and secrete toxic chemicals that after their release to soil environment impose their inhibitory effects on crop seed germination. Such a phenomenon first discovered by H. Molisch in 1937, was called "allelopathy". Dependant on the level of phytotoxicity of the secreted compounds and the concentration of the produced chemicals, environmental conditions, and the susceptibility of the crop plant in the given stage in the life cycle, it can end to the postponed germination and emergence of the seedlings, or to the death of the sensitive embryo of the crop seed or its germlings. Such allelopathic compounds may be found in the leaves, roots, flowers, fruits, and rhizomes of the producing weeds (Putnam and Weston 1986). Fischer (1986) reported that the sesquiterpene lactones found in the water after washing of the leaves of the plants belonged to the families Asteraceae, Apiaceae, and Magnuliaceae were inhibitory to the seed germination of other plants. In mixed culture systems, the allelopathic compound, juglone produced and secreted by the leaves of walnut (*Juglans regia*) trees can suppress the germination of herbaceous plant seeds.

In the evaluation of the allelopathic effects of the weeds *Chenopodium album*, *Amaranthus retroflexus* (Fig. 2) and *Cynodon dactylon* on the rapeseed germination and seedling growth, Yarnia and Rezaei (2006) found that root and shoot extracts of *A. retroflexus* and *C. dactylon* resulted in the 65% reduced seedling height, while that of *C. album* caused 38% reduction. Rapeseed seed germination percentage was decreased 30% with *C. album* extract, however, more than 90% with the extracts of other weeds. In a similar study with soybean, Farajzadeh et al. (2006) concluded that the extract prepared from the weed *C. album* reduced soybean height of seedling more than 80% (compared with control) and that of other weeds *A. retroflexus* and *C. dactylon* caused 60% reduction. They also found that soybean seed germination was decreased 70% as the result of the treatment with the extract of *C. album*, and 30% with the extracts of other two weeds. Rezaei et al. (2006) obtained similar results with the extracts of above-mentioned weeds and safflower.

Weeds that secrete their allelopathic compounds from the root system near to the depth of soil, or those that produce water-soluble compounds easily washed from the leaves and penetrate to the soil, where crop seeds are laid, will expectedly be of the most inhibitory influence on crop seed, however, the major allelopathy seems to occur by the chemical compounds released from plant residues as the result of microbial activity after weed death, the effect that has been reported by several authors (Patrick 1971; Rice 1984; Barnes et al. 1986). Although these chemicals may be non-toxic in feature, however, can potentially lead to considerable localized changes in soil pH and/other factors that can end to phytotoxic effects imposed on crop seed, germlings and seedlings. Rhizomes and residues of the graminaceous weed *Agropyron repens* affect on the growth of small-grain cereals (Ohman and Kommedahl 1960) and corn (Bandeen and Buchholtz 1967). Weeds like *Sorghum halpense*, *Setaria viridis*, *Digitaria* spp., and *Helianthus annus* impose their allelopathic effects on agronomical crops and convert environmental conditions to those favorable for their own growth and development.

Fig. 2 *Amaranthus retroflexus* (*Source* http://www.narodrecept.ru/)

2.2 *Creation of Favorable Conditions for Pathogen Growth and Development*

Some of plant pathogens require moist and humid conditions for their growth and development, and the epidemics of these diseases are in close relation to wet and high relative humid conditions and the primary foci of such diseases are found in the points of fields and gardens, where such conditions are provided. Weed plants especially those with large and broad leaves with higher densities of stomata absorb soil water and evapotranspirate it to the air, and provide the conditions conducive for pathogens.

Oomycetous airborne microorganisms like those causal of downy mildews are a good example for such a topic. Grapevine downy mildew disease caused by *Plasmopara viticola* is well encountered wherever moist conditions are provided by any biotic or abiotic factor such as irrigation water furrows, and weeds. Grey mold disease of grapes caused by *Botrytis cinerea* is more severe under humid conditions, and the same is true with the rot and blight diseases induced by the generalist pathogen *Sclerotinia sclerotiorum* (Agrios 2005). Powdery mildews another group of plant diseases develop from their foci in the points where high relative humidity exists (Agrios 2005). Bacterial pathogens need free water layer and water soaked

leaves in order to penetrate plants and cause disease (Agrios 2005), and the relative high humidity provided by weeds creates localized climates that may end to dew drop formation on sensitive host plants in dawns. Weeds grown under trees and in their crown vicinity provide humid conditions that predispose trees to the attacks by various fungi and fungus-like microorganisms. *Phytophthora* species and basidiomycetous wood-rotters like *Armillariella* spp. are among famous pathogens of tree plants to exemplify.

2.3 The Effect of Weeds on the Dissemination of Pathogens

Weeds can play their role in pathogen dissemination and disease spread by two ways, directly and indirectly.

2.3.1 Direct Effect of Weeds on the Dissemination of Pathogens

In a direct way weeds may help pathogens to be distributed through their organs like seeds which have been infected or contaminated by the pathogens such as viruses. In fact, most weed are disseminated through their wind-born seeds (Sorensen 1985), and seed dissemination crosses various groups of pathogens (Gamliel 2008). Most of the dangerous fungal diseases of sugarcane such as red rot caused by *Colletotrichum falcatum* and smut caused by *Ustilago scitaminea* survive on perennial host grasses as Kan grass (*Saccharum spontaneum*) and Munj grass (*S. munja*) often grown around sugarcane fields. Grassy weeds like wild oats (*Avena fatua* L.) and ryegrass (*Lolium* spp.) maintain cereal cyst nematode (*Heterodera avenae* Woll.) populations and should be controlled. The seed-gall nematode *Anguina tritici* parasitizes rye and the wheat ancestors, emmer, spelt, and *Aegilops* (Wiese 1987). The root-knot nematodes, *Meloidogyne* species infect an abundant number of the plants belonged to the family Solanaceae, and the produced propagules are transferred through water and soil translocations to the plots cultivated with vegetables and cucurbits. Some weeds grown among more disease resistant crop plants may be more susceptible to a pathogenic invasion, and can be infected earlier and increase the potential of pathogen inoculum that leads to further temporal and spatial distribution of the disease. The increased potential of inoculum can lead to spill over of disease and break of resistance of crops (Keesing et al. 2006). The fungal pathogen of brown leaf spot of paddy, *Bipolaris oryzae* is found also on the alternative host plants *Oryza montana* as well as *Leersia hexandra* in nature (Chattopadhyay and Chakrabarti 1953). The wounds resulted from the compact growth and harsh contacts can potentially help disease spatial progress. *Tilletia controversa*, the wheat dwarf bunt fungus can infect wheatgrass *Agropyron repens* and a variety of other wild grasses. *Bromus*, *Dactylis*, and *Poa* species are infected by *Cephalosporium gramineum*, the only true vascular fungal pathogen of wheat that cause a soilborn disease called wheat cephalosporium stripe.

Pathogens either of obligatory or facultative, specialized or non-specialized types are usually of vast host ranges. Some of the pathogens are found restricted to a plant species while others can infect very abundant plant species of different families. Highly specialized pathogens such as rust pathogens are of physiological races and they may not essentially attack from infected grasses to cultivated wheat or barley plants. However, wheat black stem rust pathogen *Puccinia graminis* f. sp. *tritici* a heteroeceous macrocyclic rust fungus infects quackgrass and wild oats as, and spreads over wheat, barley and oat cultivars grown in the farms. Thus, by this means, weeds can act as foci for the distribution and spread of the diseases. *P. g*. f. sp. *striiformis* another rust fungus causes stripe rust or yellow rust as well as glume rust on barley, wheat, and other grasses including perennial grasses that play an important role in disease establishment and development as the reservoirs for the fungus. The main problem occurs with the non-specialized pathogens of vast host ranges that take advantage of weeds, wild hosts as well as self-sown host plants for their own survival, spread and reproduction in the presence or absence of their main host plant. The direct tranfer of diseases may occur through root and/or shoot bridges where infected weeds directly involve in the spatial distribution of plant diseases. With the increase of host densities, contacts between hosts will also increase and allow more opportunities for the transmission of pathogen, which is very important in the transfer of some viruses as well as fungi and bacteria. Furthermore, the probability of a pathogen death in a host population will decrease at high population densities (Anderson and May 1991), which is of high importance in the spread of the diseases caused by biotrophic pathogens such as powdery mildews as well as facultatively saprophytic pathogens such as *Phytophthora* species which are very weak saprophytes in the absence of their hosts. Weeds may act as green bridges and facilitate the hastened development of soil-born pathogens. This is most important with the pathogens which are of vast host range. A noticeable classic example of such pathogens may be the ascomycetous fungus *Sclerotinia sclerotiorum* that invades more than 400 plant species of more than 75 plant families (Boland and Hall 1994). Similarly, *Sclerotium rolfsii* has an extensive host range, and at least 500 species in 100 families are susceptible. The most common hosts are the legumes, crucifers, and cucurbits. The fungus persists in many weed hosts as well (Punja 1985). Cucumber mosaic virus (CMV), some of powdery mildew fungi (such as *Erysiphe polygoni*), and root-knot nematodes (*Meloidogyne* spp.) each are capable to infect more than 700 host plant species. *Podosphaera xanthii* (syn. *Sphaerotheca fuliginea*; powdery mildew of cucurbits) infects 570 host plant species. The host range of the root rot causal fungus, *Phymatotrichum omnivorum* includes at least 1300 plant species (Taubenhaus and Ezekiel 1936). The main danger of the presence of additional hosts is the transfer of plant pathogens from a season to another season, as well as the provision of the conditions required for the propagation of inoculum and its development to the levels needed for an epidemic occurrence of the disease on the main culture in the early season. *P. xanthii* overwinters on wild cucurbits and also on the cultivated shade plants out of the season. Such a mechanisms seems be true with the viruses of cucurbitaceae and many other host plants. The soil-born take-all fungus

Gaeumannomyces graminis var. *tritici* although specialized for the pathogenicity on wheat, however, it also invades bromegrass (*Bromus* spp.), wheat grass, and quackgrass (*Agropyron* spp.), and the weeds can play the role of green bridges to facilitate and accelerate fungal spread in the area, wherever wheat plants have not form a dense cover of vegetation (Singh 2001).

Some pathogens may be transmitted via the absorptive organs (haustoria) of parasitic weeds involved in the suction of host plant phloem sap. Dodder (*Cuscuta*), a twining yellow, orange, sometimes tinged with purple, red, and occasionally almost white plant can feed on an infected weed and spread over crop plants in the close vicinity of its first host weed. Dodder with the very thin and threadlike or relatively stout stems is classified as a member of the Morning-Glory Family, convolvulaceae (Garcia et al. 2014). *Cuscuta* spp. have a broad host range, including many cultivated crops such as tomatoes, tobacco, clover, and dicotyledonous weeds as well as trees and shrubs, but only a few grasses or monocotyledonous weeds (Dawson et al. 1994; Albert et al. 2008). Some other dodders such as *Cuscuta campestris* (Fig. 3), and *C. pentagona* are agriculturally important, found worldwide, and can synchronously infect a broad range of higher plants (Lanini and Kogan 2005). Dodders parasitize various kinds of wild and cultivated plants, and is especially destructive to agronomic plants (alfalfa, lespedeza, flax, clover and potatoes) as well as ornamentals (chrysanthemum, dahlia, helenium, virginia-creeper, trumpet-vine, English ivy and petunias). Water, minerals and carbohydrates are absorbed from the host through haustoria (modified adventitious roots) that press up against the stem of the host plant and penetrate its tissue. Although dodder rarely ends to host plant death and leads to stunting of its host growth, however, it transfers a range of diseases. In addition to leaf hoppers, dodder can transmit phytoplasmas, the cause of more than 200 so-called yellows diseases (Swift 2010). Bennett (1940) indicated that dodder would transmit viruses from a plant to another. The transmission of the viruses probably takes place through plasmodesmata that provide transient connections sites between dodder haustorial tip and host cell cytoplasm.

Fig. 3 Vines of dodder (*Cuscuta campestris*) turned around green host stem. Note to the clusters of small white flowers each harboring five white triangular petals (*Source* http://smmflowers.org/)

Dodder-vectored transmission is similar to grafting in some aspects, however, graft compatibility is limited to quite closely related plants, usually within a genus. Dodder, on the other hand, can be used to transmit a virus between distantly related plants (Desjardins et al. 1969). While some viruses can be replicated inside dodder body (such as cucumber mosaic virus) others are transmitted in a passive manner with no replication (such as tobacco mosaic virus) (Bennett 1940). However, capability of a virus to replicate inside a dodder does not mean its ability to be transmitted by the proliferative dodder. Grapevine Leafroll associated virus 7 (GLRaV-7) can be transmitted from one host to another by *Cuscuta reflexa* and *C. europea* in which it appeared to replicate, however despite its replication in *C. campestris*, it could not be transmitted to another host via this dodder species (Mikona and Jelkmann 2010). Dodders may sometimes harbor an unsuspected virus. It has been indicated that symptomless *C. californica* is frequently infected with a virus that causes serious diseases in several unrelated plant species. The virus was called as dodder latent mosaic virus (Bennett 1944). Dodder is regarded as an insignificant factor in the transmission of economically important viruses in the field, and has rarely been used in experimental work in recent times (Hull 2014). Dodder has been shown to spread the yellows disease pear decline, aster yellows, tomato big bud, *Vinca* virescence and elm phloem necrosis. Furthermore, phloem-inhabiting "rickettsia-like" bacteria have been found to be present in dodder (Swift 2010). It is not known if the viruses can be transmitted from the holoparasitic plant bromrape (*Orobanche* sp., Fig. 4) to other hosts of this parasitic plant (Hull 2014). Cucumber mosaic virus (CMV), tomato mosaic virus (ToMV), potato virus Y (PVY), and tomato yellow leaf curl virus (TYLCV) can be translocated from infected hosts to the broomrape. CMV and possibly other viruses replicate in the parasitic plant *Phelipanche aegyptiaca* (Gal-On et al. 2009). Horizontal gene transfer between plants has been reported to take place by another parasitic plant, witchweed, *Striga hermonthica* (Yoshida et al. 2010; Fig. 4). Although there are no records of virus transmission by this important parasite in tropical countries, the capability of horizontal gene transfer indicates the parasitic weed potential as a vector (Hull 2014). The witchweed is a hemiparasitic plant that parasitizes the roots of cereals and other poaceous plants. Millets (*Panicum* spp.), finger millet (*Eleusine coracana*), rice (*Oryza sativa*), sorghum (*Sorghum bicolor*), sugarcane (*Saccharum officinarum*), and maize (*Zea mays*) are regarded as the main hosts of witchweed (www.cabi.org/isc/datasheet/51849).

2.3.2 Indirect Effect of Weeds on the Dissemination of Pathogens

Indirect role of weeds in plant disease spread is through their hosting for the organisms that feed on them and can potentially act as vectors of the pathogens, however, if weed is an alternative host for the vector but not for the pathogen, then its presence may lure vectors away from the focal host, and decrease transmission (Keesing et al. 2006). In some vector-born diseases, several vector species can transmit the pathogen, therefore, infectious diseases are considered as inherently

Fig. 4 Two examples of weeds that parasitize root of crop plants (Left) *Orobanche* sp. on tobacco (*Source* http://ephytia.inra.fr/); and (right) *Striga hermonthica* grown by its host plant, rice (*Source* Mr. Eng. HAJM Toussaint from Wageningen University & Research)

ecological systems that involve the interactions among small to large networks of species. It is then not so surprising that the potential connection between species diversity and diseases transmission has long been recognized (Keesing et al. 2006). Such a diversity can for instance be resulted either from the intercropping of different agricultural plants, or the diversity of different vectors and host weeds grown among crops, etc. The connection between diversity and disease was also recognized by Elton (1958), who suggested the possibility of plant disease amelioration in complex ecosystems provided that the existent complexity reduces host plant density for a disease. However, there are controversial results obtained by different researchers, for instance, various empirical and modeling investigations have suggested the reduction of disease risk as the consequence of the increased species diversity that occurs through regulation of the abundance of an important host species (Burdon and Chilvers 1982; Rudolf and Antonovics 2005) or through redistribution of vector meals in the case of the diseases transmitted by vectors (Van Buskirk and Ostfeld 1995; Norman et al. 1999; LoGiudice et al. 2003). However, other studies have suggested the increased disease risk resulted from the increased diversity if the added species functions as alternative sources of infection, or if the added species increase vector numbers or activity through provision of additional sources of vector meals (Holt and Pickering 1985; Norman et al. 1999; Gilbert et al. 2001; Schmidt and Ostfeld 2001; Saul 2003; Dobson 2004). High host densities are

likely to increase transmission rates both within the domestic populations and between domestic and wild or weed species (Burdon and Chilvers 1982; Anderson and May 1991; Gilbert 2002). In most multiple host systems, all host species are not equal in their epidemiologically key characters such as resistance, tolerance, and vector preference (Daszak et al. 2000; Woolhouse et al. 2001; LoGiudice et al. 2003), and consequently, the rates of transmission of a generalist pathogen with a wide host range within and between different host species are generally highly heterogenous and asymmetric (Woolhouse et al. 2001).

The effects of 21 weeds acting as hosts of tomato spotted wilt virus (TSWV), temperature, thrips population and diversity on disease progress in chrysanthemum cv. Polaris were studied. Under greenhouse conditions, only *Taraxacum officinale*, *Bidens* sp., *Reseda luteola* and *Mirabilis jalapa* were hosts for TSWV. Of 38 weeds species in the area surrounding a chrysanthemum field, *Tithonia tubaeformis* and *R. luteola* had the highest populations of adult and immature thrips. These weeds, as well as *M. jalapa* had an extensive seasonal distribution and may play a key role in the disease progress. Seventeen thrips species belonging to the genera *Bravothrips*, *Thrips* and *Frankliniella* were identified on weed flowers, with *Frankliniella occidentalis* (FOC; Fig. 5) representing 9.5% of all thrips identified. Of 123 thrips collected from chrysanthemum inflorescences, 9.75% were FOC, and only 2.5% of them transmitted TSWV. Of all the thrips species collected from chrysanthemum flowers in the field, only FOC was capable of transmitting TSWV. On 120 experimental plots established at two sites, with three transplanting dates (June, July and August), it was estimated that 1.25% of the chrysanthemum cuttings were already infected with TSWV when transplanted. Secondary spread, vectored by FOC, occurred only for the earliest transplanting date and resulted in a further 2.36% disease incidence.

Fig. 5 *Frankliniella occidentalis*, the thrips that vectors tomato spotted wilt virus, and the disease symptoms on fruits (*Source* Russell IPM Ltd, and Whitney Cranshaw from Colorado State University)

In heterogenous populations of host species, wild plants as well as weeds may act as reservoir species that maintain a relatively high population of pathogen. In such a situation, the pathogen typically reaches high prevalence in the reservoir and after then spills over into the other host maybe an agricultural crop, a process called "the spillover effect" or "pathogen spillover" (Daszak et al. 2000). The pathogen spillover may lead to the reduced abundance of the non-reservoir hosts such as of the interest agricultural crop, a pathogen-mediated phenomenon that is called "apparent competition" (Power and Mitchell 2004). There are increasing documents that support the theoretical predictions that such a phenomenon can control the outcome of the interactions among wild weed plants and crop plants that share a common natural enemy (Holt and Lawton 1994; Alexander and Holt 1998; Hudson and Greenman 1998). The pathogen might be of different kingdoms. Ergot caused by the fungus *Claviceps purpurea* is a historical disease that is repeatedly encountered on wild grasses beside its cereal hosts like wheat, rye, barley and oats, and insect vectors attracted to the secreted fungal honey dew deliver fungal conidia produced by its asexual form *Sphacelia segetum*. Grasses, especially *Agropyron repens* and *Phalaris arundinacea* are susceptible hosts for wheat chlorotic streak virus as well as for its plant hopper vector *Laodelphax striatellus* (Fallén). Maize streak virus (MSV), a *Mastrevirus* indigenous to Africa (Willment et al. 2001; Fajemisin 2003; Karavina 2014), is one of at least eight viruses that cause significant agronomic losses in maize (*Zea mays* L.) worldwide (Redinbaugh et al. 2004). It causes serious maize streak disease (MSD) of corn in sub-Saharan Africa (Martin and Shepherd 2009) and south-east Asia. A *Pennisetum* strain of MSV infects wheat in India, and others infect sugarcane, guinea grass (*Panicum maximum* Jacq.), oats, barley, and certain wild grasses. The virus is circulatively transmitted by the species of sap-feeding *Cicadulina* leaf hoppers (Homoptera: Cicadellidae: Deltocephalinae: Macrostelini) (Bosque-Perez 2000), and despite all stages of leaf hoppers transmit MSV, but transovarial passage has not been demonstrated. The virus (like other African streak viruses) is neither seed nor mechanically transmitible (Martin et al. 2008). *Cicadulina* species (Fig. 6) are generally considered as grassland leafhoppers present in wild and pasture throughout the year, but can migrate in large numbers to maize (Page et al. 1999). Agropyron mosaic virus (AgMV), a *Rymovirus* infects wheat, rye, barley, and grasses like *Elytrigia intermedia*, *Elytrigia repens* (a maintenance and propagation host in addition to bread wheat), and *Lophopyrum elongatum*. The virus is transmitted by the mite *Abacarus hystrix* distributed widely by air flows, however, it is not transmitted by seed, pollen, and even bu other mites such as *Aculus mckenziei*, and *Eriophyes tulipae* (Brunt et al. 1996; Dallwitz 1980; Dallwitz et al. 1993).

Aphids, white flies, thrips, leaf-hoppers, tree-hoppers, beetles, nematodes and mites can feed and survive on their diseased weed hosts, and increase their population feeding on them, and then transfer pathogens to crop plants. Aphids and cabbage root maggots live on mustard weed, and then attack cabbage, cauliflower, turnip, and radish plants. The barley yellow dwarf virus (BYDV) is a phloem-limited *Luteovirus* obligately transmitted in a persistent manner by several species of aphids feeding on wild grasses (Miller and Rasochova 1997) such as wild

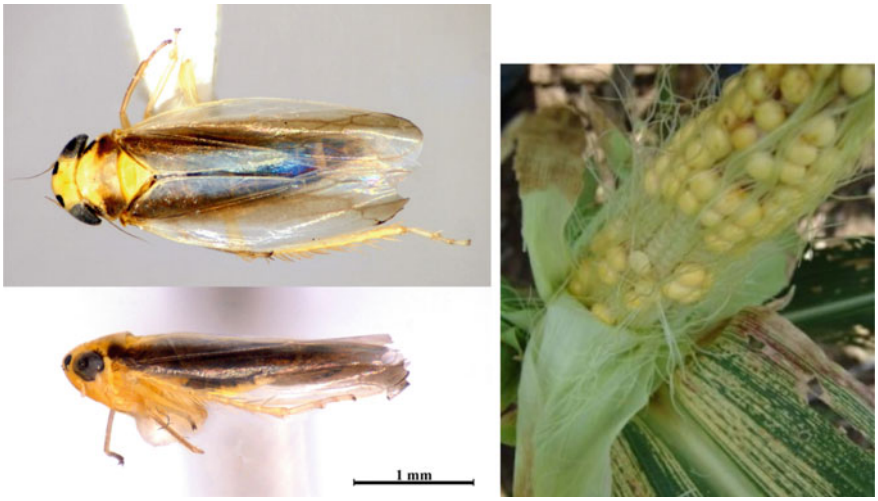


Fig. 6 From left to right: the African maize leafhopper, *Cicadulina mbila* (*Source* www.dpi.nsw.gov.au/keys/leafhop/species/cmbila.htm; credited by Holger Löcker) the most common and ubiquitous leaf hopper that transmits maize streak virus (Ruppel 1965) with a capacity higher than any other leafhopper species; Maize streak virus symptoms developed as chlorotik streaks on leaves and deformed cob development (*Source* Blackwell Publishing Ltd 2007; credited by Dr. F. Kloppers)

oats, *Avena fatua* a particularly appropriate host of BYDV that exhibits high sensitivity to infection and harbors high concentrations of the virus when infected (Power and Mitchell 2004). Power and Mitchell (2004) found no significant effect of the number of host species (richness) in the plant community on the prevalence of BYDV, but they perceived the significantly different prevalence of the virus among host communities. In particular their planned contrast indicated that the communities with *A. fatua* were of considerably higher BYDV prevalence compared with those without *A. fatua*, a result suggesting less importance of host diversity for the viral disease epidemiology versus high importance of the existence of one highly susceptible suitable host species. Furthermore, they found that in the communities with *A. fatua*, the proportion of the community made up by *A. fatua* was of no significant impact on virus prevalence among all other species, however, the simple presence of *A. fatua* affected virus prevalence in other plant species. The presence of *A. fatua* led to greatly increased prevalence of the viral pathogen in several other species demonstrating the pathogen spillover and illustrating the crucial role of host community structure in the control of the generalist pathogen dynamics. Additionally, virus spillover from wild oats decreased the abundance of two other host species *Lolium multiflorum* and *Digitaria sanguinalis* through pathogen-mediated apparent competition. Such studies indicate the role of reservoir weed species in disease prevalence. On the other hand, increased diversity by addition of host species which are less competent reservoirs may lead to reduced

disease risk if the newly added species diminish the probability of encounter between the pathogen and the focal host species that is a highly competent reservoir. The resulted encounter reduction will end to reduced disease prevalence. Such a phenomenon may occur in the multi-host plant species communities, where both weeds and crop do not effectively allow the commonly shared pathogen transmission. Most Indian wheat cultivars are susceptible to wheat mosaic streak virus transmitted by sap and by the aphids *Brachycaudus helichrysi* (Kalt.) and *Rhopalosiphum maidis* (Fitch.). The infected wheat plants occur near reservoirs of the virus in cardamom (*Amomum subulatum* Roxb.) and are predisposed to the infection by *Bipolaris sorokiniana*. Peanut mottle virus (PMV) has been isolated from a few weed hosts in nature: *Cassia obtusifolia* L., *C. leptocarpa*, *C. occidentalis*, and *Desmodium canum*. PMV is easily transmitted mechanically to and from soybean plants. Several aphid species such as *Aphis craccivora*, *A. gossypii*, *Hyperomyzus lactucae*, *Myzus persicae*, *Rhopalosiphum padi*, and *R. maidis* can readily transmit PMV in a non-persistent manner. Several other plant species, mostly legumes, can be infected with PMV: *Calopogonium mucunoides*, *Canavalia ensiformis*, *Cassia bicapsularis*, *Chenopodium amaranticolor*, *Cicer arietinum*, *Cyamopsis tetragonoloba*, *Lathyrus odoratus*, *Macroptilium atropurpureum*, *M. lathyroides*, *Phaseolus coccineus*, *Sesamum indicum*, *Trifolium hybridum*, *Trigonella foenum-graecum* L., *Vigna cylindrica*, *V. oblongifolia*, and *V. unguiculata* subsp. *unguiculata* cultigroup *sesquipedalis*. It is believed that aphids involve in the transfer of potato leaf roll virus from nightshade plants, where the virus replicates to potato plants (Hartman et al. 1999).

Hairy nightshade, *Solanum sarrachoides* is a ubiquitous weed in potato agro-ecosystems and nonagricultural lands of southeastern Idaho and the Pacific Northwest. This weed increases the complexity of the Potato leafroll virus (PLRV) (Luteoviridae: Polervirus)-potato pathosystem by serving as aphid and virus reservoir. Previous field studies showed higher densities of green peach aphid, *Myzus persicae*, and potato aphid, *Macrosiphum euphorbiae*, the two most important vectors of PLRV, on *S. sarrachoides* compared with potato plants in the same fields. Some of the *S. sarrachoides* plants sampled in these surveys tested positive for PLRV. Viral infections can alter the physiology of plant hosts and aphid performance on such plants. To understand better the potential effects of *S. sarrachoides* on the PLRV-potato-aphid pathosystem, the life histories of *M. persicae* and *M. euphorbiae* were compared on virus-free and PLRV-infected *S. sarrachoides* and potato. Individual nymphs of each aphid species were held in clip cages on plants from each treatment to monitor their development, survival, and reproductive output. Nymphal survival for both aphids across plant species was higher on *S. sarrachoides* than on potato, and, within plant species, it was higher on PLRV-infected plants than on noninfected plants. With a few exceptions, similar patterns occurred for fecundity, reproductive periods, adult longevity, and intrinsic rate of increase. The enhanced performance of aphids on *S. sarrachoides* and on PLRV-infected plants could alter the vector population dynamics and thus the PLRV-disease epidemiology in fields infested with this weed (Srinivasan et al. 2008).

In California, a kind of life and three-faced transfer of a mycoplasma disease from weeds to citrus trees has been discovered. A leaf hopper transfers the citrus stubborn disease causal microorganism *Spiroplasma citri* to the weeds periwinkle and London rocket *Sisymbrium irio*, and acquires it from them in next feeding times, so that the weeds act as the sources of the inoculum required for the infection of citrus trees. *Eragrostis*, *Bromus*, *Panicum*, and other grass genera are as susceptible as wheat plants to American wheat striate mosaic virus (AWStMV, a rhabdovirus), and durum wheat plants appear most susceptible. While the virus can not pass through eggs and plant sap, both nymphs and adults of the painted leaf hopper *Endria inimica* transmit the virus. *Elymana virescens* F. is also another vector involved in the transmission of AWStMV. European wheat striate mosaic virus (EWSMV, a tenuivirus) occurs on oats, barley, rye, corn, and grasses (*Lolium* spp.) in addition to wheat, and is transmitted persistently by plant hoppers, principally *Javesella pellucida*, and to lesser extent, by *J. dubia*, *Loadelphax striatellus*, *Psammotettix alienus*, *Delphacodes pellucida*, and *Calligypona pellucida*. The pathogen is passed through eggs and apparently is stable within its vectors for generations. Nymphs of *J. pellucida* are efficient vectors of both of oat sterile dwarf virus and European wheat striate mosaic virus, as a result, host plants may be doubly infected. Infectious nymphs overwinter on grasses and reach adulthood in early summer. Wheat infections are most numerous in autumn and spring and reflect the distribution and activity pattern of nymphs, more efficient vectors compared with adults (Wiese 1987).

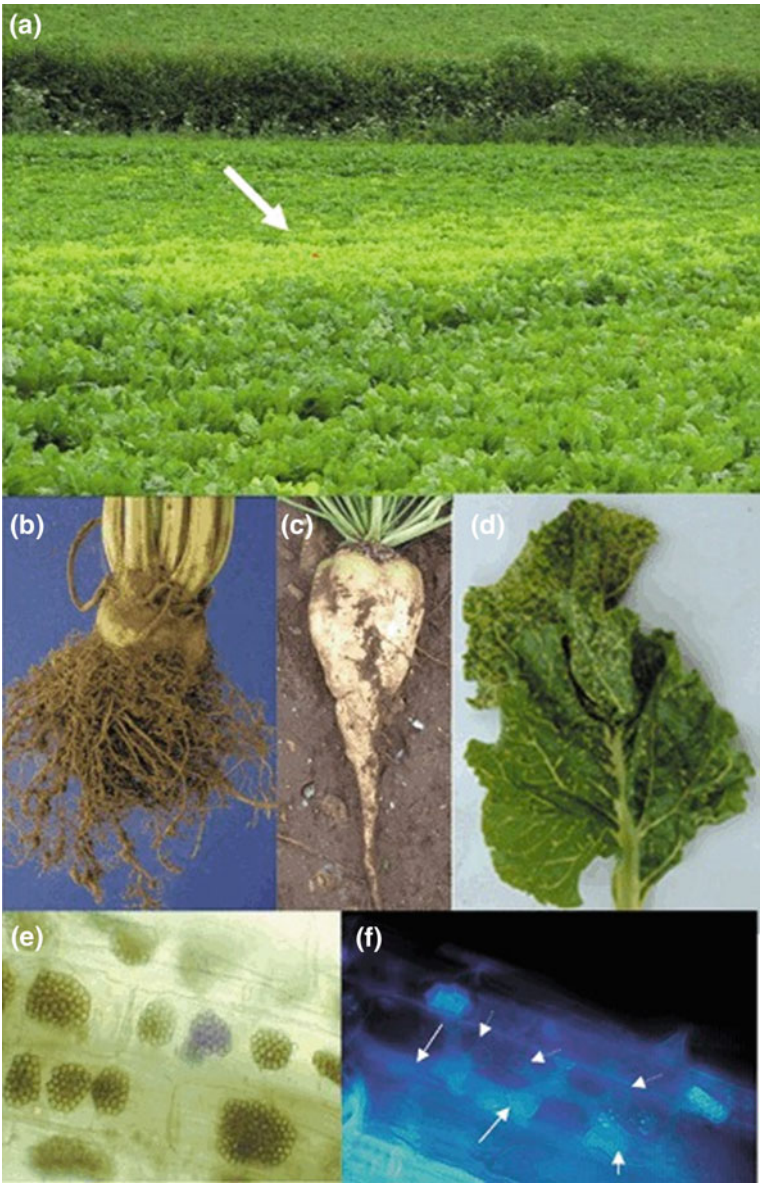
Onion thrips, *Thrips tabaci* (Fig. 7), regarded as a global pest of increasing concern in onion (Diaz-Montano et al. 2011) feeds on amarantha (*Amaranthus palmeri*), dandelion (*Taraxacum officinale*), mullin (*Verbascum thapsus*), goldenrod (*Solidago canadensis*), Kochia (*Kochia scoparia*), sage (*Salvia* sp.), sunflower (*Helianthus annuus*), smartweed (*Polygonum* spp.), yellow nutgrass (*Cyperus esculentus*), ragweeds (*Ambrosia* spp.) and mustard (*Brassica* spp.) (Chittenden 1919; Doederlein and Sites 1993), and then invades onions (Rahman and Batra 1945; Ananthakrishnan 1971).

The transmission of tobacco streak virus by thrips was investigated by Sdoodee and Teakle (1987) who mixed the adults or nymphs of *Thrips tabaci* with virus-carrying pollen from *Lycopersicon esculentum* infected with tobacco streak virus and then placed them on *Chenopodium amaranticolor* test seedlings, and found that the virus was regularly transmitted. The virus was also regularly transmitted when virus-carrying pollen was placed on the leaves of *C. amaranticolor* test seedlings and the thrips then introduced. No transmission occurred when test seedlings were exposed to virus-carrying pollen in the absence of the thrips or to the thrips without pollen. Further, no transmission occurred when the thrips were fed on virus-infected leaves and then transferred to test seedlings in the absence of virus-carrying pollen. The evidence indicates that the transmission of tobacco streak virus by *Thrips tabaci* depends on the presence of pollen-borne virus, which presumably infects via wounds made by the thrips. This method of virus transmission

Fig. 7 From left to right, *Thrips tabaci* (*Source* Thrips of California 2012, available online at keys.lucidcentral.org) the polyphagus insect known as the only vector of iris yellow spot virus; close-up of symptoms caused by IYSV on field onion leaf; and extensive leaf lesions on an onion plant heavily affected by IYSV (*Source* www.cabi.org) (Color figure online)

had not previously been reported. More importantly, *T. tabaci* is the only known vector that transmits Iris yellow spot virus (IYSV, a *Tospovirus* from the family Bunyaviridae) in many parts of the world. IYSV infects onions and induces symptoms including straw-colored, dry, tan, spindle/diamond-shaped lesions on the leaves and scales of onion plants and causes serious yield losses up to 100% in several countries (Diaz-Montano et al. 2011).

Several weed species, including the monocotyledonous plants *Alopecurus myosuroides*, *Lolium multiflorum*, *Sorghum vulgare*, *S. halepense*, and dicotyledonous plants *Calystegia sepium*, *Capsella bursa-pastoris*, *Centaurea cyanus*, *Convolvulus arvensis*, *Galinospora parviflora*, *Matricaria iodora*, and *Stellaria media* have been proved as alternative hosts for the beet necrotic yellow vein virus (BNYVV; Fig. 8), and beet soil-borne virus (BSBV), as well as their common plasmodiophorid vector, *Polymyxa betae* (Fig. 8). Also, *Chenopodium album* has been identified as a host for *P. betae*, but it is not a host for the viruses as tested by enzyme-linked immunosorbant assay (ELISA). The status of the weeds as alternative hosts has been pathologically confirmed through re-transmission of the viruses by their vector from the infected weed roots to susceptible sugar beet plants. Additionally, the use of molecular techniques (ITS sequence, and northern blot analysis) has indicated that *P. betae*, but not *P. graminis* is the vector involved in virus transmission from weed to sugar beet plants (Mouhanna et al. 2008). Several wild Hordeum species were mostly susceptible to *P. graminis* and/or barley mild mosaic virus, transmitted by *B. graminis*. An isolate of *P. betae* was used for comparison, and caused slight infection on oats but not on other cereals. The variation within and between Polymyxa spp. needs more detailed investigation (Adams and Jacquier 1994).


(a)
(b)
(c)
(d)
(e)
(f)

◂**Fig. 8** Symptoms of beet necrotic yellow vein virus (BNYVV) on sugar beet, and *Polymyxa betae*, the plasmodiophorid vector of rhizomania disease caused by the virus. **a** Rhizomania-infected patch (indicated with arrow) typical of BNYVV infection in the field; **b** classical "root madness" symptoms in BNYVV-infected sugar beet; **c** a virus-free root; **d** necrotic yellow vein symptom only observed under optimal disease conditions; **e** long-lived resting spores in sugar beet roots viewed under light microscope; **f** multilobed zoosporangium (indicated with solid arrows) inside host root cells after secondary zoospore release. Exit tubes where zoospores were released are visible (indicated with dotted arrows). The *P. betae* zoosporangium had been fixed in situ in 10% formaldehyde, pH 7.2, for at least 3 years prior to visualization and photography under the microscope with ultraviolet illumination (*Source* McGrann et al. 2009) (Color figure online)

2.4 Weeds as Alternate Hosts of the Pathogens

A number of pathogens including heteroecious rusts need two kinds of host plants in order to complete their life cycles. Some weeds have been known to play an important role in the life cycle complementation of rusts. Wheat black stem rust fungus *Puccinia graminis* f. sp. *tritici* forms its pycnia and aecidia on the leaves of barberries *Berberis vulgaris* L. and *B. canadensis*, and certain species of *Mahonia*. *Berberis* spp. are also the alternate hosts of the macrocyclic rust fungus, *P. striiformis* (Fig. 9) that causes yellow rust also known as glume rust, and stripe rust of cereals (Jin et al. 2010). With *P. g.* f. sp. *recondita* the causal fungus of a wheat disease called leaf rust, brown rust, dwarf rust as well as orange rust, aecia are formed on *Anchusa*, *Anemonella*, *Clematis*, and *Isopyrum* species (Prescott et al. 1986).

Fig. 9 Aecial pustules of the wheat yellow rust fungus *Puccinia striformis* on the abaxial side of *Berberis chinensis* leaf (*Source* Jin et al. 2010) (Color figure online)

3 Herbicides

3.1 Herbicide Groups

According to the classification system proposed by Herbicide Resistance Action Committee (HRAC) herbicides are classified in the HRAC groups (www.hracglobal.com/pages/classificationofherbicidesiteofaction.aspx) as below:

3.1.1 HRAC Group A

This group includes the herbicides that inhibit lipid biosynthesis through the inhibition of the first and key enzyme in the pathway of fatty acid biosynthesis, acetyl CoA carboxylase (ACCase). Interestingly, these herbicides only inhibit the eukaryotic form of the enzyme probably in the cytosol, but not the prokaryotic form of the enzyme found in the chloroplasts of dicotyledonous plants. As graminaceous plants lack *acc*D gene (encoding a subunit of prokaryotic form of ACCase) in their chloroplast genome, therefore do not have the insensitive prokaryotic form of the enzyme and are sensitive to the herbicides of HRAC group A (Konishi and Sasaki 1994). Malonyl-CoA, as the product resulted from ACCase activity, is both an intermediate in the de novo synthesis of primary fatty acids and also a substrate in the formation of long-chain fatty acids and flavonoids in plants (Bretschneider et al. 2007; Fig. 10).

The enzyme is important for membrane synthesis. These herbicides are primarily used in order to control post-emergence grasses in broadleaf crops. These herbicides are used solely against grasses. Broadleaf species are generally of natural resistance to the ACCase inhibitors from all three chemical families. The resistance originates from the enzyme itself which is less sensitive. However, herbicides of HRAC group A may induce symptoms on certain broadleaf crops. Natural tolerance of some grasses is because of a less sensitive enzyme or a higher rate of metabolic degradation (Mousavi et al. 2004). Some of these herbicides are active in soil to some extent, however their main activity appears on grown gramineous plants after their post-emergence application. They are effective on annual and perennial grasses, and the activity rate of each herbicide depends on the herbicide type. The translocation of these herbicides occurs through both xylem and phloem systems. The most abundant effect appears when weeds in their stage of fast growth under conditions with no stress are treated. The treated grasses decline and the complete control needs a week or more. Shoot and root growth suppression, chenages in leaf pigmentation, discoloration of leaves to red or pink colors (within 2–4 days), subsequent progressive necrosis of meristem regions and its extension to whole plant body are the symptoms encountered with the treated plants (Mousavi et al. 2004).

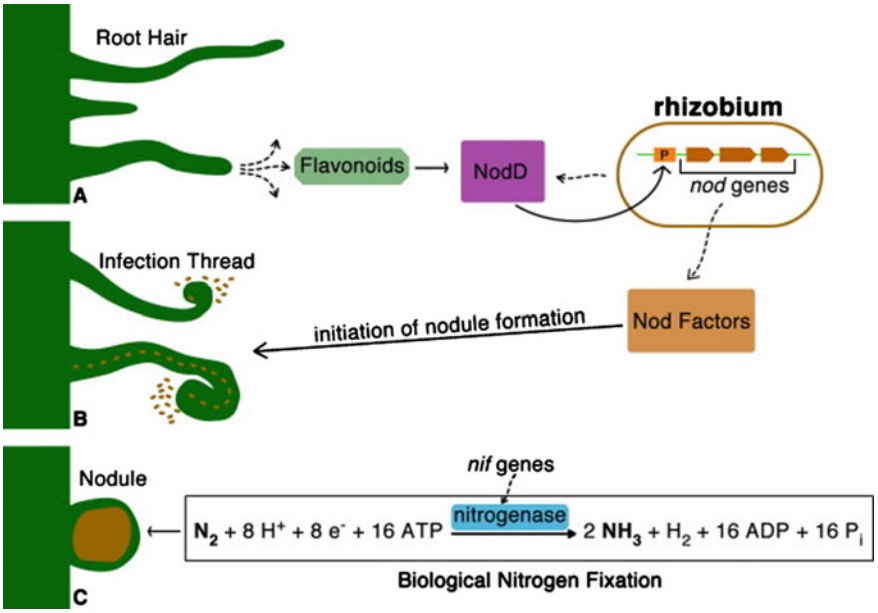


Fig. 10 The importance of the resistance of broadleaf legumes to acetyl CoA-carboxylase inhibiting herbicides (HRAC group A). Flavonoids are involved in the initial legume-*Rhizobium* recognition and subsequent induction of node genes expression (*Source* Laranjo et al. 2014)

The members of this group are from three chemically distinct families: aryloxyphenoxypropionates, FOPs (clodinafop-propargyl, cyhalofop-buthyl, diclofop-methyl, fenoxaprop-P-ethyl, fluazifop-P-buthyl, haloxyfop-R-methyl, propaquizafop, and quizalofop-P-ethyl), cyclohexanediones, DIMs (alloxydim, butroxydim, clethodim, cycloxydim, profoxydim, sethoxydim, tepraloxydim, and tralkoxydim), and phenylpyrazolines, DENs (pinoxaden). The chemical structure of three herbicides as the representatives of three chemical families has been presented in Fig. 11.

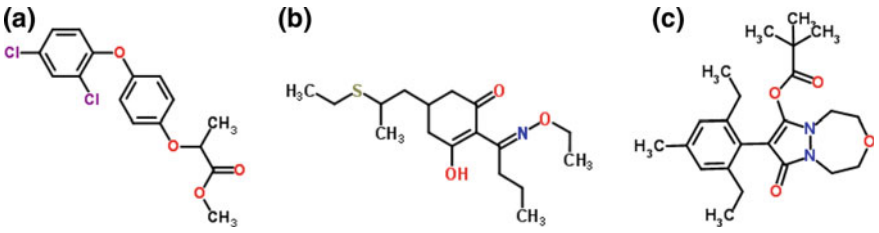


Fig. 11 Chemical structure of **a** diclofop-methyl; **b** sethoxydim; and **c** pinoxaden (*Source* www.chemspider.com)

3.1.2 HRAC Group B

The group includes those herbicides that prevent amino acid biosynthesis through the inhibition of acetolactate synthase (ALS), also known as acetohydroxyacid synthase (AHAS), in the branched-chain amino acid pathway which results in the restricted production of leucine, isoleucine, and valine. Plant death results from events that occur as the consequence of the inhibition of branched-chain amino acids, however, the actual sequence of phytotoxic process is unknown. These are potent inhibitors of plant growth effective on both dicotyledonous as well as monocotyledonous plants. The herbicides in the group exhibit either on-shoot, in-soil activity, or both activities. The growth of the treated plants is stopped after herbicidal spray. With ALS inhibitors, growing regions (terminal meristems) become chlorotic and necrotic 2–4 days after herbicide application. The treated plants are of stems with shortened internodes, reduced root growth, and converted pigments. The plant death begins from growth points and spreads to whole plant, so that it completely dies within 7–10 days (Mousavi et al. 2004).

The group includes the herbicidal compounds from five chemical families: sulfunylureas (amidosulfuron, azimsulfuron, bensulfuron-methyl, cinosulfuron, ethametsulfuron-methyl, ethoxysulfuron, flazasulfuron, flupyrsulfuron-methyl-Na, foramsulfuron, halosulfuron-methyl, imazosulfuron, iodosulfuron, mesosulfuron, metsulfuron-methyl, nicosulfuron, oxasulfuron, primisulfuron-methyl, pyrazosulfuron-ethyl, prosulfuron, rimsulfuron, sulfosulfuron, thifensulfuron-methyl, triasulfuron, trifloxysulfuron, triflusulfuron-methyl, tritosulfuron, sulfometuron-methyl, tribenuron-methyl, and cyclosulfamuron), imidazolinones (imazapic, imazamethabenz-methyl, imazamox, imazapyr, imazethapyr, and imazaquin), triazolopyrimidines (cloransulam-methyl, diclosulam, florasulam, flumetsulam, metosulam, and penoxsulam), pyrimidinyl(thio)benzoates (bispyribac-Na, pyribenzoxim, pyriftalid, pyrithiobac-Na, and pyriminobac-methyl), and sulfonylaminocarbonyltriazolinones (flucarbazone-Na, and propoxycarbazone-Na). The chemical structure of five herbicides as the representatives of the chemical families has been presented in Fig. 12.

3.1.3 HRAC Group C1

The herbicides of this HERAC group are characterized by their mode of activity, the inhibition of photosynthesis at photosystem II. Photosynthetic inhibitors control many broadleaf and some grass weeds. Generally, these herbicides inhibit photosynthesis by binding to D1 proteins of the photosystem II complex in chloroplast thylakoid membranes. Herbicide binding at D1 protein blocks electron transport and stops carbon dioxide fixation and production of energy required for plant growth. However, the death of plant is primarily not caused because of photosynthate depletion but of an indirect effect on other processes. Blockage of electron

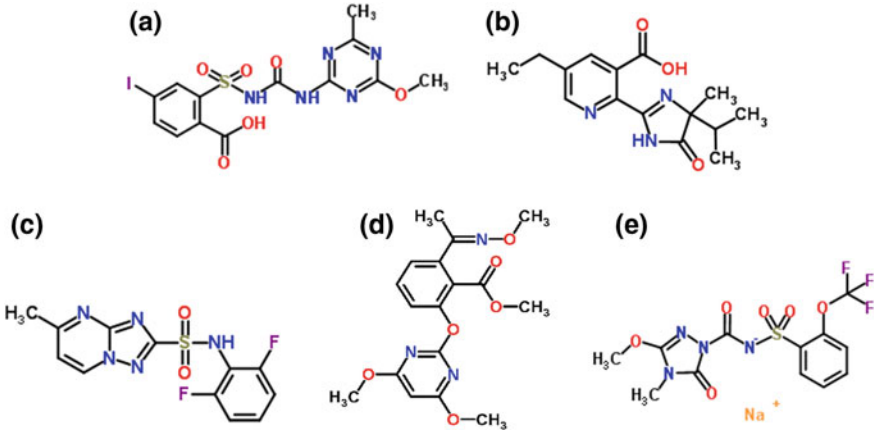


Fig. 12 Chemical structure of **a** iodosulfuron; **b** imazethapyr; **c** flumetsulam; **d** pyriminobac-methyl; and **e** flucarbazone-Na (*Source* www.chemspider.com)

transport in photosystem II promotes the formation of highly reactive molecules that initiate a chain of reactions that cause lipid and protein membrane destruction that results in membrane leakage, and leads to the desiccation and disintegration of plant cells and cellular organelles. Moreover, some PSII inhibitors affect other plant processes such as the biosynthesis of carotenoids, anthocyanins, ribonucleic acids, and proteins.

This HERAC group includes herbicidal compounds from six chemically distinct families: triazines (ametryne, atrazine, cyanazine, desmetryne, dimethametryne, prometon, prometryne, propazine, simazine, simetryne, terbumeton, terbuthylazine, trietazine, and terbutryne), triazinones (hexazinone, metamitron, and metribuzin), triazolinones (amicarbazone), uracils (bromacil, lenacil, and terbacil), pyridazinones (pyrazon, also known as chloridazon), and phenyl-carbamates (desmedipham, and phenmedipham). The chemical structure of six herbicides as the representatives of six chemical families has been presented in Fig. 13. Triazines, triazinones, uracils, and pyridazinones are soil-applied and early post-emergent herbicides. These herbicides are absorbed by both roots and shoots, but translocated only in the xylem. Phenylcarbamates are contact herbicides, primarily used as early post-emergence treatments (Mousavi et al. 2004).

3.1.4 HRAC Group C2

The group includes chemicals that inhibit photosynthesis at photosystem II but bind to protein D1 at photosystem II, however, at an attachment site different from that of the members of HERAC group C1.

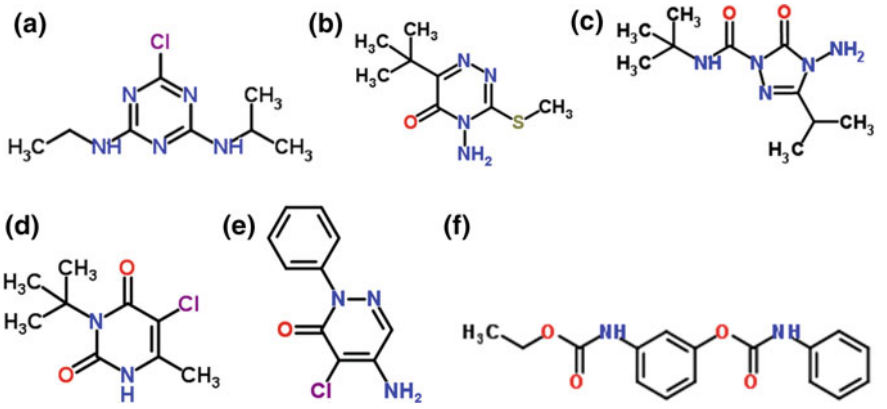


Fig. 13 Chemical structure of **a** atrazine; **b** metribuzin; **c** amicarbazone; **d** terbacil; **e** chloridazon; and **f** desmedipham (*Source* www.chemspider.com)

The members of this group are from two chemical families: ureas (chlorobromuron, chlorotoluron, chloroxuron, dimefuron, diuron, ethidimuron, fenuron, fluometuron, isoproturon, isouron, linuron, methabenzthiazuron, metobromuron, metoxuron, monolinuron, neburon, siduron, and tebuthiuron), and amides (propanil, and pentanochlor). The chemical structure of some herbicides in the group has been exhibited in Fig. 14. Ureas are soil-applied and early post-emergent herbicides. These herbicides are absorbed by both roots and shoots, but translocated only in the xylem. Amides are contact herbicides, primarily used as early post-emergence treatments (Mousavi et al. 2004).

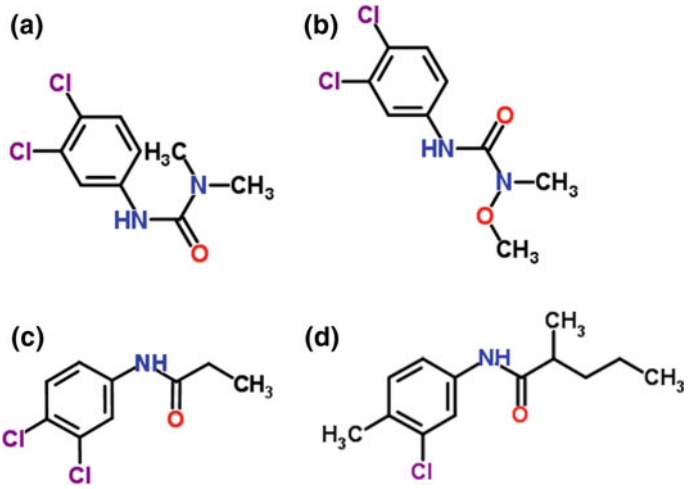


Fig. 14 Chemical structure of **a** diuron; **b** linuron; **c** propanil; and **d** pentanochlor (*Source* www.chemspider.com)

3.1.5 HRAC Group C3

The mode of action of this group of herbicides is somewhat similar to that of HRAC groups C1 and C2, however, they bind to a different attachment site of the protein D1 at the photosystem II.

