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The Influence of Atrazine (2-chloro-4-(ethylamino)-6-(isopropylamino-s-triazine) on the Severity of Gibberella zeae-Induced Seedling Blight of Corn

Louis A. Heaton
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The Influence of Atrazine (2-Chloro-4 (ethylamino)

-6- (isopropylamino-S-triazine) on the Severity

(TITLE)

Gibberella zeae-induced seedling Blight of Corn.

BY

Louis A. Heaton

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THE INFLUENCE OF ATRAZINE (2-chloro-4-(ethylamino)-6-(isopropylamino)-s-triazine) ON THE SEVERITY OF GIBBERELLA ZEAE-INDUCED SEEDLING BLIGHT OF CORN

BY

LOUIS A. HEATON

B.S., Eastern Illinois University, 1978

ABSTRACT OF A THESIS

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Abstract

Three corn inbreds were grown in artificially inoculated, steamed greenhouse soil amended with atrazine (2-chloro-4-(ethylamino)-6-(isopropylamino)-s-triazine) to determine the herbicide's effect on the severity of Gibberella zeae (Schwabe) Petch-induced seedling blight. Corn inbreds used were Gibberella-resistant (FR632), Gibberella-intermediate (FRMo17), and Gibberella-susceptible ($C123^{HT}$). Soils were amended with atrazine at rates of 2, 15, and 30 ppm.

Susceptible inbred seedlings showed no response to the herbicide with symptims equally severe in controls and all 3 concentrations of atrazine. Intermediate inbred seedlings grown in all atrazine amended soils showed significantly more severe disease symptoms than controls. Soil amended with 15 ppm atrazine produced the most severely infected plants with 30 and 2 ppm showing less severe symptoms respectively. Resistant inbred seedlings showed significantly less severe symptoms than controls when grown in soils amended with 15 and 30 ppm atrazine, while soil amended with 2 ppm produced seedlings with symptoms significantly more severe than those in the control.

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Note

For the sake of uniformity, this author has converted all units reported in the literature to parts per million (ppm).

Introduction

In the decades since the 1940's herbicides have played an increasingly important role in modern agriculture. The use of herbicides can increase yields, reduce labor costs, and, thus, reduce production costs.

Regardless of application methods employed, most herbicides eventually find their way into soils. Although much developmental research is carried out to determine the selective phytotoxicity of each chemical, the persistence of the herbicide and its toxicity to animals, little or no consideration has been given to interactions among soil microorganisms, including both saprophytes and plant pathogens and herbicides.

Audus (1964, 1970), Bollen (1961) and Fletcher (1960) indicated that certain herbicides may cause a temporary increase or decrease in total soil microbial populations, but concluded that long term effects seldom occur. Moore and Thurston (1970), however, found little experimental evidence that herbicides used on agricultural crops in England affect diseases. Katan and Eshel (1973) and Kavanagh (1969, 1974) citing many examples from the literature concluded that herbicides may interact with plant pathogens to increase or decrease plant disease.

Houseworth and Tweedy (1972) established that an interaction existed between corn seedling blight caused by Gibberella zeae (Schwabe) Petch and atrazine.

The purpose of this research was to investigate the effect of atrazine, a triazine herbicide, on the severity of Gibberella zeae- induced seedling blight of corn.

Literature Review

Herbicide-Pathogen Interactions

Crop plants form many different kinds of relationships with other organisms. One such relationship is that between crop plants and pathogens (agencies which incite disease) (59). Plant disease is the final result of a compatible interaction that occurs under suitable conditions between a particular pathogen and host (59). A third component in the association, in addition to the host and pathogen, is the surrounding microflora and fauna which may be antagonistic or synergistic to either the host or pathogen. Herbicides may interact in different ways with any one of more of the organisms involved to cause an increase or decrease in disease incidence or severity. Extreme cases are the out break of a new disease or the elimination of disease after the use of a chemical herbicide (31).

Katan and Eshel (1973) suggested four possible mechanisms involved in disease increase after the application of chemical herbicides. The first mechanism is direct stimulatory effects on the pathogen. A herbicide may stimulate the growth and reproduction of a fungus causing an increase in population density which in turn causes an increase in disease incidence. Since the effects on a single organism can be studied only in pure culture, liquid or solid culture media have been employed. Parameters used to measure fungal response to herbicides have been: (1) mycelial weight; (2) linear growth, e.g., diameter of colony; (3) size

of inhibition zone around paper discs containing the herbicide; (4) rate of physiological activities such as nutrient utilization, production of specific metabolites, respiration, enzymatic activity, etc.; and (5) reproduction, e.g., density of spores and sclerotia produced (31).

Altman (1969) measured the linear growth of Rhizoctonia solani Kuhn on solid media containing one of 25 commercial herbicides. In all instances the fungus was stimulated by herbicide concentrations of 1 to 1,000 ppm. EPTC increased mycelial production by Fusarium oxysporum Schlecht. at concentrations of 10 and 25 ppm (41). Sikka, Couch, Davis and Funderburk (1965) found that atrazine at 10 ppm in liquid culture stimulated the growth of Fusarium spp.