The group includes members from three families of chemicals: nitriles (bromofenoxim, bromoxynil, and ioxynil), benzothiadiazinones (bentazon), and phenyl-pyridazines (pyridate, and pyridafol). The chemical structure of some herbicides in the group has been exhibited in Fig. 15. The members of all three chemical families are contact herbicides, primarily used as early post-emergence treatments (Mousavi et al. 2004).

3.1.6 HRAC Group D

This group includes the herbicide that are known by the inhibition of photosynthesis through electron diversion at photosystem I (PSI). PSI electron diverters are primarily contact herbicides that are activated with light. These relatively non-selective chemicals are used to control all existing vegetation and as pre-harvest desiccants. The group includes two members (Fig. 16) of a single chemical family: bipyridyliums (diquat, and paraquat). These herbicides accept electrons from PSI and are reduced to form an herbicidal radical that reduces other molecules to form extremely reactive and hazarduous molecules that readily destroy membrane lipids, and chlorophyll.

The disintegration of cellular membranes leads to cytoplasm leakage that results in rapid leaf wilting and desiccation (Mousavi et al. 2004). These herbicides kill plants fast when applied on shoot and leaves. In order to act effectively, these herbicides need to completely cover the weed leaves. The velocity of weed eradication is very high when it is warm and very sunny. They cause the loss of water from the tissues, and lead to the drying of the leaf tissues as the consequence of cellular membrane disruption resulted from the activity of the free radicals generated as the result of the treatment with these herbicides (Mousavi et al. 2004).

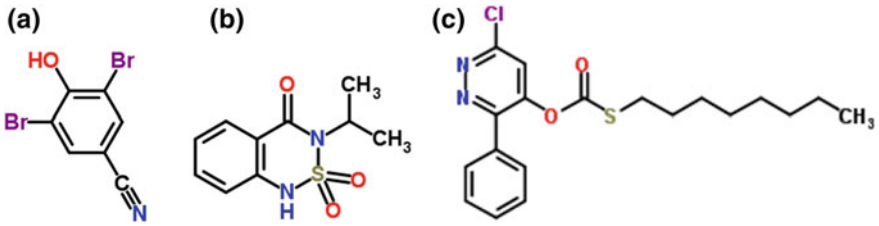


Fig. 15 Chemical structure of **a** bromoxynil; **b** bentazon; and **c** pyridate (*Source* www.chemspider.com)

Fig. 16 Chemical structure of **a** diquat; and **b** paraquat (*Source* www.chemspider.com)

3.1.7 HRAC Group E

Protoporphyrinogen oxidase (PPO) is an enzyme in the chloroplast that oxidizes protoporphyrinogen IX (PPGIX) to produce protoporphyrin IX (PPIX). PPIX is an important precursor molecule required for the biosynthesis of both chlorophyll (needed for photostnthesis) and heme (needed for electron transfer chains). The herbicides of this group inhibit PPO and block chlorophyll and heme production. Also, the inhibition of PPO results in the formation of highly reactive molecules that attack and destroy lipid and protein membranes. The affected cells and cellular organelles become leaky, dry and disintegrate rapidly (Mousavi et al. 2004). The PPO inhibitors are members of eight chemical families and one form-family: diphenylethers (acifluorfen-Na, bifenox, chlomethoxyfen, fluoroglycofen-ethyl, fomesafen, halosafen, lactofen, and oxyfluorfen), phenylpyrazoles (fluazolate and pyraflufen-ethyl), N-phenylphthalimides (cinidon-ethyl, flumioxazin, and flumiclorac-pentyl), thiadiazoles (fluthiacet-methyl, and thidiazimin), oxadiazoles (oxadiazon, and oxadiargyl), triazolinones (azafenidin, carfentrazone-ethyl, and sulfentrazone), oxazolidinediones (pentoxazone), pyrimidindiones (benzfendizone and butafenacil), and miscellaneous compounds (pyraclonil, profluazol, and flufenpyr-ethyl). The chemical structure of some herbicides in the group has been exhibited in Fig. 17. Sulfentrazone, Oxyfluorfen, and Oxadiazon are examples of the herbicides from this group that are incorporated into soil as pre-emergence soil herbicides. The herbicides of the group kill plants fast when applied on shoot and leaves. In order to act effectively, these herbicides need to completely cover the weed leaves. The velocity of weed eradication is very high when it is warm and very suny. They cause the loss of water from the tissues, and lead to the drying of the leaf tissues as the consequence of cellular membrane disruption resulted from the activity of the free radicals generated as the result of the treatment with these herbicides (Mousavi et al. 2004).

3.1.8 HRAC Group F1

This group is characterized by its particular mode of action, bleaching through the inhibition of carotenoid biosynthesis at the phytoene desaturase step (PDS). In the absence of carotenoid pigments, chlorophyll is disrupted in the presence of light, and membrane fatty acids are destructed. The treated leaves are pale white and translucent. Sometimes this paled situation is not complete but the areas between

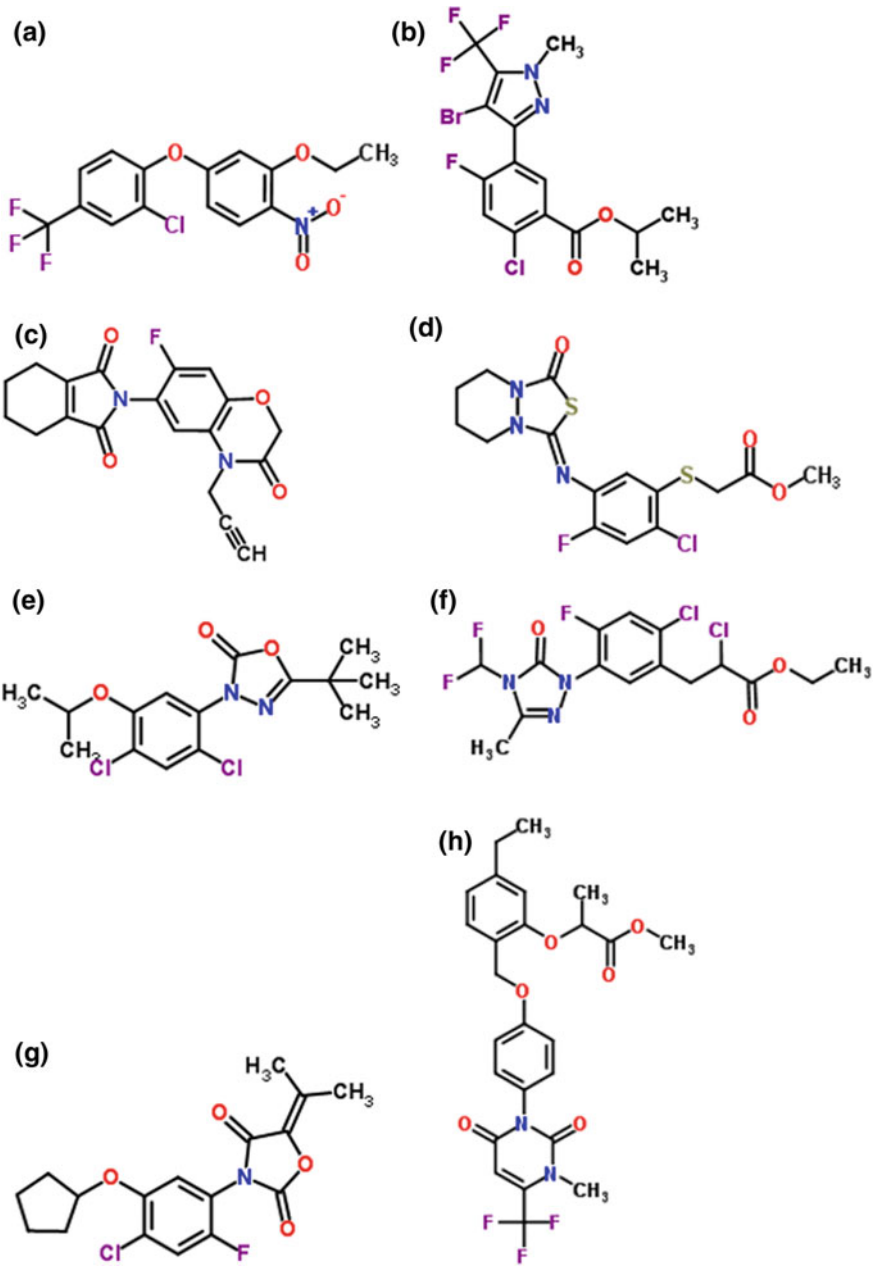


Fig. 17 Chemical structure of **a** oxyfluorfen; **b** fluazolate; **c** flumioxazin; **d** fluthiacet-methyl; **e** oxadiazon; **f** carfentrazone-ethyl; **g** pentoxazone; and **h** benzfendizone (*Source* www.chemspider.com)

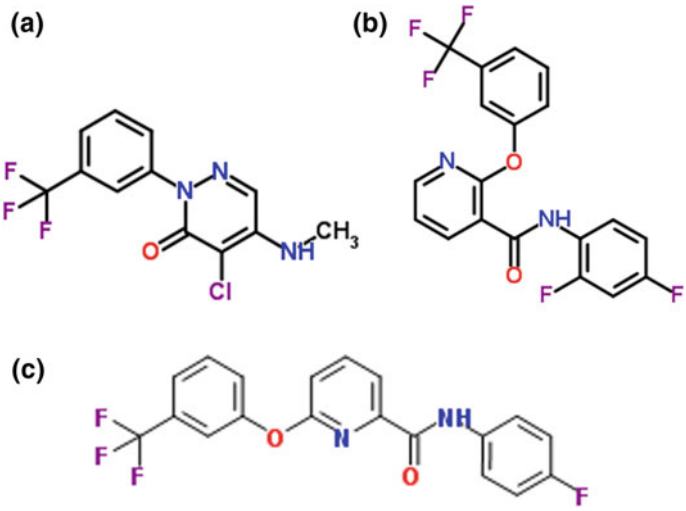


Fig. 18 Chemical structure of **a** norflurazon; **b** diflufenican; and **c** picolinafen (*Source* www.chemspider.com)

leaf veins are paled and pinkish or red hints develop in leaf margins. Norflurazon causes veinal bleaching of the affected leaves (Mousavi et al. 2004).

The HERAC group F1 includes members from two chemical families and a miscellaneous form-family: pyridazinones (norflurazon), pyridinecarboxamides (diflufenican and picolinafen), and miscellaneous compounds (beflubutamid, fluridone, flurochloridone, and flurtamone). The chemical structure of some herbicides in the group has been exhibited in Fig. 18.

3.1.9 HRAC Group F2

The group includes those chemicals that bleach treated plants through inhibition of 4-hydroxyphenyl-pyruvate-dioxygenase (4-HPPD). The enzyme catalyzes a key step in plastoquinone biosynthesis. As plastoquinone as a cofactor of phytoene desaturase, therefore, the lack of plastoquinone gives rise to bleaching symptoms resulted from an indirect inhibition of carotenoid synthesis. In the absence of carotenoid pigments, chlorophyll is disrupted in the presence of excessive light and photo-oxidation (Mousavi et al. 2004). The treated leaves are pale white and translucent. Sometimes this paled situation is not complete but the areas between leaf veins are paled and pinkish or red hints develop in leaf margins.

These herbicides are from three chemical families and a form-family: triketones (mesotrione and sulcotrione), isoxazoles (isoxachlortole and isoxaflutole), pyrazoles (benzofenap, pyrazolynate, and pyrazoxyfen) and miscellaneous compounds (benzobicyclon). The chemical structure of some herbicides in the group has been exhibited in Fig. 19.

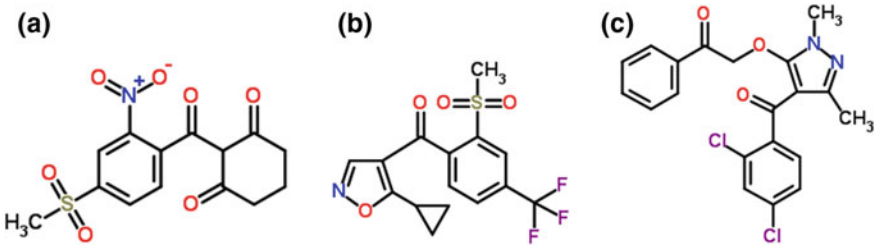


Fig. 19 Chemical structure of **a** mesotrione; **b** isoxaflutole; and **c** pyrazoxyfen (*Source* www.chemspider.com)

3.1.10 HRAC Group F3

The members of the group are from four chemical familes: triazoles (amitrole), isoxazolidinones (clomazone), ureas (fluometuron), and diphenylethers (aclonifen). The chemical structure of these herbicides has been presented in Fig. 20.

The herbicidal compounds of this group impose their bleaching effect through inhibition of carotenoid biosynthesis. In the absence of carotenoid pigments, chlorophyll is disrupted in the presence of light. The treated leaves are pale white and translucent. Sometimes this paled situation is not complete but the areas between leaf veins are paled and pinkish or red hints develop in leaf margins. Clomazone initially induces interveinal bleaching of the treated leaves (Mousavi et al. 2004). The target site of these compounds is not known, however, there is an evidence for the metabolization of clomazone to a phytotoxic form that inhibits 1-deoxy-D-xylulose-5-phosphate (DXP) synthase (Fig. 21). DXP is a key component required in plastid isoprenoid synthesis. Amitrole inhibits the accumulation of chlorophyll and carotenoids in the light (Burns et al. 1971) and it has been

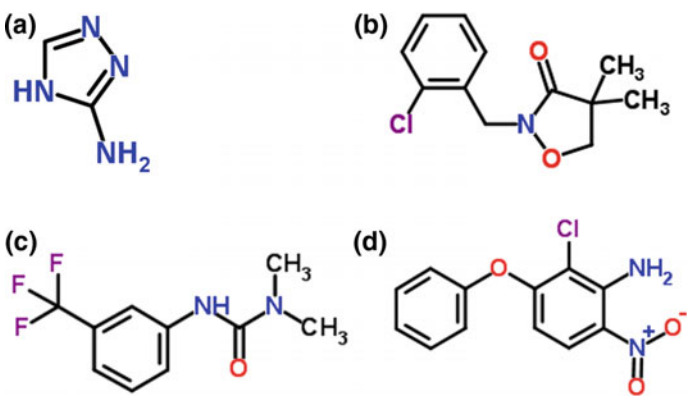
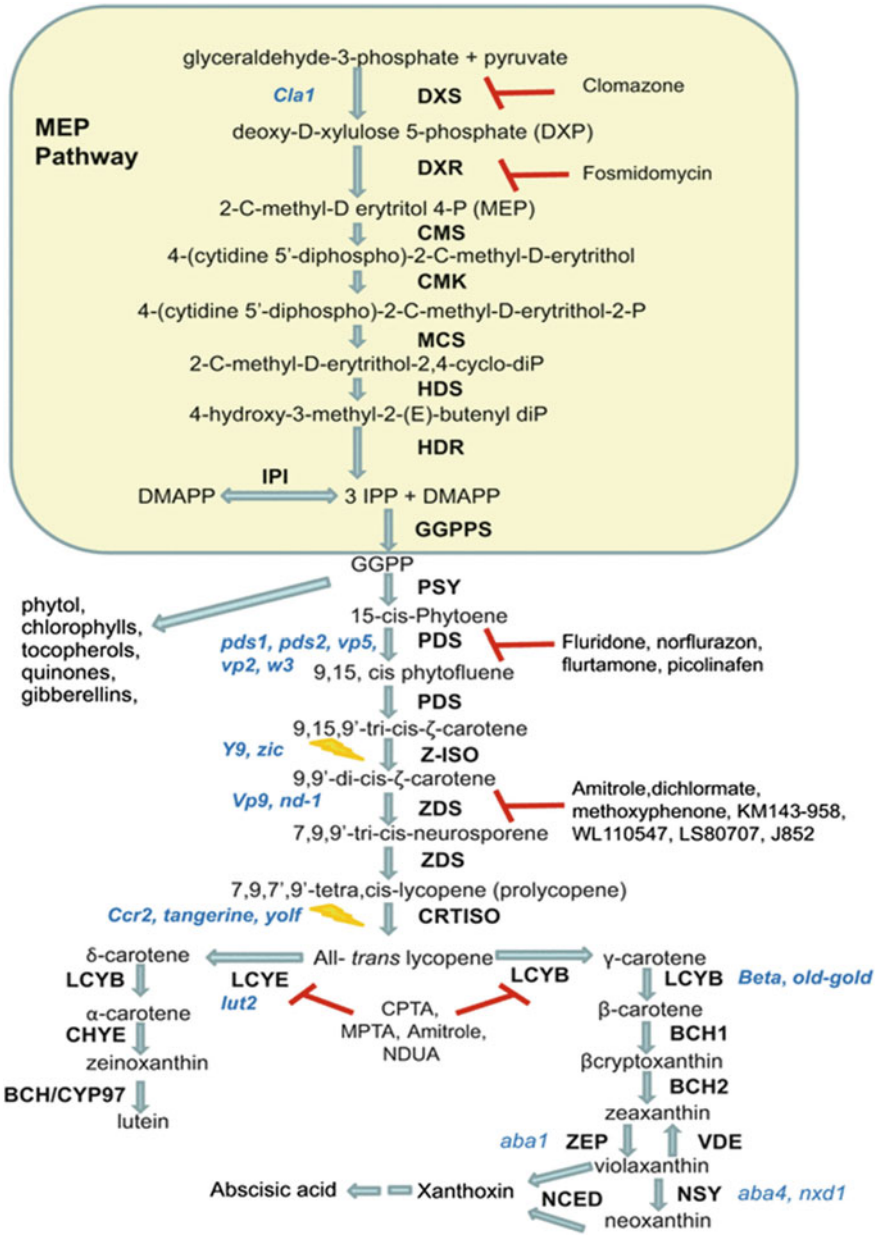


Fig. 20 Chemical structure of **a** amitrole; **b** clomazone; **c** fluometuron; and **d** aclonifen (*Source* www.chemspider.com)



reported as an inhibitor of lycopene cyclase in vivo (La Rocca et al. 1998) that can lead to the accumulation of chlorophyll precursors (La Rocca et al. 2007) and decreased salt tolerance (Chen et al. 2011). However, the specific site of its action has not been determined.

◂**Fig. 21** Isoprenoid and carotenoid pathway in plants. Schematic representation of the plastidial MEP (2-C-methyl-D-erythritol-4-P) pathway: (DXS) deoxyxylulose-5-phosphate synthase, (DXR) deoxyxylulose-5-phosphate reductoisomerase; (DXP) deoxy-D-xylulose-5-P; (CMS) 2C-methyl-D-erythritol-4-phosphate cytidyltransferase; (CMK) 4-(cytidine-5-diphospho)-2-C-methyl-D-erythritol kinase; (MCS) 2C-methyl-D-erythritol-2,4-cyclodiphosphate synthase; (HDS) 4-hydroxy-3-methylbut-2-enyl diphosphate synthase; (HDR) 4-hydroxy-3-methylbut-2-enyl diphosphate reductase; (IPP) (isopentenyl pyrophosphate); (GGPPS) geranylgeranyl pyrophosphate synthase; (DMAPP) dimethylallyl pyrophosphate; (IPI) isopentenyl pyrophosphate isomerase; (GGPP) geranylgeranyl phosphate. The relevant genes for carotenoid synthesis are: (PSY) phytoene synthase; (PDS) phytoene desaturase; (Z-ISO) ζ-carotene isomerase, (ZDS) ζ-carotene desaturase; (CRATISO) carotene isomerase; (LCYB) lycopene β-cyclase; (LCYE) lycopene ε-cyclase; (BCH) carotenoid β hydroxylase; (CHYE) carotenoid ε-hydroxylase (CYP97C1, CYP97A3); (ZEP) zeaxanthin epoxidase; (VDE) violaxanthin deepoxidase; (NSY) neoxanthin synthase; (NCED) 9-cis-epoxycarotenoid dioxygenase. Reported mutants in isoprenoid and carotenogenic genes are written in blue. Chemical inhibitors of some enzymes are included with a red T symbol. Light, referred as yellow ray can replace Z-ISO and CTISO activity in photosynthetic organs (*Source* Rosas-Saavedra and Stange 2016) (Color figure online)

3.1.11 HRAC Group G

This group is characterized by the inhibition of 5-enolpyrovyl-shikimate-3-phosphate synthase (EPSP) in shikimic acid pathway that leads to the limited production of aromatic amino acids phenylalanine, tryptophane, tyrosine, and of many important secondary compounds (Fig. 22).

The members of this group are from only a single family: glycines (glyphosate, and sulfosate). Glyphosate (relatively non-selective, Fig. 23) and sulfosate are absolutely of on-shoot activity.

The herbicides are inactivated in soil because of high soil adsorption, however, they are easily absorbed through plant foliage and translocated in the phloem to the growing points. With EPSP inhibitors, some pale colored areas may appear round the new growing regions, and plants die slowly within 1–2 weeks evenly turning to brown. The treated plants are of stems with shortened internodes, reduced root growth, and converted pigments. The plant death begins from growth points and spreads to whole plant, so that it completely dies within 7–10 days.

3.1.12 HRAC Group H

This group is known by the inhibitory impact of its members on the enzyme that converts glutamate and ammonia to glutamine, glutamine synthetase. The inhibition results in the massive accumulation of ammonia in a treated plant which destroys cells and directly inhibits both photosystem I and photosystem II reactions. Additionally, high rate of the accumulated ammonia in plants reduces the pH gradient across the membranes which inhibits energy production needed to support plant growth and development (Mousavi et al. 2004). The members of the group (Fig. 24) are from a single family: phosphinic acids (glufosinate-ammonium, and bialaphos or bilanaphos).

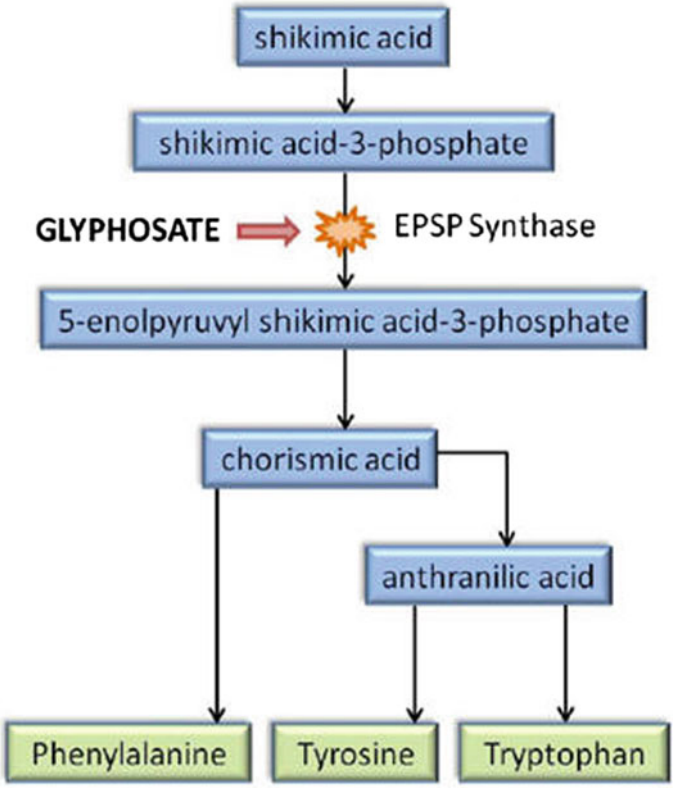


Fig. 22 The mode of action of glyphosate, a member of the HRAC group G (*Source* www.glyphosate.eu/glyphosate-mechanism-action)

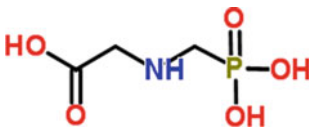


Fig. 23 Chemical structure of **a** glyphosate (*Source* www.chemspider.com)

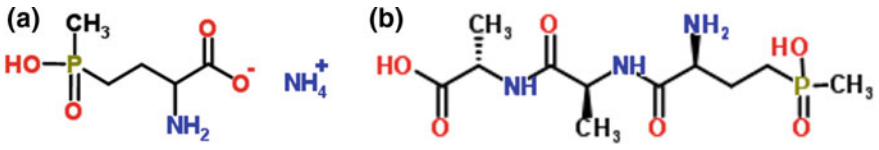


Fig. 24 Chemical structure of **a** gluphosinate-ammonium; and **b** bialaphos (*Source* www.chemspider.com)

These broad-spectrum post-emergent contact herbicides kill most annual grassy and broadleaf plants fast when applied on shoot and leaves. In order to act effectively, these herbicides need to completely cover the weed leaves. The velocity of weed eradication is very high when it is warm and severely illuminated. They cause the loss of water from the tissues, and lead to the drying of the leaf tissues as the consequence of cellular membrane disruption resulted from the activity of the free radicals generated as the result of the treatment with these herbicides (Mousavi et al. 2004).

3.1.13 HRAC Group I

The group is characterized by the inhibition of dihydropteroate (DHP) synthase. The group only includes a single member from a single family: carbamates (asulam). The chemical structure of asulam has been indicated in Fig. 25.

3.1.14 HRAC Group K1

This group includes microtubule assembly inhibitors that are generally applied pre-emergently to control annual grasses and some broadleaf weeds in many crops and turf grass. These herbicides, absorbed by both roots and shoots of emerging seedlings are not readily translocated in planta. The herbicides of HRAC group K1 are mitotic poisons that bind to tubulin, the major protein required for the polymerization of microtubules that are essential for cell division. Thus, they inhibit cell division. Hence, the meristematic regions such as growing points of stems and roots are most affected. Root tips get swollen as the cells in the region neither divide nor elongate because of the microtubule loss induced by these herbicides.

These herbicides are members of five chemical families: dinitroanilines (benefin or benfluralin, butralin, dinitramine, ethalfluralin, oryzalin, pendimethalin, and trifluralin), phosphoroamidates (amiprophos-methyl, and butamiphos), pyridines (dithiopyr, and thiazopyr), benzamides (propyzamide or pronamide, and tebutam), and benzoic acids (DCPA, also known as chlorthal-dimethyl). The chemical structure of some herbicides in the group has been presented in Fig. 26.

Fig. 25 Chemical structure of **a** asulam (www.chemspider.com)

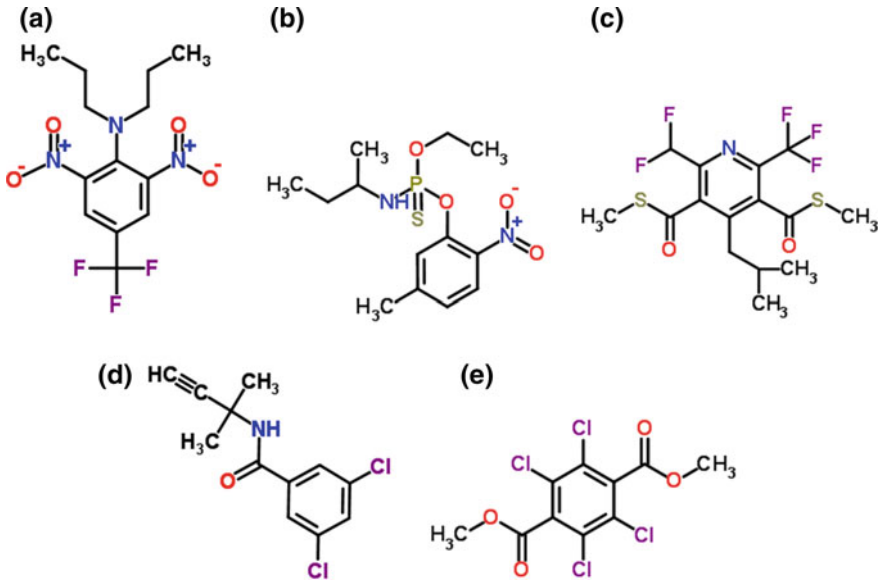


Fig. 26 Chemical structure of **a** trifluralin; **b** butamiphos; **c** dithiopyr; **d** pronamide; and **e** chlorthal-dimethyl (*Source* www.chemspider.com)

3.1.15 HRAC Group K2

The group is known through the inhibition of mitosis or microtubule organization. The members of this group are only from a single chemical family, carbamates that includes chlorpropham, propham, and carbetamide. The chemical structure of these herbicides has been exhibited in Fig. 27.

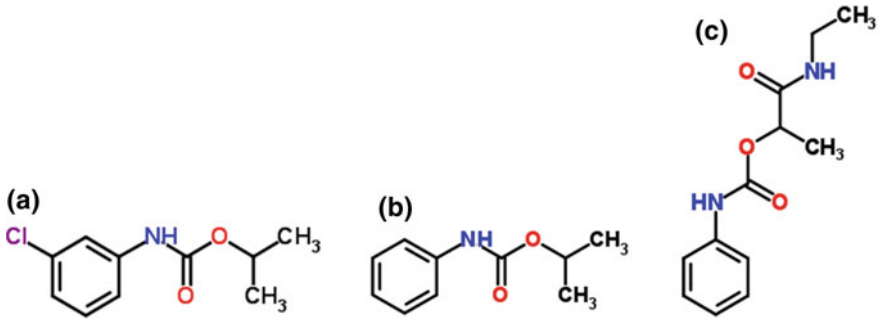


Fig. 27 Chemical structure of **a** chlorpropham; **b** propham; and **c** carbetamide (*Source* www.chemspider.com)

3.1.16 HRAC Group K3

The group is characterized by the inhibition of cell division through the inhibition of very long chained fatty acids (VLCFAs). These compounds typically affect susceptible weeds (annual grasses and some small-seeded broadleaf weeds) before emergence but do not inhibit seed germination. The primary absorption and action site of these herbicides on broadleaf species is the roots, while that on grass species is the emerging shoot (Mousavi et al. 2004).

The members of this group are from five chemical families: chloroacetamides (acetochlor, alachlor, butachlor, dimethachlor, metazachlor, metolachlor, pretilachlor, propachlor, propisochlor, thenylchlor, dimethanamid, and pethoxamid), acetamides (diphenamid, napropamide, and naproanilide), oxyacetamides (flufenacet, and mefenacet), tetrazolinones (fentrazamide), and miscellaneous compounds (anilophos, cafenstrole, and piperophos). Figure 28 represents the chemical structure of some herbicides in the group.

3.1.17 HRAC Group L

The group is characterized by the inhibition of cellulose synthesis that leads to the inhibition of cell wall synthesis. Herbicides in this group prevent cell division primarily in developing root tips and are only effective on some germinating broadleaf weeds and selected grasses. Chemically, the members of the group are classified into four families: nitriles (dichlobenil and chlorthiamid), benzamides (isoxaben), triazolocarboxamides (flupoxam), and quinoline carboxylic acid (quinclorac; for monocots; also a member of HERAC group O). An herbicide from the fifth family, alkylazines (indaziflam) is also classified in the HRAC group L (Forouzesh et al. 2015). Figure 29 represents the chemical structure of some herbicides in the group.

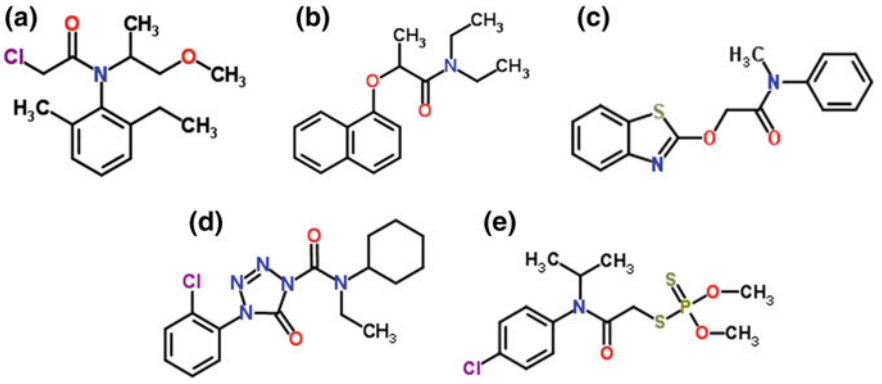


Fig. 28 Chemical structure of **a** metolachlor; **b** napropamide; **c** mefenacet; **d** fentrazamide; and **e** anilophos (*Source* www.chemspider.com)

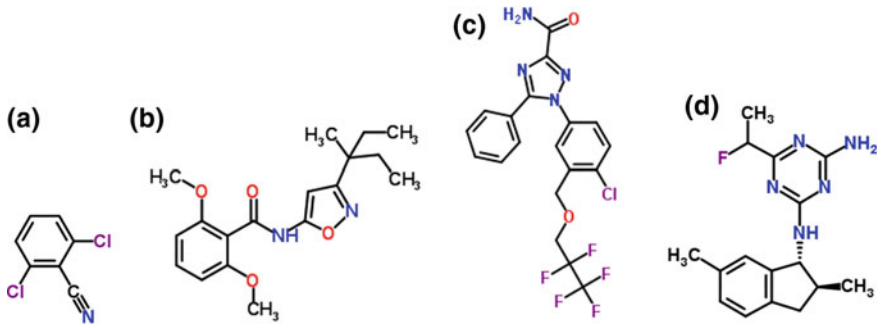


Fig. 29 Chemical structure of **a** dichlobenil; **b** isoxaben; **c** flupoxam; and **d** indaziflam (*Source* www.chemspider.com)

These compounds often used as pre-emergence herbicides include various chemical compounds that directly or indirectly inhibit the biosynthesis of cellulose and lead to reduced stability of cellular structure. They weaken the growth of weed seedlings, and induce symptoms such as root suppression and inflation. Dichlobenil and isoxaben used as pre-emergence herbicides are very effective on dicotyledonous herbicides. Quinclorac is applied in both pre-emergence and post-emergence manners and completely inhibits cellulose biosynthesis by monocotyledonous plants, while still regulates the growth of dicotyledonous plants (Mousavi et al. 2004).

3.1.18 HRAC Group M

The members of the group impose their herbicidal effect through uncoupling of oxidative phosphorylation and membrane disruption. The members of this group are classified in the chemical family of dinitrophenols (dinitro-ortho-cresol or DNOC, dinoseb, and dinoterb). Figure 30 represents the chemical structure of the herbicides in the group.

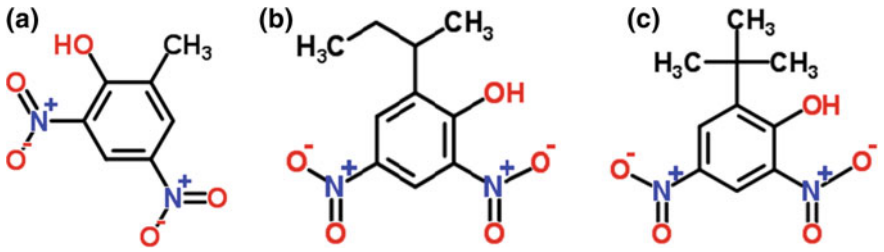


Fig. 30 Chemical structure of **a** dinitro-ortho-cresol (DNOC); **b** dinoseb; and **c** dinoterb (*Source* www.chemspider.com)

3.1.19 HRAC Group N

The members of this group inhibit lipid biosynthesis, however unlike the members of the HRAC group A and as represented in Fig. 31, they inhibit lipid biosynthesis in a target site other than acetyl coenzyme A carboxylase (ACCase). The specific mode of action of these herbicides is not well elucidated, however, there is strong evidence that these herbicides interfere with the biosynthesis of fatty acids and lipids in the newly developing shoot, which may account for the reported reductions in cuticular was deposition. Furthermore, these herbicides cause abnormal cell development or prevent cell division in germinating seedlings.

They stop the plant from growing by inhibiting cell division in the shoot and root tips while permitting other cell duplication processes to continue (Mousavi et al. 2004). These herbicides generally applied pre-planting or pre-emergence and incorporated to soil, are most effective on annual grasses and some broadleaf weeds. Most of these herbicides are volatile and need to be incorporated

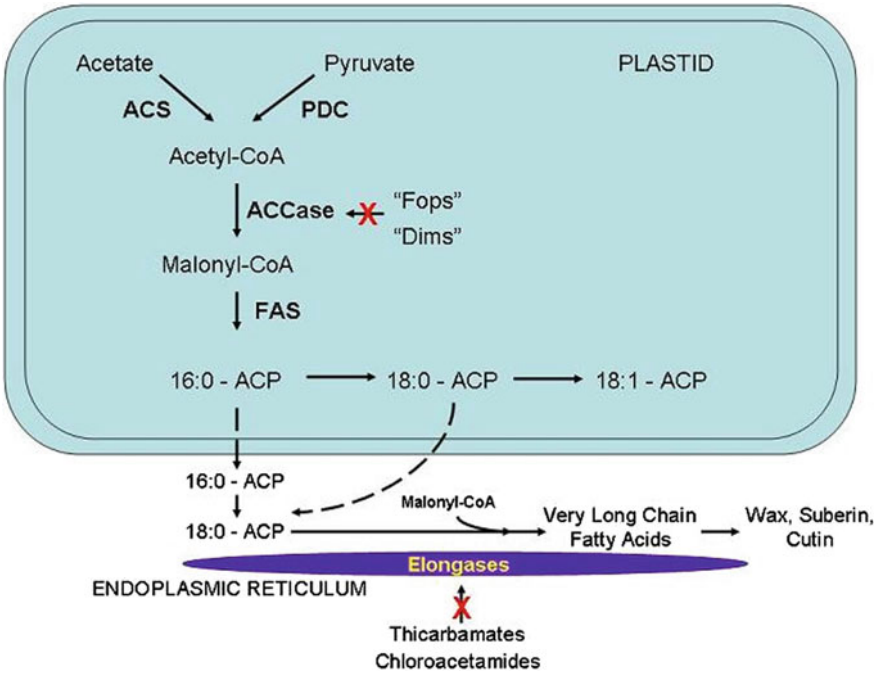


Fig. 31 Simplified schematic of fatty acid synthesis and elongation in higher plants based on the modification of Gronwald (1991). Abbreviations: ACCase, acetyl-CoA carboxylas; ACP, acyl carrie protein; ACS, acetyl-CoA synthase; CoA, coenzyme A; dims, cyclohexanedione inhibitors; FAS, fatty acid synthase; fops, aryloxyphenoxy propionate inhibitors; PDC, pyruvate dehydrogenase complex (*Source* Dyer WE, Inhibitors of fatty acid synthesis and elongation. Available: https://passel.unl.edu/pages/printinformationmodule.php?idinformationmodule=1130447055)

immediately after application to avoid excessive vapor loss. The HRAC group N herbicides are absorbed through both roots and emerging shoots but are translocated only in the xylem. The primary site of absorption and action is the emerging shoot and growing point. The members of HRAC group N are distributed in four chemical families: thiocarbamates [butylate, cycloate, diallate, dimepiperate, esprocarb, molinate, orbencarb, pebulate, prosulfocarb, thiobencarb or benthiocarb, tiocarbazil, triallate, vernolate, and S-ethyl dipropylthiocarbamate (EPTC)], phosphorodithioates (bensulide), benzofurans (benfuresate, and ethofumesate), and chloro-carbonic acids [(TCA), dalapon, and flupropanate] (Fig. 32).

3.1.20 HRAC Group O

The members of this group are known as synthetic auxins or plant growth regulators, and act as the natural plant hormone, indole acetic acid (IAA). Most of these herbicides although readily absorbable through both roots and shoots, are applied as post-emergence treatments. Their translocation to the actively growing sites occurs through both of xylem and phloem systems and their major effect is exerted on weed shoot. These herbicides include some of more effective chemicals for perennial broadleaf weed and brush control that impose their selective control effects on broadleaf weeds grown in cereal fields. With perennial weeds, most of these herbicides are transferred to the underground parts of the weed and put an end to its life. The herbicides interfere with cell formation in the meristematic regions and as the result, the primary symptoms on the newly developed leaves and shoots appear as fast turnings and shoot epinasty, cupped and shrunken leaves, inflated stems and the disruption of their phloem systems. Also, the damage in roots appears as branched and bunchy secondary roots of suppressed growth. The exact action site of this class of herbicides is not known, however, they seem to have multiple action sites and lead to hormonal imbalance, the interfered metabolism of nucleic

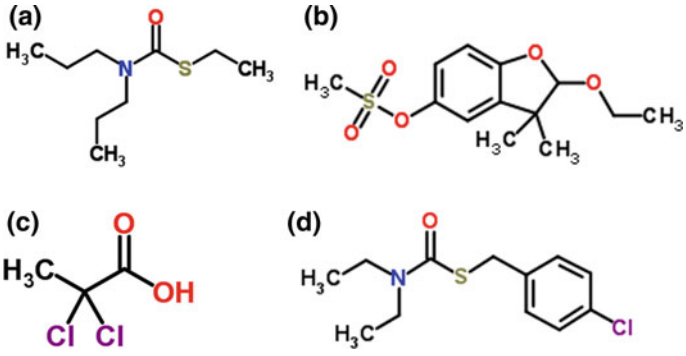


Fig. 32 Chemical structure of **a** S-ethyl dipropylthiocarbamate (EPTC); **b** ethofumesate; **c** dalapon; and **d** thiobencarb (*Source* www.chemspider.com)

acids and the interfered synthesis of proteins that end to the changes in auxine activity, weakness of cell walls, accelerated but useless cellular divisions, and final death of the plant within a few days or weeks (Mousavi et al. 2004).

The auxin-like activity is observed in the herbicidal compounds of four chemical families: phenoxy-carboxylic-acids [clomeprop, 2,4-dichlorophenoxyacetic acid (2,4-D), 4-(2,4-dichlorophenoxy)butyric acid (2,4-DB), dichlorprop or 2-(2,4-dichlorophenoxy)propionic acid (2,4-DP), 2-methyl-4-chlorophenoxyacetic acid (MCPA), and mecoprop or meta-chlorophenylpiperazine (MCPP or CMPP)], benzoic acids [chloramben, dicamba, and 2,3,6-trichlorobenzoic acid (TBA)], pyridine carboxylic acids (clopyralid, fluroxypyr, picloram, and triclopyr), and quinolone carboxylic acids (quinmerac, and quinclorac that also belongs to the HRAC group L).

There is also an herbicidal compound, benazolin-ethyl that does not belong to the chemical families mentioned above. The chemical structure of some of the members of the HRAC group O has been shown in Figs. 33 and 34.

3.1.21 HRAC Group P

Two members (Fig. 35) of the chemical family phthalamate semicarbazones (neptalam, and diflufenzopyr-Na) belong to the group characterized by the inhibition of auxin transport. Auxine transfer inhibitors like the soil herbicide neptalam, and the on-shoot herbicide diflufenzupyr inhibit auxine activity or its distribution in

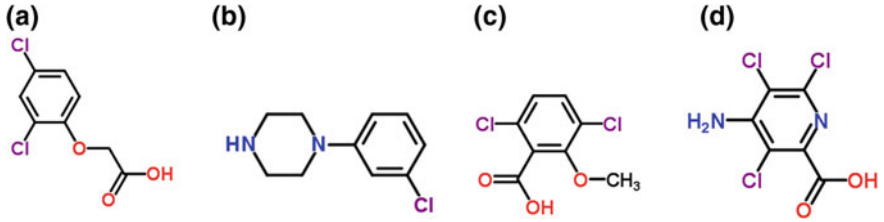


Fig. 33 Chemical structure of **a** 2,4-dichlorophenoxyacetic acid (2,4-D); **b** meta-chlorophenylpiperazine (MCPP); **c** dicamba; and **d** picloram (*Source* www.chemspider.com)

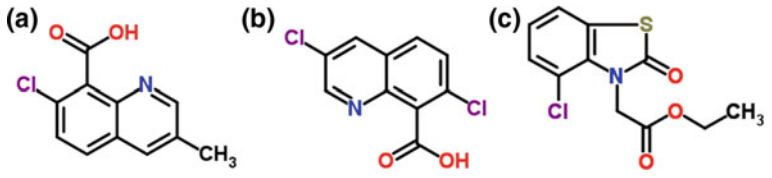


Fig. 34 Structural formula of **a** quimerac; **b** quinclorac; and **c** benazolin-ethyl (*Source* www.chemspider.com)

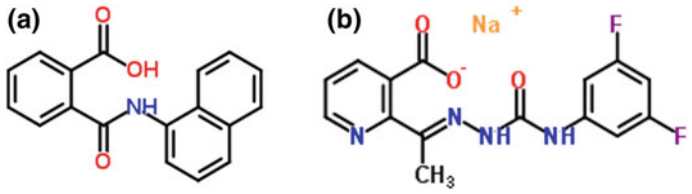


Fig. 35 Structure formula of **a** neptalam, and **b** diflufenzopyr-Na (*Source* www.chemspider.com)

plants, and as the result of the reduced growth hormone required for cellular development, bring about plant growth defects. The marked symptom of their phytoxicity in addition to reduced growth is the upward reversion of the treated plant root tips.

3.1.22 HRAC Group Z

This group includes a heterogenous collection of the herbicidal compounds of unknown mode of actions. The target site of these herbicides are not known, and may be different. The members of the group belong to three chemical families: arylaminopropionic acids (flamprop-M-methyl/-isopropyl), pyrazoliums (difenzoquat), as well as organoaresenicals including disodium methanearsonate (DSMA), and monosodium methanearsonate (MSMA). The chemical structure of these herbicides are indicated in Fig. 36.

There are also other herbicidal compounds of unknown mode of action: bromobutide, (chloro)-flurenol, cinmethylin, cumyluron, dazomet, dymron (also known as daimuron), methyl-dymron (also known as methyl-dimuron), etobenzanid, fosamine, indanofan, metam, oxaziclomefone, oleic acid, pelargonic acid, and pyributicarb. The effect of herbicides on plant diseases has been the topic of a

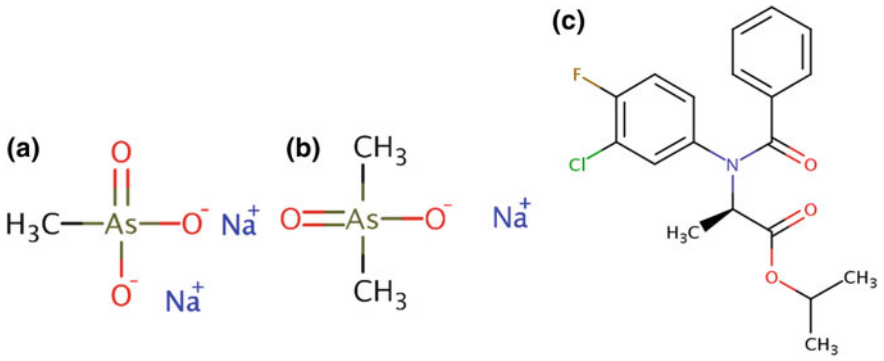


Fig. 36 Structural formulas of **a** disodium methanearsonate, **b** monosodium methanearsonate, and **c** flamprop-M-isopropyl (*Source* http://comptox.epa.gov)

number of previous reviews, either as a single topic (for example, Altman 1991; Altman and Campbell 1977a; Katan and Eshel 1973; Levesque and Rahe 1992) or as part of amore extensive review on secondary effects of pesticides (several of these are in the book by Altman 1993) or chemical effects on microbial weed biocontrol agents (Hoagland 1996).

3.2 *Effect of Herbicides on Plant Diseases*

Herbicides affect all three components of disease triangle (host plant, pathogen, and biotic as well as abiotic environmental factors). Herbicide are capable to exert their impacts on the incidence and severity of plant diseases (Altman and Campbell 1977a; Altman and Rovira 1989; Duke et al. 2007; El-Khadem and Papavizas 1984; El-Khadem et al. 1979, 1984; Heydari and Misaghi 1998; Miller et al. 1979; Moustafa-Mahmoud et al. 1993; Neubauer and Avizohar-Hershenson 1973; Pinckard and Standifer 1966; Rovira and McDonald 1986; Sanyal and Shrestha 2008). Most of the reported effects of herbicides on plant diseases have been obtained from field studies with no imply to the observed effect that if it is due to a direct herbicide-pathogen interaction or it is due to an direct effect on plant, making the plant less or more resistant to the pathogen. In general, most studies present no clue to the mechanism of the antifungal activity. In some cases, like the decrease of melon vascular wilt disease (caused by *Fusarium oxysporum*) followed by acetochlor treatment, the possibility of direct fungitoxic effects of the herbicide was rejected, but the mechanism was not revealed (Cohen et al. 1996). The decreased incidence of cotton seedling disease induced by *Fusarium oxysporum* f. sp. *vasinfectum* has been recorded as the consequence of the soil incorporation of the herbicides trifluralin, dinitramine, flumeturon, diuron, dalapon, and prometryn under field conditions (El-Khadem et al. 1984). Similarly, the use of EPTC and linuron reduces the post-emergence but not the pre-emergence incidence of cotton seedling disease caused by *Fusarium oxysporum* f. sp. *vasinfectum* (El-Khadem and Papavizas 1984). Sharma and Sohi (1983) indicated that bromacil, diuron, nitrofen, and alachlor all reduced disease severity of *Phaseolus vulgaris* induced by *Rhizoctonia solani*, but they had no data to suggest amechanism for the observed antifungal impacts. The severity of sheath blight disease of rice caused by *Rhizoctonia solani* reduced as the consequence of the application of the herbicide benthiocarb (Vasavan et al. 1980).

There are other studies showing enhancement of plant diseases by herbicides. In a survey of the effects of twelve herbicides (bentazon, acifluorfen, chlorimuron, fluazifop, diclofop, sethoxydim, imazaquin, metribuzin, oryzalin, thidiazuron, diaminozide, and mefluidide) on disease severity of four plant pathogens (*Alternaria cassiae*, *Colletotrichum coccodes*, *C. truncatum*, and *Fusarium lateritium*), all of the herbicides enhanced disease severity of at least one of the pathogens to a host plant but the mechanisms of these effects were not revealed (Caulder et al. 1987). The increased occurrence of cereal cyst disease by the nematode *Heterodera*

avenae (Altman and Rovira 1989), wheat take-all by *Gaeumannomyces graminis* var. *tritici* (Rovira and McDonald 1986), and sugar beet damping-off by *Rhizoctonia solani* (Rovira and McDonald 1986; Altman and Campbell 1977b) have been attributed to the soil application of trifluralin, chlorsulfuron, and cycloate, respectively. The application of chlorsulfuron resulted in increased levels of pythium root rot in barley seedlings (Blowes 1987). Terbacil (a uracil from HRAC group C1) increased the severity of sugarcane root rot induced by the oomycetous pathogen *Pythium arrhenomanes*, but metribuzin was found of no impact (Dissanayake et al. 1998). Heydari and Misaghi (1998) investigated the impact of herbicides on the incidence and development of cotton seedling damping-off caused by *Rhizoctonia solani*, and found that soil application of prometryn led to the significant increase of the disease incidence in soil infested at planting. Post-emergence soil infestation with the pathogen led to the considerably increased incidence of the disease in the presence of pendimethalin and prometryn, but not trifluralin. Field experiments with pendimethalin and prometryn confirmed the results obtained under controlled environmental conditions of growth chambers. Increase of various airborne as well as soilborne plant diseases have been recorded following programmed application of glyphosate (Johal and Huber 2009). These include apple canker induced by *Botryosphaeria dothidea* (Rosenberger and Fargione 2004), banana Panama disease (Fig. 37) caused by *Fusarium oxysporum* f. sp. *cubense* (Harper 2007), barley root rot induced by *Magnaporthe grisea* (Smiley et al. 1992), bean anthracnose induced by *Colletotrichum lindemuthianum* (Johal and Rahe 1984, 1988, 1990), bean damping-off, and root rot by *Pythium* spp. (Johal and Rahe 1984), pythium root rot in barley seedlings (Blowes 1987), bean root rot by *Fusarium solani* f. sp. *phaseoli* (Harper 2007), bean hypocotyl rot by *Phytophthora megasperma* (Keen et al. 1982), canola crown rot by *Fusarium* spp. (Harper 2007), canola wilt by *Fusarium oxysporum* (Harper 2007; Lange and McLaren 2002), citrus variegated chlorosis by *Xylella fastidiosa* (Yamada 2006), citrus crown rot by *Phytophthora* spp. (Yamada 2006), cotton damping-off by *Pythium* spp. (Harper 2007), cotton bunchy top manganese deficiency (Harper 2007), cotton wilt by *F. oxysporum* f. sp. *vasinfectum* (Harper 2007), grape black goo by *Phaeomoniella chlamydospora* (Harper 2007), melon root rot by *Monosporascus cannonbalus*, soybeans root rot by *Corynespora cassiicola* (Huber et al. 2005), soybeans target spot by *Corynespora cassiicola* (Huber et al. 2005), soybean sudden death syndrome by *Fusarium solani* f. sp. *glycines* (Keen et al. 1982), soybean root rot by *Phytophthora megasperma* (Keen et al. 1982), soybeans cyst nematode by *Heterodera glycines* (Geisler et al. 2002; Kremer et al. 2000), soybeans white mold by *Sclerotinia sclerotiorum* (Harper 2007), sugar beet yellows by *Fusarium oxysporum* f. sp. *betae* (Larson et al. 2006), sugar beet root rot by *Rhizoctonia solani* (Larson et al. 2006), sugarcane decline by *Marasmius* spp. (Huber, unpublished), tomato crown root rot by *Fusarium* (Bramhall and Higgins 1988), tomato wilt *Fusarium oxysporum* f. sp. *pisi* (Harper 2007), various canker diseases by *Phytophthora* spp. (Harper 2007), weeds biocontrol by *Myrothecium verrucaria* (Boyette et al. 2006), wheat bare patch by *Rhizoctonia solani* (Harper 2007), wheat glume blotch by *Septoria* spp. (Harper 2007), wheat

Fig. 37 Symptoms of Panama disease on Cavendish bananas in Jordan. External symptoms included: **a** chlorosis and/or **b** necrosis of leaves, progressing from the oldest to the youngest in a plant. Note in (**b**) that buckling of leaves also occurred before they became chlorotic or necrotic. Internally, affected xylem and the associated parenchyma was reddish to dark brown which, when viewed in **c** cross section was evident as discrete dots or contiguous sections of discoloration, and when viewed in **d** longitudinal sections encompassed much of the pseudostem length (*Source* Ploetz et al. 2015)

root rot by *Fusarium* spp. (Fernandez et al. 2005, 2007; Harper 2007), wheat head scab by *Fusarium graminearum* (Fernandez et al. 2005), and wheat take-all by *Gaeumannomyces graminis* (Hornby et al. 1998). Such an extended list of diseases affected by only an individual herbicide indicates the importance of the conscious use of herbicides in agriculture. Herbicides can directly and/indirectly affect plant diseases.