Most studies report an inhibition of pathogen growth by chemical herbicides. Some studies have shown that stimulation or inhibition is a function of herbicide concentration, with low concentrations being stimulatory while higher concentrations of the same chemical are inhibitory. Katan and Eshel (1973) warn that care must be exercised when in vitro data are translated to field conditions because: (1) the biological activity of a herbicide in soil is usually much lower than in culture due to soil physico-chemical processes; (2) since herbicides may be translocated in the plant or secreted into the rhizosphere or at the leaf surface, their biological activity will also depend on the plant species; (3) in a natural environment, the herbicide might effect the pathogen in one way and its antagonists in another; and (4) the behavior of a pathogen and its response to toxicants in its natural ecosystem might be different from those occurring when the

pathogen is grown in isolation. They feel that studies done in sterile soil may be more readily translated because the conditions more closely resemble those in the field.

The results of various experiments using sterile soil demonstrate that herbicides have highly variable effects on the soil mycoflora. In their review, Kaiser, Pochon and Cassini (1970) cited four authors reporting no effect, six authors reporting stimulation, and eight authors reporting inhibition of the soil mycoflora or certain species by triazine herbicides.

Percich and Lockwood (1975) found an increase in Fusarium solani (Mart.) Appel and Wr. populations in sterile soil after the addition of 2.5 to 25 ppm atrazines. Tang, Curl and Rodriguez-Kabana (1970) reported that chlamydospore production by Fusarium oxysporum is enhanced by low concentrations (0.6 to 5.0 ppm) of trifluralin. Mycelial growth may or may not be effected by the herbidice, but innoculum density can be effected by the stimulation of the production of the resistant spores. In the same study, spore germination was found to be enhanced by low concentrations (0.6 to 5.0 ppm) of the herbicide. Certain species may be stimulated at one concentration and inhibited at another, while others show the same effect over a range of concentrations. Carbon dioxide production by Sclerotium rolfsii Tode is stimulated by low concentrations (8.0 ppm) of atrazine but inhibited at higher concentrations (20 to 80 ppm) (50). In the same study, Trichoderma viride Pers. was found to be stimulated by 8 to 80 ppm atrazine. The same herbicide increases carbon dioxide production by Fusarium oxysporum over a range of concentrations. The fungus was especially

stimulated at higher concentrations (20 to 80 ppm) (48). The increased carbon dioxide produced in this case may be due to an atrazine-induced change in the metabolism of the fungus. It is unreported whether or not there is an accompanying increase in mycelial mass. Whether or not the altered metabolism will cause an increase in disease incidence is speculative (48).

The second mechanism of increased disease incidence after herbicide application suggested by Katan and Eshel (1973) is an increase in pathogen virulence. Since pathenogensis involves enzymatic activity and toxin production, any environmental factor that effects the pathogen metabolism can effect the virulence of that particular organism. Beam and Curl (1971) measured enzyme activity in a monoculture of Rhizoctonia solani in soil amended with 1 to 40 ppm of fluometuron and prometryne. Concentrations of 1 ppm of fluometuron and 1, 5, and 10 ppm of prometryne enhanced the activity of B-galactosidase. Carbon dioxide evolution by the fungus was found to be stimulated by 1, 5, 10, and 20 ppm of prometryne, while NO_3-N utilization efficiency was increased by 20 ppm of prometryne. Carbon dioxide evolution by Fusarium oxysporum was also stimulated by 20, 40 and 80 ppm of atrazine. Since there was a depression of mycelial production in liquid culture with the same herbicide concentrations, the increased carbon dioxide production in this case is the result of an atrazine induced change in the metabolism of the fungus (45). Whether or not these changes in metabolism will cause an increase in disease incidence is, as stated above, speculative (48). Katan and Eshel (1973) report a lack of research in the area of

herbicide effects on the virility of pathogens.

The third suggested mechanism of increased disease incidence is an increase in host susceptibility due to herbicide presence. Altman (1969) reported that glucose exudates at the host-soil interface were increased in herbicide treated soils making the plants more susceptible to attack by Rhizoctonia solani. In a seperate study, Altman (1972) reported a 50% increase in damping-off in sugar beets after pyramin application at a rate of 2 ppm. Again, there was an increased glucose exudation that predisposed the plants to increased disease. Lai and Semeniuk (1970) found that exuded carbohydrates increased as much as 430% in picloram-treated corn seedlings. Incorporated trigluralin stunted and predisposed cotton seedlings to damping-off (43).

Katan and Eshel (1973) state that chemical herbicides can increase the susceptibility of host plants by interfering with their defense mechanisms by any one of the following: (1) induction of direct or indirect morphological and anatomical changes by altering the growth pattern of plant tissues, which might facilitate and enhance the penetration and colonization of the tissues by the pathogen; (2) alteration of the composition of the tissues, rendering them more suitable for the growth of the pathogen, or for toxin production; (3) stimulation of root exudation which, in turn, stimulates soil-born pathogens; or (4) affecting the chemical defense mechanisms, preformed or induced, which enable the host to suppress growth and the metabolic activity of the pathogen.