3.2.1 The Direct Impacts of Herbicides on Pathogens

Unfortunately, there are few data available on the direct effects of herbicides on plant pathogens. However, some herbicides are directly toxic to plant pathogens at the recommended rates. Three methods are conventionally used to investigate the

effects of a number of commercial herbicides on the growth of certain soil fungi: measurements of hyphal extension across agar plates; measurements of hyphal extension along sterilized plant material; and manometric techniques. In particular, three points emerged from these studies. First, that there was no stimulation of fungal growth. Herbicide interference in growth included suppression of spore germination, inhibition of the rate of linear extension of the mycelia, and abnormalities in growth habit and in patterns of spore production. Secondly, that some herbicides (for example, linuron and paraquat) were more fungitoxic than others (for instance, MCPA and simazine) to a range of organisms. Thirdly, there were differences among fungi in their sensitivity to individual herbicides. All three methods have shown consistent differences among fungi in their ability to tolerate paraquat. Pero and Owens (1971) introduced a chromatogram-based micro-method for the detection of antifungal activity of chemicals that could also be applied with herbicides.

Direct Anti-pathogenic Effects of Herbicides

The direct influence of herbicides on pathogens has also been indicated by a number of researches (such as Altman and Campbell 1977a; Black et al. 1996; El-Khadem and Papavizas 1984; El-Khadem et al. 1979, 1984; Neubauer and Avizohar-Hershenson 1973). The results from in vitro studies performed by Roberti et al. (2006) implicated the inhibitory effect of glyphosate on mycelial growth of most of the studied soilborne fungi. Abdel-Mageed et al. (2013) isolated, identified and characterized forty-five rhizofungal isolates from 11 herbicides polluted-soil. Among the isolates, 10 fungal species proved to be the most potent and promising ones in herbicides tolerance. The herbicides exhibited severe and dramatic effect and modulation on fungal DNA and protein as represented in DNA and protein profile. Severe losses were found in the rate of total soluble cell ion (SCI) and total cell protein concentration (TCPC). With the loss of SCI by glyphosate, *Aspergillus flavus* (86.30%) was the most affected one, followed by *Penicillium spiculisporus* (76.30%), *Penicillium verruculosum* (64.40%), and *Alternaria tenuissima* (64%), respectively. For pendimethalin, *A. tenuissima* (54.01%) was the most affected fungus. For diclofop-methyl, *P. spiculisporus* (74.20%) was the most affected fungus. With the loss of TCPC by glyphosate, *A. tenuissima* (64.71%) was the most effected fungus, followed by *P. spiculisporus* (57.14%), respectively. For pendimethalin, *A. terreus* (54.29%) was the most affected fungus. For diclofop-methyl, *P. spiculisporus* (60%) was the most affected fungus, followed by *A. tenuissima* (58.82%), and *Aspergillus tamarii* (55.56%), respectively. The results proved severe reductions and alteration in protein, SCI, TCPC and DNA in fungal strains exposed to these herbicides which might reflect a degree of tolerance occurred during the assimilation of those toxic compounds from the pesticides polluted-soil. The herbicidal product Galex®, the commercially available formulated mixture of two active ingredients metolachlor (from the chemical family chloroacetamides and a member of the HRAC group K3) and motobromuron (from the chemical family

ureas and a member of the HRAC group C2) affected the growth of a few plant pathogenic fungi including *Fusarium oxysporum, F. monilliforme* and *Aspergillus flavus* and reduced the incidence of soil-borne disease in legumes (Olajire and Oluyemisi 2009). Greenhouse experiments had previously demonstrated that microbial population of cowpea soil was significantly reduced by applying the normal field application rate of a pre-emergnece herbicide, Galex®, into the soil (Fawole 2000). This reductive effect was more in fungal than bacterial population. Thus among premoninant fungal genera including *Mucor*, *Fusarium*, *Aspergillus* and *Penicillium*, *Fusarium* counts were remarkably reduced upon Galex® application into the soil (Fawole 2000). Pretreatment of muskmelon seedlings with four chloracetamide herbicides at 0.1 μg g^{-1} reduced the incidence of fusarium wilt by 22–79%. Also, acetochlor applied as a seed treatment (soaking for 5 h in 50 μg acetochlor mL^{-1}) reduced fusarium wilt in muskmelon seedlings, however, it was less effective when applied as foliar spray (Cohen et al. 1992a). The herbicides oxyfluorfen (a diphenylether from HRAC group E), butachlor (a chloroacetamide from HRAC group K3), acetochlor (a chloroacetamide from HRAC group K3), cinmethylin (of a yet unknown HRAC group), and oxadiazon (an oxadiazole from HRAC group E) inhibited the mycelial growth and sclerotial germination of *Rhizoctonia solani* on potato sucrose agar (PSA) medium, where the herbicidal concentrations required for 50% growth inhibition (IC_{50}) were 2.01, 4.16, 8.12, 11.97, and 22.01 mg L^{-1}, respectively. Sclerotial germination was inhibited by oxyfluorfen, acetochlor, cinmethylin, and oxadiazon at 100 mg L^{-1} (Hua et al. 2002). Here the literature is reviewed in the context of HRAC groups of herbicides and the chemical families in which the member herbicides of antifungal activity are classified.

HRAC Group A

Studies has indicated the direct antifungal activity of some herbicidal ACCase inhibitors from the chemical families, aryloxyphenoxy propionates (FOPs) and cyclohexanediones (DIMs). Diclofop-methyl is recorder to be of direct antifungal effects on *Colletotrichum truncatum* (Caulder et al. 1987). Clodinafop-propargyl exhibited direct antifungal activity against *Ceratocystis radicicola*, *Gaeumannomyces graminis*, *Rhizoctonia solani*, *Sclerotinia sclerotiorum*, *Verticillium dahlia*, and inhibited the growth of two oomycetous pathogens *Phytophthora* sp., as well as *Pythium* sp., however, diclofop-methyl indicated narrower range of antifungal activity and only inhibited *Bipolaris* sp., *G. graminis*, *V. dahlia*, *Phytophthora* sp., and *Pythium* sp. (Pakdaman et al. 2002). Also, studies with the herbicides applied in rapeseed or canola (*Brassica napus* var. *oleifera*) fields demonstrated that cycloxydim, sethoxydim, and haloxyfop-ethoxyethyl were of considerable antifungal impacts against *Sclerotinia sclerotiorum*, the causal fungus of canola white stem rot disease (Pakdaman and Mohammadi Goltapeh 2007). Further studies on the mechanism of antifungal activity of sethoxydim exhibited increased levels of malondialdehyde (MDA) in the biomass of the white stem rot pathogen, *S. sclerotiorum* treated with the herbicide. MDA is the main

product generated through peroxidation of polyunsaturated fatty acids and a reliable indicator of the disintegration of fungal cellular membranes (Gaweł et al. 2004). Also, hydrogen peroxide concentrations increased as the consequence of the herbicidal treatment of the pathogen (Pakdaman et al. 2007).

HRAC Group B

No information was found. However, the commercially available mixture of mesosulfuron-methyl and iodosulfuron methyl sodium (Atlantis® OD) inhibited the in vitro growth of the mycotoxigenic pathogen of fusarium head blight disease of wheat and other small grain cereals, *Fusarium graminearum* as well as the biological control agent *Trichoderma asperelloides* on potato dextrose agar when its final rate in the medium was adjusted to 5000 ppm (Pakdaman and Elahifard, unpublished data).

HRAC Group C1

There are reports of the antifungal activity of some members of the chemical families, triazines as well as phenylcarbamates. Schuldt and Wolfe (1956) proposed the toxic effects of some *s*-triazine herbicides against *Pythium* spp. as a reason for the improved emergence of soybean seedlings and decreased damping-off with the addition of atrazine. Atrazine (25, and 50 μg mL^{-1}) was of no significant effect on *Pectinobacterium carotovorum* growth (Breazeale and Camper 1972). In an study on the effect of 14 herbicides and two herbicide combinations on three microscopic soil fungus species carried out following Miller's method, Helmeczi et al. (1988) found that the triazine derivative Hungazin PK (atrazine) was of the least inhibitory impact on *Aspergillus*, *Rhizopus*, and *Fusarium*. Atrazine, simazine, and metribuzin at 500 μg mL^{-1} inhibited the mycelial development of *Sclerotinia sclerotiorum* on 1.5% Bacto agar. Additionally, the carpogenic germination of sclerotia in the soil amended with atrazine and simazine at the rate of 0.5–10.0 mg g^{-1} soil was not affected as compared with that in untreated soil, however, sclerotia in the treated soils produced abnormal apothecia, while those in the soil amended with 0.5 and 1.0 mg metribuzin g^{-1} soil could not germinate (Casale and Hart 1986). In contrast, sclerotial germination was stimulated and a higher number of stipe initials was developed in soils treated with metribuzin at 0.5 and 1.0 μg g^{-1}, and atrazine at 0.5 μg g^{-1} (Radke and Grau 1986). Atrazine reduced the colony diameter of *Macrophomina phaseolina* (Fig. 38) in sorghum (*Sorghum bicolor*; Fig. 39) but did not reduce the production and germination of microsclerotia (Russin et al. 1995). Atrazine inhibited the mycelial growth of the oomycetous species *Pythium arrhenomanes* (Dissanayake et al. 1998). Atrazine, a corn herbicide (Atrazine®, tested in vitro at two concentrations of 3 and 33 μg mL^{-1}) and cyanazine (Bladex®, tested in vitro at two concentrations of 17, and 170 μg mL^{-1}) were of no significant effect on the hatching of *Heterodera glycines* when compared with that in deionized water, however, they exerted a negative impact ($P \leq 0.05$) when compared with hatching rate in the solution of zinc sulfate (3.14 mM).

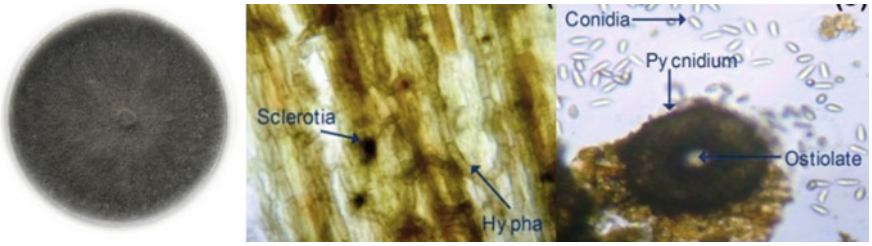


Fig. 38 Charcoal rot causal fungus, *Macrophomina phaseolina*, (Left) blackish mycelial growth on potato dextrose agar plate (*Source* Photchana Trakunsukharati, Department of Agriculture, Thailand); (Middle) hyphae and sclerotia of the fungus (*Source* Islam et al. 2012); (Right) Osteolate pycnidium of the fungus and one-celled hyaline pycnidiospores (*Source* Islam et al. 2012)

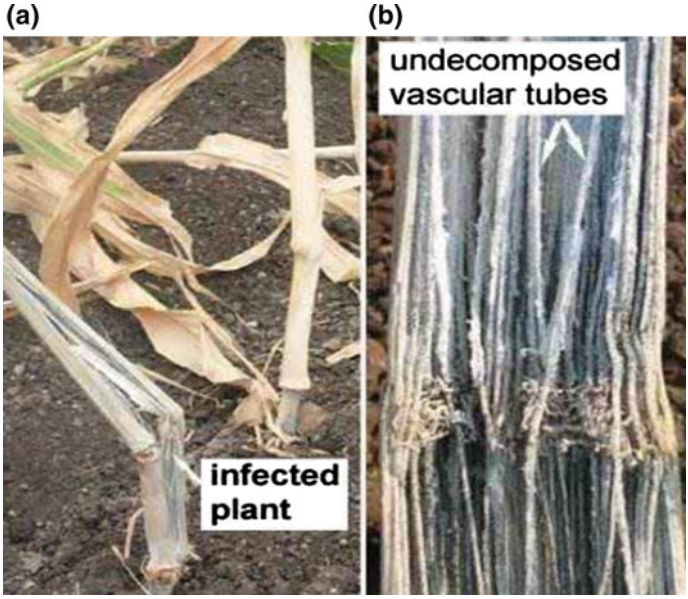


Fig. 39 Charcoal rot disease of sorghum caused by *Macrophomina phaseolina*, **a** symptoms of the disease on plant foot and foot breakage, **b** internal black rot of foot and stem and undecomposed vascular tubes (*Source* Reddy et al. 2012)

Bladex® was numerically (not statistically) a little more toxic than Atrazine® (Wong et al. 1993). Desmedipham, a phenylcarbamate, indicated direct antifungal activity against *Bipolaris* sp., *Ceratocystis radicicola*, *Fusarium graminearum*, *Gaeumannomyces graminis*, *Macrophomina phaseolina*, *Rhizoctonia solani*, *Sclerotinia sclerotiorum*, *Verticillium dahliae*, and two oomycetous pathogens *Phytophthora* sp., and *Pythium* sp. (Pakdaman et al. 2002).

HRAC Group C2

The chemical compound from the family of amides, propanil (Rogue™, and Stam-F-34) was of direct fungicidal activity against brown leaf spot fungus *Bipolaris oryzae* (Sen and Kaiser 1972). Metoxuron, a member of the chemical family of ureas, was of direct antifungal activity against *Puccinia lagenophorae*, a rust fungus pathogenic on the perennial weed common groundsel, *Senecio vulgaris* (Frantzen et al. 2005; Fig. 40).

Fluometuron and diuron, tested at both concentrations of 25, and 50 μg mL^{-1} were of no Impact on *Pectinobacterium carotovorum* growth rate (Breazeale and Camper 1972). Diuron at 50 μg mL^{-1} inhibited the mycelial development of *Sclerotinia sclerotiorum* on 1.5% Bacto agar (Casale and Hart 1986). Out from five herbicides (also including isoproturon, 2,4-D ethyl ester, atrazine, and 2,4-D ethyl sodium) tested under in vitro conditions, Tribunil® (methathiabenzuron, an urea) applied at 100 ppm (equal to 100 μg g^{-1} soil; ppm: part per million) was the most effective, and completely inhibited the sclerotial germination of *Sclerotium rolfsii*. Another urea, isoproturon exhibited complete inhibition of fungal radial growth when it was applied at the concentration of 250 ppm. Soil application of 0.4% Tribunil® (equal to 4000 ppm or 4000 μg g^{-1} soil) led to complete ceasation of the fungus saprophytic activity (Harlapur et al. 1989).

Fig. 40 Aecia of *Puccinia lagenophorae*, the rust fungus pathogenic on the perennial weed common groundsel, *Senecio vulgaris* (*Source* http://www.naturefg.com/)

HRAC Group C3

Direct antifungal activity of a member of the family benzothiadiazinones, bentazon on *Colletotrichum truncatum* (Caulder et al. 1987), and of a member of the family of nitriles, bromoxynil on *Rhizoctonia cerealis* and *Pseudocercosporella herpotrichoides* (Kataria and Gisi 1990) have been recorded. Ioxynil, another nitrile herbicide, inhibited in vitro growth of *Pectinobacterium carotovorum* when applied at the concentration of 25 μg mL^{-1} (Breazeale and Camper 1972). Bentazone (Basagran® tested in vitro at two concentrations of 50 and 500 μg mL^{-1}) was of no significant effect on the hatching of soybean cyst nematode *Heterodera glycines* when compared with that in deionized water, however, it exerted a negative impact ($P \leq 0.05$) when compared with hatching rate in the solution of zinc sulfate (3.14 mM) (Wong et al. 1993).

HRAC Group D

Paraquat, a member of the chemical family bipyridyliums was found to be of direct antifungal effect on *Dreschlera teres* (Toubia-Rahme et al. 1995). Diquat (25 μg mL^{-1}), another member of the family, and paraquat (50 μg mL^{-1}) inhibited the growth of *Pectinobacterium carotovorum* (Breazeale and Camper 1972).

HRAC Group E

Acifluorfen, as a diphenylether and protoporphyrinogen oxidase (PPO) inhibitor, was found to reduce the radial growth of *R. solani* isolates of IA colonies by more than 72% under in vitro conditions (Black et al. 1996), while fomesafen, another diphenylether seemed to be of no significant direct impact on *Sclerotinia sclerotiorum* (Dann et al. 1999). Also, acifluorfen (Blazer®, applied in vitro at the rate of 50–500 μg mL^{-1}) led to a 42–67 percentile reduction (compared with that in deionized water) and to a 61–71 percentile reduction (compared with that in zinc sulfate solution) of the hatched eggs of soybean cyst nematode (SCN) *Heterodera glycines*. Therefore, this postemergence soybean herbicide may be of a potential role in the management of SCN (Wong et al. 1993). The herbicides oxyfluorfen (a diphenylether) and oxadiazon (an oxadiazole) inhibited the mycelial growth and sclerotial germination of *Rhizoctonia solani* on potato sucrose agar (PSA) medium, where the herbicidal concentrations required for 50% growth inhibition (IC_{50}) were 2.01, and 22.01 mg L^{-1}, respectively. Sclerotial germination was inhibited by oxyfluorfen, and oxadiazon at 100 mg L^{-1} (Hua et al. 2002).

HRAC Group F1

No information was found.

HRAC Group F2

No information was found.

HRAC Group F3

Fluometuron, tested at both concentrations of 25, and 50 μg mL^{-1}, was of no Impact on *Pectinobacterium carotovorum* growth rate (Breazeale and Camper 1972). An herbicide from the chemical family isoxazolidinones, clomazone (Command®) tested in vitro at the concentrations of 50, and 500 μg mL^{-1} was of no negative impact on the hatching of soybean cyst nematode *Heterodera glycines* (Fig. 41) when compared with deionized water, however, its effect was statistically significant ($P \leq 0.05$) compared with that of zinc sulfate solution (3.14 mM), which increased hatching (Wong et al. 1993).

Fig. 41 Soybean cyst nematode, *Heterodera glycines*, (Top, Left) Juvenile larva with visible stylet (*Source* Jonathan D Eisenback, Virginia Polytechnique Institute and State University, Bugwood.org), (Top, Right) Lemon-shaped female cysts (*Source* Mactode Publications, Bugwood.org); (Down) Adult male with distinct stylet and projected spicules (*Source* Jonathan D Eisenback, Virginia Polytechnique Institute and State University, Bugwood.org)

HRAC Group G

Glyphosate as the member of the chemical family, glycines, is capabale to impose its antifungal effects on *Puccinia lagenophora* (Wyse and Müller-Schärer 2001), *Dreschlera teres* (Toubia-Rahme et al. 1995), *Calonectria crotalariae* (Berner et al. 1991), *Pythium ultimum* and *Fusarium solani* f. sp. *pisi* (Kawate et al. 1992). Glyphosate also inhibited pseudothecium formation of wheat tan spot fungus, *Pyrenophora tritici-repentis* in wheat straw (Sharma et al. 1989). Glyphosate not only inhibited aeciospore formation by the biological control rust fungus, *Puccinia lagenophora* on common groundsel (*Senecio vulgaris* L.) but also inhibited the germination of its aeciospores (Wyss and Müller-Schärer 2001). Spraying glyphosate-resistant bread wheat (*Triticum aestivum*) plants with the glyphosate commercially available formulation, Roundup® WeatherMAX at 0.84 kg a. i. ha^{-1}, inhibited plant infection in growth chamber experiments where more than 75% of check plants were infected to the rust fungus after they were sprayed with water or formulation control. The rate of rust control was maximal just after glyphosate application and proportional to the herbicide concentration in planta, where the herbicide persisted at least for a 14 day period. The antifungal activity of the symplastic herbicide was attributed to its inhibitory impact on fungal 5-enolpyruvyl shikimate 3-phosphate (EPSP) synthase (Feng et al. 2005). Similar results were also obtained when glyphosate-resistant bread wheat plants were treated with the herbicide in order to study its effect on the wheat stripe rust (*Puccinia striiformis* f. sp. *tritici*) incidence, progress, and severity (Feng et al. 2005). Interestingly, the application of glyphosate on transgenic gluphosate-resistant (GR) wheat reduced leaf rust (induced by *Puccinia recondita* f. sp. *tritici*) development, and black stem rust (caused by *Puccinia graminis* f. sp. *tritici*) infection (The level of leaf rust control decreased with the reduction in the application rates of glyphosate and the transitory effect lasted for 21–35 days after application (Anderson and Kolmer 2005). These findings indicate the antifungal activity of the widely used broad-spectrum herbicide, glyphosate that provides disease control benefits in glyphosate-resistant wheat, where glyphosate exhibits both preventive and curative activities against leaf rust pathogen, *P. triticina* (Fig. 42), and stripe rust causal fungus, *P. striiformis* f. sp. *tritici* (Fig. 43) under greenhouse as well as field conditions (Feng et al. 2005, and 2008).

A similar reduction of infection has been reported with Asian soybean rust (ASR, caused by the fungus, *Phakopsora pachyrhizi*) as the result of the glyphosate-resistant soybean plants treatment with glyphosate (Feng et al. 2005). Glyphosate at the rates between 0.84 and 1.68 kg ha^{-1} delayed the onset of ASR in glyphosate-resistant soybeans under laboratory conditions. However, field trials conducted in Argentina and Brazil under natural infestations indicated variable ASR control from the application of glyphosate in glyphosate-resistant soybeans. Therefore, further field studies are ongoing in order to define the activity of glyphosate against ASR (Feng et al. 2008).

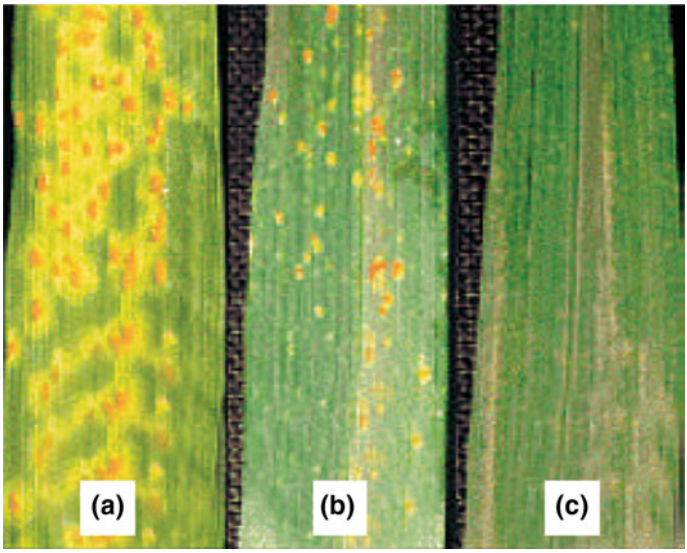


Fig. 42 Effect of glyphosate on wheat leaf rust (*Puccinia triticina*) 13 days after infection. **a** No treatment; **b** treated with 0.84 kg ae ha^{-1} Roundup® WeatherMAX 13 days before inoculation; **c** treated with 0.84 kg ae ha^{-1} Roundup® WeatherMAX 1 day before inoculation (*Source* Feng et al. 2005). Copyright 2005 National Academy of Sciences, USA

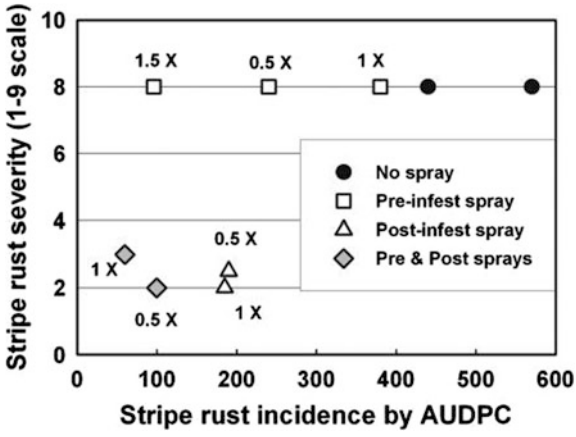


Fig. 43 Field evaluation of the effect of glyphosate treatment on stripe rust (*Puccinia striiformis* f.sp. *tritici*) in glyphosate-resistant wheat cultivar Macon from natural inoculum near Pullman, WA. Roundup® Ultra™ (0.42–1.26 kg a. i. ha^{-1}, 0.5× to 1.5× was sprayed preinfestation on May 19, 2004 and/or postinfestation on June 30, 2004. Rust symptoms first appeared on June 25, 2004. Stripe rust incidence was calculated based on Area Under Disease Progress Curve (AUDPC) from ratings on July 2nd, 8th, and 12th. The y axis describes rust severity (based on 1–9 scale) from July 12th (*Source* Feng et al. 2005)

HRAC Group H

Glufosinate and bialaphos, both glutamine synthase inhibitors belong to the single family of phosphine acids, and exhibit considerable antifungal activities against oomycetous, ascomycetous, and basidiomycetous pathogens. In a less definitive study, Ahmad and Malloch (1995) found that 1 mM glufosinate (a member of the family, phosphinic acids) reduced the bacterial population of the tested agricultural soil by 40% within 14 days after soil treatment. Additionally, it indicates direct antifungal activity against *Aspergillus flavus* (Tubajika and Damann 2002) and *Verticilium albo-atrum* (Ahmad and Malloch 1995). The effect of the non-selective herbicide glufosinate on plant diseases is known to be the result of direct fungitoxic effects. Its effects are best seen in glufosinate-resistant crops. Glufosinate has antimicrobial activity in glufosinate-resistant soybeans (Pline et al. 2001), rice (Uchimiya et al. 1993), and creeping bentgrass (Liu et al. 1998; Wang et al. 2003), protecting these crops from bacterial and fungal diseases. Liu et al. (1998) assessed the antifungal activity of the herbicides bialaphos (another member of the family, phosphinic acids) and glufosinate. Bialaphos, a natural herbicide, showed a higher level of in vitro antifungal activity against *Rhizoctonia solani*, *Sclerotinia homoeocarpa* and *Pythium aphanidermatum* than glufosinate. Glufosinate reduced the radial growth of soybean fliar blight fungus, *R. solani* by up to 61%, and reduced sclerotium formation under laboratory conditions. Glufosinate exerted a fungistatic effect on *R. solani* isolates of IA colonies, but it was fungitoxic to the isolates of IB colonies (Black et al. 1996). Glufosinate suppressed the mycelial growth of *R. solani* (Fig. 44) and *S. homoeocarpa*, but it had no inhibitory effect on *P. aphanidermatum* up to the highest tested concentration (Liu et al. 1998). In contrast, gluphosinate ammonium inhibited the growth and propagation of *Phytophthora infestanc* and *Pythium ultimum* in vitro, especially when tested under nutritionally poor conditions (Kortekamp 2008). The rate of glufosinate required for

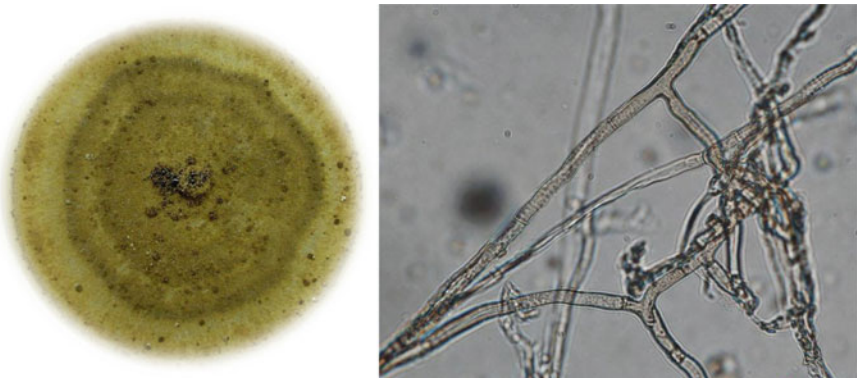


Fig. 44 *Rhizoctonia solani* (Left) mycelia and sclerotia of the fungus on agar plate (*Source* Paul Bachi, University of Kentucky Research and Education Center, Bugwood.org); (Right) mycelia of the fungus (*Source* Paul Bachi, University of Kentucky Research and Education Center, Bugwood.org)

the complete inhibition of the mycelial growth of *S. homoeocarpa* (336 mg L^{-1}) was less than that for *R. solani* (448 mg L^{-1}) (Wang et al. 2003), indicating that *S. homoeocarpa* was more sensitive to glufosinate than *R. solani* (Liu et al. 1998; Wang et al. 2003).

Additionally, glufosinate directly inhibited the mycelial growth of grapevine pathogens *Botrytis cinera*, *Guignardia bidwellii*, *Penicillium expansum*, and *Phomopsis viticola*, when it was applied at various concentrations under in vitro conditions (Albrecht and Kortekamp 2009). Interestingly, the reaction of different fungi to the presence of gluphosinate was different. Gluphosinate was found of almost a complete genostatic impact on the sporulation of *P. expansum* when it was tested at the concentration 500-fold more diluted than the recommended dose for field applications, a finding of importance in the control of late seasonal infections. However, glufosinate was less toxic to its mycelial growth, while it reduced the mycelial growth of *G. bidwellii* by about 80% at the same dilution effective on *P. expansum* sporulation. Moreover, glufosinate severely inhibited the growth of *Plasmopara viticola*, an obligate parasite and causal agent of grapevine downy mildew. While it was phytotoxic at higher doses, glufosinate hermetically stimulated chlorophyll content increase in grapevine leaves without any visible phytotoxicity at low doses (Kortekamp 2008, 2010). Various concentrations of bialaphos solutions were applied to transgenic, bialaphos- and glufosinate-resistant creeping bentgrass (*Agrostis stolonifera* var. *palustris*) inoculated with fungal pathogens. Bialaphos applications were able to significantly reduce symptomatic infection by *R. solani* and *S. homoeocarpa* on transgenic plants. Bialaphos significantly inhibited *P. aphanidermatum*, but not to the same degree that *R. solani* and *S. homoeocarpa* were inhibited. These results indicate that bialaphos may provide a means for the simultaneous control of weeds and fungal pathogens in turf areas with transgenic, bialaphos-resistant creeping bentgrass. Glufosinate ammonium specifically impeded appressorium formation of rice blast and brown leaf spot pathogens, respectively *Magnaporthe oryzae* and *Cochliobolus miyabeanus* (sexual state of *Bipolaris oryzae*) on hydrophobic surface as well as on 35S *bar* transgenic rice. In contrast, conidial germination was not affected (Ahn 2008).

HRAC Group I

No information was found.

HRAC Group K1

Studies with the herbicides applied in rapeseed or canola (*Brassica napus* var. *oleifera*) fields demonstrated that dinitroaniline herbicides trifluralin and ethalfluralin were highly toxic against *Sclerotinia sclerotiorum*, the causal fungus of canola white stem rot disease (Pakdaman and Mohammadi Goltapeh 2007). The direct antifungal activity of trifluralin against *Fusarium solani* (Yu et al. 1988), and the oomycetous pathogen *Aphanomyces euteiches* (Jacobsen and Hopen 1981) has also been recorded. Singh et al. (1999) indicated the inhibitory effect of two

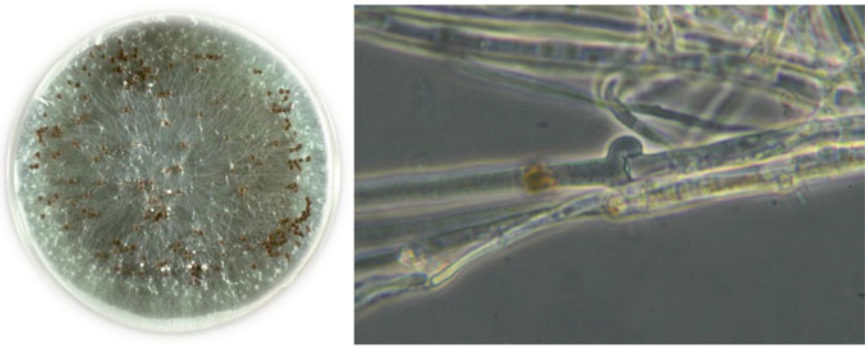


Fig. 45 *Sclerotium rolfsii*, (Left) Mycelia and sclerotia of the fungus on agar plate (*Source* Howard F Schwarz, Colorado State University, Bugwood.org); (Right) Clamp connections (hooked structures) on the mycelia of the fungus (*Source* http://www.paceturf.org/) indicating its basidiomycetous nature

pre-emergence herbicides, fluchloralin and pendimethalin on growth and development of *Phytophthora drechsleri* f. sp. *cajani*, the causal agent of pigeon pea blight. Trifluralin was found on no impact on *Pectinobacterium carotovorum* growth rate when tested at the concentrations of 25, and 50 μg mL^{-1} (Breazeale and Camper 1972). Fluchloralin drastically inhibited the growth of *Sclerotium rolfsii* (Fig. 45) and *Rhizoctonia bataticola* under in vitro conditions (Tripathi et al. 1988).

Similarly, trifluralin and basalin inhibited the growth of *S. rolfsii* in vitro (Lal and Nagarajan 1988). Trifluralin and pendimethalin were the most efficient herbicides out of the tested herbicides (also including imazethapyr, metachlor, and a mixture of acetochlor + imazethapyr) that significantly reduced the production of viable sclerotia by *S. rolfsii* under in vitro conditions (Pastro and March 1999). Also, the inhibition of the sclerotial germination of *S. rolfsii* has been reported with fluchloralin studied using soil plate technique, where its inhibitory effect (94.02%) was statistically at par ($P = 0.05$) with that of the most effective fungicides, carboxin (98.99%), and tridemorf (97.89%) (Bhoraniya et al. 2002). Trifluralin (Treflan®), and ethalfluralin (Sonalan®), both tested in vitro at two concentrations of 1 and 10 μg mL^{-1}, were of no significant effect on the hatching of soybean cyst nematode *Heterodera glycines* when compared with that in deionized water, however, they exerted a negative impact ($P \leq 0.05$) when compared with hatching rate in the solution of zinc sulfate (3.14 mM). The negative effect of Sonalan® on the nematode hatching in zinc sulfate solution was almost twice that of Treflan® (Wong et al. 1993).

HRAC Group K2

No information was found.

HRAC Group K3

In an investigation on the antifungal activity of two soil herbicides conducted by Kataria and Dodan (1983), alachlor (a chloroacetamide) was found more inhibitive to *Rhizoctonia solani* growth than fluchloralin (a dinitroaniline from HRAC group K1) in potato dextrose broth (PDB). However, the infective capacity of the pathogen was not altered by growing it in a medium containing either of the herbicides. Metolachlor inhibited of the sclerotial germination (62.78%) (Bhoraniya et al. 2002). Alachlor (LaSSO®) tested in vitro at the concentrations of 24, and 240 μg mL^{-1} was of no negative impact on the hatching of soybean cyst nematode *Heterodera glycines* when compared with deionized water, however, its effect was statistically significant ($P \leq 0.05$) compared with that of zinc sulfate solution (3.14 mM), which increased hatching (Wong et al. 1993).

HRAC Group M

The direct antifungal effect of dinoseb, an uncoupler and membrane disruptor herbicide from dinitrophenols family, on the fungal pathogens *Aspergillus niger*, and *Alternaria tenuissima* (Kovacs and Malligni 1975) as well as on the oomycetous pathogen *Aphanomyces euteiches* (Jacobsen and Hopen 1981) has been recorded.

HRAC Group N

Paul and Schönbeck (1976) studied the impact of the herbicide diallate (a thiocarbamate) on several cereal pathogens in vitro, and found that the herbicide added to agar reduced mycelial growth of *Fusarium avenaceum*, *F. culmorum*, *F. graminearum*, *F. moniliforme*, and *Bipolaris sorokiniana*. Soil-incorporated diallate reduced maize root rot caused by *F. moniliforme*, and wheat crown and root rot induced by *F. avenaceum*, and *F. culmorum*. Diallate inhibited maize root rot and also barley disease caused by *B. sorokiniana* in hydrocultural maize and barley growing systems. Diallate severely prevented *F. moniliforme* penetration in stele region, and hyphae in roots exposed to diallate often were partially deformed and of the cytoplasm seemingly very granulated. Helmeczi et al. (1988) studied the effect of 14 herbicides and two herbicide combinations on three microscopic soil fungi following Miller's method, and found that (i) none of the 14 herbicides and 2 combinations belonging to four derivative groups induced the growth of *Aspergillus niger*, *Fusarium oxysporum*, and *Rhizopus nigricans*; (ii) higher doses of the herbicides prevented the growth of all test organisms significantly, and thiocarbamate derivatives Alirox B 80 EC (EPTC), Anelda Plus 80 EC (butylate) and Anelda III (butylate) were the most inhibitory; and (iii) the sensitivity of test organisms against the examined herbicides decreased in the order of *Fusarium*, *Rhizopus*, *Aspergillus*.

The herbicides of thiocarbamates family, cycloate (Ro-Neet® tested at 15 mg mL^{-1}), pebulate (Tillam® tested at 21 mg mL^{-1}), triallate (Avadex® BW tested at 7 mg mL^{-1}), and vernolate (Vernam® tested at 12 mg mL^{-1}) eliminated the subsequent hatch of *Globodera rostochinensis* and *Heterodera schachtii* eggs in root diffusate. Tests with New Blue R (Shepherd 1962) indicated that triallate applied at the recommended dose for field application (1.7 kg a. i. ha^{-1}) killed 90% of cyst contents. The thiocarbamate family seems to be the important factor in the inhibition, and there is no link between the method of formulation and the effect on hatching of nematode eggs. Furthermore, the effect of thiocarbamate herbicides on hatch was markedly reduced by dilution, therefore, any thiocarbamate herbicide-based control strategy probably as a pre-emergence treatment would have to overcome the problem of soil incorporation to ensure that the required concentrations contact the cysts (Perry and Beane 1989).

HRAC Group O

The synthetic auxin like growth regulator, 2,4-D inhibited in vitro growth of *Ceratocystis radicicola*, *Fusarium graminearum*, *F. oxysporum*, *Gaeumannomyces graminis*, *Macrophomina phaseolina*, *Rhizoctonia solani*, *Sclerotinia sclerotiorum* (Fig. 46), *Verticillium dahliae*, and that of two oomycetous pathogens, *Phytophthora* sp., and *Pythium* sp. (Pakdaman et al. 2002). 2,4-D drastically inhibited the in vitro growth of *Sclerotium rolfsii* (Tripathi et al. 1988) and *Rhizoctonia bataticola* (Tripathi et al. 1988), and *R. solani* (Pathak et al. 1996).

HRAC Group P

No information was found.

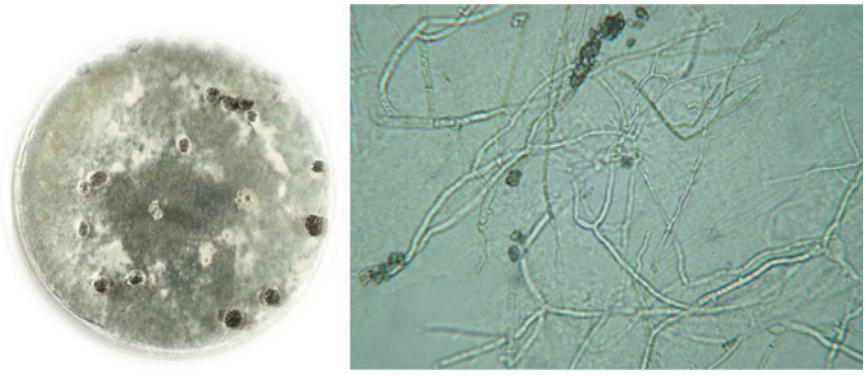


Fig. 46 *Sclerotinia sclerotiorum*, (Left) Mycelia and sclerotia on agar plate (*Source* Howard F. Schwartz, Colorado State University, Bugwood.org), (Right) mycelia of the fungus (*Source* Clarissa Balbalian, Mississippi State University, Bugwood.org)

HRAC Group Z

Because of the high diversity of the origin, chemical structure, and mode of action of these herbicides, here we ignore this group.

Direct Pro-pathogenic Effects of Herbicides

Some xenobiotic compounds such as herbicides are known as pro-pathogenic agents that either directly enhance pathogen growth and development or directly increase its virulence. Here, a new term is coined and "pathancer", the abbreviation of pathogen enhancer, is suggested. It is not unusual for low rates of herbicides to stimulate in vitro pathogen growth (for example, Yu et al. 1988). Hormesis (the stimulatory effect of a sub-toxic level of a toxin) is common with both fungicide effects on fungi and herbicide effects on plants (Duke et al. 2006). Thus, dose rates are likely to be highly important in both direct and indirect effects of herbicides on plant disease. Some of the herbicides are not of direct toxicity to the pathogenic fungi and oomycetes, and such herbicides can be used as xenobiotic sources of energy as well as materials (such as carbon, nitrogen, phosphorus, sulfur and so on) needed for the growth and development of these pathogens. For instance, 2,4-dichlorophenoxyacetic acid (2,4-D) can be biodegraded and catabolized by *Burkholderia cepacia* (Bhat et al. 1994), *B. graminis* (Dejonghe et al. 2000), *Pseudomonas* spp. (Musarrat et al. 2000), and the fungus *Phanerochaete chrysosporium* (Yadav and Reddy 1993). The effect of the herbicides, pendimethalin, and prometryn (applied to the soil at 2.4, and 3.6 μg a. i. g^{-1} soil, equivalent to the respective recommended field concentrations of 0.9, and 1.3 kg a. i. ha^{-1}, respectively) on the incidence of *Rhizoctonia solani*-induced cotton seedling damping-off was investigated in the microcosm conditions. Prometryn significantly raised up the incidence of pre-emergence damping-off, while post-emergence damping-off increased significantly after the application of pendimethalin and prometryn. Also, similar results were obtained with both herbicides, particularly with prometryn in the fields treated with the herbicides before planting (Heydari and Misaghi 1998). Arfarita et al. (2011) applied glyphosate as a screening agent used as the sole phosphorus source for the isolation of soil-born fungi from forest soil. Three fungal strains were able to grow consistently in the presence of glyphosate as the only source of phosphorus (*Fusarium* sp. strain FRP1, *Scopulariopsis* sp. strain FRP2, and *Trichoderma* sp. strain FRP3). On standard medium, ten fungal strains were isolated and identified, *Botrytis* sp. strain FR1, *Mucor* sp. strain FR2, *Acremonium* sp. strain FR3, *Trichoderma* sp. strain FR4, *Botrytis* sp. strain FR5, *Crysosporium* sp. strain FR6, *Scopulariopsis* sp. strain FR7, *Trichoderma* sp. strain FR8, *Botrytis* sp. strain FR9, and *Acremonium* sp. strain FR10. Of the three screened fungal species, *Scopulariopsis* sp. strain FRP2 and *Trichoderma* sp. strain FRP3 were of the highest ratios of growth diameter, and were selected for further studies. Some herbicides are biodegraded or bioconverted to non-toxic metabolites, and some are not lethal but restrict pathogen growth when

applied following the recommendations on the labels. These herbicides can act as pathancers. The application of glyphosate, and chlorsulfuron has been associated with increased levels of take-all fungus, *Gaeumannomyces graminis* (Mekwatanakarn and Sivasithamparam 1987). In a greenhouse study of the impact of atrazine, the populations of *Fusarium* were increased up to four folds compared with the soil not treated with atrazine added at 10, 30, and 100 μg g^{-1} soil. Additionally atrazine amendment in the soil at the rate of 10 μg g^{-1} soil led to the increased germination of *Fusarium* macroconidia, growth of germ tubes, as well as the formation of chlamydospores (Percich and Lockwood 1975). The herbicides like sethoxydim may be of potential pathancing effects on the virulence of some pathogens such as head blight causal *Fusarium* species. *Fusarium graminearum* (Fig. 47) is able to tolerate this herbicide and even to increase its rate of growth, however, its characteristic carmine red color decreases to pale pink, indicating reduced production of naphthoquinone pigments (Pakdaman and Kariman 2006) that occurs through polyketide biosynthesis pathway (Kim et al. 2005). This can lead to the fortification of trichothecene biosynthesis pathway (Pakdaman and Kariman 2006). Trichothecenes are among the main virulence factors responsible for the host plant yield loss, as well as the occurrence of mycotoxicoses in human and livestocks.

Chan Cupul et al. (2014) evaluated the effect of atrazine concentrations on mycelial growth and ligninolytic enzyme activities of eight Mexican ligninolytic macrofungi in a semi-solid culture medium. Inhibition of mycelial growth and growth rates were significantly affected ($p = 0.05$) by atrazine concentrations (468, 937, 1875, and 3750 mg L^{-1}). In accordance with the median effective concentration (EC_{50}), *Pleurotus* sp. strain 1 proved to be the most tolerant isolate to atrazine (EC_{50} = 2281.0 mg L^{-1}), although its enzyme activity was not the highest. *Pycnoporus sanguineus* (Fig. 48) strain 2, *Daedalea elegans* and *Trametes maxima* showed high laccase activity (62.7, 31.9, 29.3 U mg/protein, respectively) without

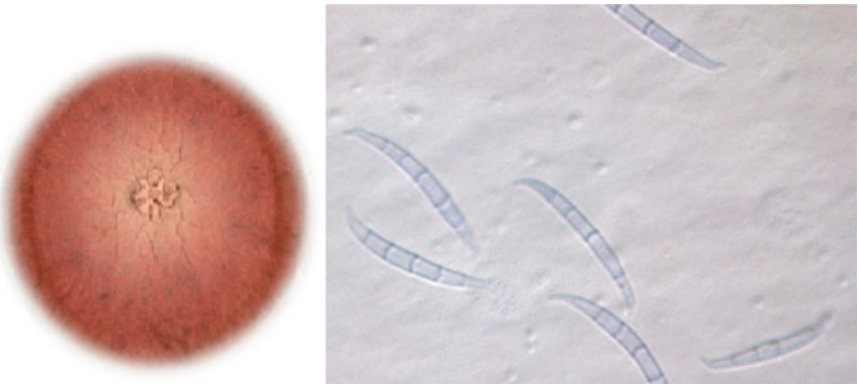


Fig. 47 *Fusarium graminearum* (Left) mycelial colony on agar plate (*Source* http://mycota-crcc.mnhn.fr/); (Right) Macroconidia (*Source* Liu et al. 2012)

atrazine (control); however, this activity considerabely increased ($p < 0.05$) (to 191.1, 83.5 and 120.6 U mg/protein, respectively) because of the effect of atrazine (937 mg L^{-1}) in the culture medium.

Pleurotus sp. strain 2 and *Cymatoderma elegans* significantly increased ($p < 0.05$) their manganese peroxidase (MnP) activities under atrazine stress at 468 mg L^{-1}. The isolates with high EC_{50} (*Pleurotus* sp. strain 1) and high enzymatic activity (*P. sanguineus* strain 2 and *T. maxima*) could be considered for future studies on atrazine mycodegradation. Furthermore, this study confirms that atrazine can increase laccase and MnP activities in ligninolytic macrofungi (Chan Cupul et al. 2014). During invasive growth of pathogenic fungi in host plants, three main enzymes get directly or indirectly involved in the modification of lignin structures that are lignin peroxidases (LiPs), manganese peroxidases (MnPs), and laccases sensu stricto (Kuo et al. 2015). In addition to lignin degradation, fungal laccases play notable roles in fungal morphogenesis (Zhu and Williamson 2004), stress defence (Thurston 1994; Gianfreda et al. 1999), as well as fungal virulence (Zhu and Williamson 2004; Kuo et al. 2015). MnP produces Mn^{3+} ions that play their major role in the oxidation and degradation of lignin (Forrester et al. 1988). The Mn^{3+} ions can directly oxidize the phenolic compounds in lignin. The Mn^{3+} ion can degrade lignin through catalysis of alkyl-aryl cleavages as well as alpha-carbon oxidation in phenols (Tuor et al. 2002). Additionally, the ions can oxidize some organic sulfur compounds and unsaturated fatty acids that leads to the formation of thiyl and peroxyl radicals. In the presence of molecular oxygen, the radicals can oxidize lignin or form hydrogen peroxide as the result of their reaction to water (Wariishi et al. 1989; Kapich et al. 1999).

Fig. 48 The laccase producing mushroom, *Pycnoporus sanguineus* (*Source* www.mycelia.be/)

3.2.2 Indirect Impact of Herbicides on Plant Pathogens

Indirect Impact of Herbicides on Pathogens Through Control of Weeds

Herbicides control of the host weeds of pathogens that otherwise could serve as survival, reservoir, alternative, alternate, and volunteer hosts and act their roles as primary infection foci, or dispersal centers for pathogens, vectors, or both. Herbicides play an important role in the control of plant diseases caused by facultative saprobes and biotrophic parasites. Soil-transient pathogens are not competitive saprophytes in a complex soil medium. Therefore, the application of non-selective herbicides will be helpful in the reduction of pathogen inoculum in the soil before the next crop plant is grown. Heteroecious rust fungi as a group of biotrophic fungi require plant hosts usually of different taxonomical groups, and soil/pre-emergence application of herbicides will be helpful in the reduction of the disease through eradication of the alternate host weeds and will lead to the plant disease cycle beakage. On the other hand, the treatment with selective post-emergence herbicides will have its own obliging effect in the combat against autoecious as well as heteroecious rust pathogen development on alternative hosts after emergence of host crop. Post-emergence herbicides would also be useful in the control of biotrophic pathogens of broad host ranges such as *Erysiphe polygoni*, and cucumber mosaic virus. However, with necrotrophic pathogens and with pathogenic facultative parasites, the conditions will be very different. The application of herbicides, either of non-selective pre-emergence type or of selective and/post-emergence type, will provide a lot of predisposed plants and dead plant materials that can easily be colonized by these saprophytically very competitive groups of pathogens. Pathogens like *Fusarium*, *Rhizoctonia*, *Alternaria*, *Botrytis cinerea* (Fig. 49) and *Pythium ultimum* lay in this group. Most of these fast-growing pathogens are armed with the genes responsible for the biosynthesis and the secretion of the toxins that are used in order to defend their territories in the competition against other microorganisms. As the result of herbicidal treatment, these necrotrophic pathogens will gain an exceptional opportunity for their fast growth and development in the treated declining weeds with disturbed defense system resistance. These conducive conditions will lead to the increased inoculum potential of the pathogen as the consequence of its increased accessibility to dead organic materials. This can result in the outbreak of these diseases if no contrivance is made with the management of the remnant weed residues.

For instance, pendimethalin and atrazine inhibit the mycelial growth of *Pythium arrhenomanes*, but neither decrease root rot severity in sugarcane. Glyphosate, pendimethalin and terbacil are injurious to sugarcane and increase root rot severity (Dissanayake et al. 1998). The increased sugarcane root rot after atrazine treatment can be explained by its especial mode of distribution *in planta* (Jachetta et al. 1986), and the method of application of atrazine in their work (spraying instead of soil application). This strongly indicates the importance of herbicide application and translocation mode in the determination of its final impact on plant disease control.