The fourth, and final, suggested mechanism of increased disease incidence is the effect of herbicides on the relationships between

pathogens and organisms other than the host. In a natural soil, pathogens co-exist with the surrounding microflora and fauna. Interactions between pathogens and other microorganisms play an important role in determining the inoculum density and the ability of the pathogen to survive in the soil in the absence of a suitable host. Certain species of microorganisms are known to be antagonistic to pathogens. Just as the pathogen can be inhibited or affected in some way by the herbicide. Alexander (1961) suggested possible mechanisms of antagonism in natural, unamended soil. They are: (1) competition for limited quantities of nutrients, oxygen, space, or other common requirements; (2) the release of toxic products (antibiotics) which inhibit the growth of the pathogen; and (3) direct parasitism or predation. The ability of a chemical herbicide to inhibit or effect in some way any one of these antagonist mechanisms makes the environment more favorable for the growth of the pathogen. Even if the pathogen is found to be inhibited in sterile soil, there may be an increase in disease incidence in the field if antagonists are inhibited to a greater degree than the pathogen.

Wilkinson and Lucas (1969) induced competition between pairs of fungi in the presence of herbicides. In the presence of paraquat, the growth of the pathogen Fusarium culmonorum (W.G. Sm.) Sacc. was favored over that of its antagonist Trichoderma viride. Other studies show the opposite effect; that is, stimulation of the antagonist and inhibition of the pathogen (3, 17, 18, 32, 47). This type of interaction will be discussed in the following paragraphs.

The opposite effect, a decrease in disease incidence, is also a possibility with the use of chemical herbicides. This type of herbi-

cidal effect deserves much attention because of its potential usefulness in disease control. The mechanisms involved are the opposites of those that operate in increased disease incidence (31).

The first of these mechanisms is the direct inhibition of a pathogen by a herbicide. The techniques used are the same as those discussed in earlier paragraphs concerning increase in disease incidence. Bever and Slife (1948) grew Gibberella zeae (Schwabe) Petch on potato dextrose agar amended with 2,4-D. They found that concentrations of 250 to 2,000 ppm of this growth regulator severely inhibited the growth of the fungus; however, the concentrations used were much greater than those found in agricultural situations. Bozarth and Tweedy (1971) grew Sclerotium rolfsii on potato dextrose agar amended with one of five different herbicides. They found thiram to be the most effective inhibitor with 1 ppm causing a 27.4% decrease in radial growth of the fungus. At 50 ppm, atrazine, fluometuron, metobromuron, trifluralin, and thiram all caused significant decreases in fungal growth with thiram being the most effective and atrazine the least effective inhibitor. They also found that all five reduced the quantity of sclerotia produced. Those sclerotia produced in the presence of the herbicides were larger than those produced by the control.

Cohen, Lattar, and Barkai-Golan (1965) measured the effects of NAA, 2,4-D, and 2,4,5-T on the growth and spore production of 14 fungi. They found that 10 to 5,000 ppm concentrations of all three growth regulators also inhibited spore germination, but there were not always correlations between effects on vegetative growth and spore germination. 2,4-D at 100 ppm in Fries nutrient solution reduced Fusarium sp. growth

by 75%. Millikan and Fields (1964) found that simazine and amitrole at 10 ppm reduced Fusarium growth by 71 and 94%, respectively. Simazine and amitrole together reduced growth 87%.

In liquid culture, atrazine at 40 to 80 ppm retards the growth of Fusarium oxysporum (46). Likewise, Sclerotium rolfsii was inhibited by atrazine concentrations greater than 40 ppm. Houseworth and Tweedy (1972) found that atrazine inhibited the growth of Gibberella zeae on potato dextrose agar by 10%.

Wilkinson and Lucas (1969) studied the effects of five herbicides on eight genera of fungi grown on liquid and solid media. They found that none of the herbicides stimulated growth, but that linuron and paraquat were more fungitoxic than MCPA and simazine.

Care must be exercised when attempting to translate pure culture data to field conditions. Studies done in sterile soil may be more readily translated since the conditions more closely resemble those in the field (31). Tang et al (1970) found that chlamydospore production by Fusarium oxysporum and germination of the spores decreased with increasing concentrations of trifluralin. Chopra, Curl, and Rodriguez-Kabana (1970) found that increasing concentrations of prometryne had the same effect on the fungus. Curl and Funderburk (1965) found that the density of fungi and bacteria decreased rapidly with increasing atrazine concentrations. Carbon dioxide evolution by Sclerotium rolfsii was restricted in sterile soil amended with 12.5 to 1,000 ppm of paraquat (49).

Many workers have conducted greenhouse studies using one particular herbicide-pathogen-host system. If there is a decrease in disease

incidence or severity in the presence of a herbicide, it's impossible to determine whether the host was made more resistant or the pathogen less virulent, or whether the growth of the pathogen was directly inhibited.

Buczacki (1973) grew cabbages in soil infested with Plasmodiophora brassicae Wor. and amended with various concentrations of trifluralin. He found that the incidence and severity of clubroot was reduced when the herbicide was present. Chandler and Santelmann (1968) found that trifluralin and prometryne reduced the effect of Rhizoctonia solani on cotton seedlings. However, at the concentrations required to supress the effect of the fungus, cotton seedlings were stunted, injured, or killed.