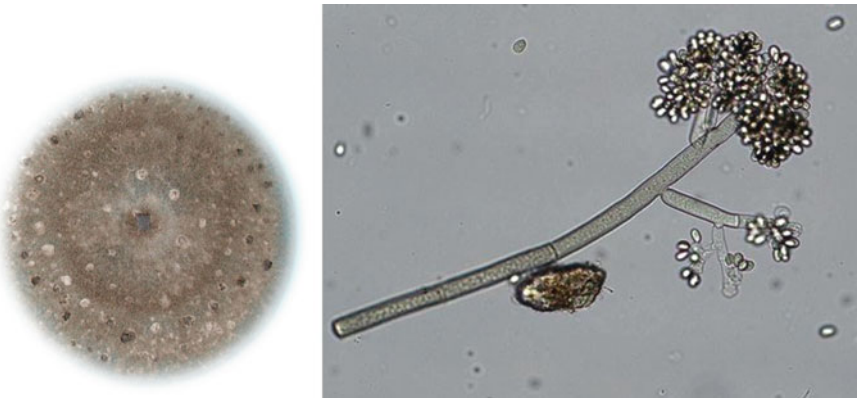


Fig. 49 Gray mold, *Botrytis cinerea* (Left) mycelial growth with gray to brown conidiation and sclerotial development on potato dextrose agar plate after 10 days of incubation at 20 C and 12 h alternating cycles of darkness and fluorescent light (*Source* Xiao 2006); (Right) Conidiophore with branches that end to fertile with denticles bearing conidia (*Source* Paul Bachi, University of Kentucky Research and Education Center, Bugwood.org)

The eradication of some weeds that host vectors of pathogens, with selective post-emergence herbicides can lead to the attack of vectors to the main crop plants. Such attacks will lead to disease transmission to the cultivated crop plants and increased damages. However, if vectors need wild weed plants to complete the primary stages of their life cycles, and if they are very vulnerable in the absence of their host weeds and mostly are in the life stages of ignorable mobility, the elimination of host weeds will create a severe break in their life cycle and will result in the reduced rate of disease transmission. Some weeds are primary attraction foci for most of the vector populations and therefore, the conscious and clever management of these weeds will lead to the synchronized extermination of weed and vectors after attraction of the first early season populations of the vectors in early growth season and before planting or seeding of the main crop or before its emergence in the field. For example, since airborne aphids are attracted to the color yellow (Agrios 2005), weeds with yellow flowers will attract the aphids, and early season control of weeds and aphids by an herbicide will be very useful in the control of the pathogens associated with aphid vectors. Furthermore, some herbicides are known to be directly entomotoxic, and may influence the insect vectors life and reproduction. For example, the mixture of glyphosate and 2,4-D was found of direct insecticidal activity and caused 100% mortality of the adult grain beetle, *Tenebrio molitor*. More beetles escaped from the herbicide as well as nitrate treatments, suggesting a kind of behavioral avoidance of toxic environments (Castilla et al. 2010). 2,4-D ethyl ester (Weedkill 80 WP) was not toxic to the larvae of *Spilarctia obliqua* (Lepidoptera: Arctiidae), while imazethapyr (Pursuit 10 EC) and quizalofop ethyl significantly reduced the pest survival at almost all doses. The concentrations required to kill 50% of the pest larvae (LC50) were calculated for

quizalofop ethyl (LC50 = 0.230%), and imazethapyr (LC50 = 0.855%) through probit analysis, and were almost equal to labeled doses (FR) of quizalofop ethyl (FR = 0.16%) and imazethapyr (FR = 0.625%). These data indicate the high toxicity of these herbicides against *S. obliqua* larvae. Additionally, imazethapyr exerted sublethal effects on the pest at almost all concentrations tested in the artificial diet. Quizalofop ethyl was of no sublethal effect, and exerted a markedly significant effect on the pest survivorship. Therefore, quizalofop ethyl incorporation into the integrated pest management module for *S. obliqua* in legumes or oilseed crops with other biorational insecticides was suggested. However, despite of entomotoxicity of imazethapyr, such a suggestion was not made with this herbicide because of its sublethal effects (Gupta and Bhattacharya 2008). Studies with the generalist endoparasitoid *Palmistichus elaeisis* (Hymenoptera: Eulophidae) indicated the negative impact of glyphosate on its performance, however it was concluded that the responsible use of this herbicide would be acceptable (Alcántara-de la Cruz et al. 2017). Glyphosate severely reduced the reproduction and fecundity of lacewing, *Chrysoperla externa* (Neuroptera: Chysopidae) the natural enemy of aphids (Schneider et al. 2009). None of glyphosate-based products (Roundup Original®, Roundup Ready®, Roundup Transorb®, Roundup WG®, and Zapp Qi®) tested at the same acid equivalent concentration under laboratory conditions according to the International Organization for Biological Control (IOBC) standard methods for testing side-effects of pesticides on egg parasitoids, were found harmful or moderately harmful to the parasitoid *Telenomus remus* (Hymenoptera: Platygastridae) when exposure occurred at the pupal or adult stages. Therefore, all mentioned glyphosate-based herbicides can be applied in agriculture with no negative influence on the parasitoid (Stecca et al. 2016), an effective biological control agent for various *Spodoptera* (Lepidoptera: Noctuidae) spp. (Cave 2000; Morales et al. 2000; Pomari et al. 2012) responsible for heavy damages in soybean, maize, and cotton crops (Cruz et al. 1999; Carmo et al. 2009; Santos et al. 2010). In addition to insects, glyphosate improved this parameter for plant mites, the broad mite, *Polyphagotarsonemus latus* (Acari: Tarsonemidae) and *Tetranychus bastosi* (Acari: Tetranychidae) at the application rate of 360 g ae ha^{-1} (Saraiva et al. 2016). On the other hand, the elimination of weeds, within or around fields, as the providers of nectar and shelter for natural enemies of insect vectors can significantly affect the abundance as well as the diversity of parasitoids and predators (Altieri 1994). The removal of natural enemies of a given pest can lead to the resurgence of vectors and the disease(s) transmitted. Map-based precision agricultural systems can be a reliable solution, especially when the primary foci of the vectors are determined and the required actions are performed in limited areas. Such an approach not only can lead to reduced crop yield losses, but also helps in the preservation of natural biodiversity and reduced environmental pollutions. With nematodes, another group of pathogenic obligate parasites of plants, the early seasonal pre-planting application of non-selective herbicides after emergence of weeds and in the absence of host plants, will result in the death of nematodes larvae because of starvation.

Indirect Impact of Herbicides on Pathogens Through the Influence on Plant Physiology

Herbicides may increase, decrease or have no effect on plant disease that depends on the herbicide, pathogen, crop and environmental conditions (Altman and Campbell 1977a). For instance, Jackson and Stack (2002) applied the herbicide dicamba (a benzoic acid from HRAC group O) to the roots of potted Siberian elm *(Ulmus pumila)* and Russian-olive *(Elaeagnus angustifolia)* trees under greenhouse conditions. They used five herbicide concentrations equivalent to the rates of 0 (0), 93 (1/12), 140 (1/8), 280 (1/4), and 1121 (1) g ha^{-1} (lb ac^{-1}) active ingredient. Two weeks after herbicide application, each tree was inoculated with a single isolate of the canker-causing fungus *Tubercularia ulmea*. The experiment was repeated using two different *T. ulmea* isolates. Leaf cupping, a symptom of dicamba exposure, was evident two to three weeks after herbicide application. Symptoms occurred at rates of 140 g ha^{-1} (1/8 lb ac^{-1}) and above in the Siberian elms and the 1121 g ha^{-1} (1 lb ac^{-1}) rate in the Russian-olives. All four *T. ulmea* isolates caused cankers, with canker size differences between fungal isolates and between tree species, however, none of the herbicide treatments increased or decreased *T. ulmea* canker size (Jackson and Stack 2002). In contrast, other herbicides applied and incorporated into soil before planting, including cycloate (a thiocarbamate from HRAC group N), diethathyl-ethyl, EPTC (a thiocarbamate from HRAC group N), and ethofumesate (a benzofuran from HRAC group N) as well as those applied after emergence, including clopyralid (a pyridine carboxylic acid from HRAC group O), desmedipham, desmedipham + phenmedipham (phenyl-carbamates from HRAC group C1), and triflusulfuron-methyl (a sulfonylurea in HRAC group B) did not affect severity of seedling disease and chronic root rot disease of sugar beet caused by the soil born oomycetous pathogen *Aphanomyces cochlioides* compared to a hand-weeded control (Roebke et al. 2002). However, herbicides may affect plant physiology and lead to changes at both physical and biochemical levels (Altman and Campbell 1977a). They may lead to the alterations in plant growth, lignin-containing substances, β-glucosides (Paul and Schönbeck 1976), wax layers on shoot and foliage (Heitefuss 1970, 1972), the rate of root exudates and the release of nutrients from plant root system (Altman and Campbell 1977b; Liu et al. 1995, 1997; Wyse et al. 1976) and altered mineral nutrition (Neumann et al. 2006). Reduced availability of important macro-, as well as micro-nutrients (potassium, magnesium, iron, and zinc) and increased availability of sodium and calcium have been reported as the result of herbicidal treatments with the recommended rates of paraquat (4 L ha^{-1}), glyphosate (4 L ha^{-1}), Primeextra (4 L ha^{-1}), and atrazine (3 kg ha^{-1}, applied for soil treatment) (Sebiomo et al. 2012). Also, low levels of residual glyphosate in soil also reduce root uptake and translocation of Fe, Mn, and Cu (Eker et al. 2006; Ozturk et al. 2008). Additionally, some herbicides may enhance defense mechanisms including the elicitation and accumulation of phytoalexin and mimicry of the hypersensitive response (Landini et al. 2003; Nelson et al. 2002a, b), and finally they may alter the level of host plant resistance to pathogens (Starratt and Lazarovits 1996).

Here, the effect of herbicides in the increase or decrease of plant diseases is discussed due to its high importance in the integrated management of plant diseases.

Increased Disease Resistance of Herbicide-Treated Crops

Increased plant resistance to disease as the consequence of certain herbicidal treatment has been reported. Huber et al. (1966) indicated that although diuron (a member of HRAC group C2) used at the rate of 1.12 kg ha^{-1} did not inhibit the initial penetration by the winter wheat foot rot disease pathogen *Cercosporella herpotrichoides*, it led to the increased winter wheat resistance to the disease. Diuron was not of direct antifungal activity when it was used at the ratio below 100 ppm on corn meal agar. Also, it was shown that the populations of soil fungi, bacteria, and actinobacteria were not affected by diuron applications. Heitefuss and Bodendörfer (1968) found that urea and triazine herbicides could significantly decrease wheat eye-spot disease caused by the fungus *C. herpotrichoides* and powdery mildew caused by *Blumeria graminis* f. sp. *tritici* that was not related to the removal of weeds, as the same reductive effect had also been observed in a weed-free crop. The extracts from simazine-treated wheat plants added to agar medium were inhibitory on *C. herpotrichoides* growth compared with control media. However, this inhibitory effect disappeared after glucose was added to the medium. They concluded that such an inhibitory fungitoxic impact was not adequate to explain the observed disease reduction resulted from the field-application of triazine and urea herbicides. Further experiments conducted by Brandes and Heitefuss (1971) on the physiological and biochemical alterations as the consequence of simazine (a triazine member of HRAC group C1) and monolinuron (an member of urea family in HRAC group C2) revealed significant changes in total nitrogen, sugar, 4-hydroxy-7 methoxy-1,4 benzoxazin-3 on-2 glucoside (DMBO-glucoside), and 2,4-dihydroxy-7 methoxy-1,4 benzoxazin-3 on-aglucan (DMBO-aglucan) that were totally related to the observed disease decrease in wheat plants exposed to the attacks by both pathogenic fungi. Two herbicides dinitrobutylacetate and ioxynil (a nitrile from HRAC group C3) were of no effect on the occurrence of both wheat diseases mentioned above.

Protoporphyrinogen oxidase inhibitors (HRAC group E) cause enough oxidative stress at sublethal levels to induce production of phytotalexins (Kömives and Casida 1983). Some protoporphyrinogen oxidase inhibitor herbicides such as azafenidin (a triazolinone; Viator et al. 2002), flumioxazin (a N-phenylphthalimide; Viator et al. 2002), lactofen (Nelson et al. 2002a), fumesafen (Nelson et al. 2002a), oxyfluorfen (Nelson et al. 2002a), carfentrazone (Nelson et al. 2002a), and sulfentrazone (a triazolinone; Nelson et al. 2002a; Viator et al. 2002) reduce the severity of sclerotinia stem rot of soybean (Dann et al. 1999; Nelson et al. 2002a, b), decrease soybean cyst nematode reproduction (Levene et al. 1998), and the severity of rhizoctonia foliar blight of soybean (Black et al. 1996) through induction of host resistance that is believed to be associated with the singlet oxygen radicals generation by PPOase inhibitor herbicides that destroy lipids in cell membranes

(Daugrois et al. 2005). High levels of glyceollin are induced by lactofen (a nitrophenoxybenzen) in soybeans leading to the protection from white mold (sclerotinia stem rot; Dann et al. 1999). The application of flumioxazin to the leaves of sugarcane resulted in the reduced severity of pythium root rot (Daugrois et al. 2005). Lactofen applied at the V3 stage of soybean, reduced the severity of sclerotinia stem rot by 40–60% in the years of high disease pressure (Dann et al. 1999). It is believed that lactofen is unlikely to directly inhibit *S. sclerotiorum* because of its rapid degradation in field, where it is of only a 3 day half-life. Therefore, the disease suppression may be because of other reasons such as alteration of the canopy environment or reduction in natural infection sites on the plant, or more possibly because of the induction of other localized defenses in plants as it was implied by the reduced lesion development resulted from the fungus in lactofen-treated detached leaflets which were of higher levels of the soybean phytoalexin, glyceollin (Dann et al. 1999). Most of the herbicides in the HRAC group E induce the synthesis of glyceollin in soybeans (Landini et al. 2003). Keen et al. (1982) indicated that glyphosate (a glycine from HRAC group G) application led to the induction of glyceollin phytoalexin production and accumulation by soybean plants, and resulted in the enhanced resistance to *Phytophthora megasperma* f. sp. *glycinea*.

Glufosinate ammonium (a phosphinic acid in HRAC group H) reduced development of rice blast and brown leaf spot diseases in *35S: bar*-transgenic rice. Both pre- and post-inoculation treatments with the herbicide decreased disease development. Glufosinate ammonium diminished mycelial growth of both pathogens however its inhibitory effect was attenuated under malnutrition conditions. Glufosinate ammonium led to slight chlorosis and lower chlorophyll content, however, these changes were almost completely restored in transgenic rice within 7 days. Glufosinate ammonium triggered transcriptions of pathogenicity-related (*PR*) genes and hydrogen peroxide accumulation in the transgenic rice and *PR1* transcription in *Arabidopsis thaliana* wild type Col-0 harboring *35S:bar* construct. All transgenic arabidopsis plants exhibited distinctive hydrogen peroxide accumulation induced by the treatment with glufosinate ammonium. Fungal infection could not alter the transcriptions of *PR* genes and the accumulation of hydrogen peroxide induced by the herbicide. Glufosinate ammonium infiltration could not affect appressorium formation of *M. grisea* in vivo, however, inhibited blast disease development. Hydrogen peroxide scavengers nullified blast protection and transcriptions of *PR* genes incited by glufosinate ammonium however, they did not influence brown leaf spot development. Both direct inhibition of pathogen infection and activation of defense systems are responsible for the protection of *bar*-transgenic rice against blast and brown leaf spot diseases (Ahn 2008; Fig. 50). Low concentrations of glufosinate, although not effective on the release and germination of the grapevine downy mildew pathogen zoospores, restricted the development of its intercellular mycelium and dramatically reduced its sporulation (Kortekamp 2010). Higher doses up to that normally applied to the field were completely effective in the inhibition of each developmental step of the disease cycle. Glufosinate ammonium indicated prophylactic as well as curative activities.

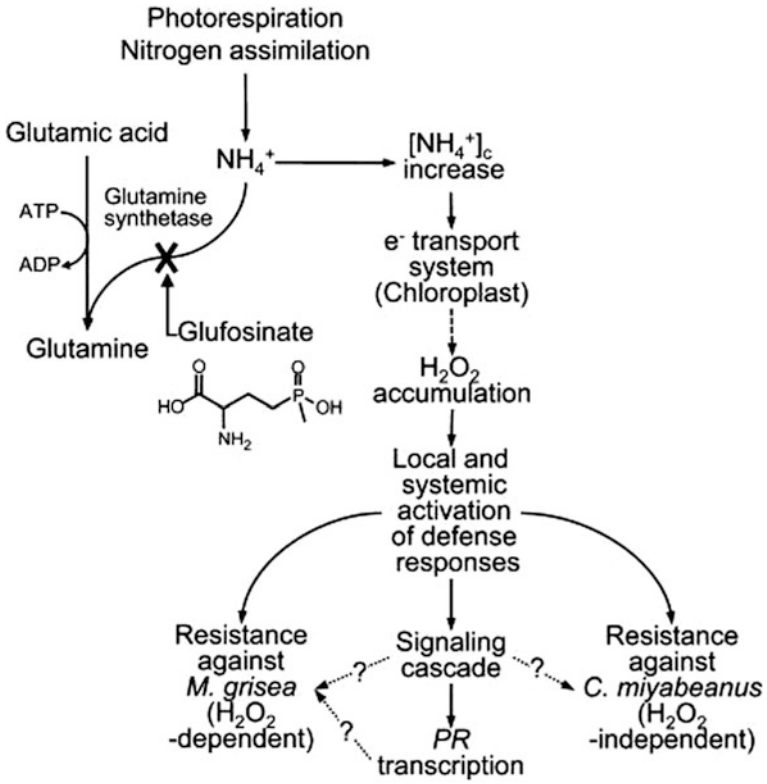


Fig. 50 Proposed model for glufosinate-induced disease resistance. Glufosinate initiates accumulation of free radicals by irreversible binding with and inactivation of Gln synthetase. Toxic ammonia derived from photorespiration or nitrogen assimilation is increased within the cell and disturbed electron transport system within chloroplast. Free radicals were produced and in turn, this molecule triggers disease resistance against *Magnaporthe oryzae* and *Cochliobolus miyabeanus* (Ahn 2008)

Pre-infectional and post-infectional treatments with the herbicide led to significant reduction of sporulation rates. The herbicide exhibited more inhibitory effect on spore production when the time intervals between inoculation and herbicidal treatment decreased, since the pathogen could not access to the opportunity required for the establishment of dense network of hyphae within the infected tissue and sporulation. However, pre-inoculation herbicidal treatment increased prophylactic impact when time intervals between treatment and inoculation increased, suggesting the activation of plant defense pathways. Alternatively and additionally, the herbicidal treatment might lead to the aggravated niche conditions due to the reduced level of amino acids, reduced nitrogen availability, an altered pH and/or an accumulation of ammonium. Especially, changes in the pH, nitrogen availability, and ammonium concentrations have been suggested as a regulatory factor for colonization of pathogenic fungi (Kortekamp 2011).

The use label for lactofen in the USA implies to its usability in soybean white mold management. Grinstein et al. (1976) reported the increased resistance to *Rhizoctonia solani*, *Fusarium*, and *Verticillium dahliae* as the result of the treatment with dinitroaniline herbicide such as fluchloralin (a dinitroaniline from HRAC group K1; Basalin®). Trifluralin decreased fusarium wilt on tomato and melon (Cohen et al. 1986), while had no effect on rhizoctonia root and crown rot of sugar beet (Ruppel et al. 1982). Trifluralin potentiates cotton and tomato to produce fungitoxic compounds treating with vascular wilt-causing fungi (Grinstein et al. 1984). Cohen et al. (1992b) found that the growth of tomato seedlings (cv. Bonny Best) in rooting substrate amended with the dinitroaniline herbicide, dinitramine (1 ppm in sand/Pro-mix, 1:1 or 1:2, V V^{-1}) caused a suppression in bacterial wilt symptoms induced by *Ralstonia solanacearum* strain K60. Dinitramine was not inhibitory to *R. solanacearum* in in vitro assays nor did it restrict the pathogen multiplication in the treated plants. Dinitroamine reduced the severity of bacterial canker caused by *Clavibacter michiganense* subsp. *michiganense* JD83-1, but had no significant impact on the severity of bacterial speck, induced by *Pseudomonas syringae* pathovar *tomato* DC894H. Resistance was not induced by similar exposure of plants to the herbicides ethalfluralin or oryzalin (Cohen et al. 1992b).

Resistance to fusarium wilt, caused by *Fusarium oxysporum* f. sp. *lycopersici*, was induced by the exposure of seedlings to the herbicides dinitramine, ethalfluralin, and oryzalin (Cohen et al. 1992b). Exposure of plants to the dinitroaniline herbicide, trifluralin, did not induce resistance towards either bacterial wilt or fusarium wilt (Cohen et al. 1992b). A related herbicide, pendimethalin, induces the synthesis of the phytoalexin tomatine in tomato (El-Shanshoury et al. 1995). Starratt and Lazarovits (1996) demonstrated the highly induced levels of tomato seedlings to *Fusarium oxysporum* f. sp. *lycopersici* as the result of dinitroaniline herbicide application. Lotan-Pompan et al. (2007) used suppression subtractive hybridization (SSH) and cDNA-amplified fragment-length polymorphism (cDNA-AFLP) techniques in order to identify the genes involved in trifluralin herbicide-induced resistance of melon to *Fusarium oxysporum* f. sp. *melonis*. From a total of 123 clones they isolated, sequenced and annotated, 60 clones had never been isolated from melon. A significant proportion (35%) of the total 123 clones was similar to genes previously described in relation to stress, or defense. They analyzed the expression of thirty-two selected clones in detail, and found that one-third of which were up-regulated in response to trifluralin treatment and/or inoculation with the fungal pathogen. They discussed over the putative roles of seven of the clones in stress. Further studies indicated enhanced expression of four stress-related and up-regulated genes in the plants under salinity stress, suggesting that trifluralin induces a general stress response which protects the plant against fusarium wilt. Herbicides with different mechanisms of action can also stimulate production of phytoalexins and thereby influence plant disease resistance. For example, pretilachlor and butachlor (chloroacetamide members of HRAC group K3) trigger the accumulation of the phytoalexins momilactone A and sakurantetin in rice leaves (Tamogami et al. 1995). Acetochlor (a chloroacetamide in HRAC group K3) induced tomato plant resistance towards fusarium wilt but did not

towards bacterial wilt of tomato caused by *Ralstonia solanacearum* strain K60 (Cohen et al. 1992b).

In field trials, Jacobsen and Hopen (1981) demonstrated that dinoseb (a dinitrophenol in HRAC group M) and trifluralin reduced aphanomyces root rot on peas caused by *A. euteiches*. They found that both dinoseb and trifluralin were highly effective against aphanomyces root rot of peas at economical field rates, with dinoseb providing consistently superior control. Trifluralin offered better control of annual grass weeds. They recommended the consideration of the interaction of weed control, phytotoxicity, and disease control in the selection of herbicides. They suggested dinoseb as an excellent choice in the case of obtainment of excellent stands with minimal weed pressures and the occurrence of root rot as a limiting factor. Where weed pressure was as great as or greater than disease control, they recommended a combination of trifluralin + dinoseb, or trifluralin alone. They wrote that the effect of these herbicides on *A. euteiches* might be beneficial in controlling fungal pathogens with similar life cycles. Diallate (a thiocarbamate from HRAC group N) a herbicide of decreasing effect on root rot diseases of cereals caused by various *Fusarium* species reduces the abundance of the lipids, globules, and spherical bodies in the cells of the treated plants, and leads to about 25% greater β-glucosidase activity in diallate-treated cereal crops compared with controls (Paul and Schönbeck 1976).

Increased Disease Susceptibility of Herbicide-Treated Crops

Resistant as well as susceptible maize hybrids became more sensitive to maize dwarf mosaic virus (MDMV) with the increasing amounts of atrazine (a triazine in HRAC group C1) so that the application of 20 ppm atrazine led to 100% infection. Inoculation of maize plants with the virus and subsequent treatment with 1 ppm atrazine inhibited maize growth (Cole et al. 1968, 1969a, b). Some herbicides decrease crop resistance to plant diseases. For instance, the spring wheat plants malformed as swollen and bulbous because of their exposition to the herbicide mecoprop (a member of HRAC group O) were very amenable for easier penetration of the take-all pathogen fungus *Gaeumannomyces graminis* (Nilsson 1973). Hsia and Christensen (1951) recorded heavier infection with *Bipolaris sorokiniana* in the wheat plants that had been weakened, stunted, and predisposed to fungal infection due to the treatment with 2,4-D (a member of HRAC group O). The herbicide 2,4-D stimulates the accumulation of protein in corn, that has been proposed as a probable reason for the favored growth of the southern corn leaf blight pathogen *Bipolaris maydis* and resulting increased disease in corns treated with 20–200 ppm 2,4-D. Accordingly, the plants treated with only 10 ppm 2,4-D as well as untreated plants were of fewer blight lesions (Oka and Pimentel 1976). Similarly, picloram (a chloropropionic acid in HRAC group O) applied to soil increased wheat and corn seedlings root rot caused by several soil-born pathogenic fungi (Semeniuk and Tunac 1968) that was positively correlated with the significant increases in the exudation of total carbohydrates and reducing sugars that might account for the

increased root damage from soil-born root pathogens in soil treated with picloram (Lai and Semeniuk 1970).

The application of great rates of the dinitroaniline herbicides (HRAC group K1) trifluralin and nitralin under both growth chamber as well as field conditions led to increased damage to cotton crop (Fig. 51). Damage increase was observed under both field conditions of the large rates of *R. solani* and large rates of nitralin, as well as of the large rates of trifluralin and small rates of the fungus (Chandler and Santelman 1968). Neubauer and Avizohar-Hershenson (1973) found that trifluralin increased susceptibility of cotton to *R. solani* and increased the saprophytic activity of the fungus in soil.

Trifluralin (a member of HRAC group K1) increased phytophthora root rot (Duncan and Paxton 1981) and fusarium foot rot (Carson et al. 1991) on soybean as well as on rhizoctonia root rot on sugar beet (Ruppel and Hecker 1982). Cowpea seedlings grown in alachlor-treated soil were more susceptible to *R. solani* than those treated with fluchloralin (a member of HRAC group K1) and the untreated

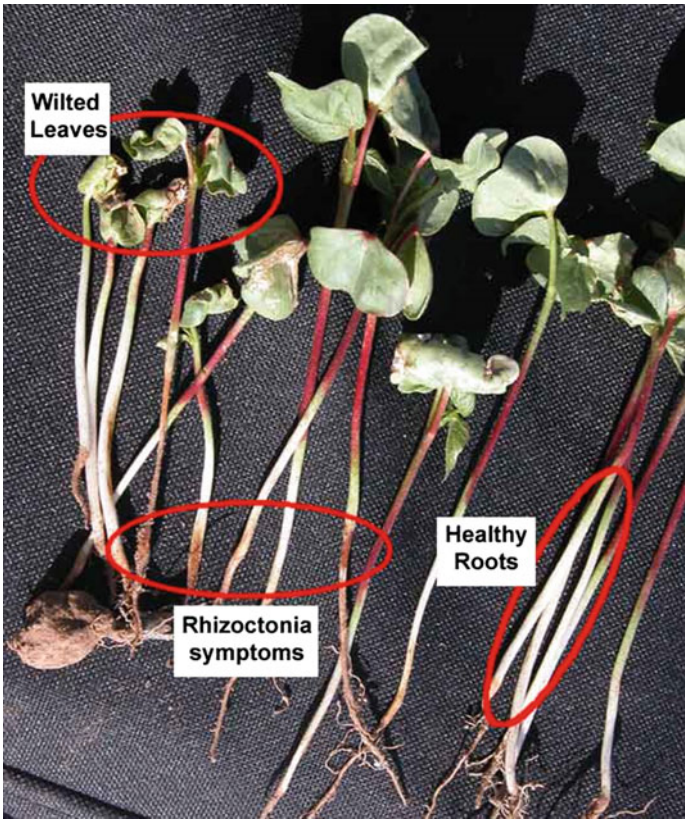


Fig. 51 Symptoms of cotton seedling infection by *Rhizoctonia solani*, root rot and wilting of leaves as the result of insufficient water uptake (*Source* http://cals.arizona.edu/)

seedlings. Pre-sowing application of alachlor (a member of HRAC group K3) in soil (5 μL a. i. kg^{-1}) aggravated damping-off whereas fluchloralin decreased the disease incidence to nearly half of that in untreated soil in greenhouse pot tests (average temperature of 31 ± 5 °C). Both herbicides reduced damping-off in pots kept at constant temperature of 30 °C and increased the disease incidence at 20 °C. Impact of fluchloralin and alachlor on *R. solani* damping-off of cowpea appears to be due to the predisposing effect by the herbicides on the susceptibility of the host and is influenced by atmospheric temperature (Kataria and Dodan 1983).

Herbicide-treated sugar beet hypocotyls leaked or exuded more substance into the soil than beet hypocotyls from beets grown in non-treated soil (Wheeler 1975). Further research indicated that greater quantities of glucose and certain mineral ions leaked out in all instances when plants were grown in soil treated with the herbicides such as cycloate (a thiocarbamate from HRAC group N) suggesting one reason for the increased disease that might be the increased availability of nutrients for the pathogens such as *Rhizoctonia solani* (Altman 1972; Altman and Campbell 1977b; Altman and Ross 1967). The herbicides EPTC (a thiocarbamate in HRAC group N) and linuron (an urea in HRAC group C2) applied to soil in the field at three different concentrations of the recommended dose reduced post-emergence, but not pre-emergence damping-off of cotton caused by *R. solani*. Both herbicides at two high concentrations considerably decreased wilt induced by *Fusarium oxysporum* f. sp. *vasinfectum*. Both herbicides reduced *F. oxysporum* f. sp. *vasinfectum* germination of chlamydospores under natural soil conditions but not in streamed soil. The 2-fold concentration of EPTC and linuron decreased *R. solani* saprophytic activity in soil. The impacts of both herbicides on both diseases were attributed to their suppressor effects on the saprophytic activity of *R. solani*, and the germinability of *Fusarium* chlamydospores in soil, respectively (El-Khadem and Papavizas 1984). The enhanced disease severity or incidence may be because of the herbicidal injury to the root system of the host plant that leads to the leakage and exudation of the nutrients put available for the soil-born pathogens. Such a phenomenon has been proved with the herbicide cycloate (a thiocabamate in HRAC group N; application rate less than 32 μg g^{-1} soil) and sugar beet damping-off pathogen, *R. solani*, where the herbicide enhances disease despite of its interference with the growth of the pathogen (Altman and Campbell 1977b).

Sharon et al. (1992) indicated glyphosate (a glycine from HRAC group G) suppression of an elicited response that led to the increased susceptibility of *Cassia obtusifolia* to a mycoherbicide. The synergistic activity of glyphosate weed control in predisposing plants to infectious organisms has been observed for many diseases, and the extensive use of glyphosate in agriculture is a significant factor in the increased severity or "re-emergence" of diseases once considered efficiently managed. Glyphosate has made crops susceptible to normally non-pathogenic isolates of *Fusarium*, and the population of *Fusarium* increases in soil after glyphosate application (Levesque et al. 1987; Kremer et al. 2000). Glyphosate predisposes tomato to fusarium crown and root rot by inhibiting the plant's structural and defense barriers (Bramhall and Higgins 1988). Cotton growers in Australia and the Western United States have seen a resurgence of fusarium wilt since the

introduction of Roundup Ready® cotton, and previously high levels of wilt resistance appear to be less effective under glyphosate management programs (Harper 2007). Glyphosate also breaks resistance to cyst nematodes in soybeans (Geisler et al. 2002). The increased fusarium yellows and *Rhizoctonia solani* diseases of Roundup Ready® sugar beets prompted Larson et al. (2006) to comment that "precautions need to be taken when certain soilborne diseases are present if weed management for sugar beet is to include post-emergence glyphosate treatments." These authors also reported that the sugar beet variety resistant to *Rhizoctonia* was as susceptible to this pathogen as the susceptible variety after glyphosate application regardless of the time of inoculation. Fusarium head scab of cereals and other diseases caused by *Fusarium* spp. increase following glyphosate applications (Fernandez et al. 2005; Larson et al. 2006), and previously established "cardinal" conditions (precipitation, flowering, and temperatures above 26°C) for head scab are modified when glyphosate is applied prior to a susceptible cereal crop (Fernandez et al. 2005, 2007). Glyphosate modifies plant nitrogen metabolism similar to high temperature-induced changes that provide susceptibility to head scab (Huber, unpublished) so that head scab and the mycotoxins produced by the causal fungi are now prevalent in cooler areas where they were rarely observed before the extensive use of glyphosate (Fernandez et al. 2005, 2007). Similar changes in nitrogen and carbohydrate metabolism provide transient resistance of wheat and soybeans to rust after glyphosate application (Anderson and Kolmer 2005; Feng et al. 2005, 2007). Plants rely on multiple components of defense to deter pathogens following infection (Hammond-Kosack and Jones 2000). Many of these active resistance components are derived from the phenylpropanoid pathway, which acquires almost all its precursors (notably phenylalanine and chorismate) from the shikimic acid pathway (Hammond-Kosack and Jones 2000; Dixon et al. 2002). A key inducible defense component associated with the shikimic acid pathway is the production of antimicrobial phytoalexins that accumulate rapidly at the site of infection. Lignification of cell walls at and around the infection site is another shikimate-derived component that functions to fortify cells and ensure isolation of the pathogen at the infection site. The production of salicylic acid (SA) following infection represents another component of inducible defense. SA is synthesized either directly from chorismic acid or indirectly through phenylalanine. Although SA is not antimicrobial per se, it functions to signal and coordinate various defenses following challenge by a pathogen; however, its direct role in plant–pathogen interactions involving root tissue remains unclear. Another defense component that relies on three final products of the shikimic acid pathway- tryptophan, tyrosine and phenylalanine- is the production of a diverse variety of pathogenesis-related (PR) proteins that function to curtail the advance of a pathogen. Many kinds of PR proteins have been identified (Hammond-Kosack and Jones 2000). Given the reliance of many plant defenses on the shikimic acid pathway, and the fact that glyphosate blocks this pathway, it is not surprising that this herbicide would render plants more susceptible to pathogens. Keen et al. (1982) were the first to show that by inhibiting the phytoalexin glyceollin, glyphosate was able to compromise resistance of soybeans to *Phytophthora sojae* (syn. *Phytophthora*

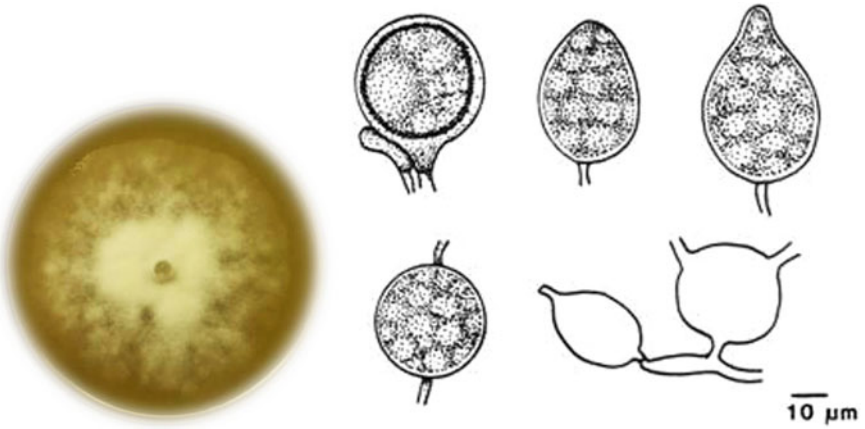


Fig. 52 The homothallic oomycetous fungus-like pathogen, *Phytophthora sajae* (Left) mycelial growth on V8 juice agar plate (*Source* Jean B Ristaino); (Right) Morphology of the oomycetous *P. sojae* (Upper row) globose and possibly subglobose oogonium with a mostly paragynous and possibly amphigenous antheridium; ovoid and obpyriform, noncaducous, nonpopillate sporangia; (Lower row) chlamydospores; empty remnants of intercalary spherical and irregular hyphal swellings formed in aqueous culture (*Source* Vaziri A, reproduced from Erwin and Ribeiro 1996)

megasperma f. sp. *glycinea*; Fig. 52). However, Ward (1984) considered the suppressive effect of glyphosate on the anti-oomycotic activity of metalaxyl as an evidence that host defence mechanisms contribute to metalaxyl inhibition of *P. sojae* to soybeans. Using the bean-anthracnose pathosystem, Johal and Rahe (1988, 1990) demonstrated that, while glyphosate did not interfere with the hypersensitive reaction (HR) of incompatible interactions, it suppressed significantly the production of all four of the bean phytoalexins. As a result, the pathogen was able to kill the plant if it escaped the localized HR, a situation that occurred only with glyphosate-treated plants.

The effect of glyphosate on the compatible bean anthracnose interaction was even more dramatic (Johal and Rahe 1990). Glyphosate almost completely suppressed the production of phytoalexins associated with susceptible lesion containment and permitted the pathogen to invade unimpededly until the entire hypocotyl collapsed. As little as 2% of the recommended herbicidal rate of glyphosate was enough to transform normally delimited lesions typical of anthracnose into constantly expanding lesions (Johal and Rahe 1990). The defense studies mentioned above were confined largely to diseases of aerial parts of host plants. There are indications that defense components may vary significantly in root tissue that are in intimate and continuous contact with potential pathogens (Hammond-Kosack and Jones 2000). For instance, roots do not rely on HR-mediated defense to contend with pathogens, although the exact defense components that keep roots pathogen-free are only partially understood. To gain an insight into what contributes to glyphosate-induced susceptibility of French beans (*Phaseolus vulgaris*) to *Pythium*, Liu et al. (1995, 1997) assessed phytoalexins as well as lignification of

root tissue in response to glyphosate treatment. By comparing phytoalexins in roots of bean seedlings grown in different media, they concluded that phytoalexins were induced by soil microorganisms. Interestingly, while phytoalexin accumulation was affected only modestly by glyphosate in response to *Pythium*, lignification (a process requiring Mn) was suppressed significantly. Thus, enhanced colonization by *Pythium* in roots of bean seedlings treated with foliar applied glyphosate occurs as a result of glyphosate interference with lignin-based defense mechanisms (Liu et al. 1997). However, these results also suggest that sustained production of phytoalexins in response to *Pythium* infection is maintained temporarily following glyphosate treatment, whereas lignification is not. 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 variousplant 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). Glyphosate is known to predispose many plants to pathogens due to its inhibition of the shikimic acid pathway (Holliday and Keen 1982), through which phytoalexins, the compounds produced by plants to defend against pathogens are produced. Mn plays an important role in plants' disease defense mechanisms (Thompson and Huber 2007). It has been proposed that glyphosate interferes with absorption and utilization of Mn, thus increasing a plant susceptibility to disease. However, the majority of research has not found reductions in Mn concentrations within plants following glyphosate applications (Bott et al. 2008; Rosolem et al. 2009; Nelson 2009). Glyphosate had no effect on charcoal rot on soybean but when plants were stressed by alachlor, chloramben or 2,4-DB and soil temperatures were greater than 26 °C, the disease increased (Canaday et al. 1986).

Effect of Herbicides Through Their Impact on Non-pathogenic Microorganisms

The effect of herbicides on soil beneficial microorganisms is of significant importance in maintainment of soil fertility, and suppressiveness against plant pathogens. Additionally, compatibility of herbicides with biological control agents is very relevant in integrated management of plant diseases and pests, as well as in development of new formulations that take advantage of compatible combination of biological as well as chemical agents. Microbial organic matter transformation is considered as a proven source of nutrient elements in nutritionally poor soils (Kang et al. 2012), and microorganisms operate as a sign of soil quality because of the key

roles they play in various soil functions (Schloter et al. 2003). Therefore, there has been long standing interest in the effects of herbicides on soil microflora (Greaves and Malkomes 1980; Simon-Sylvestre and Fournier 1979). Herbicides can affect microbial activity, soil microbiology, soil biochemistry, soil fertility, and plant disease incidence and severity. There are numerous records on the negative impacts of herbicides on soil microbiology and fertility: bromoxynil (Abbas et al. 2014; El-Ghamry et al. 2000; Pampulha and Oliveira 2006). The effect of an herbicide varies with the type and the concentration of herbicide as well as the type of soil, the experimental conditions (temperature, moisture, and the time of incubation), the method and time of herbicide application concentration, the type of herbicide formulation, the type and the rate of other materials applied in herbicide formulation. Additionally, different groups of microorganisms, and even different strains of a given species show different levels of sensitivities to an herbicide. Carfentazone-ethyl had a stimulating effect on total oligotrophic as well as organotrophic bacteria, but it inhibited the growth of *Azotobacter*, fungi, spore-forming oligotrophic bacteria and actinobacteria (Tomkiel et al. 2015). With sulfonylurea herbicides, most of the investigations carried out on the active ingredients tribenuron, primi-, tria-, and prosulfuron, metsulfuron, sulfometuron and thifensulfuron, chlorimeturon, chlorsulfuron and rimsulfuron, and nicosulfuron have indicated that the recommended field rate of sulfonylurea herbicides were generally of no significant impact on soil microbial number and activity (Allievi and Gigliotti 2001). Among the bacteria including *Bacillus*, *Pseudomonas* and *Arthrobacter*, *Bacillus* and *Arthrobacter* were primarily inhibited in the soil due to the herbicide Galex® application (Fawole 2000). *Acinetobacter calcoaceticus*, *Actinomyces bovis*, *Actinomyces viscous*, *Nocardia farcinica*, *Bacillus subtilis*, *B. licheniformis*, *B. megaterium*, *Flavobacterium aquantile*, *Pseudomonas putida*, *P. stutzeri*, *Serratia marcescens*, and *Alcaligenes eutrophs* were able to grow heavily in the soil polluted with atrazine and Primextra® (a combined formulation of atrazine and metolachlor). While the studied isolates of all the above-mentioned bacterial species were able to grow on the herbicides as the sole source of carbon, *B. subtilis* exhibited the highest growth (Sebiomo et al. 2011a). Similarly *Bacillus* spp. degrade 80–95.6% of the herbicide oxyfluorfen within 21 days, while *Pseudomonas* sp. (82.2%), *Arthrobacter* spp. (82.2%), *Aspergillus* sp. (77.8%), *Mycobacterium* sp. (75.6%), *Micrococcus* sp. (73.3%), and *Streptomyces* sp. (68.9%) were of relatively lower capabilities (Mohamed et al. 2011). The growth of *Bacillus* sp. was inhibited by diquat (25 μg mL^{-1}), paraquat (5 μg mL^{-1}), ioxynil (10 μg mL^{-1}), and pentachlorophenol (PCP, 5 μg mL^{-1}), but was not affected by the tested concentrations (25, and 50 μg mL^{-1}) of the herbicides atrazine, diuron, trifluralin, and fluometuron (Breazeale and Camper 1972). The application of the herbicides EPTC, and linuron in field soil at three doses i.e. 1/2 field recommended dose (FR), 1 FR, and 2 FR resulted in the significant reduction of the vascular wilt caused by *Fusarium oxysporum* f. sp. *vasinfectum* and of the germination rate of chlamydospores of *Fusarium* spp. in natural soil but not in the steamed soil, implying to the role of soil microflora in the disease control via

the herbicidal treatments performed with two higher doses (El-Khadem and Papavizas 1984).

Glyphosate, N-(phosphonomethyl) glycine, is the most extensively used herbicide in the history of agriculture. Glyphosate is similar to most herbicides in that when it enters the soil it differentially affects soil microorganisms (Kremer et al. 2005). Ratcliff et al. (2006) applied the herbicide glyphosate at the recommended field rate to a clay loam as well as a sandy loam forest soil that resulted in few changes in microbial community structure. Total and culturable bacteria, fungal hyphal length, bacterial: fungal biomass, carbon utilization profiles (BIOLOG), and bacterial and fungal phospholipid fatty acids (PLFA) were unaffected 1, 3, 7, or 30 days after application of a commercial formulation (Roundup®). In contrast, a high concentration of glyphosate (field rate) simulating an undiluted chemical spill substantially altered the bacterial community in both soils. Increases in total bacteria, culturable bacteria, and bacterial: fungal biomass were rapid following application. Culturable bacteria increased from about 1% of the total population in untreated soil to as much as 25% at the high concentration by day 7, indicating enrichment of generalist bacteria. Community composition in both soils shifted from fungal dominance to an equal ratio of bacteria to fungi. Functional diversity of culturable bacteria, estimated by C substrate utilization, also increased at the high glyphosate concentration, particularly in the clay loam soil. Unlike the other bacterial indices, only minor changes in bacterial PLFA resulted after the third day following the field rate application. Apparently the herbicide resulted in an across-the-board stimulation of bacteria that was not reflected by the finer-scale PLFA community structure. Changes in fungal properties (hyphae, propagules, PLFA biomarkers) were few and transient. They concluded that the commercial formulation of glyphosate had a benign affect on community structure when applied at the recommended field rate, and produces a non-specific, short-term stimulation of bacteria at a high concentration.

Using next-generation sequencing technology, it was known that glyphosate (as Roundup®) application in a soil with a high clay content (41%; favorable for sorption of the herbicide because of high surface area) and pH value of 6.9 (less favorable for sorption of glyphosate) led to no considerable change in soil diversity of bacterial populations. However, glyphosate application resulted in an increase in relative abundance of proteobacteria (in particular, the gammaproteobacterial members of Xanthomonadalean families, Xanthomonadaceae, and Sinobacteriaceae) in soybean and especially in corn cropping systems, whilst it reduced the relative abundance of acidobacteria. Further studies are required in order to get precise information on the effect of glyphosate and its metabolites, most notably, aminomethylphosphonic acid (AMPA) on soil bacteria at finer taxonomic levels. It is believed that acidobacteria are highly involved in the biogeochemical processes such as cellulose biodegradation. Decreases in the frequency of acidobacteria over long-term can lead to the impaired ability of soil to perform certain biogeochemical reactions carried out by acidobacteria, and can influence rhizosphere nutrient status. Such adverse effects in the glyphosate-tolerant (GT) corn cropping systems may be more exacerbated than in a corn-soybean rotation

(Newman et al. 2016). Further studies have shown species-dependent as well strain-dependent growth responses of *Pseudomonas* species grown on succinate (a common root exudate) and treated with different concentrations (up to 5 mM) of glyphosate. While the growth rate of both *P. protegens* Pf-5 and P. fluorescens RA12 remained unchanged, both of the tested *P. putida* strains (KT2440, and S12) exhibited growth inhibition from 0 to 100%. Monitoring disruptions in metabolic homeostasis and fluxes via application of a 13C-assisted metabolomics approach, and profiling of the whole-cell metabolome captured deviations in metabolite levels involved in the tricarboxylic acid cycle, ribonucleotide biosynthesis, and protein biosynthesis. Altered metabolite levels specifically in the biosynthetic pathwat of aromatic amino acids, the target of toxicity for glyphosate in plants, implied the same toxicity target in the soil bacterium. Kinetic flux experiments with 13C-labeled succinate revealed that biosynthetic fluxes of the aromatic amino acids were not inhibited in *P. fluorescens* Pf-5 in the presence of low and high glyphosate doses but these fluxes were inhibited by up to 60% in *P. putida* KT2440, even at sublethal glyphosate exposure. Notably, the greatest inhibition was found for the aromatic amino acid tryptophan, an important precursor to secondary metabolites. When the growth medium was supplemented with aromatic amino acids, *P. putida* S12 exposed to a lethal dose of glyphosate completely recovered in terms of both growth rate and selected metabolite levels. Collectively, the glyphosate-induced specific disruption of de novo biosynthesis of aromatic amino acids accompanied with widespread metabolic disruptions was responsible for dose-dependent adverse effects of glyphosate on sensitive soil *Pseudomonas* species (Aristilde et al. 2017).

Weed management programs in glyphosate-resiatant (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. Glyphosate applications to glyphosate-resistant (GR) crops alter the type and quantity of compounds released from crops roots into the rhizosphere, including the exudation of glyphosate. These changes in exudates can have a dramatic impact on the microbes found in the root zone. Manganese is absorbed by plants in the reduced state (Mn^{2+}). High soil pH limits Mn availability due to oxidation to the Mn^{4+} state under alkaline conditions. While the specific physiological mechanisms are poorly understood, many plants are able to absorb Mn from soils with limited Mn availability. This is accomplished either via associations between the plant and Mn-reducing bacteria, or alteration of the pH of the rhizosphere via root exudates (Rengel and Marschner 2005). Indirect effects of glyphosate on disease predisposition result from immobilization of specific micro-nutrients 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. Furthermore, even low levels of residual glyphosate in soil can decrease root uptake and translocation of iron (Fe), manganese (Mn), and copper (Cu) (Eker et al. 2006). Thompson and Huber (2007) reported that glyphosate application to GR soybean altered the balance of Mn reducing and oxidizing bacteria associated with soybean roots in a manner that suggested that Mn would be immobilized in the soil. However, published data documenting reduced soil availability of Mn due to the activity of glyphosate on soil microorganisms is lacking. Furthermore, there have been no documented reports of Mn crop deficiency symptoms in Iowa.

Manganese (Mn) deficiency symptoms occur in some regions of the cornbelt, and these areas are where interactions between glyphosate and Mn nutrition have been reported. Microorganisms proposed for biological control of this disease such as *Bacillus cereus* and *Trichoderma konigii* are all strong Mn reducers that increase Mn availability in the rhizosphere (Huber and McCay-Buis 1993; McCay-Buis 1998; Rengel et al. 1996). In contrast, the addition of Mn-oxidizing organisms increases take-all (Crowley and Rengel 1999; McCay-Buis 1998; Rengel 1999; Thompson et al. 1998; Fig. 53).

The virulence mechanism of some pathogens such as *Gaeumannomyces*, *Magnaporthe*, *Phymatotrichum*, *Corynespora*, and *Streptomyces* involves Mn oxidation at the infection site to compromise plant 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

Fig. 53 More severe take-all root rot of wheat grown following Roundup Ready® soybeans sprayed with glyphosate (left) than following Roundup Ready® soybeans grown with a non-glyphosate herbicide (right) (*Source* Johal and Huber 2009)

avirulent and disable 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). 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 the disease. 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). Similarly, various diseases caused by *Fusarium* spp. are increased by glyphosate (Fernandez et al. 2005; Sanogo et al. 2000, 2001). Here, the impact of herbicides on the microbial population of importance in agricultural soil fertility and plant pathology is reviewed.

Effect of Herbicides on Nitrogen Cycle

Herbicides affect nitrogen cycle through their impacts on different groups of microorganisms involved in nitrogen transformation in nature:

Effect of Herbicides on Symbiotic and Nitrogen-Fixing Rhizobium Species

Effect of herbicides on rhizobial nitrogen fixation depends on the variety of legume crop, soil type, environmental conditions, the strain of a rhizobial species, the herbicide, its dose and its application method and time. The effect of herbicides on rhizobial nitrogen fixation can be considered from two viewpoints, their effects on the process of legume host rhizobial nodulation, and on the process of nitrogen-fixation in the developed nodules (Singh 2005; Fig. 54). Herbicides can exert their adverse effects on legume plants and thereby prevent rhizobial nodulation of their roots. For example, terbutryn/terbutylazine, trietazine/simazine and prometryn negatively affect rhizobial nodulation of legume plants due to their adverse effects on plants, and not on the rhizobial bacterium *Rhizobium leguminosarum*, as these herbicides were of negative impact on the growth of *R. leguminosarum* only when they were tested at the concentration unexpected to occur under normal field conditions, and bentazone was safe to rhizobia even under these conditions (Singh and Wright 2002). Here, the effect of herbicide on rhizobial bacteria is the subject of our focus. Out from three rhizobial species, *Rhizobium* sp. IC 3342 (a pigeonpea nodulator), *R. leguminosarum* 2001 (nodulating lentil), and *R. meliloti* 4013 (nodulating alfalfa), the former was found sensitive to all the herbicides tested, butachlor, simazine, and oxyfluorfen. The herbicides were of more adverse effects compared to tested insecticides and fungicides. Also, the plants ifested with herbicide treated *Rhizobium* were of less dry weight and less total nitrogen content because of the decreased growth and nitrogen fixing capacity

Fig. 54 Nodules induced by rhizobial bacteria in legume roots (*Source* Jeremy Kemp, San José State University, Wikimedia.org)

of the rhizobial bacteria (Madhavi et al. 1994). Different strains of *R. leguminosarum*, *R. meliloti*, and *R. loti* were able to reproduce when they were exposed to the equivalent to bentazon (a benzothiadiazinone from HRAC group C3; 1–3 kg ha^{-1}) and glyphosate (1–3 kg ha^{-1}), and MCPA (2-methyl-4-chlorophenoxyacetic acid, 0.5–3.5 kg ha^{-1}) and bacterial exposure to the herbicides exerted no imact on the nodulating capacity of the bacteria except for glyphosate (Mårtensson 1992). Bacterial growth in aseptic culture was not influenced by the amendment of 0.55 and 5.50 μM chlorsulfuron (Mårtensson and Nilsson 1989) and it was concluded that the inhibition of nodulation and nitrogenase activity of alfalfa as well as red clover nodules probably occurred as the result of the adverse impacts of the herbicides on the plant growth and development rather than on the rhizobia. The significantly inhibiting effect of glyphosate (10 mg a. i. L^{-1}), paraquat (2 mg a. i. L^{-1}), and chlorsulfuron (2 mg a. i. L^{-1}) on the growth of *R. trifolii* in nutrient broth is recorded (Eberbach and Douglas 1989).