In several instances antagonistic organisms have been found to be stimulated by herbicides. The increased activity of an antagonist may reduce the activity of a pathogen to cause a decrease in disease incidence or severity. In flask shake cultures, the percentage of fungal and bacterial isolates inhibitory to Sclerotium rolfsii reached a peak at 20 ppm of atrazine, then declined at higher concentrations. The toxic zone of inhibition produced by a bacterium became progressively wider with increased concentration of herbicide in Czapek's atrazine agar (17). Curl, Rodriguez-kabana, and Funderburk (1968) measured carbon dioxide evolved from soil amended with atrazine and found that the pathogen S. rolfsii was inhibited while it's antagonist, Trichoderma viride, was stimulated. Rodriguez-kabana et al (1967) measured the growth of S. rolfsii and T. viride in liquid culture containing atrazine. S. rolfsii was little effected at concentrations under 40 ppm, but was

inhibited at higher concentrations, while the growth of the antagonist was stimulated at all concentrations. In a separate study, the same authors found the same fungi behave similarly when grown in soil amended with atrazine (50).

Observations from the field showed that fall applications of diuron reduced the incidence and severity of foot rot of wheat caused by Cercosporella herpotrichoides Fron. and Rhizoctonia solani. The herbicide had been applied at a rate of 1 lb/A.

Kaufman (1964) applied simazine and atrazine to corn-cropped soil, and linuron and diuron to both corn- and soybean-cropped soil. Effects on soil fungi were determined by the use of a soil dilution-plate method. Numbers of Fusarium spp. in atrazine-treated soils were lower than in simazine-treated soils. Linuron and diuron decreased the number of Fusarium spp. in soybean-cropped soil, but had no effect in corn-cropped soil. All four chemicals stimulated one or more genera of fungi known to be antagonistic to Fusarium.

As is the case with increased disease incidence, no general statements can be made concerning the effects of herbicides on decreases in disease incidence. In fact, the same chenical may reduce the incidence of one disease but increase another (31). The effect depends on the herbicide used and the pathogen in question. Each herbicide must be studied with each pathogen and host to determine what the effects are on disease incidence, if any. Reports are varied and in some cases opposite effects have been reported for the same herbicide-pathogen-host system. Davis and Dimond (1953) reported a decrease

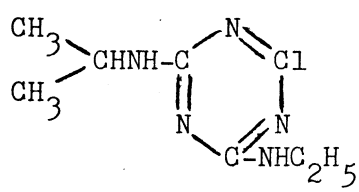
of Fusarium wilt after the application of 2,4-D, while Richardson (1959) reported an increase of the same disease after using the same chemical. It was found that the time of application was responsible for the difference in effects reported. This descrepency illustrates the necessity of controlled experiments.

Since a chemical herbicide effects different organisms in different ways, each organism must be studied separately to determine what the effects are on that particular organism, and on the relationships into which that organism enters.

Atrazine

Atrazine is the common name for 2-chloro-4-(ethylamino)-6-(isopropylamino)-s-triazine. United States trade names are: (1) AAtrex 80W; (2) AAtrex 4L; (3) AAtrex Nine-0; (4) AAtram 20G (a combination of atrazine plus sodium chlorate plus sodium metaborate); and (6) Atratol 80W (a combination of atrazine plus prometon). European designations are: (1) Gesaprim (for nonselective use); and (2) Primatol (for nonselective use).

Its structural formula is:



Its molecular formula is: $C_8J_{14}C1N_5$. It has a molecular weight of 215.7 daltons. Atrazine is a white crystalline solid with a melting point of 173-175°C. The AAtrex brand of atrazine is subject to decomposition by ultraviolet irradiation, but under normal field conditions this effect is small. Atrazine is manufactured by the Ciba-Geigy Corporation (2).

Atrazine is a widely used selective herbicide for control of broadleaf and grassy weeds in corn, sorghum, sugarcane, macadamia orchards, pineapple, and turf grass sod. It is used also in some areas for selective weed control in conifer reforestation, Christmas tree plantations and grass seed fields, as well as for nonselective control of vegetation in chemical fallow. Atrazine also is used widely as a nonselective herbicide for vegetation control in non-crop land. Future use of atrazine is envisioned in the following areas among others: establishment and renovation of rangeland, chemical fallow, and in minimum tillage corn and sorghum. Granular formulations containing only atrazine for herbicidal use are not marketed at the present time. Combination granules of atrazine plus propachlor, atrazine plus alachlor and atrazine plus Sutan are presently marketed for weed control in corn. In addition, the granular combination of AAtrex plus sodium chlorate and sodium metaborate is currently marketed for nonselective vegetation control. Sugarbeets, tobacco, oats, and many vegetable crops are very sensitive to atrazine (2).

Depending upon the crop or intended use, atrazine sprays may be applied preplant, preemergence, or postemergence, but before weed seedlings are more than $1\frac{1}{2}$ inches high with few exceptions. These exceptions include postemergent application for yellow nutsedge and Canada thistle control. Preemergence use is generally the preferred application method where it can be used. Under dry conditions, a shallow incorporation may increase the degree of weed control. A single layby cultivation is sometimes useful to prevent relatively tolerant late-season grasses from developing. Aerial applications have been very success-

ful, especially when wet weather prevents the use of ground equipment and in cases where rough terrain such as in conifer reforestation makes ground applications impractical. A liquified formulation containing 4 lb ai/gal. has been developed and is currently registered for weed control in corn and sorghum. Future registration of the 4L formulation is scheduled for sugarcane and other crops. Postemergent application of either the wettable powder or liquified formulation of atrazine are usually made in combination with a nonphytotoxic crop oil, crop oil concentrate or surfactant. These additions enhance the uptake of atrazine and hence its activity (2).

Rates the equivalent of 2 to 4 lb/A. are required for selective weed control for most situations. Higher rates are used for nonselective weed control. Lower rates will effectively control cheatgrass and most other weeds in chemical fallow or rangeland uses, and many common annual broadleaf weed species (2).

Water at 10 gpa or more is the usual carrier for unigorm ground application. Nitrogen solution and other liquid fertilizers have been widely and successfully used as carriers. The major advantage is in applying both herbicide and fertilizer in one operation. Agitation in the spray tank is necessary to keep the chemical in suspension. Recently it has been recommended that aerial application of atrazine be applied in a minimum of 2 gallons of water per acre. Use is widely made of nonphytotoxic oils in combination with postemergent application of atrazine for weed control in corn and sorghum. The typical volume of oil applied by ground means is one gallon per acre. This volume is reduced to one-half gallon per acre for aerial application.

An attempt is now in progress to obtain registration of oil concentrates for use with postemergent applications of atrazine. These oil concentrates will be applied at approximately one-fourth the volume of that of the nonphytotoxic oils (2).

The use of surfactants with postemergent application of atrazine for weed control in sorghum is recommended under certain conditions (2).

Atrazine is absorbed through both roots and foliage, although foliar absorption often is small in most plants under field conditions, depending on factors as species and environmental conditions. The herbicide can be washed off plant foliage by rain. Following absorption through roots and foliage, it is translocated acropetally in the sylem and accumulated in the apical meristems and leaves of plants. It is a photosynthetic inhibitor, but may have additional effects (2).

Atrazine is readily metabolized by tolerant plants to hydroxy-atrazine and amino conjugates. The hydroxy-atrazine can be further degraded by dealkylation of the side chains and by hydrolysis of resulting amino groups on the ring and some CO_2 production. These alterations of atrazine are major protective mechanisms in most tolerant crop and weed species. Soil placement selectivity is also important in the care of some deep-rooted perennial crops. Unaltered atrazine accumulated in sensitive plants, causing chlorosis and death (52).

Atrazine is more readily absorbed on muck or clay soils than on soils of low clay and organic matter content (56). The downward movement or leaching is limited by its absorption to certain soil constituents. Absorption is not irreversible and desorption often occurs readily, depending on such factors as temperature, moisture, and pH (55). Atrazine normally is not found below the upper foot of soil in detectable quantities, even after years of continuous use (5).

Microbial activity possibly accounts for decomposition of a significant portion of atrazine in the soil (12). A range of soil microorganisms can utilize it as a source of energy and nitrogen. The effect of atrazine on these and other organisms appears to be small (58).

The significance of photodecomposition and/or volatilization of atrazine from soil is not fully understood. Available data indicate that both occur to some extent if high temperatures and prolonged sunlight follow application before precipitation, but that these factors are of little significance in atrazine dissipation under most field conditions (29). Atrazine is more subject to ultraviolent and volatility losses than simazine, but probably about equal or less subject to these losses compared to the commercial methylmercapto- or methoxytriazines (27).

The residual activity of atrazine in soil at selective rates for specific soil types is such that most rotational crops can be planted one year after applications, except under an arid or semi-arid climate. Atrazine will persist longer under dry and cold conditions or conditions not conducive to maximum chemical or biological activity. Broadcast rates needed in some of the heavier and relatively higher organic matter solid of the north central states result in enough residue carryover, under some donditions, to injure small grains, alfalfa, and soybeans planted 12 months later. Plant removal and chemical alteration are also factors in dissipation (2).

Effects of Atrazine on Soil Fungi

Sikka, Couch, Davis, and Funderburk (1965) determined the effect of various concentrations of atrazine on 4 common soil fungi. At 1 ppm

atrazine in liquid culture medium, the herbicide had no significant effect on any of the fungi studied. Geotrichum, Fusarium, and Trichoderma were significantly stimulated by 10 ppm of atrazine, while Penicillium showed no response at the same concentration. Further experiments showed that concentrations of 2 to 64 ppm stimulated the growth of Trichoderma. In general, Sikka et al found that the mycelial growth of Trichoderma increased with increasing concentration of the herbicide.

Rodriguez-Kabana and Curl (1970) found that higher concentrations of atrazine inhibited the mycelial growth of Fusarium oxysporum. They reported that exposure of the fungus to 40 to 80 ppm of atrazine retarded growth after 6 days but not thereafter. Soil studies have shown that there is an increase of carbon dioxide production by the fungus in the presence of 20, 40, and 80 ppm of atrazine (48). These results seem to indicate that atrazine stimulates the respiration of the fungus without an accompanying increase in mycelial weight.

In the field, Kaufman (1964) found that there were fewer Fusarium species in atrazine-treated than in simazine-treated soils. The treated soils were corn-cropped. Atrazine, as well as three other herbicides, stimulated one or more genera of soil fungi known to be antagonistic to Fusarium.

On the other hand, Percich and Lockwood (1975) reported a four-fold increase of Fusarium populations in soils treated with 10 to 100 ppm of atrazine over nonamended soil. Atrazine increased the numbers of Fusarium solani in artificially infested, steamed greenhouse soil at all concentrations studied (10, 30, and 100 ppm). At 30 ppm atrazine in artificially infested soil, the incidence of pea root rot was increased three times, and corn seedling blight twice.