The application or residues of acetolactate synthase inhibitor (ALS) herbicides can reduce the growth and nitrogen-fixing ability of legumes (Anderson 2001), and further studies indicate that common ALS herbicides such as Spinnaker® (imazethapyr as ammonium), Broadstrike™ (flumetsulfam), and Glean® (chlorsulfuron) can reduce the amount of biologically fixed nitrogen in chickpeas by up to 70%, leading to evidently slowed growth and reduced grain yields in alkaline soils. Reduced productivity has also been observed for pea, medic, subclover, and Lucerne. Apparently, the herbicides can interfere with rhizobial nodule formation process through their influence on *Rhizobium* bacteria, rather than the growth of the bacteria, or legume plant (Anderson 2001). Imazatapyr was rather non-toxic to *Rhizobium* growth and higher doses more than 0.34 mM were required to cause only slight effects on the bacterium growth in a defined medium. Additionally, imazethapyr did not affected the nodulation ability of *Rhizobium* on pea roots. Therefore, it was concluded that the herbicide would not inhibit rhizobial growth under normal field conditions (Gonzalez et al. 1996). It was proposed that the high acetolactate synthase (ALS) activity expressed by *Rhizobium*, both as free-living

bacteria and as bacterioids, was related to the growth tolerance of rhizobia to imazethapyr and it was related to the relative tolerance of symbiotic pea plants (Royuela et al. 1998). Herbicides can affect *Rhizobium*-legume symbiosis in four ways (Kalia and Gosal 2011), of which three ways are related to their effects on *Rhizobium* symbionts: (i) herbicides can negatively affect the growth and survival of the rhizobia, and reduce their nodulation capabilities (Anderson et al. 2004), (ii) herbicides can reduce the efficacy of rhizobia in terms of their ability to nodulate or form an effective symbiosis (Anderson et al. 2004), and (iii) herbicides can reduce the nitrogen-fixing effectiveness of the symbiosis through inhibition or interruption of biochemical pathways in the bacterioids (Drew et al. 2007). Sawicka and Selwet (1998) indicated that seed treatment with imazethapyr (an imidazolinone from HRAC group B) could lead to the reduced nitrogenase activity of root-nodule rhizobia and stimulate the development of resistant bacteria. Also, they reported that the effect depended on the herbicide, its concentration, as well as on the weather conditions. Accordingly, a reduction in nitrogenase activity of the active strain KGL of *R. leguminosarum* biovar *trifolii* was reported in red clovers as the result of Pivot® 100 SL (imazethapyr) application under both pot and field conditions. The herbicide inhibited the proliferation of the microorganisms in the soil under red clover plantations in the first days after treatment, but later stimulated their multiplication (Niewiadomska 2004).

Yueh and Hensley (1993) reported that trifluralin though not significantly effective on nitrogen fixation of soybean and limabean, adversely influenced nodulation. Trifluralin was not toxic to *Bradyrhizobium* and *Rhizobium* species in a study carried out using disc inhibition method (Durgesha 1994). The inhibition of nodulation as the result of the application of trifluralin (a dinitroaniline from HRAC group K1) and metribuzin (a triazinone from HRAC group C1) has been recorded (Bertholet and Clark 1985). The triazine herbicides of the HRAC group C1 (photosystem II inhibitors), terbutryn, propyzamide, terbutryn + propyzamide, and metribuzin were not of any significant effect on the rhizobial nodulation of pea plants, however, carbetamide significantly increased the number of nodules per plant. Hand weeding with no use of the herbicides also significantly raised rhizobial nodulation rate (Zaid et al. 2014). Thiobencarb (a thiocarbamate from HRAC group N), when applied at the rates of 2 and 4 mg kg^{-1} soil, inhibited *Azospirillum* populations, anaerobic nitrogen fixers, and *Azotobacter* in an alluvial soil (Jena et al. 1990).

Investigation of the in vitro effect of nine glyphosate commercial formulations (Zapp QI®, Roundup®, Roundup Multiação®, Roundup Transorb®, Roundup® WG, Trop®, and Agrisato®) on three *Bradyrhizobium* strains (*B. japonicum* SEMIA 5079, and two strains of *B. elkanii*, SEMIA 5019, and SEMIA 587) revealed the different toxicity of the formulations and different vulnerability of the strains. Zapp Qi® was the least toxic formulation to the strains, while Roundup Transorb® was of the highest toxicity and reduced growth over 94% for all the strains tested. No correlation was found among the type of salt, isopropylamine, ammonium or potassic present in the formulations, and the toxicity degree to the strains. *B. elkanii* strain SMIA 587 was known as the least tolerant to most formulation while the

strain SEMIA 5019 was the most sensitive to the control treatment N-(phosphonomethyl) glycine, without salts or other additives (Santos et al. 2004). Reduction in growth of the strains in the lowest tested glyphosate concentration (5.4 μg L^{-1}) was 18% for SEMIA 5079, 29% for SEMIA 5019, and 35% for SEMIA 587. In general, the higher the concentration of the herbicide in the culture medium, the greater the growth inhibition. The strains depicted differential sensitivity only at the lowest concentration of glyphosate, and all exhibited undifferentially severe growth reduction in the presence of the highest concentration of the herbicide (43.2 μg L^{-1}) (Jacques et al. 2010).

Effect of Herbicides on Free-Living Nitrogen-Fixing Bacteria

There are a number of free-living bacterial species from different genera that are able to fix the atmospheric nitrogen (Lal 2006) out of which, the species from two genera *Azospirillum* and *Azotobacter* have been regarded as potential nitrogen fertilizers (Martín et al. 1993) and tested as plant growth promoting bacteria (Baltensperger et al. 1978; Tejera et al. 2005; Yasari et al. 2008; Mirzae et al. 2010; Naderifar and Daneshian 2012).

Effect of Herbicides on Azotobacter Species

Azotobacter is a genus of usually motile, oval or spherical bacteria that form thick-walled cysts and may produce large quantities of capsular slime. The bacteria in the genus are aerobic, free-living soil microbes that play an important role in atmospheric nitrogen (a form of nitrogen inaccessible to plants) fixation and its release as ammonia into soil (Kizilkaya 2009). The common characteristics of the species in the genus *Azotobacter* are presented in Table 1.

Table 1 Biochemical characteristics of *Azotobacter* species

Biochemical tests	Azotobacter species
Gram-staining	Positive
Motility	Motile
Catalase test	+
Nitrate reduction	+
Pigment production	+
H_2S production	+
Urease test	+
Citrate test	+
Utilization of carbon source	+
Glucose	+
Fructose	+
Maltose	–
Indole test	+

Source Kasa et al. (2017)

The significance of the bacterium contribution to nitrogen fixation was understood knowing that atmospheric nitrogen fixation by the bacterium occurs much more efficiently in the rhizosphere, and the bacterium exists in larger numbers in the rhizosphere than in "open" non-rhizospheric soil (Abd-El-Malik 1971). Beside nitrogen fixation, *Azotobacter* spp. also synthesize some biologically active substances including some phytohormones (auxins, cytokinins, and gibberellin-like substances) (Azcorn and Barea 1975) and stimulate plant growth (Jnawali et al. 2015). Additionally, the bacteria facilitate the mobility of heavy metals in the soil, and help in the bioremediation of soil from heavy metals such as cadmium, mercury, and lead. *Azotobacter* spp. produce siderophores that bind to the available form of iron (Fe^{3+}) in the rhizosphere, and make it unavailable to plant pathogens and protect the plant health. Also, most of *Azotobacter* spp. produce and secret antimicrobial compounds such as hydro cyanine (HCN, Althaf and Srinivas 2013), and an antifungal antibiotic (Jnawali et al. 2015). *Azotobacter* in sufficient numbers will outcompete pathogens for food. Some of the pathogens controlled by *Azotobacter* spp. in the soil and on the leaf include: *Alternaria*, *Fusarium*, *Colletotrichum*, *Rhizoctonia*, *Macrophomina*, *Diplodia*, *Botryodiplodia*, *Cephalosporium*, *Curvularia*, *Helminthosporium*, and *Aspergillus* (Jnawali et al. 2015). *Azotobacter* spp. can also biodegrade chlorine-containing aromatic compounds such as 2,4,6-trichlorophenol (Li et al. 1991), a mutagenic and carcinogenic compound previously applied as an insecticide, fungicide, and herbicide. However, the bacteria in the genus *Azotobacter* are extremely sensitive to soil contamination and they are considered as the effective indicators of the pollution with crop protection products (Milošević and Govedarica 2002). It is, therefore, a reliable indicator of the biological value of soil. The numbers of this group of nitrogen-fixing bacteria decrease considerably in the period of 7–14 days after herbicide application (Milošević and Govedarica 2002). Some herbicides such as carfentrazone-ethyl (the most inhibitory effect at only 42.240 μg kg^{-1} soil; Tomkiel et al. 2015), a mixture of diflufenican + mesosulfuron methyl + iodosulfuron-methyl-sodium (4.560 mg kg^{-1} soil; Baćmaga et al. 2015), Successor T (pethoxamid + terbuthylazine; Wyszkowska et al. 2016), sulfometuron (0.41 mM; Burnet and Hodgson 1991), chlorsulfuron (0.56 mM; Burnet and Hodgson 1991) inhibit the growth of *Azotobacter*. The herbicides Agronex 50SC, Agroxone, and 2,4-Damine were toxic to *Azotobacter vinelandii*, and the latter was of the most toxicity. Also, there was a reduction in LC_{50} of herbicides with increased number of days, and the percentage survival decreased with increased concentration of herbicides and days (Adeleye et al. 2004). Dalapon and 2,4,5-T were neither effective on growth of *A. vinelandii*, nor on its nitrogenase activity in pure culture (Mackenzie and Macrae 1972). The reaction of Azotobacter towards different herbicides was highly variable. Some herbicides such as metribuzin 70% WP (applied at the rate of 300 g ha^{-1}) are significantly toxic to *Azotobacter*. Similar results on the toxicity of metribuzin to *Azotobacter* were also reported (Radivojevic et al. 2003). Certain herbicides such as clodinafop propargyl 15% WP (applied at the rate of 400 g ha^{-1}; Hussain et al. 2014), and quizalofop (Das et al. 2012) induce its growth as if they are favourably exploited by the bacterium. 2,4-D (Balajee and Mahadevan 1990; Musarrat et al.

2000), p-chlorophenoxy-acetic acid (Balajee and Mahadevan 1990), p-chlorophenol (Balajee and Mahadevan 1990), and simazine (Martinez-Toledo et al. 1991) were biodegraded by *Azotobacter chroococcum* as carbon sources, which ultimately stimulated nitrogenase enzyme. Other herbicides such as isoproturon 75% WP (applied at the rate of 1333 g ha^{-1}) are neutral and do not influence it at all (Hussain et al. 2014). Similarly, linuron did not inhibit the growth of any of fourteen strains of *A. chroococcum* (Lenart 2012), and pendimethalin (Stomp 330 EC, applied at the rate of 400 mL da^{-1}) as well as s-metolachlor (Dual Gold 960 EC, applied at the rate of 150 mL da^{-1}) were of no negative impact on the development of *A. chroococcum* (Kalinova et al. 2014). Furthermore, the sulfonylurea herbicides such as metsulfuron-methyl (He et al. 2006), mesosulfuron-methyl and iodosulfuron methyl sodium (Atlantis® 3.6% WG, 400 g ha^{-1}; Hussain et al. 2014), and Sulfosulfuron® 75% WG (applied at the rate of 33.33 g ha^{-1}; Hussain et al. 2014) initially enhance its growth, and then depress it. With UPH-110 54% WG (clodinafop propargyl and metribuzin) applied at the rates of 400, 500, 600, and 1000 g ha^{-1}, at all the doses barring the lowest dose, a fall in the population of *Azotobacter* was observed at varying time intervals. With UPH-110, the magnitude of toxicity and duration of hazard increased as with the increase of the dose. The lower concentration of UPH-110 might have been metabolized rapidly leading to no toxic effect, while higher concentration might have persisted for a longer period and thereby led to the inhibition of *Azotobacter* population (Hussain et al. 2014). A similar behavior has been reported with bentazon that was not detected in soil after a few months when applied at 10 ppm concentration, whereas higher amounts persisted for several months (Drescher and Otto 1973; Marsh et al. 1978; Gaynor and Hamill 1983). With UPH-110 (500 g ha^{-1}), the *Azotobacter* count was well short of control at 3rd and 7th day of incubation. The inhibitory impact of the higher doses of UPH-110 (600, and 1000 g ha^{-1}) was observable up to one month after which it neutralized and paralleled with control (Hussain et al. 2014). Glyphosate not only inhibits nitrogen fixation process in *A. chroococcum* but also reduces the bacterium respiration rate by 40–60% and thereby precludes its positive effects (Chennappa et al. 2014). Cinosulfuron, a sulfonylurea herbicide (applied at recommended field rate of 42 μg kg^{-1} of soil) influenced acetolactate synthase (ALS) activity and inhibits the biosynthesis of three branched chain amino acids, however, its effect was limited even at the doses higher than that recommended for field applications. Also, there were apparent differences between the sensitivity of *A. chroococcum* strains (Allievi and Gigliotti 2001).

Effect of Herbicides on Azospirillum Species

Azospirillum species are found in much larger numbers in the rhizosphere of some cereals and grass seed crops than in the soil itself (Döbereiner and Pedrosa 1987; Cárdenas et al. 2010). Newer findings have indicated the possibility of its adaptation in the soils of the highland (Aguirre-Cadena et al. 2014). Furthermore, the beneficial impact of *Azospirillum* spp. is not restricted to monocotyledonous crops,

and they seem as universally well-distributed general root colonizers and not the plant-specific bacteria restricted to poaceous crops and grasses. In contrast to *Azotobacter* spp., these bacteria are not so sensitive to environmental pollutions (Bashan and Holguin 1997). *Azospirillum* spp. enhance the growth of various plants including sorghum, sugarcane, triticale, barley, wheat, corn (in a genotype-dependent manner), rice, sunflower, carrot, mustard (in a genotype-dependent manner), tomato, eggplant, cotton, pepper, chick-pea, and oak seedlings (Bashan and Holguin 1997; Bashan et al. 1989; Del Gallo and Fabbri 1990; Pedraza et al. 2009; Saha et al. 1985). *Azospirillum* spp. are of other interestingly positive characters (Bashan and Holguin 1997). Seed application of cobalt and molybdenum together with seed inoculation with *A. brasilense* promoted the highest leaf nitrogen content, hundred-grain weight, yield, and profitability with soybean crop (Galindo et al. 2017). Several studies have marked the ability of *Azospirillum* spp. to stimulate plant growth, yield, and nitrogen content (Díaz-Zorita and Fernández-Canigia 2009), dinitrogen fixation, radical proliferation, and hormone development activities (Dart 1986; Bashan and de-Bashan 2010). Inoculation of corn seed with *A. brasilense* reduced iron concentration in leaf and increased leaf chlorophyll index (LCI), and leaf concentration of phosphorus, agronomic efficiency, and grain yield. Therefore, the use of *A. brasilense* was found viable even when high rates of nitrogen were applied (Galindo et al. 2016). Similarly, plant growth promotion was observed with potato seed tubers inoculated with an *Azospirillum* sp. studied beside other plant growth promoting bacteria (Naqqash et al. 2016; Fig. 55).

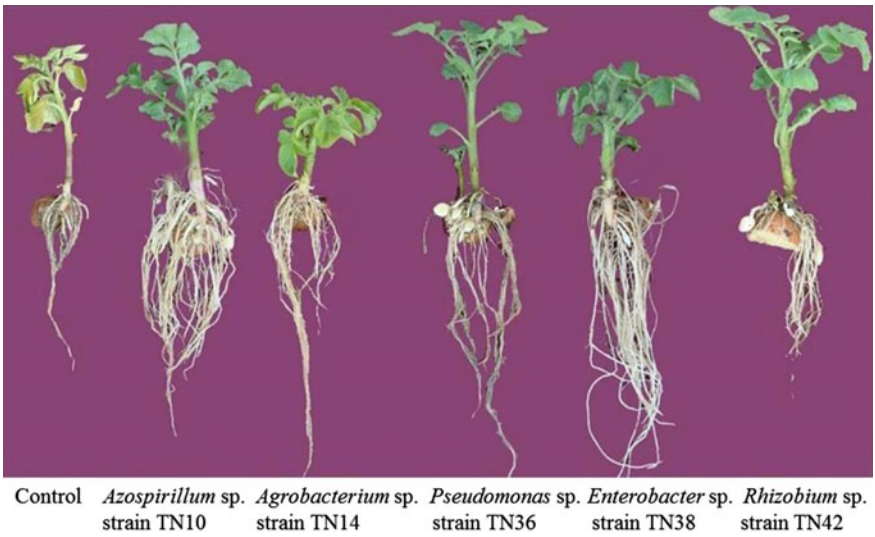


Fig. 55 Effect of bacterial inoculation of seed tubers on the health and root system of potato (variety Kuroda) plants. Plants were harvested 60 days after sowing (Naqqash et al. 2016)

A number of factors such as plant type and age, soil type, composition of microbial community, agricultural practice, root constituents and chemical compounds applied to the soil and plants, may influence the ecology and dinitrogen fixation of *Azospirillum* (Balandreau 1986). Herbicides may influence the growth and activity of *Azospirillum* in vitro, and *in soilum*. For instance, in an in vitro assay, all tested herbicides led to an initial decrease in the growth of *A. lipoferum* (after 24 h) followed by a recovery in the later stages of incubation. The negative effect of butachlor on the growth of the *A. lipoferum* in N-free malic acid broth (5.128 log CFU mL^{-1}) was recorded compared with 2,4-diphenoxy-2-ethylhexyl ester (2,4-DEE; 5.188 log CFU mL^{-1}), pretilachlor (5.226 log CFU mL^{-1}), and pyrazosulfuron ethyl (5.258 log CFU mL^{-1}). Among the different concentrations, the maximum inhibition of growth was recorded at a dose 100 fold of the recommended dose for field application (100 FR; 5.059 log CFU mL^{-1}), while the dose of 1 FR caused the least inhibition (5.244 log CFU mL^{-1}). 2,4-DEE, butachlor, pretilachlor, and pyrazosulfuron ethyl were found to reduce the growth and nitrogenase activity of *A. lipoferum* when the herbicides were applied at the concentrations of 0.375, 0.50, 0.15, and 0.125 μg mL^{-1} (Latha and Gopal 2010). It seems that no of the tested herbicides including butachlor can exert a hazardous impact on *A. lipoferum* populations *in soilum*, where herbicides are biodegraded by some members of soil microflora, or they lose their bioactivity being sequestered by organic and inorganic components *in soilum*. With *A. brasilense*, the reduction of dinitrogen fixation and adenosine triphosphate (ATP) content of the bacterium has been reported in a chemically defined medium as well as in a dialyzed soil medium as the result of their exposure to either of the herbicides, alachlor or metolachlor. However, the adverse effects due to metolachlor disappeared after 48 h indicating the tolerance of high concentrations of metolachlor by *A. brasilense* (Salmeron et al. 1991). The sulfonylurea herbicides, chlorosulfuron and rimsulfuron inhibited the growth of *Azospirillum*, and the significantly enhanced toxicity of the commercial formulations of rimsulfuron was attributed to the surfactants applied (Forlani et al. 1995). Another in vitro study indicated the inability of 2,4-diphenoxyacetic acid (2,4-D) to affect the growth and nitrogenase activity of *A. brasilense* when the herbicide was amended at 100, 200, and 300 μg mL^{-1} (Martinez-Toledo et al. 1990). However, 2,4-D influenced growth and protein, DNA and RNA synthesis of *A. brasilense*. At a concentration of 1 mM, 2,4-D inhibited bacterial cell growth tat was reversed via transferring the bacteria to a control 2,4-D-free medium or to a 2,4-D treated medium supplemented with polyamines. The inhibitory effect of 2,4-D on in vitro protein synthesis was also reversed by the addition of polyamines to the 2,4-D treated medium (Rivarola et al. 1992). The influence of the herbicides metamitron, metribuzin, ethiozin, and paraquat on the growth and nitrogenase activity of *A. lipoferum* and *A. brasilense* indicated that metamitron (35, and 70 μg mL^{-1}) and ethiozin (20 μg mL^{-1}) were not of any inhibitory effect on nitrogenase activity of the tested bacteria, while metribuzin (7, and 14 μg mL^{-1}) and ethiozin (50 μg mL^{-1}) caused a marked decrease in the enzyme activity (Gadkari and Klingmuller 1988). In vitro screening of the field recommended doses of the herbicides applied on sugarcane for those

that do not affect neither growth nor the biological nitrogen fixation (BNF) process of the diazotrophic bacterium *A. brasilense* indicated that out of eighteen herbicides including paraquat (8.49 μg L^{-1}), ametryn (56.60 μg L^{-1}), amicarbazone (19.81 μg L^{-1}), diuron (45.28 μg L^{-1}), metribuzin (27.17 μg L^{-1}), hexazinone + diuron (5.60 + 19.87 μg L^{-1}), clomazone (15.57 μg L^{-1}), hexazinone + clomazone (3.54 + 14.15 μg L^{-1}), isoxaflutole (3.71 μg L^{-1}), sulfentrazone (11.32 μg L^{-1}), oxyfluorfen (16.98 μg L^{-1}), imazapic (3.47 μg L^{-1}), imazapyr (7.08 μg L^{-1}), trifloxysulfuron-sodium + ametryn (0.52 + 20.70 μg L^{-1}), S-metolachlor (27.17 μg L^{-1}), glyphosate (25.47 μg L^{-1}), MSMA (40.75 μg L^{-1}), and 2,4-D (14.22 μg L^{-1}), some herbicides could reduce the bacterial growth in liquid DIG medium. This effect was found with the herbicides paraquat, oxyfluorfen, trifloxysulfuron-sodium + ametryn, and glyphosate. With (trifloxysulfuron-sodium + ametryn), the effect was associated with increases in both the length of lag phase as well as the generation time, whereas with glyphosate, it was only associated with the increased generation time. MSMA, amicarbazone, and specially paraquat reduced the BNF of *A. brasilense*. Other herbicides were effective on neither growth nor the BNF of *A. brasilense* (Procópio et al. 2011). In a similar study, nitrogenase activity of *A. lipoferum* was assayed in the presence of glyphosate, 2,4-D, mecoprop + dichlorprop, and the commercial products Roundup® (glyphosate), Nurmikko-Hedonal® (2,4-D), Mepro® (mecoprop), and Dipro® (MCPA) tested at different doses of 0, 5, 25, and 100 mg L^{-1}. In average field soils, where d = 1 kg dm^{-3}, the highest recommended dose of herbicides, affecting the 5 cm-thick top layer is 5 mg L^{-1}. Apart from Mepro® and mecoprop, no phenoxy acid herbicide inhibited the nitrogenase activity of *A. lipoferum*. Glyphosate and Roundup® were of no impact on the growth of *A. lipoferum*, but stimulated its nitrogenase activity (Haahtela et al. 1988).

Studies performed under greenhouse conditions indicated the occurrence of a significantly positive synergistic effect on the growth of a local cultivar of maize, Galal, when the potted soil inoculated with *A. lipoferum* strain ATCC29145 (2 mL of a bacterial suspension with the concentration of 3.2×10^8 cells mL^{-1}) was treated with one of the herbicides bromoxynil (0.42 L ha^{-1}), or particularly afalon S (a mixture of linuron and monolinuron; 0.42 kg ha^{-1}). The incorporation of the recommended field dose of either bromoxynil or afalon S into the soil was seemingly of no significant effect on nitrogenase and dehydrogenase activities (Fayez et al. 1983). In another study under greenhouse conditions, the seed of wheat cultivar, Altiplano were inoculated with *A. brasilense* before planting, and the herbicides were applied 40 days after planting. The treatments including (i) uninoculated plants (absolute control), (ii) inoculated with *A. brasilense* (inoculated control), (iii) inoculated with *A. brasilense* + application of an organochlorine herbicide, 2,4-D, (iv) inoculated with *A. brasilense* + application of an organophosphate herbicide, glyphosate, (v) treatment with 2,4-D without any inoculation, and (vi) treatment with glyphosate without any inoculation were applied in a completely randomized design with ten replicates. The survival of the bacteria in the root system was recorded via the use of the most probable number technique, recording plant height and weight. Two herbicides were of no significant

influence on the population of *A. brasilense*. Wheat plants inoculated with *A. brasilense*, with or without herbicide treatment indicated increased biomass compared with the non-inoculated plants. The application of organochlorine herbicide, 2,4-D (250 g ha^{-1}) resulted in the increased biomass production of the wheat cultivar planted in an unsterilized sandy (Bouyoucus) soil mixed with organic soil waste and filled into 1 kg pots with perforated bases to promote drainage. The pH of the mixture was 7.5 (2:1 potentiometer), and its total nitrogen and phosphorus contents were respectively 0.13 ppm (Kjeldahl), and 7.5 mg kg^{-1} (Olsen/spectrophotometer). While both herbicide induced *Azospirillum* proliferation in inoculated as well as uninoculated unsterilized soil mixture, glyphosate was more inductive than 2,4-D, but still of more negative impact on wheat growth (Aguirre-Cadena et al. 2014). In contrast, the herbicides, 2,4-D and atrazine negatively influenced *Azospirillum* populations in the rhizospheric soil of groundnut crop, as well as soil enzymatic activities (Mohiuddin and Mohammed 2014). In another study on the effect of fertilizers (urea, diammonium phosphate and potassium chloride) and herbicides (glyphosate and 2,4-D) on the viability of *A. brasilense* strain C16 performed with three different concentrations for each agrochemical in a minimal salt medium at 0, 12, and 24 h of growth, the herbicides indicated more toxicity than fertilizers. In addition to type of the tested agrochemical, the concentration and time of exposure for each agrochemical were directly correlated with their antibacterial effects. After 24 h exposure to the maximal concentration, glyphosate, 2,4-D and potassium chloride reduced C16 viability by 43, 27, and 26%, respectively. Instead, urea (23%), and diammonium phosphate (50%) promoted its growth by 12 and 6% (Romero-Perdomo et al. 2015). The study on the effect of thiobencarb (also known as benthiocarb) on two *Azospirillum* spp. in pure cultures as well as in association with rice under both laboratory and field conditions indicated that the herbicide was of no negative effect on *A. lipoferum* 4B, but it inhibited the growth of *A. brasilense* N040. Also, despite of its negative impact on the growth of aseptically grown rice plantlets, thiobencarb did not influence dinitrogen fixation when rice plantlets were first inoculated with *Azospirillum* strains in gnotobiotic conditions containing the herbicide (Omar et al. 1992). However, *Azospirillum* populations were inhibited with low levels of thiobencarb applied to three tropical rice soils incubated under non-flooded conditions (Jena et al. 1990). The inoculation of corn seeds with *A. brasilense* together with the application of nitrogen fertilizer (140 kg urea ha^{-1}; applied at 26 days after sowing) minimized the harmful effects of the herbicide mesotrione (192 g ha^{-1}; applied at 21 days after sowing) in the initial development of corn (Bulegon et al. 2017).

Effect of Herbicides on Cyanobacteria (Green-Blue Algae)

Heterocystous and filamentous cyanobacteria are considered as a considerable part of soil microflora of paddy fields in tropical countries that significantly contribute to soil fertility (Singh 1961; Venkataraman 1981) through photosynthetic and photoheterotrophic dinitrogen fixation (Stewart et al. 1975). Biofertilization with

cyanobacteria and green algae is able to increase rooting of grapes cuttings and germination of sunflower seeds, and improve plant growth as observed in rice, barley, oats, tomato, radish, cotton, sugarcane, maize, chili, lettuce, wheat, gilly-flower, grapevine (Spiller and Gunasekaran 1990; Romanowska-Duda et al. 2004, 2010; Saadatnia and Riahi 2009; Tajuddin and Subramanian 2005; Song et al. 2005; Nilsson et al. 2005; Karthikeyan et al. 2007; Abd El-Moniem and Abd-Allah 2008; Shanan and Higazy 2009; Sahu et al. 2012; Shariatmadari et al. 2013; Grzesik and Romanowska-Duda 2014, 2015). Additionally, the effectiveness of the foliar application of cyanobacteria and green algae in enhancing the photosynthetic performance and growth of willow (*Salix viminalis* L.) plants has newly been indicated under limited synthetic fertilizers application (Grzesik et al. 2017). Cyanobacteria possess photosynthetic machinery similar to that of the chloroplasts of higher plants (Fogg et al. 1973; Stewart 1973) and therefore, can be influenced by the certain groups of the herbicides that affect photosynthesis or respiration (Dodge 1975). The herbicides can also exert an indirect negative influence on cyanobacterial dinitrogen fixation through inhibition of cyanobacterial photosynthesis (Vaishampayan 1984). The effect of herbicides amitrole, a derivative of amitrole (3,5-diamino-1,2,4-triazole), diquat, paraquat, linuron, MCPA, and monuron on nitrogen-fixing blue-green algae, *Anabaena cylindrica*, *Aulosira* sp., *Calothrix elenkenii*, *Chlorogloeae frischii*, *Cylindrospermum muscicola*, a phycobiont *Nostoc* sp. from the lichen *Collema tenax*, *N. muscorum*, *Tolypothrix tenuis*, and *Westiellopsis* sp. was studied. The responses were recorded as an initial period of depression followed either by an increased activity, or by a distinct decrease on prolonged incubation. Some herbicides could severely restrict the nitrogen-fixing capacities of the cyanobacteria, and thereby generally influenced the overall nitrogen economy of soils (DaSilva et al. 1975).

Pre-emergence herbicides applied at field recommended doses, 3 days after transplanting (DAT) rice plants inhibited cyanobacterial growth and dinitrogen fixation inoculated 10 DAT. The inhibition lasted up to 20 DAT. Butachlor and oxadiazon were more toxic than benthiocarb and pendimethalin. The application of 0.5 kg ha^{-1} active ingredient of 2,4-DNa (Na-2,4-dichlorophenoxy acetate) inhibited cyanobacterial growth, but the post-emergence herbicide 2,4-DEE (2,4-dichlorophenoxyacetic acid ethyl ester) applied 30 DAT inhibited cyanobacterial growth as well as dinitrogen fixation. The inoculation of 10 kg ha^{-1} of dry mixture of blue green algae 10 DAT could produce the maximum biomass 60 and 80 DAT in control and herbicide treated plots, respectively. The biomass and nitrogen produced by cyanobacteria were higher in controls than in the herbicide treated plots. The application of cyanobacterial biofertilizer along with herbicides increased the grain and straw yields as well as panicle number and nitrogen uptake by rice over no cyanobacterial treatment. The cyanobacterial treatment even without weeding resulted in the increased rice yield up to that of herbicide and biofertilizer treatments (Singh et al. 1988). The herbicides Saturn and Knockweed inhibited the growth of blue-green alga at relatively higher concentrations, while exerted no significant effect on heterocyst frequency. Saturn depicted more toxicity than knockweed as reflected by their lethal doses of 20 ppm and 1000 ppm, respectively

(Ahluwalia 1988). Herbicides chlorotoluron and diuron effectively induced mutants of *Nostoc muscorum* to 50–100 times more than background level. The mutants indicated a range of characters including pigment deficiency, no fixation of dinitrogen, as well as cyanophage resistance (Amla and Kochhar 1982). Prolonged cultivation of the cyanobacterium *A. variabilis* in the presence of sublethal concentrations of diuron indicated a two-stage adaptation to the herbicide action. In the relatively short-time first stage, physiological adaptation was observed as the increased ratio of phycobilin/chlorophyll. In the more prolonged second stage, cells with low sensitivity to diuron were selected in the population. These cells were of a low ratio of phycobilin/chlorophyll (Andreev and Maslov 1988). Monuron inhibited growth and heterocyst formation in the nitrogen-fixing blue-green alga *Nostoc muscorum* (Fig. 56), where the inhibitory impact was reversible in both nitrogen-free and nitrate media (Vaishampayan 1984).

The herbicides DCMU [3-(3,4-dichlorophenyl0-1,1-dimethylurea], isopropturon, and ioxynil not only blocked photosynthetic photosystem II in the aquatic cyanobacterium, *Synechocytis salina*, but also more strongly inhibited the photosynthetic oxygen evolution. Phenylurea herbicides (DCMU, and isoproturon) were found of stronger impact on cyanobacterial PSII activity compared to the phenol-type ioxynil (Yotsova et al. 2017). Interestingly, herbicides can increase the rate of nitrogen fixation, wherever they omit the impact of green algae interactions with the effective herbicide-resistant cyanobacterial strains. This has been indicated by developing a variant of the nitrogen-fixing cyanobacterium, *Aulosira* strain 68 resistant to the herbicide simetryne at the concentrations that prevented the development of indigenous algae. Interference from indigenous algae may hamper the development of nitrogen-fixing cyanobacteria introduced into rice fields in attempts to increase biological nitrogen fixation (Wilson et al. 1979). Monosulfuron, applied at 0.03–0.3 nMol L^{-1} under laboratory conditions, stimulated the growth of the nitrogen-fixing cyanobacteria *Anabaena flos-aquae* (a

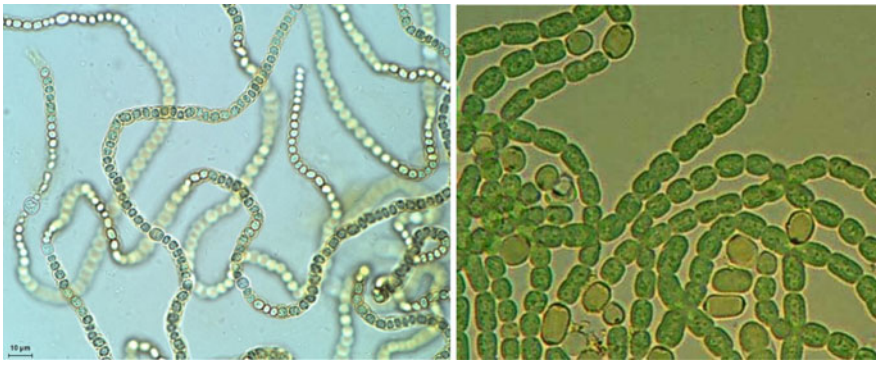


Fig. 56 (Left) *Nostoc muscorum*, filamentous structures called trichomes with enlarged cells called heterocysts (*Source* http://dbmuseblade.colorado.edu/); (Right) *Nostoc* (*Anabaena*) *azollae* trichomes and heterocysts (*Source* http://www.pinterest.com/)

free-living soil cyanobacterium that mat be toxic to some organisms; Fig. 57), *A. azollae* (a cyanobacterial symbiont with the water fern, *Azolla* spp., Fig. 56), and *A. azotica* (a free-living soil cyanobacterium with a high nitrogen-fixing capability; Fig. 57), but applied at higher concentrations of 30–300 nMol L^{-1} inhibited protein. The production of 16 amino acids was reduced in *A. flos-aquae* from 7 to 69% with increasing the herbicide concentration. The application of monosulfuron at 3–300 nMol L^{-1} substantially inhibited in vitro activity of acetolactate synthase (ALS) enzyme as indicated by 50% inhibition index values of 3.3, 65.2, and 101.3 nMol L^{-1} for *A. flos-aquae*, *Nostoc (Anabaena) azollae*, and *A. azotica*, respectively. Apart from *A. flos-aquae* at higher concentrations of 30–300 nM L^{-1}, the activity of the extracted ALS was not affected in the algal species treated with monosulfuron when applied at the rate of 0.03–300 nMol L^{-1}. Thus, *A. flos-aquae* was found as the species most sensitive to monosulfuron.

The toxicity of the herbicide to three cyanobacterial nitrogen-fixers was attributed to its interference with protein metabolism via the inhibition of branch-amino acid biosynthesis and particularly ALS activity. In rice cropping systems where monosulfuron is applied at low concentration (3 nMol L^{-1}), the use of the beneficial nitrogen-fixing cyanobacteria *A. azotica* and *A. azollae* may be suitable as "biofertilizers" given their high tolerance to the sulfonylurea herbicide (Shen et al. 2009). The sensitivity of cyanobacteria to PSII inhibitors such as s-triazines, and phenylurea has been reported (Lockert et al. 2006), and blue-green algae exhibit less sensitivity than green algae to the phenylura herbicide diuron applied at 0.16 mg L^{-1} (Abdel-Aty and El-Dib 2016).

Effect of Herbicides on Nitrifying Bacteria

Studies on the effects of selected herbicides on the parameters of soil nitrification processes (Fig. 58) led to the separation of four distinct groups of herbicides. The formulated octanoates of bromoxynil and ioxynil (NPH1320 and Totril, respectively) were the most toxic herbicides. Because of their very high toxicity, ioxynil and bromoxynil might also, in spite of their low practical field rates of

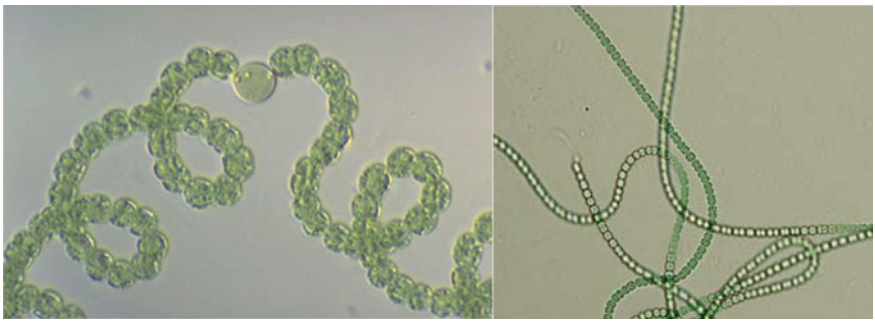


Fig. 57 Trichome of (Left) *Anabaena flos-aquae* with heterocyst, and (Right) *A. azotica*

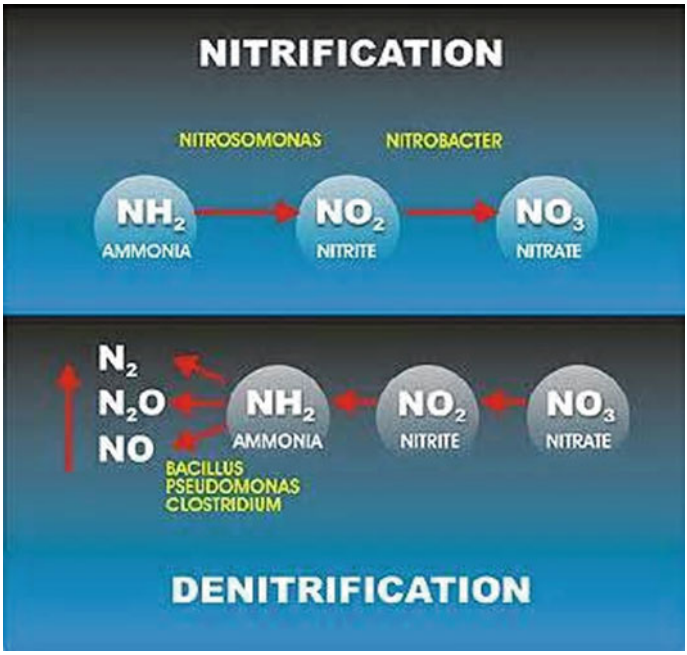


Fig. 58 Nitrification and denitrification and the bacteria involved in the phenomena (*Source* http://nsspo.com/p1/Nitrification.htm/)

0.25–0.5 kg ha^{-1}, exert a small though temporary depressing effect (Debona and Audus 1970). Next in order of toxicity were chlorbufam, phenmedipham, formulated oxadiazon, formulated legurame, ioxynil, formulated trifluralin, and bromoxynil. Terbacil, dicamba, and tricamba were of low toxicity, whereas asulam and related experimental herbicide MB9555 influenced some parameters only when they were tested at the very high concentrations. Bromoxynil application resulted in the decreased population of the bacteria involved in nitrification. *Nitrobacter winogradskii* was more sensitive than *Nitrosomonas europaea* to four groups of herbicides.

Legurame and oxadiazon were relatively more toxic to *N. europaea* in culture, but this differential toxicity was not found in the soil. Dicamba, tricamba, trifluralin and chlorbufam were more toxic to *N. winogradskii* than to *N. europaea* in the soil. However, the formulated trifluralin seemingly stimulated the growth of both nitrifiers but only in the soil, suggesting the suppression of antagonistic organisms as a possible reason. It was extrapolated that terbacil was the only herbicide that might cause small inhibition of nitrification at the rates recommended for field applications. However, terbacil was found disproportionately toxic at low concentration. The formulated octanoates of bromoxynil and ioxynil, and possibly dicamba and oxadiazon can cause small inhibitions when applied at the rates somewhat in excess of normal (Ratnayake and Audus 1978). The bacteria involved

in nitrification process show highest sensitivity to bromoxynil herbicide (Topp et al. 1992) and the inhibition of the activity of ammonium oxidation by the bacteria has also been reported in soils treated with the herbicide (Pampulha and Oliveira 2006). The actions of these two herbicides are nearly similar, and both effectively inhibit nitrification to the 50% level even at the concentrations much lower than 50 ppm (Debona and Audus 1970). Chlorflurazole was not an inhibitor of nitrification as complete as propanil, and even at 100 ppm inhibited only a little more than 80% of nitrification. Chlorflurazole had much more striking effect on the growth of bacterial nitrifiers and no growth was detected at only at 50 ppm. No indication of adaptation was found with chlorflurazole in contrast to propanil, bromoxynil and ioxynil. Chlorflurazole might exert a small depressing effect under field conditions, however unlike bromoxynil and ioxynil, its effect might be more persistent (Debona and Audus 1970). Chlorthiamid depicted a marked suppression of nitrification, 50% inhibition when tested at the concentration of 100 ppm. The herbicide was found to be of a strict impact on the proliferation of nitrifiers. Its effect on the proliferation of nitrifying bacteria was more than that of propanil, bromoxynil, ioxynil, and chlorflurazole, and led to 50% inhibition of proliferation at the concentrations below 50 ppm. The inhibition of growth clearly occurs through an action independent of that through nitrification itself. Under field conditions, chlorthiamid with application rates up to 10 kg ha^{-1}, might lead to a very slight depression of nitrification processes at the highest rates (Debona and Audus 1970).

2,3,6-TBA completely inhibits nitrification processes at the application rate of 1000 ppm, however, the toxicity of this herbicide is much less in soil (50% inhibition at the rate of 1000 ppm). Despite of the inhibition of proliferation, the nitrifying bacteria extensively adapt to 2,3,6-TBA in soil (Debona and Audus 1970). The lowest concentrations used in the perfusers (50 ppm) correspond with field rates of approximately 25 kg ha^{-1} (see Fletcher 1960), therefore, it seems very unlikely that endothal at its normal field rate could have any significant impact on nitrification (Debona and Audus 1970). Picloram had a much less impact on nitrification process, and its virtually total inhibitory impact was found only at its concentration of 500 ppm, while it reached the 50% level at 150 ppm. Picloram, even in lower concentrations, significantly inhibited the proliferation of bacterial nitrifiers. No growth was detected at the rate of 1000 ppm, where nitrification was completely inhibited. Picloram influenced *Nitrosomonas* activity and the process of ammonia to nitrite conversion (Debona and Audus 1970). Similarly, partial inhibition of the conversion of ammonia to nitrite was found in soil when picloram was applied at the rate of 1000 ppm. However, the treatment was of no effect on nitrite to nitrate conversion suggesting that *Nitrosomonas* was much more sensitive than *Nitrobacter* (Goring et al. 1967). The lowest concentrations used in the perfusers (50 ppm) correspond with field rates of approximately 25 kg ha^{-1} (see Fletcher 1960), therefore, it seems very unlikely that endothal at its normal field rate could have any significant impact on nitrification (Debona and Audus 1970). The toxicity of paraquat is low in soil because of its extensive adsorption on clay colloids. Biologically, the adsorbed paraquat is totally inactive, therefore, only a relatively

small fraction of unadsorbed herbicide in equilibrium in the soil solution is effective in inhibition of nitrification in the soil (Debona and Audus 1970). However, paraquat is highly inhibitory of the oxidation of ammonia to nitrite and nitrate by *Nitrosomonas* and *Nitrobacter* in culture (Debona 1967). The effect of paraquat on the nitrification seems to be a progressive inhibition, with increasing concentrations reaching 50% level of inhibition at about 750 ppm. Paraquat was of no significant effect on the rate of nitrifying bacteria proliferation, and its action would seem to be only on nitrification, not directly on cell growth (Debona and Audus 1970). Paraquat (4 M) inhibited the growth of *Nitrobacter agilis* but did not influence the growth of *N. europaea*. Also, nitrite did not accumulate in the presence of paraquat (4 M) when ammonia was oxidized by a mixed culture of the two nitrifiers (Yamanaka 1983). As the toxicity of paraquat on *N. gracilis* is known as the result of superoxide anion radical formation (Moody and Hassan 1982), the difference in sensitivity to paraquat between the two nitrifiers may be attributable to the differences in the content of superoxide dismutase in the two bacteria (Yamanaka 1983). Additionally, the relative insensitivity of *N. europaea* to paraquat has been attributed to hydroxylamine (Yamanaka 1983), a metabolic intermediate of ammonia (Hofman and Lees 1953), which may function as a scavenger of superoxide anion radicals (Elstner et al. 1975). The lowest concentrations used in the perfusers (50 ppm) correspond with field rates of approximately 25 kg ha^{-1} (see Fletcher 1960), therefore, it seems very unlikely that endothal at its normal field rate could have any significant impact on nitrification (Debona and Audus 1970). Dichlobenil inhibited nitrification but its effect was somewhat smaller than that of paraquat, and the 50% level of inhibition was reached at the application rates over 1000 ppm. In contast to paraquat, dichlobenil not only reduced nitrification, but also significantly reduced their proliferation. The lowest concentrations used in the perfusers (50 ppm) correspond with field rates of approximately 25 kg ha^{-1} (see Fletcher 1960), therefore, it seems very unlikely that endothal at its normal field rate could have any significant impact on nitrification (Debona and Audus 1970).

Endothal stimulated nitrification, and the greatest effect was obtained at the highest concentration tested (1000 ppm). Endothal only caused a slight reduction in the rate of cell proliferation at all its concentrations tested. The rapid microbial breakdown of endothal in the soil (Jensen 1964), the ability of soil bacterial nitrifiers in the complete breakdown of the herbicide to carbon dioxide and in the use of carbon dioxide as the principal carbon source, and the possible existence of other growth-stimulants in the formulated product have been discussed as the reasons for stimulatory impact of endothal. The lowest concentrations used in the perfusers (50 ppm) correspond with field rates of approximately 25 kg ha^{-1} (see Fletcher 1960), therefore, it seems very unlikely that endothal at its normal field rate could have any significant impact on nitrification (Debona and Audus 1970). Studies seven herbicides, indicated that sodium pentachlorophenate (PCP), 4,6-dinitro-ortho-secondary-butyl phenol (DNBP), isopropyl N-(3-chlorophenyl)-carbamate (CIPC), and monuron were the strongest inhibitors. Sesone (sodium 2,4-dichlorophenoxyrthyl sulfate also known as 2,4-DES) and dalapon were of the least inhibitory impact on the respiration of nitrifying bacteria, while PCP exhibited

the greatest level of inhibition. A comparison of the rates of nitrification as measured by formation of nitrate in the percolation method and oxygen uptake in the monometric method showed fair agreement for CIPC, but not for monuron. A comparison of the approximate concentrations of herbicides used in the field for chemical weed control and concentrations which caused a 50% inhibition of oxygen uptake by the nitrifying microorganisms indicated that the concentrations of herbicides which inhibited respiration by 50% would not occur at usual field application rates and that there would be little or no detrimental effect on soil nitrification (Hale et al. 1957). Propanil extremely inhibits nitrification, and gives virtually complete inhibition even in the lowest concentrations (50 ppm). Propanil, with normal practical rates of 5–15 kg ha^{-1}, is likely to cause serious and persistent depressions of nitrification in the field (Debona and Audus 1970).

Effect of Herbicides on Mycorrhizal Fungi

Mycorrhiza (plural, Mycorrhizae) is a term that means "fungus root", widely applied to describe the mutually beneficial symbiotic relationship between fungal partner (mycobiont) and plant partner (phytobiont) roots. These mycobionts colonize the root of their phytobionts and then form a vast hyphal network throughout the soil and thereby greatly increase absorptive surface area, and improve phytobiont nutrition through increased absorption of phosphorus (P), zinc (Zn), manganese (Mn), copper (Cu), and water. The phytobiont supplies carbohydrates for mycorrhizal mycobiont. More than 150 species of mycorrhizal mycobionts have been identified in all types of soils and climates throughout the world. There are several classes wherein these fungi are categorized to, however, here we will pay to the effect of herbicides on mycobionts involved in the two most common classes of mycorrhizae, endomycorrhizal and ectomycorrhizal fungi.

Effect of Herbicides on Endomycorrhizal Fungi

Endomycorrhizal fungi are regarded as obligate mycobionts that cannot aseptically be cultivated on growth media. These glomeromycotan true fungi are morphologically identified with chitinized walled aseptate (without cross-walls) hyphae, that form arbuscules (dendroid haustorium-like structures, Fig. 59) in the periplasmic region of plant root cortical cells, and reproduce asexually through production of thick-walled and long-surviving spores that are very resistant to freezing and intense heat. Also, some genera produce intercalary or terminal sac-like cellular structures in their hyphae that contain lipids and primarily serve as storage organs for the fungus. These structures called vesicles (little sacs, Fig. 59) can also act as propagules and colonize other parts of the plant root. As the formation of arbuscules is a common morphological character, these fungi are also known as arbuscular mycorrhizal fungi (AMF).