Rodriguez-Kabana, Curl, and Funderburk (1966) found that the total mycelial dry weight of Rhizoctonia solani was considerably less for concentrations of 10 to 70 ppm of atrazine. The degree of growth inhibition was directly related to increased herbicide concentration. The fungus was grown in Czapek's solution.

Curl and Funderburk (1965) and Bozarth and Tweedy (1971) found that atrazine increasingly inhibited the growth of Sclerotium rolfsii with increasing concentration. The fungus was grown on solid medium containing 0 to 80 ppm of atrazine. Rodriguez-Kabana, Curl, and Funderburk (1967) also reported inhibition of S. rolfsii in the presence of atrazine, and found that an antagonist, Trichoderma viride, was stimulated by 8 to 80 ppm of atrazine.

Klyuchnikov, Petrova, and Polesko (1964) and Fink, Fletchall, and Calvert (1968) reported reductions in Aspergillus and Penicillium populations in field soils treated with atrazine; however, total fungal populations remained constant before and after the application of the herbicide.

Spiridonov and Yakovlev (1968) found that 10 to 20 kg/ha of atrazine increased the activity of cellulose-decomposing microorganisms by 30% in comparison with the control.

Gibberella zeae

The mycelium of Gibberella zeae (Syn=G. roseum, f. sp. cerealis (Cke.) Synd. & Hans.; G. saubinetti (Mont.) Sacc.) is septate, branching, and white to pink enmasse. The macroconidia are typical of Fusarium sickle-shaped, hyaline, up to five-septate, 4.3 to 5.5 by 41 to 60 u. They are produced very promptly under favorable conditions on sporodochium-like clusters of short conidiophores, where they form acrogenously. Septation often is completed after the conidium is pushed off to make room for a succeeding spore. After an initial crop of conidia is formed, the latter may germinate to form a stroma from which, after a few weeks, typical sporodochia arise (20). Microconidia and chlamydospores do not occur. The perithecia are ovoid to subconical, purplish black to dark blue, scattered over the host surface, somewhat embedded, smooth at the base with protuberant projections near the apex. Asci are cylindrical, tapering toward the base, slightly curved, hyaline. Ascospores are usually eight per ascus, fusiform, slightly curved, hyaline, mostly three-celled, 3.4 to 5.0 by 20 to 30 u. (59).

Disease Cycle

Gibberella zeae overwinters in seed or on infected debris, particularly in infected cornstalks. Perithecia may appear in the early fall, but in northern states they mature most commonly in the spring. Airborn ascospores thus serve as the chief primary inoculum. Macro- and microconidia are formed abundantly on infected plant parts in moist warm weather and serve as the airborn secondary inoculum. (59).

The details of penetration and host-parasite relations in the corn seedling have been worked out by Pearson (1931). As the adventitious roots arise in the pericycle of the stele and traverse the cortex, they form channels through which the fungus invades. The latter proceeds through the intercellular spaces of the cortex, invading the cells in the older portion of the lesion. The intercellular mycelium causes swelling of the cell walls in some regions. When a hypha penetrates from cell to cell, it often becomes greatly constricted at the point of penetration; a fine filament passes through the wall, and the mycelium then regains the original diameter as it enters the next cell cavity. In the coleorhiza, plug-like structures occur at the surface between the epidermal cells. They appear not to be cuticularized as is the rest of the epidermal layer. The pathogen penetrates the coleorhiza through the plugs, which are apparently readily dissolved by fungus secretions. The fungus develops in the epidermal wall, which swells considerable, and it also preceeds intercellularly. Cellular penetration follows after the coleorhiza has been rather completely invaded, at which time the fungus may proceed into the primary root. (59)

Soil temperature is important in determining the extent of seedling blight of corn incited by G. zeae. Dickson (19) showed that the fungus grew on agar at a range of 3 to 32 C. with an optimum at 24 to 28 C. The most favorable range for seedling blight is 8 to 20 C, while none occurs above 24 C. It is obvious that the effect of temperature on disease development is dependent upon the reaction of the host rather than of the pathogen. When the temperature is most favorable for the host, it withstands the pathogen most effectively. Eckerson and Dickson (1923) attributed the resistance or susceptibility of the plants, as influenced by temperature, to the chemical composition of cell walls and the nature of food available to the pathogen. Wheat seedlings grown at low soil temperature are high in available carbohydrates and are high in available nitrogen, while the cell walls are composed of pectic materials, cellulose being absent until after photosynthesis begins. The cell walls are, therefore, the most resistant and the food reserves least attractive to the fungus in the low-temperature plants. Corn seedlings show the reverse situation at high and low temperatures with reference to cell walls and reserves, and high-temperature plants are correspondingly the most resistant (59). When the environment is favorable for seedling blight development, control can be achieved by the use of healthy seed and fungicide treatment of the seed (39).

Materials and Methods

Soil

Steamed greenhouse soil was used in all experiments. The texture was that of sandy loam. Organic content was approximately 5.0%.

Cultural Techniques

An isolate of Gibberella zeae (Schwabe) Petch in soil was obtained from Sharon Onken of Purdue University, West Lafayette, Indiana. Soil from the original isolate was used to inoculate potato-dextrose agar (PDA) at pH 5.6 in sterile petri-plates. The inoculated agar was incubated for 3 days at 14 C. After the incubation period, 3 mm diameter plugs were taken from the margins of actively growing colonies with a sterile cork borer. Sterile greenhouse soil contained in 6-inch culture tubes was inoculated with 1 plug per tube. All tubes were stored at room temperature.

Inoculum

Inoculum for soil work was prepared by sprinkling several grains of infested soil (described above) over PDA (pH 5.6) in sterile petri plates. The plates were incubated for 10 days at 14 C. After the incubation period, the contents of 7 plates was homogenized with 350 ml sterile distilled water for 30 seconds in a disinfected Waring blender. Steamed greenhouse soil was divided into eight, 5 kg aliquots per experiment. Seventy-five grams of the homogenate plus 200 ml sterile distilled

water was thoroughly mixed with each aliquot by hand in plastic dish pans. The pans were covered with cellophane and incubated for 6 days at room temperature before each experiment. When herbicide was needed it was added immediately prior to planting.

Herbicide

The herbicide AAtrex Nine-0 (90% atrazine) was used in all herbicide tests. The chemical was supplied by the Agricultural Division of the Ciba-Geigy Corporation.

Herbicide was applied to the soil by bringing an atrazine-water stock solution to a volume of 200 ml with sterile distilled water. The solution was mixed thoroughly with 5 kg of dry soil by hand. Controls consisted of 200 ml sterile distilled water mixed with 5 kg aliquots of soil.

Corn Inbreds

Corn inbreds used were: FR 632, a Gibberella-resistant strain (39); FRMo17, a Gibberella-intermediate strain (39); and $Ci23^{HT}$, a Gibberella-susceptible strain (39). Seeds were obtained from Illinois Foundation Seeds, Urbana, Illinois.

Seedling Blight Severity

Three corn inbreds were grown in treated and control soils to determine the effect of atrazine on severity of seedling blight of corn caused by G. zeae. Treated soils consisted of those amended with atrazine, those inoculated with G. zeae, and those both amended with atrazine and inoculated with G. zeae. The control soil consisted of

soil without atrazine or G. zeae. The atrazine-soil were amended at rates of 2, 15, and 30 ppm atrazine to soil on w:w basis. The soils inoculated with G. zeae were prepared as described in previous paragraphs. Soils both amended with atrazine and inoculated with G. zeae were amended with 2, 15, and 30 ppm atrazine and inoculated as described above. The herbicide was added immediately prior to planting in all cases.

Enough of each soil was prepared to fill 3 disinfected four-inch clay pots per inbred, except in the control, where there were 4 pots planted. Clean clay pots were disinfected by soaking in a 5.0% NaOCl solution for 5 minutes, followed by 2 changes of sterile distilled waterof 5 minutes each. Five seeds were planted per pot. With 3 inbreds, there was a total of 75 pots planted per experiment. The experiment was run 3 times.

All seeds were soaked for 8 hours in distilled water before planting. They were surface disinfected in a 0.5% NaOCl solution for 45 seconds and rinsed in sterile distilled water for 15 seconds immediately prior to planting. Sterile forceps and dibbles were used during the planting process, and were resterilized between treatments. All seeds were planted to a depth of 2.5 cm with the radicle end down.

The pots were placed randomly (25) into a walk-in refrigerator whose temperature oscillated between 11 and 16 C. Light was provided by Duro-lite grow lights having a color rendering index of 91 and approximate color temperature of 5500 D. Light intensity ranged from 350 to 500 footcandles. The photoperiod was 16 hours.

The plants were harvested after 3 weeks, and evaluated for disease severity. Evaluation was done on a scale of 0-10 adapted from Onken

(1979). The scale was as follows: 0 points for no disease symptoms; 2 points for localized lesions on the stem and/or root; 4 points for lesions larger, almost running together; 6 points for lesions completely surrounding stem and/or root, but still superficial; 8 points for cortical rot; and 10 points for plants killed.

The data was analyzed statistically using the Kruskal-Wallis test (51) and rank sums were separated using a modification of the Newman-Keul's test (62).

Although all precautions were taken to keep experimental conditions constant, there was variation between runs according to the Kruskal-Wallis test. Since this was the case, data from all three experiments were pooled and subjected to statistical analysis.

Results and Discussion

According to the Kruskal-Wallis test, there was no significant difference in the severity of Gibberella-induced seedling blight in the susceptible strain of corn ($C123^{HT}$) used in these experiments. The disease was equally severe in plants grown in the presence of only the fungus and plants grown in the presence of the fungus and 2, 15, or 30 ppm atrazine. The herbicide caused neither an increase nor decrease in disease severity at any of the concentrations tested using the susceptible inbred. Rank sums are listed in Table 1.

There were significant differences in disease severity in the intermediate strain (FRMo17). Seedlings grown in all three herbicide concentrations tested were significantly more severely diseased than those grown in the presence of the fungus only. Seedlings grown in the presence of 2 ppm atrazine and G. zeae were more severely diseased than those grown in the presence of only the fungus. Seedlings grown in the presence of 30 ppm atrazine and the fungus were more severely diseased than those grown in 2 ppm atrazine and those grown in 15 ppm atrazine were more severely diseased than those grown in 30 ppm of the herbicide. Rank sums are listed in Table 1.

Seedlings of the resistant inbred (FR632) also showed significant differences in disease severity between treatments. Seedlings grown in both 15 and 30 ppm atrazine and the fungus were significantly less severely diseased than those grown in the presence of the fungus only.