AMF are of interest for their reported roles in preserving soil fertility in agroecosystem, which form mutualistic symbiosis with the roots of most agricultural plants. These associations are common in terrestrial ecosystems, including

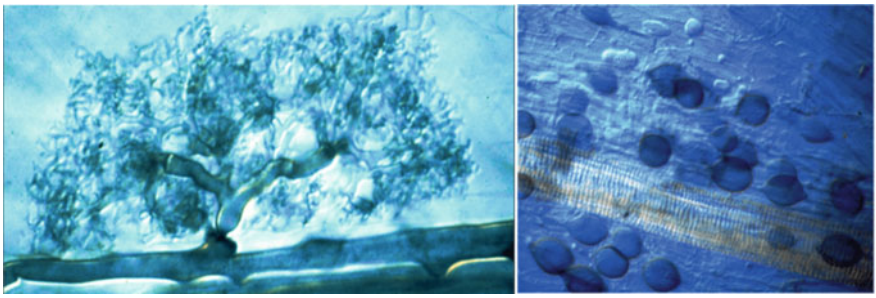


Fig. 59 Morphological characters of arbuscular fungi. (Left) an arbuscule (a little tree-like organ) formed by the fungus in the periplasmic region of plant root cortex cell and involved in bidirectional exchange of metabolites between two symbionts; (Right) vesicles, round sac-like structures developed by the fungus in the intercellular spaces in plant root (Buechel and Bloodnick 2016)

those of anthropogenic origin, such as agroecosystems (Smith and Read 2008). AMF stimulate host plant growth, and improve its resistance to hazardous biotic agents such as pathogens, as well as pests, as well as abiotic agents such as drought, salinity, heavy metals, hot temperatures, and chemical stressors (Smith and Read 2008; Gianinazzi et al. 2010; Vannette and Hunter 2009). AMF secret a glycoproteinaceous substance, glomalin and thereby play an important role in the formation and stabilization of soil aggregates (Smith and Read 2008; Gianinazzi et al. 2010). Additionally, they act as crop nutrition facilitators through increment of nutrient uptake and crop growth promotion that results in non-mycorrhizal weed suppression. Therefore, AMF represent potential alternatives to costly and environmentally hazardous herbicides (Cameron 2010; Rinaudo et al. 2010). Hence, AMF are crucially relevant in the maintenance of soil sustainability and plant production (Gosling et al. 2006; Gianinazzi et al. 2010; Raviv 2010). In the past, most research on AMF has been focused on possible responses to fungicides, rather than on the potential effects on mycorrhizal symbiosis following herbicide application. Because of obligately endosymbiotic nature of endomycorrhizal associations between host plants and AMF, any severe herbicidal phytotoxicity to the host can result in a severely damaged AMF and endomycorrhizal association. Pasaribu et al. (2013) studied the effect of three application rates of two widely used herbicides (1.8, 3.6, and 5.4 μg a. i. g^{-1} for alachlor and 1.08, 2.16, and 3.24 μg a. i. g^{-1} for glyphosate) on sporulation and infection of peanut plant by *Glomus mosseae* under greenhouse conditions. The result of their study indicated that the mycobiont *G. mosseae* responds in a differential way to two different herbicides applied, and the fungus sensitivity to alachlor proved significantly higher than glyphosate. None of the herbicide treatments could affect the external hyphal length and succinate dehydrogenase activity. However, the spore number, total and active infection intensity of internal hyphae was significantly reduced with the increasing rates of alachlor application, while glyphosate had no significant impact at all tested application rates. Consequently, phosphorus inflow through mycorrhizal hyphae

was considerably increased with the application rates of glyphosate, with the highest value (41.48 and 479.72 $\times$ 10^{-13} mol Phosphorus m^{-1} s^{-1} hyphal inflow and hyphal uptake, respectively) obtained at recommended rates for field application of glyphosate (2.16 μg a. i. g^{-1}). Therefore, symbiotic functions of *G. mosseae* with host plant could be affected by the depressive effects of the herbicides, apparently related to the types of herbicides and their application rates (Pasaribu et al. 2013). Similarly, the application of glyphosate (Roundup®) in a model ecosystem resulted in 40% reduction in mycorrhization in the soil amended with *G. mosseae* (Zaller et al. 2014). The reduced total and active mycorrhizal infection intensity following certain herbicidal treatments is attributed to the decreased supply of host photosynthates as the result of negative effect of the herbicide on plant photosynthesis (Smith 1980). Accordingly, glyphosate did apparently not influence the spore germination of *G. mosseae* even when applied at highest doses but when added to culture medium, it reduced AMF spore germination and germ tube growth only at the concentrations higher then those recommended for application in field (Giovannetti et al. 2006). However, the negative impact of glyphosate on endomycorrhization of carrot roots by *Rhizophagus irregularis* has been observed in vitro (Wan et al. 1998). Moreover, the evaluation of three rates of glyphosate (0, 0.26 and 1 $\times$ recommended field rate) 10 and 30 days after treatment (DAT), indicated that spre viability in herbicide untreated control soils was between 5.8-and 7.7-fold higher than in treated soils, even when the lower rate was applied. Significant reductions in root mycorrhization were found in *Lolium multiflorum* plants grown in glyphosate treated soil than in control soils. The reduced number of arbuscules (but not vesicles) indicated the affected functionality of symbiosis 30 DAT (Druille et al. 2013). Arbuscules are considered as the main sites for nutritional exchanges between host plant and endomycorrhizal mycobiont (Smith and Gianinazzi-Pearson 1988). The results indicated that soil residence time of glyphosate and/or its degradation products were enough to reduce AMF spore viability and their ability to colonize host roots. The decrease in the viability and symbiotic capability of AMF propagules may affect plant diversity, taking into account that different plant species are of different degree of mycorrhizal dependencies (Druille et al. 2013). Similarly, soil treatment with glyphosate (0.4, and 4 L ha^{-1}) notably reduced endomycorrhizal colonization of pepper (*Capsicum annuum* L.) roots (Ronco et al. 2008). In contrast, no adverse impact on soybean root endomycorrhization was found when glyphosate was applied in high dose of 10 L ha^{-1} (Malty et al. 2006). These controversial differences are possibly due to the differences in the studied host plant species, AMF species, dose and formulation of the herbicide, agricultural practices (such as irrigation), soil texture, soil chemistry and microbiology, and weather conditions. Herbicide effects on vesicular AMF in *Citrus* spp. under greenhouse as well as grove conditions indicated that the mixture of bromacil + diuron was not of significant impact on mycorrhization of *Citrus* spp. in cultivated plots. Trifluralin, bromacil, and diuron were of no apparent effect on *Glomus etunicatus*, and plant growth. However, the mixture of simazine + paraquat led to a slightly reduced mycorrhization of grove trees roots. Paraquat, and simazine were of adverse impacts on plant growth as well as the

mycobiont (Nemec and Tucker 1983). Simazine did not influence the mycorrhizal hyphal elongation in vitro, but paraquat, and dichlobenil, even at the lowest concentrations, were found to be of significantly inhibitory impact on hyphal elongation, however, different application rates of these three herbicides could not affect mycorrhizal root colonization under greenhouse conditions (Hamel et al. 1994). The significant inhibition of endomycorrhization of plant root as well as reduction of the number of spores in legumes have been reported following treatments with Brominal® (bromoxynil) and Gramoxone® (paraquat; Abd-Alla et al. 2000). Terbuthylazine (5.2, 10, 20.4, 40.4, and 81.2 mg L^{-1}), and MCPA (2-methyl-4-chlorophenoxyacetic acid; 1.2, 2.8, 5.2, 10.8, and 21.2 mg L^{-1}) significantly inhibited the mycelial growth of *G. mosseae* in vitro, and the fungus indicated a clear dose-effect response exclusively in the presence of the herbicidal active ingredient, pendimethalin (9.6, 19.2, 38, and 76 mg L^{-1}). Pendimethalin, when applied at the rate of 4.8 mg L^{-1}, exhibited a hormetic effect and induced the mycelial growth of *G. mosseae* under in vitro conditions (Giovannetti et al. 2006).

Ocampo and Barea (1984) found that carbamate herbicides which are responsible for inhibited photosynthesis mostly did not have any negative effect on arbuscular mycorrhiza (AM). Among three carbamate herbicides including chlorpropham, sulfallate and phenmedipham, only phenmedipham caused the reduction of root sugars and fungal metabolism when applied as foliar spray. However, these herbicides were of no effect on the amount of AM infection when applied through either foliar spraying or direct addition to the soil. Although the application of high concentration of phenmedipham greatly reduced plant growth, plants could recover with the help of AM demonstrating the beneficial effects of AM for its host plant. The results strongly suggest that plants which benefit from AM are less affected by the deleterious effects of herbicides, especially when used at high concentrations, than those without AM. Moreover, it seems that carbamate herbicides cannot significantly affect the infection of AM which makes them proper choices in the chemical control of weeds. The negative impact of sulfentrazone (0.7 mg kg^{-1} soil) application on soybean symbiotic processes (Vieira et al. 2007), and of soil treatment with herbicides on crop root colonization by AMF (Santos et al. 2006) have been indicated. The pre-emergence herbicide, isoxaflutole (Converge Pro®; applied at the rate of 19.8, 39.6, 79.2, and 158.4 μg a. i. L^{-1} of soil) did not influence corn root colonization by the mycorrhizal fungus, *Rhizophagus irregularis* (Stoklosa et al. 2011).

In vitro studies on the effect of two herbicides, prometryn and acetochlor on the endomycorrhization of carrot hairy roots [induced through transformation by root inducing tumor-inducing DNA (Ri T-DNA)] by the AM fungus, *G. etunicatum* indicated that acetochlor at all tested concentrations (0.1, 1, and 10 mg L^{-1}) and low concentrations of prometryn (0.1, 1 mg L^{-1}) were not of any significant impact on spore germination, while all treatments notably inhibited hyphal growth of the fungus. When exposed to the herbicides at certain concentrations, the initial growth of the fungus spores was directly affected. Prometryn and acetochlor detrimentally influenced the formation and function of AM, se well as the rate of mycorrhizal colonization, regardless of concentration. The colonization of host plant roots is a

prerequisite for growth and development of AMF, whereas, the rate of mycorrhizal colonization is an index of the affinity of AMF for the host plants. Hence, the limitation of hazardous effects of agricultural chemicals (such as various types of pesticides as well as fertilizers) on mycelial growth and metabolism, as well as the formation, function, and colonization of AM, is a key factor in maximizing the positive effects of AM symbiosis on plants (Li et al. 2013). Also, both herbicides significantly reduced succinate dehydrogenase activity at all concentrations (but not 0.1 mg acetochlor L^{-1}), indicating their inhibitory impact on the respiratory electron transfer within fungal mitochondria, where the enzyme forms a part of complex II. Prometryn and acetochlor also inhibited the activity of alkaline phosphatase (Li et al. 2013), the enzyme associated with active metabolism of phosphates (Tisserant et al. 1993).

Interestingly, AMF help host plants to escape from the phytotoxic impacts of herbicides. Bethlenfalvay et al (1996) conducted a greenhouse study to determine if AM fungi, whose hyphae interconnect the roots of adjacent plants, modify herbicide effects by enhancing nutrient fluxes between associated plants. Soybean (*Glycine max* (L.) Merr.), and common cocklebur (*Xanthium strumarium* L.) plants were grown together in pots (1.5 L) in a high-P (28 mg kg^{-1}) soil. They were sprayed with the herbicide, bentazon (BEN, 3-isopropyl-lH-2,1,3-benzothidiazine-(4)3H-one 2,2-dioxide), at dose rates of 0, 1/3, 2/3, 3/3, and 4/3 of field recommendation (FR, 1.12 kg a. i. ha^{-1}) while in the 7- to 8-leaf stage. Labelled N (1 mL of 100 mM tSNHaNO_3, 98 atom percent 15 N) was applied to the cocklebur leaves 4 days before spraying and then assayed in the soybean leaves at harvest. Growth and nutrient contents of +AM soybean shoots were enhanced only at the intermediate FR levels, while shoot growth in adjacent cocklebur was inhibited beyond the extent measured in −VAM plants. Labelled N was at natural abundance in both +AM and −AM soybean leaves at 4/3-FR, but at 1/3-FR to 3/3-FR 15 N abundance was significantly higher in +AM than in −VAM plants. These results suggest that shifts in source-sink relations occurred both within each plant and between plants as a result of the selective stress imposed on cocklebur. Moreover, this shift in competitiveness permitted an AM-mediated flux of nutrients from weed to crop.

It is interesting that the inhibitory effect of an herbicide on a given AM fungus can differ in a host-dependent manner. For instance, diclofop inhibited root colonization by *G. deserticola* in wheat, but not in ryegrass. Its inhibitory effect on wheat root endomycorrhization directly increased as its dose (10%, 50%, 100%, and 1000% of the dose recommended for field application, 0.9 kg ha^{-1}) rose (Rejon et al. 1997).

Effect of Herbicides on Ectomycorrhizal Fungi

Ectomycorrizal fungi (EMF) can help in plant establishment in nutritionally poor or degraded soils. They increase volume of the exploited soil by roots through the improvement of water and nutrient absorption, in special those with low mobility such as phosphorus (Marx and Cordell 1989). EMF can also provide great

resistance to high temperatures and extreme pH values (Marx and Cordell 1989), increase root longevity (Allen 1991), stimulate dry matter production (Souza et al. 2004), and provide greater tolerance to soil toxicity conditions and pathogens (Allen 1991; Grazziotti et al. 2003). Weeds harm seedling establishment and growth via allelopathy (Toledo et al. 2003) or competition over water, nutrient and light. Additionally, weeds can raise fire risks, interfere with, and even hinder other forestry practices (Tuffi Santos et al. 2006). Therefore, herbicides are broadly applied in silviculture and forestation, where the use of EMF requires some knowledge on the impact of herbicides on these fungi (Fernandes et al. 2014). Ectomycorrhizal associations depend on interaction with soil, environment, and host plants and can be affected by adopted crop management (Campos et al. 2011). Herbicide can directly affect EMF in three ways: (i) some herbicides may exert no influence on EMF (Wardle and Parkinson 1991); (ii) some herbicides may stimulate the growth and/sporulation of EMF (Roslycky 1982); and (iii) some herbicides may inhibit EMF growth and/sporulation as well as ectomycorrhization of host plant roots (Iloba 1978; Culdlin et al. 1983; Trappe et al. 1984). A few herbicides commonly used in foresty were tested for their possible effects on ectomycorhizal development and seedling growth of lodgepole pine (*Pinus contorta* var. *latifolia*) and white spruce (*Picea glauca*), and it was shown that hexazinone, glyphosate and triclopyr are able to reduce seedling growth and mycorhizal development of the plants (Sidhu and Chakravarty 1990). Among these foresty herbicides, triclopyr exhibited the most toxic effect and hexazinon was significantly effective when applied at high concentrations (2 and 4 kg ha^{-1}). Recovery from adverse effects of hexazinone was observed over the time and even no negative effect could be found when the herbicide was applied at low concentration (1 kg ha^{-1}) highlighting the importance of natural adaptation and herbicide dosage applied to the soil. Interestingly, it was found that seedlings with mycorrhizal fungus (*Suillus tomentosus*) are more sensitive to herbicides than those without any type of mycorrhiza. The adverse effect of hexazinone was less in field experiments as only high concentration (4 kg ha^{-1}) of the herbicide could reduce the seedling growth and mycorrhizal infections (Sidhu and Chakravarty 1990). Triclopyr, imazapyr, and sulfometuron methyl did not inhibit ectomycorrhizal formation at concentrations as high as twice the recommended field rates (Busse et al. 2004). Experiments with the isolates of a *Pisolithus* sp. indicated that they were of different tolerances to the herbicides glyphosate as well as isoxaflutole, and the rate of an isolate tolerance depended on the tested herbicide, its concentration, and physical status of the culture medium (liquid or solid). A given herbicide exhibited more toxicity on the growth of a given isolate of the fungus when it was amended into a solid culture medium. Also, no uniformity was found in the behavior of different isolates of the fungus. While the isolate Pt24 was the most tolerant of glyphosate, the isolate UFVJM04 exhibited the highest tolerance to isoxaflutole. The lowest effect of isoxaflutole suggested the possibility of its use in the areas where seedlings inoculated with EMF are applied (Fernandes et al. 2014). Furthermore, glyphosate was found more toxic to the isolates of *Pisolithus* than isoxaflutole.

The growth of *Pisolithus tinctorius* (Fig. 60) on a solid medium decreased because of 1 mg L^{-1} of the herbicides triclopyr, glyphosate, hexazinone, and 2,4-dichlorophenoxyacetic acid and was totally inhibited in 5000 mg L^{-1} of the same herbicides (Estok et al. 1989). On a solid medium, an isolate of *P. tinctorius* exhibited growth reduction by 59% at 50 mg L^{-1} concentration of glyphosate, and complete growth inhibition in same concentration of oxyfluorfen (Paula et al. 1995). However in an experiment with another isolate of *P. tinctorius*, no growth reduction was found in broth medium with glyphosate concentrations up to 10 mg L^{-1} (Lake et al. 1981). Triclopyr and 2,4-D when tested at the rate of 1000 mg L^{-1} completely inhibited the growth of *Hebeloma logicaudum*, while hexazinone and glyphosate were found more toxic and inhibited its growth in 55% at the concentration of 100 mg L^{-1} (Estok et al. 1989). Studies on the effect of herbicides on the growth of ectomycorrhizal fungi under in vitro conditions revealed that hexazinone was highly toxic to *P. tinctorius* (ED_{50} = 1 μg a. i. mL^{-1}), *Suillus hirtellus* (ED_{50} = 1 μg a. i. mL^{-1}), and *Suillus cothurnatus* (ED_{50} = 5 μg a. i. mL^{-1}). Bifenox decreased the in vitro growth of *P. tinctorius* (ED_{50} = 1 μg a. i. mL^{-1}), and

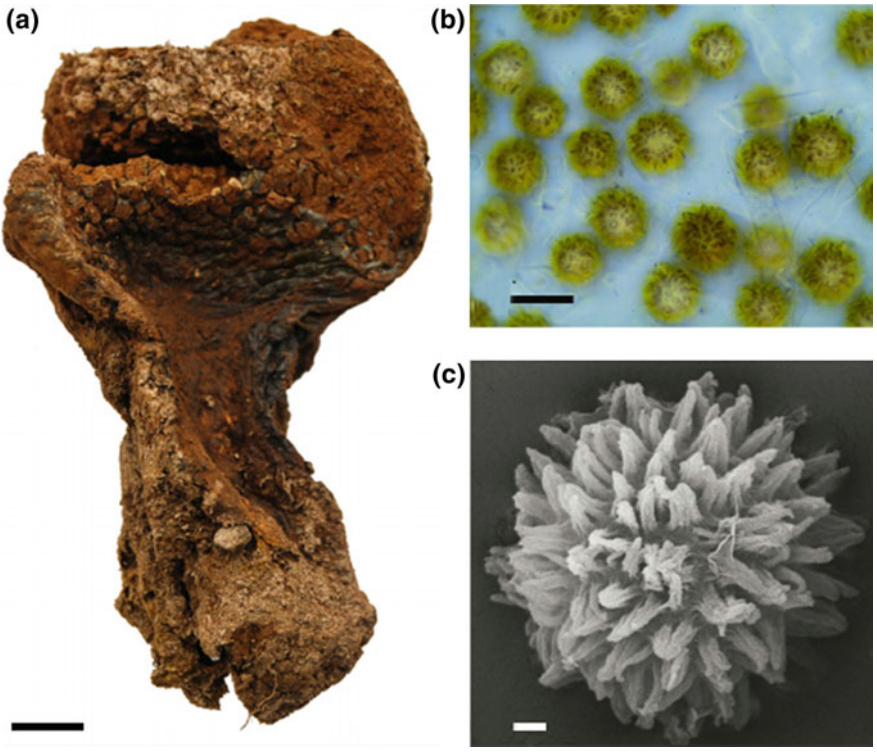


Fig. 60 *Pisolithus tinctorius* (05MCF5506). **a** Basidioma, scale bar = 1 cm; **b** spores under light microscope, scale bar = 10 μm; **c** spores under scanning electron microscope, scale bar = 1 μm (*Source* Rusevska et al. 2015)

S. cothurnatus (ED_{50} = 5 μg a. i. mL^{-1}). Oxyfluorfen had an inhibitory impact on the growth of *P. tinctorius* (ED_{50} = 1 μg a. i. mL^{-1}), *S. cothurnatus* (ED_{50} = 1 μg a. i. mL^{-1}), and *Laccaria laccata* (ED_{50} = 3 μg a. i. mL^{-1}). Oryzalin had a negative impact on the growth of *L. laccata* (ED_{50} = 3 μg a. i. mL^{-1}), *S. cothurnatus* (ED_{50} = 3 μg a. i. mL^{-1}), and *P. tinctorius* (ED_{50} = 10 μg a. i. mL^{-1}). Nitrofen (ED_{50} = 3 μg a. i. mL^{-1}), dipropetryn (ED_{50} = 3 μg a. i. mL^{-1}), trifluralin (ED_{50} = 10 μg a. i. mL^{-1}), and napropamide (ED_{50} = 10 μg a. i. mL^{-1}) were effective in the inhibition of the growth of *P. tinctorius*, while atrazine, propazine, simazine, and perfluidone did not seem to affect its growth at the doses recommended for field applications. Propazine (ED_{50} = 1 μg a. i. mL^{-1}) had a considerable effect on the growth of *S. luteus* under in vitro conditions. Interestingly when applied at different rates of 1, 3, and 500 μg a. i. mL^{-1}, simazine stimulated the growth of *P. tinctorius* and *S. hirtellus*, whereas perfluidone induced the growth of *Thelefora terrestris* (Kelley and South 1980). The ectomycorrhizal species *Rhizopogon vulgaris* and *Gautieria crispa* could metabolize and biodegrade atrazine (Donnelly et al. 1993). *Cenococcum geophilum* was known as an ectomycorrhizal fungus of high potential to biodegrade hexazinone (Donnelly and Fletcher 1994), and its was stimulated in 53% up to 10 mg L^{-1} dose of triclopyr and hexazinone (Estok et al. 1989). The ability to degrade an herbicide apparently depends on the herbicide as well as the mycorrhizal fungus (Donnelly et al. 1993; Donnelly and Fletcher 1994) and the rate of bidegradation of an herbicide by an ectomycorrhizal fungus varies with the ratio of C/N and pH (Donnelly and Fletcher 1994). The ectomycorrhizal fungi *Hebeloma cylindrosporum*, *Suillus bellini*, and *Suillus variegatus* were known as potent biodegraders of the herbicide chlorpropham (Rouillon et al. 1990).

Recently the ectomycorrhizal species *Hebeloma cylindrosporum* (Fig. 61) has been introduced as a model fungus (Fig. 62). Its full genome has been sequenced and annotated at the Joint Genome Institute, and is now available at http://genome.igi-psf.org/Hebcy2.home.htm for public information. Functional studies on the symbiosis are carried out using transcriptomic and proteomic approaches, while the fungus is still useful in secretomic studies and targeted analysis of secreted proteins carried out to evaluate the controversial "saprotrophic potential" of symbiotic fungi and their capacity to mobilise nutrients from forest soils. Its entire life cycle, from spore to spore, can be obtained under in vitro conditions (Debaud and Gay 1987). Just recently, a protocol has been developed for the establishing a symbiotic interface between cultured ectomycorrhizal fungi and plants to follow fungal phosphate metabolism (Becquer et al. 2017). It can easily be transformed using *Agrobacterium tumefaciens* (Combier et al. 2003), and a collection of mutant strains is available, including non-mycorrhizal ones (Combier et al. 2004). Furthermore, the fungus harbors a dominant carboxin resistance gene (*Hc.Sdh*^{R}; Ngari et al. 2009) that may be useful in development of selective media for the fungus. Therefore, the fungus provides an excellent opportunity for studies on the effect of agrochemicals (such as herbicides) on the biology of ectomycorrhizal fungi.

Fig. 61 *Hebeloma cylinrosporum*, an ectomycorrhizal fungus that is frequently and abundantly born in autumn and easily located under pines and holm oaks in pastures of sandy soil. Its descriptive characteristics are as follow: caps, 2–4 cm of diameter, at beginning convex, soon flattened, slightly umbonate, margin slightly striated and somewhat incurved; cuticle, viscous in wet weather, smooth, brown in the center with the lightest edge; stalks, cylindrical, relatively long, of the same color or somewhat lighter than cap, and without appreciable remnants of the curtain, when tearing it retains the sand; meat, thin, whitish with a weak smell; basidiospores (*Source* Mythological Society Extremena, MICOEX, http://micoex.org/2016/09/17/hebeloma-cylindrosporum/) (Color figure online)

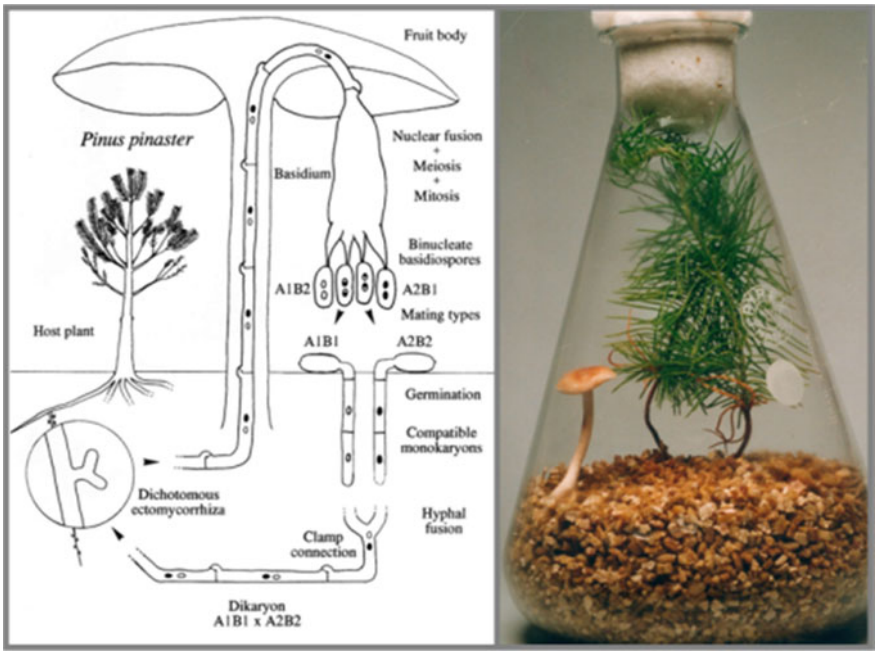


Fig. 62 The basidiomycetous fungus, *Hebeloma cylindrosporum* associated with *Pinus pinaster* as a model to infer the genetic and molecular bases of differentiation and functioning of the ectomycorrhizal symbiosis (*Source* http://www.ecologiemicrobiennelyon.fr/)

Effect of the Herbicides on Microbial Biological Control Agents

Effect of the Herbicides on Antagonistic Fungi

As *Trichoderma* spp. are the most important fungi applied in the biological control of plant diseases, therefore, here we focus on the impact of herbicides on *Trichoderma* spp. Studies with *Clonostachys rosea* 47 (CR47), *Trichoderma atroviride* 59 (TA59), *T. atroviride* 312 (TA312), *T. harzianum* 24 (TH24), *T. longibrachiatum* 9 (TL9), *T. longibrachiatum* 144 (TL144) and *T. viride* 15 (TV15) to evaluate their in vitro sensitivity to four herbicides (chlorsulfuron, chlorotoluron, flufenacet and pendimethalin) indicated that except the isolate TL144, none of the antagonists were sensitive from the stand point of their mycelia radial growth inhibition in the presence of the herbicides applied at field doses. Most antagonists did not exhibit any inhibition of conidial germination as the result of herbicide fungitoxicity. The predisposing impact of diphenamid on the host has been shown to be due to its adverse effect on soil microbial antagonists (Eshel and Katan 1972). Meriles et al. (2006) transplanted bean (*Phaseolus vulgaris* L.) seedlings to soil with previous corn crop residues, previous groundnut crop residues, and no agricultural soil, treated with a range of glyphosate concentrations. Population dynamics of soilborne fungi *Trichoderma*, *Gliocladium*, *Fusarium* and *Pythium* were monitored during a 24-day period after glyphosate treatment to study the effects of the herbicide and previous crop residue on fungal populations. In addition, those genera of soilborn fungi were tested to study in vitro fungitoxicity of glyphosate. Independent on glyphosate concentration, the highest population of *Trichoderma* spp. and *Gliocladium* spp. were noted on soil with previous corn residue. *Fusarium* and *Pythium* populations increased proportionally to the increase in glyphosate concentration. Glyphosate did not have any effect on *Trichoderma* and *Gliocladium* populations. *Trichoderma* isolates can be adversely affected by some herbicides commonly used for tea plantation (Islam et al. 2008). Among the tested herbicides, Bimaster was the only one which completely inhibited the growth of *Trichoderma.* Other herbicides including glyphosate, paraquat, kem-Amin and butachlor also negatively affected the fungal growth in the media. As *Trichoderma* isolates are of well-known antagonistic fungal microorganisms in tha soil, it is necessary that to precisely manage the incorporation of these herbicides into the soil. The in vitro effect of the herbicide ohinol 50 SC (50% napropamide) on *Trichoderma harzianum* and in vivo influence on colony forming units per gram of soil, also, the interaction between its quantity and the intensity of the root rot disease induced by *Rhizoctonia solani* were studied by Gveroska (1999). Different doses and methods were tested. The inhibition of dry biomass yield and radial growth proportionally increased with the increase of the applied dose of the herbicide. The highest decreases of *T. harzianum* quantity was found in 0.2 g m^{-2} before +0.3 g m^{-2} herbicide application after sowing. The greater quantity led to reduced intensity of the disease. The most suitable approach of *Trichoderma* application with tobacco seedlings was determined as the sowing of the seeds

already kept in pure culture of the biocontrol agent followed by after sowing application of the herbicide in a dose of 0.5 g m^{-2} (Gveroska 1999). Additionally, our simple experiments exhibit the possibility of the application of some of biological control agents such as *Talaromyces flavus* and various *Trichoderma* species in integrated disease management programs synchronous to or following the intelligent application of herbicides. Furthermore, our unpublished data from the experiments with sethoxydim and *Fusarium graminearum* and *Trichoderma* isolate No. 100 have well indicated that despite of the increased growth rate of *F. graminearum* on herbicide-amended culture media, the pathogen is still amenable to the biological control with the *Trichoderma* isolate No. 100, inferring to high capacity of its application in the integrated plant disease management programs against two major pathogens of wheat and rapeseed crops, namely *F. graminearum* and *Sclerotinia sclerotiorum*. To apply herbicides, the positive effects of some weeds shall also be considered, for example their role in the preservation of the beneficial parasitoids useful in control of pests. An experiment with the commercially available herbicidal formulation, Atlantis® OD 42 (mesosulfuron + iodosulfuron) was performed in order to study its potential effect on the interaction of the biological control fungus, *Trichoderma asperelloides* and the mycotoxigenic fungal pathogen *Fusarium graminearum* on potato dextrose agar (PDA) medium amended with the Atlantis® OD 42 to the final concentrations of 0, 50, 500, and 5000 ppm (Pakdaman and Elahifard, unpublished data). Interestingly, Atlantis® OD 42 inhibited the growth of both fungi when it was applied at the highest concentration (a dose higher than that recommended for field applications). *T. asperelloides* could finally control the mycotoxigenic fungus in the presence of lower concentrations of the herbicide (Fig. 63) under in vitro conditions (darkness, 26 °C). Also, the herbicide increased the pathogen resistance (R) and its linear growth (p) parameter in the presence of *T. asperelloides*, and reduced Pakdaman's Biological Control Index (PBCI), an index already introduced to study the effect of various biotic and abiotic factors (Pakdaman et al. 2013).

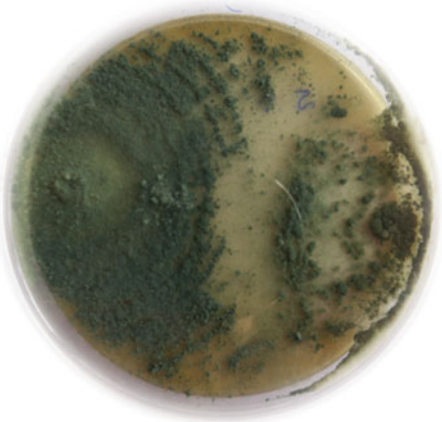


Fig. 63 Growth and sporulation of *Trichoderma asperelloides* over the mycotoxigenic pathogen *Fusarium graminearum* in potato dextrose agar medium amended with Atlantis® OD 42 (500 ppm) (*Source* Pakdaman and Elahifard, unpublished data)

In another study, it was shown that fungal strains from three genera including *Penicillium* and *Trichoderma* could use an herbicide, metsulfuron-methyl (MM) as a sole source of carbon and energy (Vázquez and Bianchinotti 1999). Among these genera, *Trichoderma* isolates exhibited the highest capacity for using the herbicide. These isolates could use the herbicide during their growth. Fungal spores could germinate in the media containing MM and use it as a carbon and energy source. These isolates were introduced as promising bioremediation agents (mycoremediants) to eliminate the herbicidal pollution from the environment (Vázquez and Bianchinotti 1999). *Trichoderma viride*, in particular, has been found to be sensitive to paraquat. The inhibitory effects were observed at concentrations well within the range likely to be experienced in the field (Wilkinson and Lucas 1969). Romero et al. (2014) indicated that a few soil fungi such as *Gliocladium roseum,* were able to degrade one of the widely used herbicides, atrazine in both pure cultures and polluted soil sediments. As atrazine residue in soil and water imposes lasting environmentally noxious effects, using living microorganisms to degarade this chemical would be promising in the removal of these pollutant residues from the natural habitats.

Effect of the Herbicides on Antagonistic Actinobacteria and Bacteria

Actinobacteria with their capacity to produce a broad spectrum of antibiotics and extracellular enzymes represent a high proportion of the soil microbial biomass (Doumbou et al. 2001), especially in suppressive soils (Postma et al. 2008). As an important part of soil microflora, actinobacteria are regarded as (i) a source of agroactive biological compounds, (ii) a group of plant growth promoting soil microorganisms, and (iii) tools for the biological control of plant diseases (Doumbou et al. 2001; Palaniyandi et al. 2013). Taxonomy, physiology, and natural products of *Actinobacteria* have recently been reviewed (Barka et al. 2016). Microbial community is significantly affected by the application of herbicides into the soil. When a few herbicides including atrazine, Primeextra (a combination of atrazine plus metolachlor), paraquat and glyphosate were incorporated into soil, bacterial, and actinobacterial populations decreased immediately. However, the soil microorganisms adapted to this chemical stress as their population was recovered over time (Sebiomo et al. 2011a). Microorganisms degrade herbicides and they may serve as bioindicators of soil changes following herbicide application. Simultaneously, the numbers of actinobacteria and less so of fungi increase, indicating that these microorganisms use herbicides as sources of biogenous elements. Rate of herbicidal decomposition depends on the properties of the preparation applied, herbicide dose as well as on the physical and chemical soil properties, soil moisture and temperature, ground cover, agrotechnical measures applied and the resident microbial population (Milošević and Govedarica 2002). Some herbicides such as isoproturon (Hussain et al. 2014), carfentrazone-ethyl (Tomkiel et al. 2015), sulfosulfuron (Kucharski and Wyszkowska 2008), terbutryn (Zaid et al. 2014), carbetamide (Zaid et al. 2014), propyzamide (Zaid et al. 2014), terbutryn + propyzamide (Zaid et al. 2014), metribuzin (Zaid et al. 2014; Hussain et al. 2014), Successor T (pethoxamid

and terbuthylazine; Wyszkowska et al. 2016), ametryn (Bera and Ghosh 2013), atrazine (Sebiomo et al. 2011b), Primextra (a mixture of atrazine plus metolachlor; Sebiomo et al. 2011b), paraquat (Sebiomo et al. 2011b), glyphosate (Sebiomo et al. 2011b), and glufosinate (Pampulha et al. 2007) are of negative impact on the growth of actinobacteria. Interestingly, hand weeding reduced actinobacterial populations highly significantly, and its effect on actinobacteria was higher than terbutryn, but less than that of other tested herbicides (Zaid et al. 2014). However, pre-emergent herbicides atrazine, metsulfuron methyl, metolachlor, and anilofos were of no negative impacts on soil actinobacteria in rain-fed maize field (Ramesh and Nadanassababady 2005). Other herbicides, for instance sulfentrazone (Martinez et al. 2008), glyphosate (in a dose of 2.16 mg kg^{-1} soil dry weight; Araújo et al. 2003), a mixture of diflufenican + mesosulfuron-methyl + iodosulfuron-methyl-sodium (Baćmaga et al. 2015) stimulate the growth of actinobacteria. The stimulation of the growth and development of actinobacteria by herbicides has been known as a general phenomenon by Milošević and Govedarica (2002) occurring with dimethenamide and metolachlor applied to sugarbeet (Govedarica et al. 2001), prometryn applied to soybean and sunflower (Milošević and Govedarica 2001), flumetsulam + trifluralin (Rival®), and alachlor + linuron (Linuron®), imazethapyr and clomazone (Milošević and Govedarica 2002). The population of actinobacteria did not increase significantly with any type or concentration of the employed herbicides including clodinafop propargyl 15% WP (applied at the rate of 400 g ha^{-1}), metribuzin 70% WP (applied at the rate of 300 g ha^{-1}), isoproturon 75% WP (applied at the rate of 1333 g ha^{-1}), mesosulfuron-methyl and iodosulfuron methyl sodium (Atlantis® 3.6% WG, 400 g ha^{-1}), Sulfosulfuron® 75% WG (applied at the rate of 33.33 g ha^{-1}), and UPH-110 54% WG (clodinafop propargyl and metribuzin) applied at the rates of 400, 500, 600, and 1000 g ha^{-1}. At field recommended dose, isoproturon (up to 15th day) and metribuzin (up to 7th day) reduced the actinobacterial population but other treatments did not alter the populations. UPH-110 was not of any significant impact at lower application rates, but it could significantly depress actinobacterial populations at higher doses, persisting up to 7th and 15th day of application. The toxicity of UPH-110 was directly correlated with its dose of application (Hussain et al. 2014).

Paraquat (25 μg mL^{-1}), pentachlorophenol (PCP, 25 μg mL^{-1}), and picloram (50 μg mL^{-1}) inhibited *Pseudomonas fluorescens*, while nitralin (25 μg mL^{-1}), and gluphosate (Gloria 2010) stimulated its growth. Atrazine, diuron, trifluralin, and fluometuron were found of no significant impact on *P. fluorescens* growth rate when tested at the concentrations of 25, and 50 μg mL^{-1} (Breazeale and Camper 1972). Sulfometuron methyl (0.41 mM) was found more toxic to in vitro growth of *P. aeruginosa* than *P. fluorescens* (93% compared to 33% growth inhibition; Burnet and Hodgson 1991). Similarly, chlorsulfuron (0.56 mM) was of more toxicity to in vitro growth of *P. aeruginosa* than *P. fluorescens* (93% compared to 24% growth inhibition; Burnet and Hodgson 1991).

Effect of Herbicides Through Their Interactions with Pesticides

Herbicides interact with each other as well as with other pesticides. Here, we focus on the interactions of herbicides with other group of pesticides. Such interactions may lead to synergistic, additive, neutral, or antagonistic effects on the pathogens, vectors, as well as biological control agents. Therefore, these interactions can influence plant disease incidence and severity.

Effect of Herbicides Through Their Impacts on the Antifungal Activity of Fungicides

Herbicides may have enhancing impact on the antifungal activity of the fungicides applied in the chemical control of fungal plant diseases, and vice versa. The effect of two herbicides (paraquat and simazine on the antifungal activity of two fungicides (captan and mounsrin) against *Rhizoctonia solani* was studied. When the herbicides paraquat and simazine were applied to soil they altered the effectiveness of both fungicides in controlling *R. solani*, thus causing damping-off of cotton. Both herbicides increased the toxicity of both fungicides against mycelial growth of the fungus. In pot tests, seed/soil treatment with captan or mounsrin gave better control of *R. solani* damping-off disease when the soil treated with paraquat or simazine was compared to untreated soil. Captan was, however, found to be more effective in controlling the disease than mounsrin (Awadalla and El-Refaie 1994).

The herbicides fluchloralin and alachlor applied to soil altered the effectiveness of fungicide treatments to seed and soil for controlling cowpea damping-off. These herbicides also modified the in vitro toxicity of the fungicides to the mycelial growth of *Pythium butleri* and *Rhizoctonia solani* in a nutrient medium. Both herbicides reduced the toxicity of 2-methoxyethylmercury chloride (MEMC) and propamocarb to the growth of *P. butleri*, and of carbendazim to the growth of *R. solani*, but enhanced the toxicity of captafol and quintozene to *P. butleri* and *R. solani*, respectively. In pot tests, quintozene gave better control of *R. solani* damping-off in soil treated with fluchloralin or alachlor than in untreated soil, whereas disease control by carbendazim was decreased in similarly treated soils. Both herbicides attenuated the effectiveness of MEMC and captafol applied to control the damping-off caused by *P. butleri*. The efficacy of propamocarb was increased by alachlor but was decreased by fluchloralin. The implications of herbicide-fungicide interactions are discussed in the context of fungicidal control of root diseases in herbicide-treated soil (Kataria and Dodan 1982). In vitro studies have proved the influence of herbicides on the toxicity of fungicides. Ward (1984) found that glyphosate suppresses the anti-oomycotic activity of metalaxyl. Singh et al. (1999) indicated the synergistic effect of fluchloralin on the toxicity of matalaxyl against the oomycetous fungus-like *Phytophthora*, despite it reduced the fungicidal activity of captafol. Singh et al. (1999) indicated that two pre-emergence soil fungicides, fluchloralin and pendimethalin affected the in vitro toxicity of some fungicides against pigeaon pea blight pathogen, *Phytophthora drechsleri* f. sp. *cajani*. Both herbicides enhanced the antifungal activity of Apron® 35 WS, and Ridomil® MZ-72, while reduced the fungitoxicity of captafol. Moreover, when

combinations of the herbicides and pesticides were used, most of them (10 combinations) acted synergistically while only two combinations acted antagonistically. The researchers concluded that these herbicides can be mixed with these fungicides in order to efficiently achieve both herbicidal and fungicidal effect.

The combination of the fungicide cyproconazole and four herbicides DNOC (dinitro-ortho-cresol), dicamba, ioxynil and bromoxynil were tested singly as well as in mixtures against *Rhizoctonia cerealis* and *Pseudocercosporella herpotrichoides*, and cyproconazole-herbicide mixtures exhibited synergistic activity against both fungi in vitro as well as in bread wheat (*Triticum aestivum* L.), where the level of synergism depended on the relative proportion of fungicide and herbicide components in the mixture (Kataria and Gisi 1990).

Effect of Herbicides Through Their Impacts on the Insecticidal Activity of Insecticides

The search of literature for the interactive effect of herbicides and insecticides on insect pests did not lead to any result. The only study on the interactive effects of herbicides and insecticides have been carried out with the honey bee, *Apis mellifera*, where no additive/synergistic interaction was found with Advise® (58.6 mg a. i. L^{-1} imidacliprid) + Roundup® (1217.5 mg a. i. L^{-1} glyphosate). However, this does not mean the infeasibility of the incidence of herbicide-insecticide interactive effects on insect vectors of plant diseases, and there are reports on the direct entomotoxicity of at least some herbicides (Castilla et al. 2010).

Effect of Herbicides Through Their Impacts on the Nematicidal Activity of Nematicides

The effects of nematicides carbofuran and fenamiphos and the herbicides metribuzin and trifluralin, alone and in combination, on hatching, penetration, development, and reproduction of root knot nematode *Meloidogyne incognita* race 3 was studied under laboratory conditions. To study hatching, entire egg masses were exposed to nematicides (6 μg mL^{-1}), herbicides (0.5 μg mL^{-1}), and their combinations over a 16 day period. The hatched juveniles were extracted and counted every 24 h. Second-stage juveniles that hatched from day 6 to day 8 were used as inoculum to determine the effect of the chemicals on penetration, development, and reproduction of the nematode on tomato 4, 16, and 32 days after inoculation. Fenamiphos, alone and combined with either of the herbicides effectively inhibited hatching, therefore, it was not possible to get enough juveniles for post-infection development studies. However, carbofuran, trifluralin, metribuzin, carbofuran combined with either of the herbicides were of no impact on hatching, penetration, and development of females, or reproduction. Apparently, the herbicides were of no antagonistic effect on the nematicidal activity of both nematicides (Payan et al. 1987). Interestingly, while alachlor, vernolate, metribuzin, and trifluralin were of pathancing effects on the population development of *Heterodera glycines* when applied at recommended rates under field conditions (Bostian et al. 1986; Kraus

et al. 1982), metribuzin and trifluralin improved the efficacy of aldicarb, when the nematicide was combined with each of the herbicides (Kraus et al. 1982). The combined application of the herbicide alachlor with phenamiphos led to reduced efficacy of phenamiphos against the number of eggs of *H. glycines*. Also, linuron and alachlor synergistically increased the nematicidal activity of aldicarb, where application of the nematicide in bands was of higher impact than in furrow (Schmitt et al. 1983). In an experiment with sugarcane plants grown in two irrigated, and non-irrigated systems, no interaction was found between herbicides (diuron, ametrin, oxyfluorfen, and pendimethalin) and systemic nematicides (aldicarb, and terbufos) in the sense of reducing nematicide efficiency, except in an irrigated experiment for the number of stalks in herbicide treatments and the number of stems and stalks in nematode treatments. Nematicides and herbicides did not affect (Pol) and (PCC) levels at harvesting. However, a synergistic interaction between nematicide and herbicide was found with *Pratylenchus zeae* in the rhizosphere in irrigated system, where aldicarb and pendimethalin interactive effect brought down the nematode population to the lowest rate (Barros et al. 2006). While incorporation of Avadex® (40% diallate) into the soil at 3.5 L ha^{-1} just before planting led to high levels of soil infestation with *Heterodera schachtii* in sugar beet field, the combined application of the herbicide together with the nematicide Temik® 10 G (10% aldicarb) applied at 10 kg ha^{-1} in the furrow at planting time resulted in the greatest reduction in cyst production at all sampling dates ($P = 0.05$) and stabilized the enhanced effect of Temik® 10 G against sugar beet cyst nematode (Kraus and Sikora 1983). The pathancing effect of Avadex® has been attributed to its effect on the lipid layer of the nematode egg shell (Perry and Beane 1989), the presence of which was demonstrated by Perry and Trett (1986).

4 The Share of Herbicides in the Integrated Plant Disease Management

There are two point of views in the control of plant diseases. One emphasizes on the control of a disease on a plant species, and the other intends to control major diseases of a plant in an area (Singh 2001). Here, a third view point is introduced, that is to control of the diseases of crop plants that are grown in rotation in a given area. While the previously introduced viewpoints focus on a crop, the new viewpoint focus on the disease of the crops in a given altering agroecosystem wherein different crops are grown in rotation. With an eye to what mentioned above, there remains no doubt that herbicides, if intelligently chosen, can play an importantly great role in the integrated plant disease management programs. With enough ecological and toxicological information on the consequences of a special herbicide application in an agricultural program, it will be possible to control weeds of the grown crop in field and in the mean time, to reduce disease risks imposed by different pathogens, and the population of vectors through the removal of their wild

host plants. Herbicides can be selected in a manner that reduce the inocula of the pathogens expected to be problematic for the current and/next crop due to be grown in a given rotation program. All these will help farmers to reduce their costs for the production of agricultural products, and will provide a way to diminish the probability of the rapid emergence of resistant pathogenic populations, and to reduce the need for more repeated application of antimicrobials. Herbicides with a mode of action different from that of fungicides can help in the decrease of fungal pathogen population and the risk of fungicide-resistance development. The herbicides of chemical structures other than those of antimicrobials are expected to be useful in the control of the pathogenic microbes adapted to the pesticides (in its general meaning) due to the increased activity of the plasma membrane transporters. It seems possible to decrease population of some important pathogens through the intelligent application of well-selected herbicides against weeds.

The formulation/and co-application of the herbicides together with the biological control agents not only will help to control weeds but also will aid plant residue management for pest and disease risk diminishment as well as recyclization of the nutrients and their re-incorporation into soil environment for the preservation of soil fertility. The application of mycoherbicides and other biological products may help to the control of the plant diseases caused by the pathogenic microorganisms of close taxonomic relationship, although their mixed application with chemical herbicides will ensure higher control output against resistance development in weeds. The herbicides effective against necrotrophic pathogens but still allowing the bioactivity of biological control agents are suitable for crop residue management. Alternatively, such herbicides can be co-formulated/mixed with proper fungicides that are more toxic to pathogens than biological control agents. However, more studies are required in this neglected research area, and a research performed with imazapic and the biocontrol fungus *Pyrenophora semeniperda* in order to control winter annual grass, *Bromus tectorum* (Ehlert et al. 2014) well indicates the necessity of further information required for the integrated control of weeds. The compatibility of herbicidal formulation and biological control agents, the timing as well as method of application, the host range of the biological control agent are among important factors that determine the result. Herbicides effective against pathogens or pests are of advantage and can lead to reduced control costs and environmental damages such as soil erosion. Such a view point is expected to have an effective impact in the subsequent conversion of disease conducive soils to disease suppressive and or ideally composite soils, as has been discussed before (Higa and Parr 1994).

With the herbicides like glyphosate, there are strategies to ameliorate their predisposing effects on disease. These strategies include judicious selection of herbicide application rates, micro-nutrient amendment, glyphosate detoxification in meristematic tissues and soil, changes in cultural practices to enhance micro-nutrient availability for plant uptake, and biological amendment with GR microbes for nitrogen fixation and nutrient availability. Given that the recommended doses of glyphosate are often many times higher than that needed to control weeds, it is believed that 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. Toxicity of glyphosate to Mn-reducing and synergistic nitrogen-fixing organisms in the rhizosphere can have serious consequences for sustainability of legume production. Regular inoculation of legume crops with synergistic nitrogen-fixing organisms may be required in many areas for maximal productivity where extended applications of glyphosate have eliminated them from the soil profile. Development of glyphosate-tolerant nitrogen-fixing and Mn-reducing organisms would be beneficial in many of these situations, and especially for perennial Roundup Ready® legume crops such as alfalfa (Huber et al. 2004).

There is little information on the precise effects of herbicides on the pathogens, biological control agents, and their interactions under in vitro, in vivo, and *in soilum* conditions. Such information is very essential for effective integrated management of plant diseases as well as for the companies that intend to continuously improve their products in the world of increasing concerns, knowledge, and competitive marketing intensified by continuous decrease of agricultural lands due to increasing population and climatic changes. It is a global need to not only preserve the current fertility of agricultural lands but also increase the fertility of the ever-decreasing available lands, and keep them free of pollutants. Finally, I want here to finish this

Fig. 64 An old Indian proverb that well indicates the way we should take for future (*Source* http://www.geckoandfly.com/)

chapter with an Indian proverb (Fig. 64) once I knew it by a poster on the wall in the office room in Martin Luther University, Halle-Wittenberg, Halle (Saale), Germany.