Table 1. Rank sums from Kruskal-Wallis test using pooled data.

Cultivar	Rank sums			
	G. zeae[1]	2[2]	15[2]	30[2]
FR632[3]	2785.5	3006.1	2557.9	2437.7
FRMol7[3]	2474.1	2746.8	3243.9	2950.0
C123[HT]	2625.0	2772.0	2629.0	2414.0

[1]Plants grown in soil inoculated with G. zeae.

[2]Plants grown in soil inoculated with G. zeae and amended with the concentration of atrazine indicated.

[3]Inbreds showing significant difference between rank sums.

There was no significant difference in severity between the 15 and 30 ppm treatments. However, seedlings grown in the presence of 2 ppm atrazine and the fungus were significantly more severely diseased than those grown in the presence of only the fungus. Rank sums are listed in Table 1.

The herbicide-pathogen-host system is a complex one, and, as these results show, responses to different concentrations of herbicide may vary depending on the corn inbred and herbicide concentration used. As mentioned earlier, the susceptible variety showed no response at all, with the disease equally severe at all concentrations tested. Using the intermediate inbred, the disease was more severe with herbicide present at all concentrations tested. In this case the herbicide stimulates some part of the infection process to cause more severe symptoms in the host plants. The response was still different in the resistant inbred. In this case only low concentrations of the herbicide (2 ppm) stimulated the disease process, while higher concentrations inhibited some part of the process.

Several mechanisms of disease increase due to herbicide presence have been proposed (31). They are: (1) direct stimulatory effects on the pathogen; (2) an increase in pathogen virulence; (3) an increase in host susceptibility; and (4) an effect on the relationships between pathogens and organisms other than the host.

The proposed mechanisms of disease decrease due to herbicide presence are the opposites of those concerning disease increase cited above. Since steamed soil was used in this study only the fourth mechanism may be eliminated as a factor in response to atrazine presence.

Table 2. Separation of intermediate inbred seedling rank sums using a modification of the Newman-Keul's test

Comparison Difference						
(B vs. A)	$(R_B - R_A)$	SE	q	p	q0.05,00,p	Conclusion
1 vs. 2[1]	272.7	36.25	7.5228	2	4.501	Rej. H_0
1 vs. 3[2]	475.9	54.25	8.7724	3	5.910	Rej. H_0
1 vs. 4[3]	769.8	72.25	10.6547	4	6.825	Rej. H_0
2 vs. 3	203.2	36.25	5.6055	2	4.501	Rej. H_0
2 vs. 4	497.1	54.25	9.1631	3	5.910	Rej. H_0
3 vs. 4	293.9	36.25	8.1076	2	4.501	Rej. H_0

[1] 1=seedlings grown in the presence of the fungus only; 2=seedlings grown in the presence of the fungus and 2 ppm atrazine.

[2] 3=seedlings grown in the presence of the fungus and 30 ppm atrazine.

[3] 4=seedlings grown in the presence of the fungus and 15 ppm atrazine.

Table 3. Separation of resistant inbred seedling rank sums using a modification of the Newman-Keul's test.

(B vs. A)	Comparison Difference (R_B--R_A)	SE	q	p	$q_{0.05,00,p}$	Conclusion
1 vs. 2[1]	120.2	36.25	3.3159	2	4.501	Accept H_0
1 vs. 3[2]	347.8	54.25	6.4111	3	5.910	Rej. H_0
1 vs. 4[3]	568.4	72.25	7.8671	4	6.825	Rej. H_0
2 vs. 3	227.6	36.25	6.2786	2	4.501	Rej. H_0
2 vs. 4	448.2	54.25	8.2618	3	5.910	Rej. H_0
3 vs. 4	220.6	36.25	6.0855	2	4.501	Rej. H_0

[1] 1=seedlings grown in the presence of the fungus and 30 ppm atrazine; 2=seedlings grown in the presence of the fungus and 15 ppm atrazine.

[2] 3=seedlings grown in the presence of the fungus only.

[3] 4=seedlings grown in the presence of the fungus and 2 ppm atrazine.

The increases in disease severity observed in the intermediate strain at 2, 15, and 30 ppm and that in the resistant strain at 2 ppm atrazine may be due to direct stimulation of the fungus, and increase in the virulence of the fungus, an increase in corn susceptibility or any combination of these three mechanisms.

The decrease in severity in the resistant strain at 15 and 30 ppm atrazine may be the inverse of the above. Again, altered relationships between the fungus and organisms other than the host can be eliminated since steamed soil was used.

The mechanism or combination of mechanisms involved in differences of disease severity was not elucidated in this study.

Summary

The effect of atrazine on Gibberella zeae-induced seedling blight of corn was tested using resistant, intermediate, and susceptible corn inbreds and 2, 15, and 30 ppm atrazine. Tests were run using artificially inoculated, steamed greenhouse soil.

No significant difference was found in disease severity when susceptible seedlings were grown in soils amended with 2, 15, and 30 ppm atrazine.

When intermediately susceptible seedlings were grown, disease severity was significantly increased in soils amended with 2, 15, and 30 ppm atrazine.

Seedling blight symptoms were significantly reduced when seedlings of the resistant inbred were grown in soils amended with 15 and 30 ppm atrazine. Disease symptoms were significantly increased when resistant seedlings were grown in soil amended with 2 ppm atrazine.

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