References

Abbas Z, Akmal M, Saifullah Khan K, Fayyaz-ul-Hassan (2014) Effect of Buctril Super (bromoxynil) herbicide on soil microbial biomass and bacterial population. Braz Arch Biol Technol 57:9–14

Abd-Alla MH, Shukry AO, Sokol K (2000) The impact of pesticides on arbuscular mycorrhizal and nitrogen-fixing symbioses in legumes. Appl Soil Ecol 14:191–200

Abdel-Aty AM, El-Dib MA (2016) Changes in the community structure and growth of fresh water microalgae as a consequence of diuron exposures. Int J Chem Tech Res 9:114–122

Abdel-Mageed A, Areej AAS, Eman A, Sholkamy E (2013) In vitro detection of herbicide-tolerant fungi isolated from pesticide polluted soil. J Agric Sci and Technol A 3:960–972

Abd-El-Malik Y (1971) Free-living nitrogen-fixing bacteria in Egyptian soils. In: Lie TA, Mulder G (eds) Biological nitrogen fixation in natural and agricultural habitats. Nijoff, The Hague, pp 442–459

Abd El-Moniem E, Abd-Allah ASE (2008) Effect of green alga cells extract as foliar spray on vegetative growth, yield and berries quality of superior grapevine. Am Eurasian J Agric Environ Sci 4:427–433

Adams MJ, Jacquier C (1994) Infection of cereals and grasses by isolates of *Polymyxa graminis* (Plasmodiophorales). Ann Appl Biol 125:53–60. https://doi.org/10.1111/j.1744-7348.1994.tb04946.x

Adeleye IA, Okorodudu E, Lawal O (2004) Effect of herbicides used in Nigeria on *Rhizobium phaseoli*, *Azotobacter vinelandii* and *Bacillus subtilis*. J Environ Biol 25:151–156

Agrios GN (2005) Plant pathology. Elsevier Academic Press, Burlington, MA

Aguirre-Cadena JF, Téllez SR, Cuautle M, Aguirre-Medina JF (2014) Survival of *Azospirillum brasilense* after applying herbicides on *Triticum aestivum* L. Var. Altiplano. Rev Mex Cienc Agríc 5:1549–1555

Ahluwalia AS (1988) Influence of Saturn and knockweed on the growth and heterocyst formation in nitrogen-fixing blue-green alga. Pestic 22:43–44

Ahmad I, Malloch D (1995) Interaction of soil microflora with the bioherbicide phosphinothricin. Agric Ecosyst Environ 54:165–174

Ahn IP (2008) Glufosinate ammonium-induced pathogen inhibition and defense responses culminate in disease protection in *bar*-transgenic rice. Plant Physiol 146:213–227

Albert M, Belastegui-Macadam X, Bleischwitz M, Kaldenhoff R (2008) *Cuscuta* spp.: parasitic plants in the spotlight of plant physiology, economy, and ecology. Prog Bot 69:267–277. https://doi.org/10.1007/978-3-540-72954-9-11

Albrecht M, Kortekamp A (2009) The in vitro effect of the herbicide Basta® (Glufosinate ammonium) on potential fungal grapevine pathogens. Eur J Hortic Sci 74:112–117

Alcántara-de la Cruz R, Zanuncio JC, Lacerda MC, Wilcken CF, Fernandes FL, de Souza Tavares W, Soares MA, Sediyama CS (2017) Side-effects of pesticides on the generalist endoparasitoid *Palmistichus elaeisis* (Hymenoptera: Eulophidae). Sci Rep 7:e10064. https://doi.org/10.1038/s41598-017-10462-3

Alexander HM, Holt RD (1998) The interaction between plant competition and disease. Perspect Plant Ecol Evol Syst 1:206–220. https://doi.org/10.1078/1433-8319-00059

Allen MF (1991) The ecology of mycorrhizae. Cambridge University Press, Cambridge

Allievi L, Gigliotti C (2001) Response of the bacteria and fungi of two soils to the sulfonylurea herbicide cinosulfuron. J Environ Sci Health B 36:161–175. https://doi.org/10.1081/PFC-100103741

Althaf HS, Srinivas P (2013) Evaluation of plant growth promoting traits by *Pseudomonas* and *Azotobacter* isolated from rhizotic soils of two selected agroforestry tree species of Godavari belt region, India. Asian J Exp Biol Sci 4:431–436

Altieri M (1994) Biodiversity and pest management in agroecosystems. Haworth Press, New York

Altman J (1972) Increased glucose exudate and damping-off in sugar beets in soils treated with herbicides. Phytopathol 62:743

Altman J (1991) Herbicide-pathogen interaction in plant disease. Pestic Outlook 2:17–21

Altman J (1993) Pesticide interactions in crop production: beneficial and deleterious effects. CRC Press, Boca Raton

Altman J, Campbell CL (1977a) Effect of herbicides on plant diseases. Annu Rev Phytopathol 15:361–385

Altman J, Campbell CL (1977b) Pesticide-plant disease interactions. Effects of cycloate on sugar beet damping-off induced by *Rhizoctonia solani*. Phytopathol 67:1163–1165

Altman J, Ross M (1967) Plant pathogens as a possible factor in unexpected preplant herbicide damage in sugarbeets. Plant Dis Rep 51:86–88

Altman J, Rovira AD (1989) Herbicide-pathogen interactions in soil-born root diseases. Can J Plant Pathol 11:166–172

Amla DV, Kochhar VK (1982) Physiological and genetic effects on some common phenylalkylureas on nitrogen fixing cyanobacterium *Nostoc muscorum*. In: Proceedings of the national symposium on biological nitrogen fixation, IARI, New Delhi, p 606 (abstract)

Ananthakrishnan TN (1971) Thrips in agriculture, horticulture and forestry-diagnosis, bionomics and control. Pak J Sci Ind Res 30:113–146

Anderson A (2001) The effects of acetolactate synthase (ALS) inhibiting herbicides on the growth, yield, nodulation and nitrogen fixation of selected legumes, a thesis submitted for the degree of Doctor of Philosophy in the Faculty of Agricultural and Natural Resource Sciences at Adelaide University. Department of Agronomy and Farming Systems, Roseworthy Campus, Adelaide University, Sept 2001. http://digital.library.adelaide.edu.au/dspace/handle/2440/61244

Anderson JA, Kolmer JA (2005) Rust control in glyphosate tolerant wheat following the application of the herbicide glyphosate. Plant Dis 88:1136–1142

Anderson RM, May RM (1991) Infectious diseases of humans: dynamics and control. Oxford University Press, Oxford

Anderson A, Baldock JA, Rogers SL, Bellotti W, Gill G (2004) Influence of chlorsulfuron on rhizobial growth, nodule formation, and nitrogen fixation with chickpea. Aust J Agric Res 55:1059–1070

Andreev VP, Maslov YI (1988) Adaptation to diuron by the cyanobacterium *Anabaena variabilis* Fd. During long periods of cultivation. Vestn Leningr Univ Biol 0:71–75

Araújo ASF, Monteiro RTR, Abakerli RB (2003) Effect of glyphosate on the microbial activity of two Brazilian soils. Chemosphere 52:799–804

Arfarita N, Imai T, Kanno A, Higuchi T, Yamamoto K, Sekine M (2011) Screening of soil-born fungi from forest soil using glyphosate herbicide as the sole source of phosphorus. J Water Environ Technol 9:391–400

Aristilde L, Reed ML, Wilkes RA, Youngster T, Kukurugya MA, Katz V, Sasaki CRS (2017) Glyphosate-induced specific and widespread perturbations in the metabolome of soil *Pseudomonas* species. Front Environ Sci 5:34. https://doi.org/10.3389/fenvs.2017.00034

Awadalla OA, El-Refaie IM (1994) Effect of herbicides on the toxicity of fungicides against *Rhizoctonia solani* causing damping-off of cotton. J Phytopathol 140:187–192

Azcorn R, Barea JM (1975) Synthesis of auxins, gibberellins and cytokinins by *Azotobacter vinelandii* and *Azotobacter beijerinckii* related to effects produced on tomato plants. Plant Soil 43:609–619

Balajee S, Mahadevan A (1990) Influence of chloroaromatic substances on the biological activity of *Azotobacter chroococcum*. Chemosphere 21:51–56

Balandreau J (1986) Ecological factor and adaptive processes in N_2-fixing bacteria populations of the plant environment. Plant Soil 90:73–93

Baltensperger AA, Schank SC, Smith RL, Littell RC, Bouton JH, Dudeck AE (1978) Effect of inoculation with *Azospirillum* and *Azotobacter* on tuff-type Bermuda genotypes. Crop Sci 18:1043–1045. https://doi.org/10.2135/cropsci1978.0011183X010800180035x

Bandeen JD, Buchholtz K (1967) Competitive effects of quackgrass upon corn as modified by fertilization. Weeds 15:220–227

Barka EA, Vatsa P, Sanchez L, Gaveau-Vaillant N, Jacquard C, Klenk HP, Clément C, Ouhdouch Y, van Wezel GP (2016) Taxonomy, physiology, and natural products of *Actinobacteria*. Microbiol Mol Biol Rev 80:1–43. https://doi.org/10.1128/MMBR.00019-15

Barnes JP, Putnam AR, Burke BA (1986) Allelopathic activity of rye (*Secale cereale* L.). In: Putnam AR, Tang CS (eds) The science of allelopathy. Wiley, New York, pp 271–286

Barros ACB, Moura RM, Pedrosa E (2006) Studies on the effect of simultaneous application of herbicide and systemic nematode on nematicide efficacy in sugarcane fields. Fitopathol Bras 31:291–296

Bashan Y, de-Bashan LE (2010) How the plant growth-promoting bacterium *Azospirillum* promotes plant growth—a critical assessment. Adv Agron 108:77–136

Bashan Y, Holguin G (1997) *Azospirillum*-plant relationships: environmental and physiological advances (1990–1996). Can J Microbiol 43:103–121

Bashan Y, Ream Y, Levanony H, Sade A (1989) Nonspecific responses in plant growth, yield, and root colonization of noncereal crop plants to inoculation with *Azospirillum brasilense* Cd. Can J Bot 67:1317–1324

Becquer A, Torres-Aquino M, Le Guernevé C, Amenc LK, Trives-Segura C, Staunton S, Quiquampoix H, Plassard C (2017) Establishing a symbiotic interface between cultured ectomycorrhizal fungi and plants to follow fungal phosphate metabolism. Bio-Protocol 7:2577. https://doi.org/10.21769/BioProtoc.2577

Bennett CW (1940) Acquisition and transmission of viruses by dodder (*Cuscuta subinclusa*). Phytopathol 30:2 (abstract)

Bennett CW (1944) Latent virus of dodder and its effect on sugar beet plants. Phytopathol 34:77–91

Bera S, Ghosh R (2013) Soil microflora and weed management as influenced by Atrazin 50% WP in sugarcane. Univers J Agric Res 1:41–47

Bernards ML, Thelen KD, Muthukumaran RJ, McCracken JL (2005) Glyphosate interaction with manganese in tank mixtures and its effect on glyphosate absorption and translocation. Weed Sci 53:787–794

Berner DK, Berggren GT, Snow JP (1991) Effects of glyphosate on *Calonectria crotalariae* and red crown rot of soybean. Plant Dis 75:809–813

Bertholet J, Clark W (1985) Effect of trifluralin and metribuzin on faba bean growth, development and symbiotic nitrogen fixation. Can J Plant Sci 65:9–20

Bethlenfalvay GJ, Schreiner RP, Mihara KL, McDaniel H (1996) Mycorrhizae, biocides, and biocontrol. 2. Mycorrhizal fungi enhance weed control and crop growth in a soybean-cocklebur association treated with the herbicide bentazon. Appl Soil Ecol 3:205–214

Bhat MA, Tsuda M, Horiike K, Nozaki M, Vaidyanathan CS, Nakazawa T (1994) Identification and characterization of a new plasmid carrying genes for the degradation of 2,4-dichlorophenoxyacetate from *Pseudomonas cepacia* CSC90. Appl Environ Microbiol 60:307–312

Bhoraniya MF, Khandar RR, Khunti JP (2002) Evaluation of pesticides against *Sclerotium rolfsii* on chilli by soil plate technique. Plant Dis Res 17:145–146

Black BD, Russin JS, Griffin JL, Snow JP (1996) Herbicide effects on *Rhizoctonia solani* in vitro and rhizoctonia foliar blight of soybean (*glycine max*). Weed Sci 44:711–716

Blackwell Publishing Ltd (2007) First all-African GM crop is resistant to maize streak virus. ScienceDaily. 16 Aug 2007. http://www.sciencedaily.com/releases/2007/08/070815105030.htm/

Blowes WM (1987) Effect of ryegrass root residues, knock-down herbicides, and fungicides on the emergence of barley in sandy soils. Aust J Exp Agric 27:785–790

Boland GJ, Hall R (1994) Index of plant hosts of *Sclerotinia sclerotiorum*. Can J Plant Pathol 16:93–108

Bosque-Perez NA (2000) Eight decades of maize streak virus research. Virus Res 71:107–121. https://doi.org/10.1016/S0168-1702(00)00192-1

Bostian AL, Schmitt DP, Barker KR (1986) In vitro hatch and survival of *Heterodera glycines* as affected by alachlor and phenamiphos. J Nematol 18:22–26

Bott S, Tesfamariam T, Candan H, Cakmak I, Römheld V, Neumann G (2008) Glyphosate-induced impairment of plant growth and micronutrient status in glyphosate-resistant soybean (*Glycine max* L.). Plant Soil 312:185–194

Boyette CD, Reddy KN, Hoagland RE (2006) Glyphosate and bioherbicide interaction for controlling kudzu (*Pueraria lobata*), redvine (*Brunnichia ovata*), and trumpet creeper (*Campsis radicans*). Biocontrol Sci Technol 16:1067–1077

Bramhall RA, Higgins VJ (1988) The effect of glyphosate on resistance of tomatoto fusarium crown and root rot disease and on the formation of host structural defensive barriers. Can J Bot 66:1547–1555

Brandes W, Heitefuss R (1971) Nebenwirkung von Herbiziden auf *Erysiphe graminis* und *Cercosporella herpotrichoides* an Weizen. II. Physiologische und biochemische Ursachen des veränderten Befalls der Pflanze. J Phytopathol 72:34–52

Breazeale FW, Camper ND (1972) Effect of selected herbicides on bacterial growth rates. Appl Microbiol 23:431–432. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC380360

Bretschneider T, Fischer R, Nauen R (2007) Inhibitors of lipid synthesis (acetyl-CoA-carboxylase inhibitors). In: Krämer W, Schrmer U (eds) Modern crop protection compounds, vol 3. Willey-VCH Verlag GmbH & Co. KGaA, Weinheim, pp 909–926

Bromilow RH, Chamberlain K, Tench AJ, Williams RH (1993) Phloem translocation of strong acids—glyphosate, substituted phosphonic, and sulfonic acids—in *Ricinus communis* L. Pestic Sci 37:39–47

Brunt A, Crabtree K, Dallwitz M, Gibbs A, Watson L (1996) Viruses of plants: descriptions and lists from the VIDE database. CAB International, UK

Buechel T, Bloodnick E (2016) Mycorrhizae: description of types, benefits and uses. Plant Health. http://www.gpnmag.com/article/mycorrhizae-description-of-types-benefits-anduses/

Bulegon LG, Inagaki AM, Moratelli G, da Costa NV, Guimarães VF (2017) Fitotoxidez de mesotriona em milho inoculado com *Azospirillum brasilense* associado a adubação nitrogenada. Braz J Agric Sci 12:325–331. https://doi.org/10.5039/agrarian.v12i3a5459

Burdon JJ, Chilvers GA (1982) Host density as a factor in plant disease ecology. Annu Rev Phytopathol 20:143–166

Burnet M, Hodgson B (1991) Differential effects of sulfonylurea herbicides chlorsulfuron and sulfometuron methyl on microorganisms. Arch Microbiol 155:521–525. https://doi.org/10.1007/BF00245344

Burns ER, Buchanan GA, Carter M (1971) Inhibition of carotenoid synthesis as a mechanism of action of amitrole, dichlormate, and pyriclor. Plant Physiol 47:144–148. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC365828/

Busse MD, Fiddler GO, Ratcliff AW (2004) Ectomycorrhizal Formation in Herbicide-Treated Soils of Differing Clay and Organic Matter Content. Water Air Soil Poll 152(1–4):23–34

Cameron DD (2010) Arbuscular mycorrhizal fungi as (agro)ecosystem engineers. Plant Soil 333:1–5

Campos DTS, da Silva MCS, da Luz JMR, Telesfora RJ, Kasuya MCM (2011) Colonização micorrízica em plantios de eucalipto. Rev Árvore 35:965–974

Canaday CH, Helsel DG, Wyllie TD (1986) Effects of herbicide-induced stress on root colonization of soybeans by *Macrophomina phaseolina*. Plant Dis 70:863–866

Cárdenas DM, Garrido MF, Bonilla RR, Baldani VL (2010) Aislamiento e identificación de cepas de *Azospirillum* sp. En pasto guinea (*Panicum maximum* Jacq.) del Valle del Cesar. Pastos Forrajes 33:285–300

Carmo EL, Bueno AF, Bueno RCOF, Vieira SS, Gobbi AL, Vasco FR (2009) Seletividade de diferentes agrotóxicos usados na cultura da soja ao parasitoide de ovos *Telenomus remus*. Cienc Rural 39:2293–2300

Carson ML, Arnold WE, Todt PE (1991) Predisposition of soybean seedlings to fusarium roo rot with trifluralin. Plant Dis 75:342–347

Casale WL, Hart LP (1986) Influence of four herbicides on carpogenic germination and apothecium development of *Sclerotinia sclerotiorum*. Phytopathol 76:980–984

Castilla AM, Dauwe T, Mora I, Malone J, Guitart R (2010) Nitrates and herbicides cause higher mortality than the traditional organic fertilizers on the grain beetle, *Tenebrio molitor*. Bull Environ Contam Toxicol 84:101–105. https://doi.org/10.1007/s00128-009-9883-5

Caulder JD, Gotlieb AR, Stowell AR, Towell L, Watson AK (1987) Herbicidal compositions comprising microbial herbicides and chemical herbicides or plant growth regulators. Eur Pat Appl. http://patft.uspto.gov/netacgi/nph-Parser?Sect2=PTO1&Sect2=HITOFF&p=1&u=/netahtml/PTO/search-bool.html&r=1&f=G&l=50&d=PALL&RefSrch=yes&Query=PN/4808207

Cave RD (2000) Biology, ecology and use in pest management of *Telenomus remus*. Biocontrol 21:21–26

Cerdeira AL, Duke SO (2006) The current status and environmental impacts of glyphosate-resistant crops: a review. J Environ Qual 35:1633–1658

Chan Cupul W, Heredia Abarca G, Rodríguez Vázquez R, Salmones D, Gaitán Hernández R, Alarcón Gutiérrez E (2014) Response of lignolytic macrofungi to the herbicide atrazine: dose-response bioassays. Rev Argent Microbiol 46:348–357

Chandler JM, Santelman PW (1968) Interactions of four herbicides with *Rhizoctonia solani* on seedling cotton. Weed Sci 16:453–456

Chattopadhyay SB, Chakrabarti NK (1953) Occurrence in nature of an alternative host (*Leersia hexandra* Sw.) of *Helminthosporium oryzae* Breda de Haan. Nature 172:550

Chen X, Han H, Jiang P, Nie L, Fan P, Lv S, Feng J, Li Y (2011) Transformation of β-lycopene cyclase genes from *Salicornia europaea* and arabidopsis conferred salt tolerance in arabidopsis and tobacco. Plant Cell Phyiol 52:909–921. https://doi.org/10.1093/pcp/pcr043

Cheng MW (2005) Manganese transition states during infection and early pathogenesis in rice blast. M.S. thesis. Purdue University, West Lafayette, IN

Chennappa G, Adkar-Purushothama CR, Naik MK, Sreenivasa MY (2014) Impact of pesticide on PGPR activity of *Azotobacter* sp. isolated from pesticide flooded paddy soils. Greener J Agric Sci 4:117–129. https://doi.org/10.15580/GJAS.2014.4.010314003

Chittenden FH (1919) Control of onion thrips. USDA Farm Bull 1007:16

Cohen R, Riov J, Lisker N, Katan J (1986) Involvement of ethylene in herbicide-induced resistance to *Fusarium oxysporum* f. sp. *melonis*. Phytopathol 76:1281–1285

Cohen R, Blair B, Katan J (1992a) Chloroacetamide herbicides reduce incidence of *Fusarium* wilt in melons. Crop Prot 11:181–185

Cohen R, Cuppels DA, Brammall RA, Lazarovits G (1992b) Induction of resistance towards bacterial pathogens of tomato by exposure of the host to dinitroaniline herbicides. Phytopathol 82:110–114

Cohen R, Blaier B, Schaffer AA, Katan J (1996) Effect of acetochlor treatment on fusarium wilt and sugar content in melon seedlings. Eur J Plant Pathol 102:45–50

Cole H, MacKenzie DR, Ercegovich CD (1968) Maize dwarf mosaic-interactions between virus-host-soil pesticides for certain inoculated hybrids in Pennsylvania field plantings, SEries 1. Main effects of virus and chemicals on yield. Plant Dis Rep 52:545–549

Cole H, MacKenzie DR, Boyle JS, Ercegovich CD (1969a) Maize dwarf mosaic-effects of virus strains A and B on yield and interaction effects of atrazine and the virus on yield. Plant Dis Rep 53:340–344

Cole H, MacKenzie DR, Smith CB, Ercegovich CD (1969b) Influence of maize dwarf mosaic virus and trace levels of atrazine on weight and foliar element accumulation by two single cross maize hybrids in the greenhouse. Plant Dis Rep 53:652–655

Combier JP, Melayah D, Raffier C, Gay G, Marmeisse R (2003) *Agrobacterium tumefaciens* mediated transformation as a tool for insertional mutagenesis in the symbiotic ectomycorrhizal fungus *Hebeloma cylindrosporum*. FEMS Microbiol Lett 220:141–148. https://doi.org/10.1016/S0378-1097(03)00089-2

Combier JP, Melayah D, Raffier C, Pépin R, Marmeisse R, Gay G (2004) Non mycorrhizal (Myc^{-}) mutants of *Hebeloma cylindrosporum* obtained through insertional mutagenesis. Mol Plant Microbe Interact 17:1029–1038. https://doi.org/10.1094/MPMI.2004.17.9.1029

Craciun MA, Borozan AB, Bordean DM, Verdes D, Popescu R (2013) The influence of herbicides on fungus communities from soil. Ann Rom Soc Cell Biol 18:164–169

Crowley DE, Rengel Z (1999) Biology and chemistry of nutrient availability in the rhizosphere. In: Rengel Z (ed) Mineral nutrition of crops: fundamental mechanisms and implications. Food Products Press, London, pp 1–40

Cruz I, Figueiredo MLC, Oliveira CE, Vasconcelos CA (1999) Damage of *Spodoptera frugiperda* (Smith) in different maize genotypes cultivated in soil under three levels of aluminium saturation. Int J Pest Manag 45:293–296

Culdlin P, Mejstrik V, Skoupy J (1983) Effects of pesticides on ectomycorrhizae of *Pinus sylvestris* seedlings. Plant Soil 17:353–361

Dallwitz MJ (1980) A general system for coding taxonomic descriptions. Taxon 29:41–46

Dallwitz MJ, Paine TA, Zurcher EJ (1993) User's guide to the DELTA system: a general system for processing taxonomic descriptions, 14th edn. CSIRO Division of Entomology, Canberra

Dann EK, Diers BW, Hammerschmidt R (1999) Suppression of sclerotinia stem rot of soybean by lactofen herbicide treatment. Phytopathol 89:598–602

Dart PJ (1986) Nitrogen fixation associated with non-legumes in agriculture. Plant Soil 90: 303–334

Das AC, Nayek H, Chakravarty A (2012) Environ Monit Assess 184:7453. https://doi.org/10.1007/s10661-011-2512-x

DaSilva EJ, Henriksson LE, Henriksson E (1975) Effect of pesticides on blue-green algae and nitrogen-fixation. Arch Environ Contam Toxicol 3:193–204

Daszak P, Cunningham AA, Hyatt AD (2000) Emerging infectious diseases of wildlife: threats to biodiversity and human health. Science 287:443–449

Daugrois JH, Hoy JW, Griffin JL (2005) Protoporphyrinogen oxidase inhibitor effects on pythium root rot of sugarcane, *Pythium* species, and the soil microbial community. Phytopathol 95: 220–226

Dawson JH, Musselman LJ, Wolswinkel P, Dörr I (1994) Biology and control of *Cuscuta*, Musselman. Rev Weed Sci 6:265–317

Debaud JC, Gay G (1987) In vitro fruiting under controlled conditions of the ectomycorrhizal fungus *Hebeloma cylindrosporum* associated with *Pinus pinaster*. New Phytol 105:429–435. https://doi.org/10.1111/j.1469-8137.1987.tb00880.x

Debona AC (1967) Studies on the effects ofsome herbicides on soil nitrification, Ph.D. thesis. University of London

Debona AC, Audus LJ (1970) Studies on the effects of herbicides on soil nitrification. Weed Res 10(3):250–263

Dejonghe W, Goris J, Fantroussi S, Hofte M, de Vos P, Verstraete W, Top EM (2000) Effect of dissemination of 2,4-dichlorophenoxyacetic acid (2,4-D) degradation plasmids on 2,4-D degradation and on bacterial community structure in two different soil horizons. Appl Environ Microbiol 66:3297–3304

Del Gallo M, Fabbri P (1990) Inoculation of *Azospirillum brasilense* Cd on chickpea (*Cicer arietinum*). Symbiosis 9:283–287

Del Sorbo G, Schoonbeek HJ, De Waard M (2000) Fungal transporters involved in efflux of natural toxic compounds and fungicides. Fungal Genet Biol 30:1–15

Desjardins PR, Drake RJ, French JV (1969) Transmission of citrus ringspot virus to citrus and non-citrus hosts by dodder (*Campestris subinclusa*). Plant Dis Rep 53:947–948

Diaz-Montano J, Fuchs M, Nault BA, Fail J, Shelton AM (2011) Onion thrips (Thysanoptera: Thripidae): a global pest of increasing concern in onion. J Econ Entomol 104:1–13. https://doi.org/10.1603/EC10269

Díaz-Zorita M, Fernández-Canigia MV (2009) Field performance of a liquid formulation of *Azospirillum brasilense* on dryland wheat productivity. Eur J Soil Biol 45:3–11

Dissanayake N, Hoy JW, Griffin JL (1998) Herbicide effects on sugarcane growth, pythium root rot, and *Pythium arrhenomanes*. Phytopathol 88:530–535

Dixon RA, Achnine L, Kota P, Liu CJ, Reddy MS, Wang L (2002) The phenylpropanoidpathway and plant defense—a genomics perspective. Mol Plant Pathol 3:371–390

Döbereiner J, Pedrosa FO (1987) Nitrogen-fixing bacteria in nonleguminous crop plants. Sciencia Tech Publishers, Madison

Dobson AP (2004) Population dynamics of pathogens with multiple host species. Am Nat 164: S64–S78

Dodge AD (1975) Some mechanisms of herbicide action. Sci Prog 62:447–466

Doederlein TA, Sites RW (1993) Host plant preferences of *Frankliniella occidentalis* and *Thrips tabaci* (Thysanoptera: Thripidae) for onion and associated weeds on the southern high plains. J Econ Entomol 86:1706–1713. https://doi.org/10.1093/jee/86.6.1706

Donnelly PK, Fletcher JS (1994) Potential use of mycorrhizal fungi as bioremediation agents. In: Anderson TA, Coats JR (eds) Bioremediation through rhizosphere technology, ACS symposium series. American Chemistry Society, Washington DC, pp 93–99 (Chapter 8). http://dx.doi.org/10.1021/bk-1994–0563.ch008

Donnelly PK, Entry JA, Crawford DL (1993) Degradation of atrazine and 2,4-dichlorophenoxyacetic acid by mycorrhizal fungi at three nitrogen concentrations in vitro. Appl Environ Microbiol 59:2642–2647

Doumbou CL, Hamby Salove MK, Crawford DL, Beaulieu C (2001) Actinomycetes, promising tools to control plant diseases and to promote plant growth. Phytoprotection 82:85–102. https://doi.org/10.7202/706219ar

Drescher N, Otto S (1973) Ober den Abbau von Bentazon im Boden. Mitt Biol Bundes Anst Land Forstwirtsch 151:181

Drew EA, Gupta VVSR, Roget DK (2007) Herbicide use, productivity, and nitrogen fixation in field pea (*Pisum sativum*). Aust J Agric Res 58:1204–1214. https://doi.org/10.1071/ar06394

Druille M, Cabello MN, Omacini M, Golluscio RA (2013) Glyphosate reduces spore viability and root colonization of arbuscular mycorrhizal fungi. Appl Soil Ecol 64:99–103

Duke SO, Cerdeira L (2005) Potential environmental impacts of herbicide-resistant crops. Collect Biosaf Rev 2:66–143

Duke SO, Cedergreen N, Velini ED, Belz RG (2006) Hormesis: is it an important factor in herbicide use and allelopathy. Outlook Pest Manag 17:29–33

Duke SO, Wedge DE, Cerdeira AL, Matallo MB (2007) Herbicide effects on plant disease. Outlooks Pest Manag 36–40, http://dx.doi.org/10.1564/18feb13

Duncan DR, Paxton JD (1981) Trifluralin enhancement of phytophthora root rot of soybean. Plant Dis 65:435–436

Durgesha M (1994) Effect of dinitroaniline herbicides on rhizobia, nodulation and $N_2(C_2H_2)$ fixation of four groundnut cultivars. Ann Appl Biol 124:75–82. https://doi.org/10.1111/j.1744-7348.1994.tb04117.x

Eberbach PL, Douglas LA (1989) Herbicide effects on the growth and nodulation potential of *Rhizobium trifolii* with *Trifolium subterraneum* L. Plant Soil 119:15–23. https://doi.org/10.1007/BF00010421

Ehlert KA, Mangold JM, Engel RE (2014) Integrating the herbicide imazapic and the fungal pathogen *Pyrenophora semeniperda* to control *Bromus tectorum*. Weed Res 54:418–424. https://doi.org/10.1111/wre.12089

Eker S, Ozturk L, Yazici A, Erenoglu B, Romheld V, Cakmak I (2006) Foliar applied glyphosate substantially reduced uptake and transport of iron and manganese in sunflower (*Helianthus annuus* L.) plants. J Agric Food Chem 54:10019–10025

El-Ghamry AM, Huang CY, Xu JM (2000) Influence of chlorsulfuron herbicide on size of microbial biomass in soil. J Environ Sci 12:138–142

El-Khadem M, Papavizas GC (1984) Effect of the herbicides EPTC and linuron on cotton diseases caused by *Rhizoctonia solani* and *Fusarium oxysporum* f. sp. *vasinfectum*. Plant Pathol 33:411–416

El-Khadem M, Zahran M, El-Kazzaz MK (1979) Effect of the herbicides trifluralin, dinitamine and fluometuron on Rhizoctonia disease in cotton. Plant Soil 51:463–470

El-Khadem M, El-Kazzaz MK, Hassan MA (1984) Influence of different pre-emergence herbicides on cotton diseases caused by *Rhizoctonia solani* and *Fusarium oxysporum* f. sp. *vasinfectum*. Plant Soil 79:29–36

El-Shanshoury A, El-Raheem R, Abu El-Sououd SM, Awadalla OA, El-Bandy NB (1995) Formation of tomatine in tomato plants infected with *Streptomyces* species and treated with herbicides, correlated with reduction of *Pseudomonas solanacearum* and *Fusarium oxysporum* f. sp. *lycopersici*. Acta Microbiol Pol 44:255–266

Elstner EF, Stoffer C, Heupel A (1975) Determination of superoxide free radical ion and hydrogen peroxide as products of photosynthetic oxygen reduction. Z Naturforsch 30:53–56

Elton CS (1958) The ecology of invasions by animals and plants. Methuen & Co., London

Erwin DC, Ribeiro OK (1996) Phytophthora diseases worlwide. American Phytopathological Society, St Paul

Eshel Y, Katan J (1972) Effect of time of application of diphenamid on pepper weeds and disease. Weed Sci 20:468–471

Estok D, Freedman B, Boyle D (1989) Effects of the herbicides 2,4-D, glyphosate, hexazinone, and triclopyr on the growth of three species of ectomycorrhizal fungi. Bull Environ Contam Toxicol 42:835–839

Fajemisin JM (2003) Overview of maize viruses in sub-Saharan Africa. In: Hughes JH, Odu J (eds) Plant virology in sub-Saharan Africa. International Institute of Tropical Agriculture Conference, IIesa, Nigeria, pp 158–171

Farajzadeh EM, Yarnia M, Rezaei F (2006) Evaluation of allelopathical effects of *Chenopodium album*, *Amaranthus retroflexus*, and *Cynodon dactylon* on germination and seedling growth of soybean. In: Nezamabadi N (ed) Proceedings of the 17th Iranian plant protection congress, volume III, weeds. 2–5 Sept 2006, campus of agriculture and natural resources, University of Tehran, Karaj, p 16

Fawole OB (2000) Effect of pre-emergence herbicide, Galex on fungal and bacterial flora of a cowpea cropped soil. J Biosci Res Commun 2:195–200

Fayez M, Emam NF, Makboul HE (1983) Interaction of the herbicides bromoxynil and afalon S with *Azospirillum* and growth of maize. J Plant Nutr Soil Sci 146:741–751

Feng PCC, Baley J, Clinton WP, Bunkers GJ, Alibhai MF, Paulitz TC, Kidwell KK (2005) Glyphosate inhibits rust diseases in glyphosate-resistant wheat and soybean. Proc Natl Acad Sci U S A 102:17290–17295. https://doi.org/10.1073/pnas.0508873102

Feng PCC, Clark C, Andrade GC, Balbi MC, Caldwell P (2007) The control of Asian rust by glyphosate in glyphosate-resistant soybeans. Pest Manag Sci 63:15–26

Feng PCC, Clark C, Andrade GC, Balbi MC, Caldwell P (2008) The control of Asian rust by glyphosate in glyphosate-resistant soybeans. Pest Manag Sci 64:353–359. https://doi.org/10.1002/ps.1498

Fernandes MCS, Costa LSD, Grazziotti PH, Grazziotti DCFS, Santos JBD, Rossi MJ (2014) *Pisolithus* sp. tolerance to glyphosate and isoxaflutole in vitro. Rev Árvore 38:461–468

Fernandez MR, Selles F, Gehl D, DePauw RM, Zentner RP (2005) Crop production factors associated with fusarium head blight in spring wheat in eastern Saskatchewan. Crop Sci 45:1908–1916

Fernandez MR, Zentner RP, DePauw RM, Gehl DT, Stevenson FC (2007) Impacts of crop production factors on fusarium head blight in barley in eastern Saskatchewan. Crop Sci 47:1574–1584

Fischer NH (1986) The function of mono and sesquiterpenes as plant germination and growth regulators. In: Putnam AR, Tang CS (eds) The science of allelopathy. Wiley, New York, pp 203–218

Fletcher WH (1960) The effect of herbicides on soil micro-organisms. In: Woodford EK, Sagar GR (eds) Herbicides and the Soil, Blackwell Scientific Publications, Oxford, England, p 20–62

Fogg GE, Stewart WDP, Fay P, Walsby AE (1973) The Blue-Green Algae. Academic Press, London

Forlani G, Mantelli M, Branzoni M, Nielson E, Favilli F (1995) Differential sensitivity of plant-associated bacteria to sulfonylurea and imidazolinone herbicides. Plant Soil 176:243–253

Forouzesh A, Zand E, Soufizadeh S, Samadi Foroushani S (2015) Classification of herbicides according to chemical family for weed resistance management strategies—an update. Weed Res 55:334–358. https://doi.org/10.1111/wre.12153

Forrester IT, Grabski AC, Burgess RR, Leatham GF (1988) Manganese, Mn-dependent peroxidases, and the biodegradation of lignin. Biochem Biophys Res Commun 157:992–999

Frantzen J, Rossi F, Müller-Schärer H (2005) Integration of biological control of common groundsel (*Senecio vulgaris*) and chemical control. Weed Technol 787–793

Gadkari D, Klingmuller W (1988) Influence of herbicides on growth and nitrogenase activity of *Azospirillum*. In: Klingmüller W (ed) *Azospirillum* IV. Genetics, physiology, ecology. Proceedings of the fourth Bayreuth *Azospirillum* workshop. Springer, Berlin, pp 150–158

Galindo FS, Teixeira Filho MCM, Buzetti S, Santini JMK, Alves CJ, Nogueira LM, Ludkiewicz MGZ, Andreotti M, Bellotte JLM (2016) Corn yield and foliar diagnosis affected by nitrogen fertilization and inoculation with *Azospirillum brasilense*. Rev Bras Ciênc Solo 40: e0150364. https://doi.org/10.1590/18069657rbcs20150365

Galindo FS, Teixeira Filho MCM, Buzetti S, Santini JMK, Ludkiewicz MGZ, Baggio G (2017) Modes of application of cobalt, molybdenum and *Azospirillum brasilense* on soybean yield and profitability. Rev Bras Eng Agríc Amb 21:180–185. https://doi.org/10.1590/1807-1929/agriambi.v21n3p180-185

Gal-On A, Naglis A, Liebman D, Ziadna H, Kathrivan K, Papayiannis L, Holdengreber V, Guenoune-Gelbert D, Lapidot M, Aly R (2009) Broomrape can acquire viruses from its host. Phytopathol 99:1321–1329. https://doi.org/10.1094/PHYTO-99-11-1321

Gamliel A (2008) High consequence plant pathogens. In: Gullino, ML (ed) Crop Biosecurity, Springer Science+Business Media BV

Garcia MA, Costea M, Kuzmina M, Stefanovic S (2014) Phylogeny, character evolution, and biogeography of *Cuscuta* (dodders; Convolvulaceae) inferred from coding plastid and nuclear sequences. Am J Bot 101:670–690. https://doi.org/10.3732/ajb.1300449

Gaweł S, Wardas M, Niedworok E, Wardas P (2004) Malondialdehyde (MDA) as a lipid peroxidation marker. Wiad Lek 57:453–455

Gaynor JD, Hamill AS (1983) Bentazon preemergence activity on velvetleaf (Abutilon theophrasti Medic) and persistence in southwestern Ontario soils. Can J Plant Sci 63(4):1015–1022

Geisler L, Graef G, Wilson J, Schimelfenig J (2002) Interaction of glyphosate tolerancewith soybean cyst nematode resistance. Phytopathol 92:S529

Gianfreda L, Xu F, Bollag JM (1999) Laccases: a useful group of oxidoreductive enzymes. Bioremediat J 3:1–26. https://doi.org/10.1080/10889869991219163

Gianinazzi S, Gollotte A, Binet MN, van Tuinen D, Redecker D, Wipf D (2010) Agroecology: the key role of arbuscular mycorrhizas in ecosystem services. Mycorrhiza 20:519–530

Gilbert GS (2002) Evolutionary ecology of plant diseases in natural ecosystems. Ann Rev Phytopathol 40:13–43

Gilbert L, Norman R, Laurenson KM, Reid HW, Hudson PJ (2001) Disease persistence and apparent competition in a three-host community: an empirical and analytical study of large-scale, wild populations. J Anim Ecol 70:1053–1061

Giovannetti M, Turrini A, Strani P, Sbrana C, Avio L, Pietrangeli B (2006) Mycorrhizal fungi in ecotoxicological studies: soil impact of fungicides, insecticides and herbicides. Prev Today 2:47–62

Gloria ON (2010) Studies on biodegradation of glyphosate herbicide by bacterial species isolated from rice fields. A dissertation submitted to the Department of Microbiology in the Faculty of Biological Sciences in Partial Fulfillment of the Requirement for the Award of a Master's degree (M.Sc.) in Environmental Microbiology, University of Nigeria, Nsukka, Sept 2010

Gonzalez A, Gonzalez-Murua C, Royuela M (1996) Influence of imazethapyr on *Rhizobium* growth and its symbiosis with pea (*Pisum sativum*). Weed Sci 44:31–37

Goring CAR, Griffith JD, Q'Helia L, Scott HB, Youngson CR (1967) The effect of Tarden on microorganisms and soil biological processes. Down to Earth 22:14–17

Gosling P, Hodge A, Goodlass G, Bending GD (2006) Arbuscular mycorrhizal fungi and organic farming. Agric Ecosyst Environ 113:17–35

Govedarica MM, Milošević N, Jarak M, Konstantinović B (2001) Uticaj dimetenamida I metalachlora na mikrobiološka svojstva zemljišta pod šećernom repom. V Jug Sav Zašt Bil 12:3–8

Grazziotti PH, Siqueira JO, Moreira FMS (2003) Espécies arbóreas e ectomicorrizas em relação ao excesso de metais pesados. In: Cerreta CA, Silva LS, Reichert JM (eds) Tópicos em ciência do solo. Braz Soil Sci Soc, 55–105

Greaves MP, Malkomes MP (1980) Effects on soil microflora. In: Hance RJ (ed) Interactions between herbicides and the soil. Academic Press, New York, pp 223–253

Grinstein A, Katan J, Eshel Y (1976) Effect of dinitroaniline herbicides on plant resistance to soil-borne pathogens. Phytopathol 66:517–522

Grinstein A, Lisker N, Katan J, Eshel Y (1984) Herbicide-induced resistance to wilt diseases. Physiol Plant Pathol 24:347–356

Gronwald JW (1991) Lipid biosynthesis inhibitors. Weed Sci 39:435–449

Grossbard E, Atkinson D (1985) The herbicide glyphosate. Butterworths, London

Grzesik M, Romanowska-Duda Z (2014) Improvements in germination, growth, and metabolic activity of corn seedlings by grain conditioning and root application with cyanobacteria and microalgae. Pol J Environ Stud 23:1147–1153

Grzesik M, Romanowska-Duda Z (2015) Ability of cyanobacteria and green algae to improve metabolic activity and development of willow plants. Pol J Environ Stud 24:1003–1012

Grzesik M, Romanowska-Duda Z, Kalaji HM (2017) Effectiveness of cyanobacteria and green algae in enhancing the photosynthetic performance and growth of willow (*Salix viminalis* L.) plants under limited synthetic fertilizers application. Photosynthetica 55:510–521. https://doi.org/10.1007/s11099-017-0716-1

Gupta G, Bhattacharya AK (2008) Assessing toxicity of post-emergence herbicides on the *Spilarctia obliqua* Walker (Lepidoptera: Arctiidae). J Pest Sci 81:9–15

Gveroska B (1999) *Trichoderma harzianum* in tobacco seedlings with the use of a herbicide. Int J Agric Innov Res 3:950–955

Haahtela K, Kilpi S, Kari K (1988) Effects of phenoxy acid herbicides and glyphosate on nitrogenase activity (acetylene reduction) in root-associated *Azospirillum*, *Enterobacter*, and *Klebsiella*. FEMS Microbiol Ecol 53:123–127

Hale MG, Hulcher FH, Chappell WE (1957) The Effects of Several Herbicides on Nitrification in a Field Soil under Laboratory Conditions. Weeds 5(4):331–341

Hamel C, Morin F, Fortin A, Graner RL, Smith DL (1994) Mycorrhizal colonization increases herbicide toxicity in apple. Hort Sci 119:1255–1260

Hammond-Kosack K, Jones JDG (2000) Responses to plant pathogens. In: Buchanan BB, Gruissem W, Jones RL (eds) Biochemistry and molecular biology of plants. ASPP, Rockville, MD, pp 1102–1156

Harlapur SI, Kulkarni S, Nargund VB, Hedge RK (1989) Bioassay of herbicides and their efficacy on saprophytic survival of *Sclerotium rolfsii* Sacc.—a causal agent of food rot of wheat. Karnataka J Agric Sci 2:47–50

Harper M (2007) The review of the moratorium on GM canola, Australia. http://www.dpi.vic.gov.au/dpi/nrenfa.nsf/LinkView/5477226A88881F86CA-2572E300074EEF89E6C67B4668BD2A7CA256FB70001BAB8

Hartman GE, Sinclair JB, Clark JR (1999) Compendium of soybean diseases, 4th edn. American Phytopathological Society, St Paul
He Y, Shen D, Fang C, He R, Zhu Y (2006) Effects of Metsulfuron-Methyl on the Microbial Population and Enzyme Activities in Wheat Rhizosphere Soil. J Environ Sci Heal B 41 (3):269–284
Heitefuss R (1970) Nebenwirkungen von Herbiziden auf Pflanzenkrankheiten und deren Ereger. Z Pflanzenkr Pflanzenpathol Pflanzenschutz Sondernheft V:117–127
Heitefuss R (1972) Ursachen der Nebenwirkungen von Herbiziden auf Pflanzenkrankheiten. Z Pflanzenkr Pflanzenpathol Pflanzenschutz Sondernheft V:79–87
Heitefuss R, Bodendörfer H (1968) Der Einfluss von Herbiziden und Kalstickstoff auf der durch *Cercosporella herpotrichoides* Fron. verursachten Halmbruch des Weizens. Z Pflanzenkr Pflanzenpathol Pflanzenschutz 75:641–659
Helmeczi B, Kátai J, Bessenyei M (1988) Effect of herbicides on growth of some microscopic soil fungus species. Acta Microbiol Hung 35:429–432
Heydari A, Misaghi IJ (1998) The impact of herbicides on the incidence and development of *Rhizoctonia solani*-induced cotton seedling damping-off. Plant Dis 82:110–113
Hickman MV, Dodds DM, Huber DM (2002) Micronutrient interactions reduce glyphosate efficacy on tall fescue. Proc Weed Sci Soc Am 42:18
Higa T, Parr JF (1994) Beneficial and effective microorganisms for a sustainable agriculture and environment. International Nature Farming Research Center, Atami. http://www.emro-asia.com/data/66.pdf
Hoagland RE (1996) Chemical interactions with bioherbicides to improve efficacy. Weed Technol 10:651–674
Hofman T, Lees H (1953) The biochemistry of the nitrifying organisms. 4. The respiration and intermediary metabolism of Nitrosomonas. Biochem J 54(4):579–583
Holliday MJ, Keen NT (1982) The role of phytoalexins in the resistance of soybean leaves to bacteria: effect of glyphosate on glyceollin accumulation. Phytopathol 72:1470–1474
Holt RD, Lawton JH (1994) The ecological consequencesof shared natural enemies. Annu Rev Ecol Syst 25:495–520
Holt RD, Pickering J (1985) Infectious disease and species coexistence: a model of Lotka-Volterra form. Am Nat 126:196–211
Hornby D, Bateman GL, Gutteridge RJ, Lucas P, Osbourn AE, Ward E, Yarham DJ (1998) Take-all disease of cereals: a regional Pperspective. CAB International, Wallingford
Hsia YT, Christensen JJ (1951) Effect of 2,4-D on seedling blight of wheat caused by *Helminthosporium sativum*. Phytopathol 41:1011–1020
Hua Z, Huang BL, Ni GH, Wu JC, Yuan SZ (2002) Influence of some herbicides on *Rhizoctonia solani*. J Yangzhou Univ, Agric Life Sci Ed 23:71–73
Huber DM, McCay-Buis TS (1993) A multiple component analysis of the take-all disease of cereals. Plant Dis 77:437–447
Huber DM, Seely CL, Watson RL (1966) Effects of the herbicide diuron on foot rot of winter wheat. Plant Dis Rep 50:852–854
Huber DM, Cheng MW, Winsor BA (2005) Association of severe corynespora root rot of soybean with glyphosate-killed giant ragweed. Phytopathol 95:S45
Huber DM, Leuck JD, Smith WC, Christmas EP (2004) Induced manganese deficiency in GM soybeans. In: Northcentral Fert. Extension Conf., Des Moines, IA, November 2004
Hudson P, Greenman J (1998) Competition mediated by parasites: biological and theoretical progress. Trends Ecol Evol 13:387–390
Hull R (2014) Plant virology, 5th edn. Elsevier Academic Press, Tokyo
Hussain AL, Raverkar KP, Pareek N (2014) In vitro effects of herbicides on soil microbial communities. Bioscan 9:11–16
Iloba C (1978) The influence of 2,4-D on ectomycorrhizal symbiosis in pine and spruce seedlings. Eur J Forest Pathol 8:379–383

Islam MS, Saha AK, Mosaddeque HQM, Amin MR, Islam MM (2008) In vitro studies on the reaction of fungi *Trichoderma* to defferent herbicides used in tea plantation. Int J Sustain Crop Prod 3:27–30

Islam MS, Haque MS, Islam MM, Emdad EM, Halim A, Hossen QM, Hossain MZ, Ahmed B, Rahim S, Rahman MS, Alam MM, Hou S, Wan X, Saito JA, Alam M (2012) Tools to kill: genome of one of the most destructive plant pathogenic fungi *Macrophomina phaseolina*. BMC Genom 13:493. https://doi.org/10.1186/1471-2164-13-493

Jachetta JJ, Appleby AP, Boersma L (1986) Apoplastic and symplastic pathways of atrazine and glyphosate transport in shoots of seedling sunflower. Plant Physiol 82:1000–1007

Jackson MB, Stack RW (2002) Effects of dicamba herbicide on *Tubercularia ulmea* canker development. J Arboric 28:94–98

Jacobsen BJ, Hopen HI (1981) Influence of herbicides on aphanomyces root rot of peas. Plant Dis 65:11–16

Jacques RJS, Procópio SDO, Santos JBD, Kasuya MCM, Silva AAD (2010) Sensitivity of *Bradyrhizobium* strains to glyphosate. Rev Ceres 57:29–33. https://doi.org/10.1590/S0034-737X2010000100006

Jaworski EG (1972) Mode of action of N-phosphonomethyl-glycine: inhibition ofaromatic amino acid biosynthesis. J Agric Food Chem 20:1195–1198

Jena PK, Adhya TK, Rao VR (1990) Nitrogen-fixing bacterial population as influenced by butachlor and thiobencarb in rice soils. Zentralbl Mikrobiol 145:457–460

Jensen HL (1964) Studies on soil bacteria (Arthrobacter globiformis) capable of decomposing the herbicide endothal. Acta Agri Scand 14(2–3):193–207

Jin Y, Szabo LJ, Carson M (2010) Century-old mystery of *Puccinia striiformis* life history solved with the identification of *Berberis* as an alternate host. Phytopathol 100:432–435. https://doi.org/10.1094/PHYTO-100-5-0432

Jnawali AD, Ojha RB, Marahatta S (2015) Role of *Azotobacter* in soil fertility and sustainability—a review. Adv Plant Agric Res 2:00069. http://dx.doi.org/10.15406/apar.2015.02.00069

Johal GS, Huber DM (2009) Glyphosate effects on diseases of plants. Eur J Agron 31:144–152

Johal GS, Rahe JE (1984) Effect of soilborne plant-pathogenic fungi on the herbicidal action of glyphosate on bean seedlings. Phytopathol 74:950–955

Johal GS, Rahe JE (1988) Glyphosate, hypersensitivity and phytoalexin accumulation in the incompatible bean anthracnose host-parasite interaction. Physiol Mol Plant Pathol 32:267–281

Johal GS, Rahe JE (1990) Role of phytoalexins in the suppression of resistance of *Phaseolus vulgaris* to *Colletotrichum lindemuthianum* by glyphosate. Can J Plant Pathol 12:225–235

Kalia A, Gosal SK (2011) Effect of pesticide application on soil microorganisms. Arch Agron Soil Sci 57:569–596. https://doi.org/10.1080/03650341003787582

Kalinova S, Tahsin N, Hristeva T (2014) Study of oriental tobacco weed infestation in the region of Svilengrad and the influence of some herbicides on soil microflora. Plant Sci LI:63–66 (in Russian)

Kang SM, Khan AL, Hamayun M (2012) *Acinetobacter calcoaceticus* ameliorated plant growth and influenced gibberellins and functional biochemical. Pak J Bot 44:365–372

Kapich AN, Jensen KA, Hammel KE (1999) Peroxyl radicals are potential agents of lignin biodegradation. FEBS Lett 461:115–119. https://doi.org/10.1016/s0014-5793(99)01432-5

Karavina C (2014) Maize streak virus: a review of pathogen occurrence, biology and management options for smallholder farmers. Afr J Agric Res 9:2736–2742. https://doi.org/10.5897/AJAR2014.8897

Karthikeyan N, Prasanna R, Nain L (2007) Evaluating the potential of plant growth promoting cyanobacteria as inoculants for wheat. Eur J Soil Biol 43:23–30

Kasa P, Modugapalem H, Battini K (2017) Isolation, screening, and molecular characterization of plant growth promoting rhizobacteria isolates of *Azotobacter* and *Trichoderma* and their beneficial activities. J Nat Sci Biol Med 6:360–363. https://doi.org/10.4103/0976-9668.160006

Katan J, Eshel Y (1973) Interactions between herbicides and plant pathogens. Residue Rev 45:145–177

Kataria HR, Dodan DS (1982) The influence of two herbicides on the antifungal activity of some fungicides against *Pythium butleri* and *Rhizoctonia solani* causing damping-off of cowpea. Pestic Sci 13:583–588

Kataria HR, Dodan DS (1983) Impact of two soil-applied herbicides on damping-off of cowpea caused by *Rhizoctonia solani*. Plant Soil 73:275–283. https://doi.org/10.1007/BF02197723

Kataria HR, Gisi U (1990) Interactions of fungicide-herbicide combinations against plant pathogensand weeds. Crop Prot 9:403–409

Kawate SC, Kawate S, Ogg AG, Kraft JM (1992) Response of *Fusarium solani* f. sp. *pisi* and *Pythium ultimum* to glyphosate. Weed Sci 40:497–502

Keen NT, Holliday MJ, Yoshikawa M (1982) Effects of glyphosate on glyceollin production and the expression of resistance to *Phytophthora megasperma* f. sp. *glycinea* in soybean. Phytopathol 72:1467–1470

Keesing F, Holt RD, Ostfeld RS (2006) Effects of species diversity on disease risk. Ecol Lett 9:485–498

Kelley WD, South DB (1980) Effects of herbicides on in vitro growth of mycorrhizae of pine (*Pinus* spp.). Weed Sci 28:599–602

Kim YT, Lee YR, Jin J, Han KH, Kim H, Kim JC, Lee T, Yun SH, Lee YW (2005) Two different polyketide synthase genes are required for synthesis of zearalenone in *Gibberella zeae*. Mol Microbiol 58:1102–1113

Kizilkaya R (2009) Nitrogen fixation capacity of *Azotobacter* spp. strains isolated from soils in different ecosystems and relationship between them and the microbiological properties of soils. J Environ Biol 30:73–82

Kömives T, Casida JE (1983) Acifluorfen increases the leaf content of phytoalexins and stress metabolites in several crops. J Agric Food Chem 31:751–755

Konishi T, Sasaki Y (1994) Compartmentalization of two forms of acetyl-CoA carboxylase in plants and the origin of their tolerance toward herbicides. Proc Natl Acad Sci U S A 91:3598–3601. http://www.ncbi.nlm.nih.gov/pmc/articles/PMC43627

Kortekamp A (2008) Knocked out with Basta®!—are herbicides effective against downy mildew of grapevine? J Plant Dis Prot XXI:107–112

Kortekamp A (2010) Side effects of the herbicide glufosinate ammonium on *Plasmopara viticola* and other fungal pathogens. In: Calonnec A, Delmotte F, Emmet B, Gadoury D, Gessler C, Gubler D, Kassemeyer HH, Magarey P, Raynal M, seem R (eds) Proceedings of the 6th international workshop of grapevine downy and powdery mildew, Bordeaux, pp 13–15

Kortekamp A (2011) Herbicides and environment. InTech, Rijeka, Croatia. https://library.umac.mo/ebooks/b28109740.pdf

Kovacs A, Malligni C (1975) Effect of herbicides on diseases of cultivated plants. Notiziario sulle Malattie delle Piante 92–93:239–252

Kraus R, Sikora RA (1983) Effects of herbicide diallate, alone and in combination with aldicarb, on *Heterodera schachtii* population levels in sugar beet. J Plant Dis Prot 90: 132–139, http://www.jastor.org/stable/43382933

Kraus RG, Noel GR, Edwards DI (1982) Effect of preemergence herbicides and aldicarb on *Heterodera glycines* population dynamics and yield of soybean. J Nematol 14:452 (abstract)

Kremer RJ, Donald PA, Keaster AJ, Minor HC (2000) Herbicide impact on *Fusarium* spp. and soybean cyst nematode in glyphosate-tolerant soybean. Agron Abstr, 257

Kremer RJ, Means NE, Kim S (2005) Glyphosate affects soybean root exudation and rhizosphere micro-organisms. Int J Environ Anal Chem 85:1165–1174

Kucharski J, Wyszkowska J (2008) Biological properties of soil contaminated with the herbicide Apyros 75 WG. J Elementol 13:357–371

Kuo HC, Détry N, Choi J, Lee YH (2015) Potential roles of laccases on virulence of *Heterobasidion annosum s.s*. Microb Pathog 81:16–21

La Rocca N, Bonora A, Dalla Vecchia F, Barbato R, Rascio N (1998) Effects of amitrole and norflurazon on carotenogenesis in barley plants grown at different temperatures. In: Garab G (ed) Photosynthesis: mechanisms and effects, vol V. Kluwer, Dordrecht, pp 3459–3462

Lenart AM (2012) J Environ Sci Heal B 47(1):7–12

La Rocca N, Rascio N, Oster U, Rüdiger W (2007) Inhibition of lycopene cyclase results in accumulation of chlorophyll precursors. Planta 225:1019–1029. https://doi.org/10.1007/s00425-006-0409-7

Lai M, Semeniuk G (1970) Picloram-induced increase of carbohydrate exudation from corn seedlings. Phytopathol 60:563–564

Lake DB, Ippoliti DJ, Brandow CC, Otrosina WJ (1981) Effect of herbicides on the growth of *Pisolithus tinctorius* and *Scleroderma aurantium* in pure culture. Québec, Fifth N Am Conf Mycorrhizae, p 62

Lal R (2006) Encyclopedia of soil science, vol 1, 2nd edn. CRC Press, Taylor & Francis Group, New York

Lal R, Nagarajan K (1988) In vitro evaluation of pesticides against *Sclerotium rolfsii* Sacc. Causing collar rot of tobacco. Pestol 11:8

Landini S, Graham MY, Graham TL (2003) Lactofen induces isoflavin accumulation and gyceollin elicitation competency in soybean. Phytochem 62:865–874

Lange R, McLaren D (2002) Fusarium wilt—a new disease of canola. http://www.665umanitoba.ca/afs/agronomists conf/2002/pdf/lange.pdf

Lanini W, Kogan M (2005) Biology and management of *Cuscuta* in crops. Cienc Invest Agrar 32:165–179

Laranjo M, Alexandre A, Oliveira S (2014) Legume growth-promoting rhizobia: an overview on the *Mesorhizobium* genus. Microbiol Res 169:2–17. https://doi.org/10.1016/j.micres.2013.09.012

Larson RL, Hill AL, Fenwick A, Kniss AR, Hanson LE, Miller SD (2006) Influence of glyphosate on rhizoctonia and fusarium root rot in sugar beet. Pest Manag Sci 62:182–192

Latha PC, Gopal H (2010) Effects of herbicides on the growth and the activity of *Azospirillum lipoferum* and *Bacillus megaterium* var *phosphaticum* under in vitro conditions. Int J Plant Prot 3:50–54

Levene BC, Owen MDK, Tylka GL (1998) Response of soybean cyst nematode on soybeans (*Glycine max*) to herbicides. Weed Sci 46:264–270

Levesque CA, Rahe JE (1992) Herbicide interactions with fungal root pathogens, with special reference to glyphosate. Annu Rev Phytopathol 30:579–602

Levesque CA, Rahe JE, Eaves DM (1987) Effects of glyphosate on *Fusarium* spp.: its influence on root colonization of weeds, propagule density in the soil, and on crop emergence. Can J Microbiol 33:354–360

Li DY, Eberspcher J, Wagner B, Kuntzer J, Lingens F (1991) Degradation of 2,4,6-trichlorophenol by *Azotobacter* sp. Strain GP1. Appl Environ Microbiol 57:1920–1928

Li X, Miao W, Gong C, Jiang H, Ma W, Zhu S (2013) Effects of prometryn and acetochlor on arbuscular mycorrhizal fungi and symbiotic system. Lett Appl Microbiol 57:122–128. https://doi.org/10.1111/lam.12084

Liu L, Punja ZK, Rahe JE (1995) Effect of *Pythium* spp. and glyphosate on phytoalexin production and exudation by bean (*Phaseolus vulgaris* L.) roots grownin different media. Physiol Mol Plant Pathol 47:391–405

Liu L, Punja ZK, Rahe JE (1997) Altered root exudation and suppression of induced lignification as mechanisms of predisposition by glyphosate of bean roots (*Phaseolus vulgaris* L.) to colonization by *Pythium* spp. Physiol Mol Plant Pathol 51:111–127

Liu CA, Zhong H, Vargas J, Penner D, Sticklen M (1998) Prevention of fungal diseases in transgenic, bialaphos- and glufosinate-resistant creeping bentgrass (*Agrostis palustris*). Weed Sci 46:139–146

Liu B, Geisler LJ, Jackson-Ziems TA, Wegulo SN, Harveson RM, Korus KA, Klein RN (2012) Major fusarium diseases on corn, wheat, and soybeans in Nebraska. NebGuide G2181. http://extensionpublications.unl.edu/assets/html/g2181/build/g2181.htm

Lockert CK, Hoagland KD, Siegfried BD (2006) Comparative sensitivity of freshwater algae to atrazine. Bull Environ Contam Toxicol 76:73–79

LoGiudice K, Ostfeld RS, Schmidt KA, Keesing F (2003) The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. Proc Natl Acad Sci U S A 100:567–571

Lotan-Pompan M, Cohen R, Yarden O, Portnoy V, Burger Y, Katzir N (2007) Trifluralin herbicide-induced resistance of melon to fusarium wilt involves expression of stress- and defense-related genes. Mol Plant Pathol 8:9–22

Mackenzie KA, Macrae IC (1972) Tolerance of the N-fixing system of *Azotobacter vinelandii* to four commonly used pesticides. Antonie Leeuwenhoek 38:529–535

Madhavi B, Anand CS, Bharathi A, Polasa H (1994) Biotoxic effects of pesticides on symbiotic properties of rhizobial species. Bull Environ Contam Toxicol 52:87–94

Malty JD, Siqueira JO, Moreira FMD (2006) Effects of glyphosate on soybean symbiotic microorganisms, in culture media and in greenhouse. Pesqui Agropecu Bras 41:285–291

Mårtensson AM (1992) Effects of agrochemicals and heavy metals on fast-growing rhizobia and their symbiosis with small-seeded legumes. Soil Biol Biochem 24:435–445

Mårtensson AM, Nilsson ÅK (1989) Effects of chlorsulfuron on *Rhizobium* grown in pure culture and in symbiosis with alfalfa (*Medicago sativa*) and red clover (*Trifolium pretense*). Weed Sci 37:445–450

Marsh JA, Wingfield GI, Da Vies HA, Grossbaed E (1978) Simultaneous assessment of various responses of the soil microflora to bentazone. Weed Res 18(5):293–300

Martin DP, Shepherd DN (2009) The epidemiology, economic impact and control of maize streak disease. Food Sec 1:305–315. https://doi.org/10.1007/s12571-009-0023-1

Martin DP, Shepherd DN, Rybicki EP (2008) Maize streak virus. In: Mahy BWJ, van Regenmortel MHV (eds) Desk encyclopedia of plant and fungal virology. Academic Press, Elsevier, pp 209–217

Martín A, Moreno M, Marin P (1993) *Azotobacter* and *Azospirillum* as potential nitrogen fertilizers. Commun Soil Science Plant Anal 24:255–260. https://doi.org/10.1080/00103629309368796

Martinez CO, Silva CMMS, Fay EF, Maia AHN, Abakerli RB, Durrant LR (2008) Degradation of the herbicide sulfentrazone in a Brazilian Typic haploudox soil. Soil Biol Biochem 40:879–886

Martinez-Toledo MV, Salmeron MV, Gonzalez JL (1990) Effect of phenoxy and benzoic acid herbicides on nitrogenase activity and growth of *Azospirillum brasilense*. Soil Biol Biochem 22:879–881. https://doi.org/10.1016/0038-0717(90)90172-V

Martinez-Toledo MV, Salmeron MV, Golzalez L (1991) Effect of simazine on the biological activity of *Azotobacter chroococcum*. Soil Sci 151:459–467

Marx DH, Cordell CE (1989) The use of specific ectomycorrhizas to improve artificial forestation practices. In: Whipps JM, Lumsden RD (eds) Biotechnology of fungi for improving plant growth. Academic Press, New York, pp 1–25

McCay-Buis TS (1998) Ramifications of microbial interactions conditioning take-all of wheat. Ph. D. thesis. Purdue University, West Lafayette, IN

McGrann GRD, Grimmer MK, Mutasa-Göttgens ES, Stevens M (2009) Progress towards the understanding and control of sugar beet rhizomania disease. Mol Plant Pathol 10:129–141. https://doi.org/10.1111/j.1364-3703.2008.00514.x

Mekwatanakarn P, Sivasithamparam K (1987) Effect of certain herbicides on saprophytic survival and biological suppression of the take-all fungus. New Phytol 106:153–159

Meriles JM, Vargas Gil S, Haro RJ, March GJ, Guzmán CA (2006) Glyphosate and previous crop residue effect on deleterious and beneficial soil-borne fungi from a peanut-corn-soybean rotations. J Phytopathol 154:309–316

Merrill AR, Lembi CA (2009) Applied weed science: including the ecology and management of invasive plants. Pearson Prentice Hall

Mikona C, Jelkmann W (2010) Replication of Grapevine leafroll-associated virus-7 (GLRaV-7) by *Cuscuta* species and its transmission to herbaceous plants. Plant Dis 94:471–476

Miller WA, Rasochova L (1997) Barley yellow dwarfviruses. Annu Rev Phytopathol 35:167–190

Miller JH, Carter CH, Garber RH, DeVay JE (1979) Weed and disease responses to herbicides in single- and double-row cotton (*Gossypium hirsutum*). Weed Sci 27:444–449

Milošević NA, Govedarica MM (2001) Uticaj prometrina na mikrobiološka svojstva zemljišta pod sojom i suncokretom. V Jug Sav Zašt Bil 12:3–7
Milošević NA, Govedarica MM (2002) Effect of herbicides on microbiological properties of soil. Proc Nat Sci, Matica Srpska Novi Sad 102:5–21
Mirzae A, Vazan S, Naseri R (2010) Response of yield and yield components of safflower (*Carthamus tinctorius* L.) to seed inoculation with *Azotobacter* and *Azospirillum* and different nitrogen levels under dry land conditions. World Appl Sci J 11:1287–1291
Moody CS, Hassan HM (1982) Mutagenicity of oxygen free radicals. P Nat A Sci 79(9):2855–2859
Mohamed AT, El-Hussein AA, El Siddig MA, Osman AG (2011) Degradation of oxyfluorfen herbicide by soil microorganisms, biodegradation of herbicides. Biotechnol 10:274–279. https://doi.org/10.3923/biotech.2011.274.279
Mohiuddin M, Mohammed MK (2014) Fungicide (carbendazim) and herbicides (2,4-D and atrazine) influence on soil microorganisms and soil enzymes of rhizospheric soil of groundnut crop. Int J Recent Sci Res 5:585–589
Molisch H (1937) Der einfluss einer pflanze auf die andere- allelopathie. Fischer, Jena
Morales J, Gallardo S, Vásquez C, Ríos Y (2000) Patrón de emergência, longevidad, parasitismo y proporción sexual de *Telenomus remus* (Hymenoptera: Scelionidae) com relación al cogollero Del maíz. Bioagro 12:47–54
Mouhanna AM, Langen G, Schlösser E (2008) Weeds as alternative hosts for BSBV, BNYVV, and vector *Polymyxa betae* (German isolate). J Plant Dis Prot 115:193–198. https://doi.org/10.1007/BF03356263
Mousavi SK, Zand E, Saremi H (2004) Physiological function and application of herbicides. Zanjan University Press, Zanjan, p 286
Moustafa-Mahmoud SM, Sumner DR, Ragab MM, Ragab MM (1993) Interactions of fungicides, herbicides and planting date with seedling diseases of cotton caused by *Rhizoctonia solani* AG-4. Plant Dis 77:79–86
Musarrat J, Bano N, Rao RAK (2000) Isolation and characterization of 2,4-dichlorophenoxyacetic acid-catabolizing and their biodegradation efficiency in soil. World J Microbiol Biotechnol 16:495–497
Naderifar M, Daneshian J (2012) Effect of seed inoculation with *Azotobacter* and *Azospirillum* and different nitrogen levels on yield and yield components of canola (*Brassica napus* L.). Iranian J Plant Physiol 3:619–626
Naqqash T, Hameed S, Imran A, Hanif MK, Majeed A, van Elsas JD (2016) Differential response of potato toward inoculation with taxonomically diverse plant growth promoting rhizobacteria. Front Plant Sci 7:144. https://doi.org/10.3389/fpls.2016.00144
Nelson N (2009) Manganese response of conventional and glyphosate-resistant soybean in Kansas. International Plant Nutrition Institute, Southern and Central Great Plains Region, Insights, p 3
Nelson KA, Renner KA, Hammerschmidt R (2002a) Effects of protoporphyrinogen oxidase inhibitors on soybean (*Glycine max* L.) response, *Sclerotinia sclerotiorum* disease development, and phytoalexin production by soybean. Weed Technol 16:353–359
Nelson KA, Renner KA, Hammerschmidt R (2002b) Cultivar and herbicide selection affects soybean development and the incidence of sclerotinia stem rot. Agron J 94:1270–1281
Nemec S, Tucker D (1983) Effects of herbicides on endomycorrhizal fungi in Florida citrus (*Citrus* spp.). Weed Sci 31:427–431. https://doi.org/10.1017/S0043174500069320
Neubauer R, Avizohar-Hershenson Z (1973) Effect of the herbicide, trifluralin, on rhizoctonia disease in cotton. Phytopathol 63:651–652
Neumann G, Kohls S, Landsberg E, Stock-Oliveira KS, Yamada T, Römheld V (2006) Relevance of glyphosate transfer to non-target plants via the rhizosphere. J Plant Dis Prot 20:963–969
Newman MM, Hoilett N, Lorenz N, Dick RP, Liles MR, Ramsier C, Kloepper JW (2016) Glyphosate effects on soil rhizosphere-associated bacterial communities. Sci Total Environ 543:155–160. https://doi.org/10.1016/j.scitotenv.2015.11.008

Ngari C, Combier JP, Doré J, Marmeisse R, Gay G, Melayah D (2009) The dominant *Hc.Sdh*^{R} carboxin-resistance gene of the ectomycorrhizal fungus *Hebeloma cylinrosporum* as a selectable marker for transformation. Curr Genet 55:223231. https://doi.org/10.1007/s00294-009-0231-4

Niewiadomska A (2004) Effect of carbendazim, imazetapir, and thiram on nitrogenase activity, the number of microorganisms in soil and yield of red clover (*Trifolium pretense* L.). Pol J Environ Stud 13:403–410

Nilsson HE (1973) Influence of the herbicide mecoprop on *Gaeumannomyces graminis* and take-all of spring wheat. Swed J Agric Res 3:105–113

Nilsson M, Rasmussen U, Bergman B (2005) Competition among symbiotic cyanobacterial *Nostoc* strains forming artificial associations with rice (*Oryza sativa*). FEMS Microbiol Lett 245:139–144. https://doi.org/10.1016/j.femsle.2005.03.010

Norman R, Bowers RG, Begon M, Hudson PJ (1999) Persistence of tick-borne virus in the presence of multiple host species: tick reservoirs and parasite-mediated competition. J Theor Biol 200:111–118

Ocampo JA, Barea JM (1984) Effect of carbamate herbicides on VA mycorrhizal infection and plant growth. Plant Soil 85:375–383

Ohman J, Kommedahl T (1960) Relative toxicity of extracts from vegetative organs of quackgrass to alfalfa. Weeds 8:660–670

Oka IN, Pimentel D (1976) Herbicide (2,4-D) increases insect and pathogen pests on corn. Science 193:239–240

Olajire DM, Oluyemisi FB (2009) In vitro effects of some pesticides on pathogenic fungi associated with legumes. Aust J Crop Sci 3:173–177

Omar MN, Berge O, Hassanein EE, Shalan SN (1992) In vitro and in situ effects of herbicide thiobencarb on rice - Azospirillum association. Symbiosis 13:55–63

Ozturk L, Yazici A, Eker S, Gokmen O, Romheld V, Cakmak I (2008) Glyphosate inhibition of ferric reductase activity in iron deficient sunflower roots. New Phytol 177:899–906

Page WW, Smith MC, Holt J, Kyetere D (1999) Intercrops, *Cicadulina* spp., and maize streak virus disease. Ann Appl Biol 135:385–393. https://doi.org/10.1111/j.1744-7348.1999.tb00865.x

Pakdaman BS, Kariman K (2006) Sethoxydim, an herbicide with potentially increasing effects on the virulence of fusarium head blight causal *Fusarium* species. In: 8th conference of European foundation for plant pathology & British society of plant pathology presidential meeting, Copenhagen, p 99

Pakdaman BS, Mohammadi Goltapeh E (2007) In vitro studies on the integrated control of rapeseed white stem rot disease through the application of herbicides and *Trichoderma* species. Pak J Biol Sci 10:7–12

Pakdaman BS, Khabbaz H, Goltapeh EM, Afshari HA (2002) In vitro studies on the effects of sugar beet field prevalent herbicides on the beneficial and deleterious fungal species. Pak J Plant Pathol 1:23–24

Pakdaman BS, Mohammadi Goltapeh E, Sepehrifar R, Pouiesa M, Rahimi Fard M, Moradi F, Modarres SAM (2007) Cellular membranes, the sites for antifungal activity of the herbicide sethoxydim. Pak J Biol Sci 10:2480–2484

Pakdaman BS, Mohammadi Goltapeh E, Soltani BM, Talebi AA, Naderpoor M, Kruszewska JS, Piłsyk S, Sarrocco S, Vannacci G (2013) Toward the quantification of confrontation (dual culture) test: a case study on the biological control of *Pythium aphanidermatum* with *Trichoderma asperelloides*. J Biofertil Biopestici 4:2. https://doi.org/10.4172/2155-6202.1000137

Palaniyandi SA, Yang SH, Zhang L, Suh JW (2013) Effects of actinobacteria on plant disease suppression and growth promotion. Appl Microbiol Biotechnol 97:9621–9636. https://doi.org/10.1007/s00253-013-5206-1

Pampulha M, Oliveira A (2006) Impact of an herbicide combination of bromoxynil and prosulfuron on soil microorganisms. Curr Microbiol 53:238–243

Pampulha ME, Ferreira MASS, Oliveira A (2007) Effects of a phosphinothricin based herbicide on selected groups of soil microorganisms. J Basic Microbiol 47:325–331. https://doi.org/10.1002/jobm.200610274

Pasaribu A, Mohamad RB, Hashim A, Rahman ZA, Omar D, Morshed MM (2013) Effect of herbicide on sporulation and infectivity of vesicular arbuscular mycorrhizal (*Glomus mosseae*) symbiosis with peanut plant. J Anim Plant Sci 23:1671–1678

Pastro S, March GJ (1999) In vitro effect of residual herbicides used in peanut on *Sclerotium rolfsii*. Fitopatologia 34:116–121

Pathak D, Roy AK, Deka SC (1996) Effect of herbicides on the growth and sclerotial survival of *Rhizoctonia solani* Kuhn. Ann Biol 12:245–251

Patrick ZA (1971) Phytotoxic substances associated with decomposition in soil of plant residues. Soil Sci 111:13–18

Paul V, Schönbeck F (1976) Untersuchungen über den Einfluss des Herbizide Diallat auf einige Getreidekrankheiten. J Phytopathol 85:1289–1297

Paula TR Jr, Comerlato AG, Gavassoni WL, Oliveira VL, Zambolim L (1995) Efeito de glifosate e oxyfluorfen sobre *Pisolithus tinctorius* e sobre a micorrização de raízes de *Eucalyptus grandis*. Rev Árvore 19:241–248

Payan LA, Johnson AW, Littrell RH (1987) Effects of nematicides and herbicides alone or combined on *Meloidogyne incognita* egg hatch and development. Ann Appl Nematol 1:67–70

Pedraza RO, Bellone CH, de Bellone SC, Sorte PMFB, dos Santos Teixeira KR (2009) *Azospirillum* inoculation and nitrogen fertilization effect on grain yield and on the diversity of endophytic bacteria in the phyllosphere of rice rainfed crop. Eur J Soil Biol 45:36–43. https://doi.org/10.1016/j.ejsobi.2008.09.007

Percich JA, Lockwood JL (1975) Influence of atrazine on the severity of fusarium root rot in pea and corn. Phytopathology 65(2):154

Pero RW, Owens RG (1971) Simple micromethod for detecting antifungal activity. Appl Microbiol 21:546–547

Perry RN, Beane J (1989) Effects of certain herbicides on the in vitro hatch of *Globodera rostochinensis* and *Heterodera schachtii*. RevNématol 12:191–196

Perry RN, Trett MW (1986) Ultrastructure of the eggsell of Heterodera schachtii and H. glycines (Nematoda: Tylenchida). Rev Nématol 9: 399

Pinckard JA, Standifer LC (1966) An apparent interaction between cotton herbicidal injury and seedling blight. Plant Dis Rep 50:172–177

Pline WA, Lacy GH, Stromberg V, Hatzios KK (2001) Antibacterial activity of the herbicide glufosinate on *Pseudomonas syringae* Pathovar *Glycina*. Pestic Biochem Physiol 71:48–55

Ploetz R, Freeman S, Konkol J, Israeli Y (2015) Tropical race of Panama disease in the Middle East. Phytoparasitica 43:283–293. https://doi.org/10.1007/s12600-015-0470-5

Pomari AF, Bueno AF, Bueno RCOF, Menezes Junior AO (2012) Biological characteristics and thermal requirements of the biological control agent *Telenomus remus* (Hymenoptera: Platygastridae) reared on eggs of different species of the genus *Spodoptera* (Lepidoptera: Noctuidae). Ann Entomol Soc Am 105:73–81

Postma J, Schilder MT, Bloem J, van Leeuwen-Haagsma WK (2008) Soil suppressiveness and functional diversity of the soil microflora in organic farming systems. Soil Biol Biochem 40:2394–2405

Power AG, Mitchell CE (2004) Pathogen spillover in disease epidemics. Am Nat 164:S79–S89

Praveena R, Naseema A, George S (2007) Effect of herbicides on *Fusarium pallidoroseum*- a potential biocontrol agent of water hyacinth [*Eichhornia crassipes* (Mart.) Solms]. J Trop Agric 45:55–57

Prescott JM, Burnett PA, Saari EE, Ransom J, Bowman J, de Milliano W, Singh RP, Bekele G (1986) Wheat diseases and pests: a guide for field identification. CIMMYT, Mexico

Procópio SO, Fernandes MF, Teles DA, Sena Filho JG, Cargnelutti Filho A, Vargas L, Sant'anna SAC (2011) Toxicity of herbicides applied on sugarcane to the diazotrophic bacterium *Azospirillum brasilense* (in Portugese). Planta Daninha 29:1079–1089

Punja ZK (1985) The biology, ecology and control of *Sclerotium rolfsii*. Ann Rev Phytopathol 23:97–127

Putnam AR, Weston LA (1986) Adverse impacts of allelopathy in agricultural systems. In: Putnam AR, Tang CS (eds) The science of allelopathy. Willey, New York, pp 43–56

Radivojevic L, Santric L, Stankovic-Kalezic R, Brkic D, Janjic V. (2003) Effects of Metribuzin on the abundance and activity of some groups of soil microorganisms. Pesticidi 18(2):99–107

Radke VL, Grau CR (1986) Effects of herbicides on carpogenic germination of *Sclerotinia sclerotiorum*. Plant Dis 70:19–23

Rahe JE, Levesque CA, Johal GS (1990) Synergistic role of soil fungi in the herbicidal efficacy of glyphosate. In: Hoagland RE (ed) Biological weed control using microbes and microbial products as herbicides. Symposium, 9–14 Apr 1989. American Chemical Society, Washington DC, pp 260–275

Rahman KA, Batra AL (1945) The onion thrips, *Thrips tabaci* (Lindeman) (Thripidae: Terebrantia: Thysanoptera). Indian J Agric Sci 14:308–310

Ramesh G, Nadanassababady T (2005) Impact of herbicides on weeds and soil ecosystem of rainfed maize (*Zea mays* L.). Indian J Agric Res 39:31–36

Ratcliff AW, Busse MD, Shestak CJ (2006) Changes in microbial community structure following herbicide (glyphosate) additions to forest soils. Appl Soil Ecol 34:114–124

Ratnayake M, Audus LJ (1978) Studies on the effects of herbicides on soil nitrification. II. Pestic Biochem Physiol 8:170–185. https://doi.org/10.1016/0048-3575(78)90037-8

Raviv M (2010) The use of mycorrhiza in organically-grown crops under semi arid conditions: a review of benefits, constraints and future challenges. Symbiosis 52:65–74

Reddy BVS, Sanjana Reddy P, Sadananda AR, Dinakaran E, Ashok Kumar A, Deshpande SP, Srinivasa Rao P, Sharma HC, Sharma R, Krishnamurthy L, Patil JV (2012) Postrainy season sorghum: constraints and breeding approaches. J SAT Agric Res 10:1–12

Redinbaugh MG, Jones MW, Gingery RE (2004) The genetics of virus resistance in maize (*Zea mays* L). Maydica 49:183–190

Rejon A, Garcia-Romera I, Ocampo JA, Bethlenfalvay GJ (1997) Mycorrhizal fungi influence competition in a wheat-ryegrass association treated with the herbicide diclofop. Appl Soil Ecol 7:51–57

Rengel Z (1999) Mineral nutrition of crops: fundamental mechanisms and implications. Food Products Press, London

Rengel Z, Marschner P (2005) Nutrient availability and management in the rhizosphere: exploiting genotypic differences. New Phytol 168:305–312

Rengel Z, Gutteridge R, Hirsch P, Hornby D (1996) Plant genotype, micronutrient fertilization and take-all infection influence bacterial populations in therhizosphere of wheat. Plant Soil 183:269–277

Rezaei F, Yarnia M, Farajzadeh EM (2006) Evaluation of allelopathical effects of *Chenopodium album*, *Amaranthus retroflexus*, and *Cynodon dactylon* on germination and seedling growth of safflower. In: Nezamabadi N (ed) Proceedings of the 17th Iranian plant protection congress, vol III, Weeds, 2–5, Sept 2006, Campus of Agriculture and Natural Resources, University of Tehran, Karaj, p 16

Rice EL (1984) Allelopathy, 2nd edn. Academic Press, Orlando, FL

Rinaudo V, Brberi B, Giovannetti M, van der Heijden M (2010) Mycorrhizal fungi suppress aggressive agricultural weeds. Plant Soil 333:1–5

Rivarola V, Fabra A, Mori G, Balegno H (1992) In vitro protein synthesis is affected by the herbicide 2,4-dichlorophenoxyacetic acid in *Azospirillum brasilense*. Toxicol 73:71–79

Roberti R, Badiali F, Pisi A, Veronesi A, Pancaldi D, Cesari A (2006) Sensitivity of *Clonostachys rosea* and *Trichoderma* spp. as potential biocontrol agents to pesticides. J Phytopathol 154:100–109

Roebke TW, Windels CE, Dexter AG (2002) Lack of herbicide effect on severity of aphanomyces root rot of sugar beet. J Sugar Beet Res 39:75–87

Romanowska-Duda Z, Wolska A, Malecka A (2004) Influence of blue-green algae as nitrogen fertilizer supplier in regulation of water status in grapevines under stress conditions. In:

Medrano H (ed) Book of abstracts, COST 858, water transport and aquaporins in grapevines, 20–23 Oct 2004. University of the Balearic Islands, Palma, Alcudia, Spain, p 10

Romanowska-Duda Z, Grzesik M, Owczarczyk A, Mazur-Marzec H (2010) Impact of intra and extracellular substances from cyanobacteria on the growth and physiological parameters of grapevine (*Vitis vinifera*). In: Arola L, Carbonell J (eds) 20th international conference on plant growth substance (IPGSA), book of abstracts, vol 28. Universitat Rovira I Virgili, Tarragona, p 118

Romero MC, Urrutina MI, Reinoso EH, Della Vedova R, Reynaldi FJ (2014) Atrazine degradation by wild filamentous fungi. Global Res J Microbiol 4:10–16

Romero-Perdomo F, Campos PJC, Rodríguez AJ, Rusinque MC, Buitrago RRB (2015) Efecto de fertilizantes y herbicidas sobre el crecimiento in vitro de *Azospirillum brasilense* C16. Rev Soc Ven Microbiol 35:117–120

Ronco MG, Ruscitti MF, Arango MC, Beltrano J (2008) Glyphosate and mycorrhization induce changes in plant growth and in root morphology and architecture in pepper plants (*Capsicum annuum* L.). J Hortic Sci Biotechnol 83:497–505

Rosas-Saavedra, C, Stange C (2016) Biosynthesis of carotenoids in plants: enzymes and color. In: Stange C (ed) Carotenoids in nature. Subcellular biochemistry, vol 79. Springer, Cham. http://doi.org/10.1007/978-3-319-39126-7_2

Rose MT, Cavagnaro TR, Scanlan CA, Rose TJ, Vancov T, Kimber S, Kennedy IR, Kookana RS, Zwieten LV (2016) Impact of herbicides on soil biology and function. Adv Agron 136:133–220. https://doi.org/10.1016/bs.agron.2015.11.005

Roseman TS, Graham RD, Arnott HJ, Huber DM (1991) The interaction of temperature with virulence and manganese oxidizing potential in the epidemiology of *Gaeumannomyces graminis*. Phytopathol 81:S1215

Rosenberger D, Fargione M (2004) Apple pest report 12, 6. University of Maine Cooperative Extension Service

Roslycky EB (1982) Glyphosate and the response of the soil microbiota. Soil Biol Biochem 14:87–92

Rosolem CA, Gabriel GJM, Lisboa IP, Zoca SM (2009) Manganese uptake and distribution in soybeans as affected by glyphosate. Proc Int Plant Nutrition Colloquium XVI. http://escholarship.org/uc/item/3f53794z?query=Rosolem#page-4

Rouillon R, Poulon C, Bastide J, Coste CM (1990) Degradation of the herbicide chlorpropham by some ectomycorrhizal fungi in pure culture. Agric Ecosyst Environ 28:421–424. https://doi.org/10.1016/0167-8809(90)90073-M

Rovira AD, McDonald HJ (1986) Effect of the herbicide chlorsulfuron on rhizoctonia bare patch and take-all of barley and wheat. Plant Dis 70:879–882

Royuela M, Gonzalez A, Arrese-Igor C, Aparicio-Tejo PM, Gonzalez-Murua C (1998) Imazethapyr inhibition of acetolactate synthase in *Rhizobium* and its symbiosis with pea. Pestic Sci 52:372–380

Rudolf VH, Antonovics J (2005) Species coexistence and pathogens with frequency-dependent transmission. Am Nat 166:112–118

Ruppel RF (1965) A review of the genus *Cicadulina* (Hemiptera, Cicadellidae). Michigan State Univ Biol Ser 2:385–428

Ruppel EG, Hecker RJ (1982) Increased severity of rhizoctonia root rot in sugar beet treated with systemic insecticides. Crop Prot 1:75–81

Ruppel EG, Hecker RJ, Schweizer EE (1982) Rhizoctonia root rot of sugarbeet unaffected by herbicides. J Am Soc Sugar Beet Technol 21:203–209

Rusevska K, Karadelev M, Phosri C, Duenas M, Teresa Telleria M, Watling R, Martín MP (2015) DNA barcoding is an effective tool for differentiating *Pisolithus* species from Macedonia. Mycotaxon 130:1007–1016. https://doi.org/10.5248/130.1007

Russin JS, Carter CH, Griffin JL (1995) Effects of grain sorghum (*Sorghum bicolor*) herbicides on charcoal rot fungus. Weed Technol 9:343–351

Saadatnia H, Riahi H (2009) Cyanobacteria from paddy fields in Iran as a biofertilizer in rice plants. Plant Soil Environ 55:207–212

Saha KC, Sannigrahi S, Mandal LN (1985) Effect of inoculation of *Azospirillum lipoferum* on nitrogen fixation in rhizosphere soil, their association with roots, yield and nitrogen uptake by mustard (*Brassica juncea*). Plant Soil 87:273–280

Sahu D, Priyadarshani I, Rath B. (2012) Cyanobacteria–as potential biofertilizer. CIB Tech J Microbiol 1:20–26

Salmeron V, Martinez-Toledo MV, Gonzalez-Lopez J (1991) Effects of alachlor and metolachlor on the biological activity of *Azospirillum brasilense* grown in chemically defined and dialyzed-soil media. Environ Toxicol Chem 10:493–499

Sanogo S, Yang XB, Scherm H (2000) Effects of herbicides on *Fusarium solani* f. sp. *glycines* and development of sudden death syndrome in glyphosate-tolerant soybean. Phytopathol 90:57–66

Sanogo S, Yang XB, Scherm H (2001) Field response of glyphosate-tolerant soybean to herbicides and sudden death syndrome. Plant Dis 85:773–779

Santos JB, Jacques RJS, Procópio SO, Kasuya MCM, Silva AA, Santos EA (2004) Effects of different glyphosate commercial formulations on *Bradyrhizobium* strains. Planta Daninha 22:293–299. https://doi.org/10.1590/S0100-83582004000200017

Santos JB, Jakelaitis A, Silva AA, Costa MD, Manabe A, Silva MCS (2006) Action of two herbicides on the microbial activity of soil cultivated with common bean (*Phaseolus vulgaris*) in conventional-till and no-till systems. Weed Res 46:284–289

Santos KB, Meneguim AM, Santos WJ, Neves PMOJ, Santos RB (2010) Caracterização dos danos de *Spodoptera eridania* (Cramer) e *Spodoptera cosmioides* (Walker). Neotrop Entomol 39:626–631

Sanyal D, Shrestha A (2008) Direct effect of herbicides on plant pathogens and disease development in various cropping systems. Weed Sci 56:155–160. https://doi.org/10.1614/WS-07-081.1

Saraiva AS, Sarmento RA, Pedro-Neto M, Teodoro AV, Erasmo EA, Belchior DC, de Azevedo EB (2016) Glyphosate sub-lethal toxicity to non-target organisms occurring in *Jatropha curcas* plantations in Brazil. Exp Appl Acarol 70:179–187. https://doi.org/10.1007/s10493-016-0078-6

Saul A (2003) Zooprophylaxis or zoopotentiation: the outcome of introducing animals on vector transmission is highly dependent on the mosquito mortality while searching. Malar J 2:32–50

Sawicka A, Selwet M (1998) Effect of active ingredients on *Rhizobium* and *Bradyrhizobium* legume dinitrogen fixation. Pol J Environ Stud 7:317–320

Schloter M, Dilly O, Munch JC (2003) Indicators for evaluating soil quality. Agric Ecosyst Environ 98:255–262

Schmidt KA, Ostfeld RS (2001) Biodiversity and the dilution effect in disease ecology. Ecol 82:609–619

Schmitt DP, Corbin FT, Nelson LA (1983) Population dynamics of *Heterodera glycines* and soybean response in soils treated with selected nematicides and herbicides. J Nematol 15:432–437

Schneider MI, Sanchez N, Pineda S, Chi H, Ronco A (2009) Impact of glyphosate on the development, fertility and demography of *Chrysoperla externa* (Neuroptera: Chrysopidae): ecological approach. Chemosphere 76:1451–1455

Schuldt PN, Wolfe CN (1956) Fungitoxicity of substituted *s*-triazines. Contrib Boyce Thompson Inst 15:377–393

Schulze DG, McCay-Buis TS, Sutton SR, Huber DM (1995) Manganese oxidation states in *Gaeumannomyces*-infested wheat rhizospheres probedby micro-XANES spectroscopy. Phytopathol 85:990–994

Sdoodee R, Teakle DS (1987) Transmission of tobacco streak virus by *Thrips tabaci* a new method of plant virus transmission. Plant Pathol 36:377–380

Sebiomo A, Ogundero VW, Bankole SA (2011a) Utilisation and biodegradation of atrazine and primextra. J Microbiol Antimicrob 3:64–76

Sebiomo A, Ogundero VW, Bankole SA (2011b) Effect of four herbicides on microbial population, soil organic matter and dehydrogenase activity. Afr J Biotechnol 10:770–778

Sebiomo A, Ogundero VW, Bankole SA (2012) Effect of four herbicides on soil minerals. Res J Environ Earth Sci 4:617–624
Semeniuk G, Tunac JB (1968) Tordon increase of root rot severity in wheat and corn. Proc S Dak Acad Sci 47:346
Sen C, Kaiser SAKM (1972) The role of herbicides in the control of brown spot of paddy caused by *Helminthosporium oryzae*. Indian J Mycol Plant Pathol 2:152
Shanan NT, Higazy AM (2009) Integrated biofertilization management and cyanobacteria application to improve growth and flower quality of *Matthiola incana*. J Agric Biol Sci 5:1162–1168
Shannon CE, Weaver EW (1949) A mathematical theory of communication. University of Illinois Press, Urbana, IL
Shariatmadari Z, Riahi H, Hashtroudi MS, Ghassempour AR, Aghashariatmaday Z (2013) Plant growth promoting cyanobacteria and their distribution in terrestrial habitats of Iran. Soil Sci Plant Nutr 59:535–547. https://doi.org/10.1080/00380768.2013.782253
Sharma SR, Sohi HS (1983) Influence of herbicides on root rot of French beans (*Phaseolus vulgaris* L.), caused by *Rhizoctonia solani*. Zentbl Mikrobiol 138:357–361
Sharma U, Adee EA, Pfender WF (1989) Effect of glyphosate herbicide on pseudothecia formation by *Pyrenophora tritici-repentis* in infested wheat straw. Plant Dis 73:647–650
Sharon A, Amsellem Z, Gressel J (1992) Glyphosate suppression of an elicited response. Increased susceptibility of *Cassia obtusifolia* to a mycoherbicide. Plant Physiol 98:654–659
Shen J, DiTommaso A, Shen M, Lu W, Li Z (2009) Molecular basis for differential metabolic responses to monosulfuron in three nitrogen-fixing cyanobacteria. Weed Sci 57:133–141. https://doi.org/10.1614/WS-08-024.1
Shepherd AM (1962) New blue R, a stain that differentiates between living and dead nematodes. Nematologica 8:201–208
Sidhu SS, Chakravarty P (1990) Effect of selected foresty herbicides on ectomycorrhizal development and seedling growth of lodgepole pine and white spruce under controlled and field environment. Eur J Forest Pathol 20:77–94
Simon-Sylvestre G, Fournier JC (1979) Effects of pesticides on the soil microflora. Adv Agron 31:1–92
Singh RN (1961) The role of blue-green algae in nitrogen economy of Indian agriculture. Indian Council of Agricultural Reseach, New Delhi
Singh RS (2001) Plant disease management. Science Publishers, New Hamshire
Singh G (2005) Effects of herbicides on biological nitrogen fixation in grain and forage legumes—a review. Agric Rev 26:133–140
Singh G, Wright D (2002) In vitro studies on the effects of herbicides on the growth of rhizobia. Lett Appl Microbiol 35:12–16. https://doi.org/10.1046/j.1472-765X.2002.01117.x
Singh AL, Singh PK, Singh PL (1988) Effects of different herbicides on the Azolla and blue-green algal biofertilization of rice. J Agric Sci 111:451–458. https://doi.org/10.1017/S0021859600083623
Singh B, Chatutvedi SN, Singh RH (1999) Influence of the herbicides on the antifungal activity of some fungicides against phytophthora blight of pigeonpea. Indian Phytopathol 52:59–62
Smiley RW, Ogg AG, Cook RJ (1992) Influence of glyphosate on rhizoctonia root rot, growth, and yield of barley. Plant Dis 76:937–942
Smith SE (1980) Mycorrhizas of autotrophic higher plants. Biol Rev 55:475–510
Smith SE, Gianinazzi-Pearson V (1988) Physiological interactions between symbionts in vesicular-arbuscular mycorrhizal plants. Ann Rev Plant Physiol Plant Mol Biol 39:221–244
Smith SE, Read DJ (2008) Mycorrhizal symbiosis, 3rd edn. Academic Press, London
Song T, Martensson L, Eriksson T (2005) Biodiversity and seasonal variation of the cyanobacterial assemblage in a rice paddy field in Fujian, China. FEMS Microbiol Ecol 54:131–140. https://doi.org/10.1016/j.femsec.2005.03.008
Sorensen AE (1985) Seed dispersal and the spread of weeds. In: Delfosse ES (ed) Proceedings of VI international symposium of biological control of weeds, vol 19, Vancouver, Canada, 19–25 Aug 1984

Souza LAB, Silva Filho GN, Oliveira VL (2004) Eficiência de fungos ectomicorrízicos na absorção de fósforo e na promoção do crescimento de eucalipto. Pesq Agropec Bras 39:349–355

Spiller H, Gunasekaran M (1990) Ammonia-excreting mutant strain of the cyanobacterium *Anabaena variabilis* supports growth of wheat. Appl Microbiol Biotechnol 33:477–480. https://doi.org/10.1007/BF00176670

Srinivasan R, Alvarez JM, Bosque-Pérez NA, Eigenbrode SD, Novy RG (2008) Effect of an alternate weed host, hairy nightshade, *Solanum sarrachoides*, on the biology of the two most important potato leafroll virus (Luteoviridae: *Polerovirus*) vectors, *Myzus persicae* and *Macrosiphum euphorbiae* (Aphididae: Homoptera). Environ Entomol 37:592–600

Starratt AN, Lazarovits G (1996) Increase in free amino acid levels in tomato plants accompanying herbicide-induced disease resistance. Pestic Biochem Physiol 54:230–240

Stecca CS, Bueno AF, Pasini A, Silva DM, Andrade K, Filho DM (2016) Side-effects of glyphosate on the parasitoid *Telenomus remus* Nixon (Hymenoptera: Platygastridae). Neotrop Entomol 45:192–200. https://doi.org/10.1007/s13744-016-0363-4

Stewart RY (1973) Nitrogen fixation by photosynthetic microorganisms. Annu Rev Microbiol 27:283–318

Stewart WDP, Haystead A, Dharmawardene MWN (1975) Nitrogen assimilation and metabolism in blue-green algae. IBP nitrogen fixation by free-living microorganisms, vol 6. Cambridge University Press, Camberidge, pp 129–159

Stoklosa A, Nandanavanam R, Puczel U, Upadhyaya MK (2011) Influence of isoxaflutole on colonization of corn (*Zea mays* L.) roots with arbuscular mycorrhizal fungus *Glomus intraradices*. Can J Plant Sci 91:143–145

Swift C (2010) Dodder. http://www.colostate.edu/Dept/CoopExt/4dmg/Weed/dodder.htm

Tajuddin N, Subramanian G (2005) Cyanobacterial biodiversity and potential applications in biotechnology. Curr Sci 89:47–57

Tamogami S, Kodama O, Hirose K, Akatsuka T (1995) Pretilachlor [2-chloro-N-(2,6-diethylphenyl)-N-(2-propoxyethyhl) acetamide]- and butachlor [N-(butoxymethyl)-2-chloro-N-(2,6-diethlphenyl)acetamide]-induced accumulation of phytoalexin in rice (*Oryza sativa*) plants. J Agric Food Chem 43:1695–1697

Taubenhaus JJ, Ezekiel WN (1936) A rating of plants with reference to their relative resistance or susceptibility to *Phymatotrichum omnivorum*. Texas Agric Exp Stn Bull 527:52

Tejera N, Lluch C, Martínez-Toledo MV, González-López J (2005) Isolation and characterization of *Azotobacter* and *Azospirillum* strains from sugarcane rhizosphere. Plant Soil 270:223–232. https://doi.org/10.1007/s11104-004-1522-7

Thompson IA, Huber DM (2007) Manganese and plant disease. In: Datnoff LE, Elmer WE, Huber DM (eds) Mineral nutrition and plant disease. American Phytopathological Society, St. Paul, pp 139–153

Thompson IA, Guest CA, Schulze DG, Huber DM (1998) Manganese reduction and uptake in wheat rhizospheres as influenced by manganese reducing and oxidizing bacteria. Phytopathol 88:S118

Thompson IA, Huber DM, Guest CA, Sculze DG (2005) Fungal manganese oxidation in a reduced soil. Environ Microbiol 7:1480–1487

Thurston CF (1994) The structure and function of fungal laccases. Microbiol 140:19–26

Tisserant B, Gianinazzi-Pearson V, Gianinazzi S, Gollotte A (1993) In planta histochemical staining of fungal alkaline phosphatase activity for analysis of efficient arbuscular mycorrhizal infections. Mycol Res 97:245–250

Toledo REBD, Victoria Filho R, Alves PLDCA, Pitelli RA, Lopes MAF (2003) Faixas de controle de plantas daninhas e seus reflexos no crescimento de plantas de eucalipto. Sci Forum 26:78–92

Tomkiel M, Baćmaga M, Wyszkowska J, Kucharski J, Borowik A (2015) The effect of carfentrazone-ethyl on soil microorganisms and soil enzymes activity. Archiv Environ Protect 41:3–10. https://doi.org/10.1515/aep-2015-0025

Topp ED, Xun LY, Orser CS (1992) Biodegradation of the herbicide bromoxynil (3, 5-dibromo-4-hydroxybenzonitrile) by purified pentachlorophenol hydroxylase and whole cells of Flavobacterium sp. strain ATCC 39723 is accompanied by cyanogenesis. Appl Environ Microbiol 58:502–506

Toubia-Rahme H, Ali-Haimoud DE, Barrett G, Albertini L (1995) Inhibition of *Dreschlera teres* sclerotioid formation in barley straw by application of glyphosate or paraquat. Plant Dis 79:595–598

Trappe JM, Molina R, Castellano M (1984) Reactions of mycorrhizal fungi and mycorrhizal formation to pesticides. Annu Rev Phytopathol 22:331–359

Tripathi SK, Sathie KB, Vyas SC (1988) Chemical control of collar rot of pea. Indian Phytopathol 41:143–144

Tubajika KM, Damann KE (2002) Glufosiante-ammonium reduces growth and aflatoxin B1 production by *Aspergillus flavus*. J Food Prod 65:1483–1487

Tuffi Santos LD, Ferreira FA, Ferreira LR, Duarte WM, Tiburcio RAS, Santos MV (2006) Intoxicação de espécies de eucalipto submetidas à deriva do glyphosate. Planta Daninha 24:359–364

Tuor U, Wariishi H, Schoemaker HE, Gold MH (2002) Oxidation of phenolic arylglycerol .beta.-aryl ether lignin model compounds by manganese peroxidase from Phanerochaete chrysosporium: oxidative cleavage of an .alpha.-carbonyl model compound. Biochemistry 31(21): 4986–4995

Uchimiya H, Iwata M, Nojiri C, Samarajeewa PK, Takamatsu S, Ooba S, Anzai H, Christensen AH, Quail PH, Toki S (1993) Bialaphos treatment of transgenic rice plants expressing the *bar* gene prevents infection by the sheath leaf blight pathogen (*Rhizoctonia solani*). Nat Biotechnol 11:835–836

Vaishampayan A (1984) Biological effects of an herbicide on a nitrogen-fixing cyanobacterium (blue-green alga): an attempt for introducing herbicide-resistance. New Phytol 96:7–11, PMID: 808179, http://dx.doi.org/10.1111/j.1469-8137.1984.tb03537.x

Van Buskirk J, Ostfeld RS (1995) Controlling Lyme disease risk by modifying the density and species composition of tick hosts. Ecol Appl 5:1133–1140

Vannette RL, Hunter MD (2009) Mycorrhizal fungi as mediators of defence against insect pests in agricultural systems. Agric Forest Entomol 11:351–358. https://doi.org/10.1111/j.1461-9563.2009.00445.x

Vasavan MG, Rajan KM, Thomas MJ (1980) Herbicides in plant disease control. Int Rice Res New 5:18

Vázquez MB, Bianchinotti MV (1999) Fungi degrading metsulfuron methyl in agricultural soils of Argentina. Int J Exp 52:59–62

Venkataraman GS (1981) Algal biofertilizer for rice. IARI, New Delhi, India

Viator BJ, Griffin JL, Ellis JM (2002) Red morning glory (*Ipomoea coccinea*) control with sulfentrazone and azafenidin applied at Layby in sugarcane (*Saccharum* spp.). Weed Technol 16:142–148

Vieira RF, Silva CMMS, Silveira APD (2007) Soil microbial biomass C and symbiotic processes associated with soybean after sulfentrazone herbicide application. Plant Soil 300:95–103

Wan MT, Rahe JE, Watts RG (1998) A new technique for determining the sublethal toxicity of pesticides to the vascular-arbuscular mycorrhizal fungus *Glomus intraradices*. Environ Toxicol Chem 17:1421–1428

Wang Y, Browning M, Ruemmele BA, Bridger A, Chandlee JM, Kausch AP, Jackson N (2003) Glufosinate reduces fungal diseases in transgenic glufosinate-resistant bentgrasses (*Agrostis* spp.). Weed Sci 51:130–137

Ward EWB (1984) Suppression of metalaxyl activity by glyphosate. Evidence that host defence mechanisms contribute to metalaxyl inhibition of *Phytopthora megasperma* f. sp. *glycinea* to soybeans. Physiol Plant Pathol 25:381–386

Wardle DA, Parkinson D (1991) Relative importance of the effect of 2,4-D, glyphosate, and environmental variables on the soil microbial biomass. Plant Soil 134:209–219

Wariishi H, Valli K, Renganathan V, Gold MH (1989) Thiol-mediated oxidation of nonphenolic lignin model compunds by manganese peroxidase of *Phanerochyte chrysosporium*. J Biol Chem 264:14185–14191

Wheeler H (1975) Plant pathogenesis. Springer, Berlin

Wiese MV (1987) Compendium of wheat disease, 2nd edn. APS Press, The American Phytopathological Society

Wilkinson V, Lucas RL (1969) Effects of herbicides on the growth of soil fungi. New Phytol 68:709–719

Willment JA, Martin DP, Rybicki EP (2001) Analysis of the diversity of African streak mastreviruses using PCR-generated RFLP's and partial sequence data. J Virol 93:75–87. https://doi.org/10.1094/PHYTO.2002.92.1.81

Wilson JT, Greene S, Alexander M (1979) Effect of interactions among algae on nitrogen fixation by blue-green algae (cyanobacteria) in flooded soils. Appl Environ Microbiol 38:916–921

Wisler GC, Norris RF (2005) Interactions between weeds and cultivated plants as related to management of plant pathogens. Weed Sci 53:914–917. https://doi.org/10.1614/WS-04-051R.1

Wong ATS, Tylka GL, Hartzler RG (1993) Effects of eight herbicides on in vitro hatching of *Heterodera glycines*. J Nematol 25:578–584

Woolhouse MEJ, Taylor LH, Haydon DT (2001) Population biology of multihost pathogens. Science 292:1109–1111

Wyse DL, Meggitt WF, Penner D (1976) Effect of herbicides on the development of root rot on navy bean. Weed Sci 24:11–15

Wyss GS, Müller-Schärer H (2001) Effects of selected herbicides on the germination and infection process of *Puccinia lagenophora*, a biocontrol pathogen of *Senecio vulgaris*. Biol Control 20:160–166

Wyszkowska J, Tomkiel M, Baćmaga M, Borowik A, Kucharski J (2016) Response of microorganisms and enzymes to soil contamination with a mixture of pethoxamid terbuthylazine. Environ Earth Sci 75:1285. https://doi.org/10.1007/s12665-016-6092-5

Xiao CL (2006) Postharvest fruit rots in d'Anjou pears caused by Botrytis cinerea, *Potebniamyces pyri*, and *Sphaeropsis pyriputrescens*. Plant Health Prog. https://doi.org/10.1094/PHP-2006-0905-01-DG

Yadav JS, Reddy CA (1993) Mineralization of 2,4-D and 2,4,5-trichlorophenoxyacetic acid by *Phanerochaete chrysosporium*. Appl Environ Microbiol 59:2904–2908

Yamada T (2006) Informac¸ ões Agronomicas 116. IPNI-International Plant Nutrition Institute, Piracicaba-SP, Brazil. http://www.ipni.org.br/ppiweb/pbrazil.nsf/$webindex/article=93F356080325 70B5004E419AD0187284!opendocument

Yamanaka T (1983) Effect of paraquat on growth of Nitrosomonas europaea and Nitrobacter agilis. Plant Cell Physiol 24:1349–1352

Yarnia M, Rezaei F (2006) Evaluation of allelopathical effects of *Chenopodium album*, *Amaranthus retroflexus*, and *Cynodon dactylon* on germination and seedling growth of rapessed. In: Nezamabadi N (ed) Proceedings of the 17th Iranian plant protection congress, vol III, Weeds, 2–5 Sept 2006, Campus of Agriculture and Natural Resources, University of Tehran, Karaj, p 16

Yasari E, Esmaeli A, Pidashti AM, Mozafari S (2008) *Azotobacter* and *Azospirillum* inoculants as biofertilizers in canola (*Brassica napus* L.) cultivation. Asian J Plant Sci 7:490–494. https://doi.org/10.3923/ajps.2008.490.494

Yoshida S, Maruyama S, Nozaki H, Shirasu K (2010) Horizontal gene transfer by the parasitic plant *Striga hermonthica*. Science 328:1128. https://doi.org/10.1126/science.1187145

Yotsova EK, Stefanov MA, Dobrikova AG, Apostolova EL (2017) Different sensitivities of photosystem II in green algae and cyanobacteria to phenylurea and phenol-type herbicides: effect on electron donor side. Z Naturforsch C 72:315. https://doi.org/10.1515/znc-2016-0089

Yu SM, Templeton GE, Wolf DC (1988) Trifluralin concentration and the growth of *Fusarium solani* f. sp. *cucurvitae* in liquid medium and soil. Soil Biol Biochem 20:607–612

Yueh LY, Hensley DL (1993) Pesticide effect on acetylene reduction and nodulation by soybean and lima bean. J Am Soc Hortic Sci 118:73–76

Zaid AM, Mayouf M, Said YF (2014) The effect of pre-emergent herbicides on soil microflora and N-fixing bacteria in pea field. Int J Sci: Basic and Appl Res 15:131–138

Zaller JG, Heigl F, Ruess L, Grabmaier A (2014) Glyphosate herbicide affects belowground interactions between earthworms and symbiotic mycorrhizal fungi in a model ecosystem. Sci Rep 4:5634. http://dx.doi.org/5610.1038/srep05634

Zhu X, Williamson PR (2004) Role of laccases in the biology and virulence of *Cryptococcus neoformans*. FEMS Yeast Res 5:1–10