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Allelopathy

Organisms, Processes, and Applications

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Chapter 19

Potential of Hydroxamic Acids in the Control of Cereal Pests, Diseases, and Weeds

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Hydroxamic acids derived from 2-hydroxy-1,4-benzoxazin-3-one are secondary metabolites present in major agricultural crops such as wheat, maize and rye. Progress in research related to their ecological role in plants is summarized, as well as to potential uses in the control of pests, diseases and weeds. Problems presently limiting the exploitation of these compounds in plant protection are discussed.

In the last few decades, the dependence on fossil-fuel-based agrochemicals such as fertilizers and pesticides to produce agricultural and forestry products, has increased. This increased input of agrochemicals in arable crops can not be sustained in time, since agrochemicals pollute the environment and their production depends on non-renewable resources. Additionally, resistant strains of pest insects are emerging and herbicide tolerant weeds are appearing. Hence, new alternatives are needed which do not lead to the problems mentioned above and are of lower cost. Host plant resistance to pests, disease and weeds should play an increasingly important role in integrated pest management systems.

Cereals such as wheat (*Triticum aestivum* L.), maize (*Zea mays* L.) and rye (*Secale cereale* L.), which are major agricultural crops in the world, produce hydroxamic acids (Hx), a family of secondary metabolites discovered over three decades ago in relation to fungal diseases of rye (*1*), and thought to play a part in conferring resistance in some Gramineae to a wide range of pests and diseases (*2*). In addition, Hx have also been associated with detoxification of triazine herbicides (*3-5*) and with iron acquisition by plant roots (*6-8*). Hx are known to occur not only in maize, rye and wheat, but also in triticale and in several wild Gramineae (*2,9-14*), and to be absent from barley, oats and rice (*2*). Hx occcur in the plant as 2-ß-O-D-glucopyranosides which are hydrolyzed by endo-ß-glucosidases when the plant is injured

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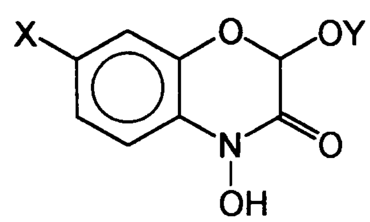
(*15,16*). The most abundant aglucone in wheat and maize extracts is 2,4-dihydroxy-7-methoxy-1,4-benzoxazin-3-one (DIMBOA). In rye, it is the demethoxylated analogue DIBOA (*2*).

Since Hx are present in the leaves, stems and roots of cereal plants, they can play a leading role in resistance against a variety of external agents. In this review, we will describe the potential uses of Hx in the control of pests, diseases and weeds of cereal crops, and discuss some of the problems that presently limit their exploitation in plant protection.



X	Y	Compound
H	H	DIBOA
H	Glucosyl	DIBOA glucoside
CH_3O	H	DIMBOA
CH_3O	Glucosyl	DIMBOA glucoside

Accumulation of Hx in Plants

Hx are not present in the grain but start accumulating as glucosides during the first stages of seedling development. The pattern of accumulation in aerial parts and roots varies between species and also between cultivars. The dynamics of Hx accumulation has been well studied in wheat and maize. In wheat, Hx are absent from the seed, increase upon germination (peaking at the young seedling stage) and decrease thereafter (*17*). In mature plants, the youngest tissue still retains a high concentration of Hx (*9-11,17-20*). Hx are present in all organs of the plant (*17*). Within the aerial parts of wheat and maize seedlings, they are present in the mesophyll as well as in the vascular bundles (*21,22*). Recently, it has been shown that the phloem sap of wheat seedlings, collected through excised aphid stylets, contained Hx glucosides (Givovich, A., Sandström, J., Niemeyer, H.M. and Pettersson, J., *J. Chem. Ecol.*, in press).

The information available on Hx accumulation in rye is still scarce. Barnes and Putnam (*23*) reported their presence in thirty five day-old greenhouse- and field- grown rye plants. Analysis of one rye cultivar grown under field conditions indicated that Hx are present throughout the life cycle of the plant (*24*). According to this limited information it seems that rye shows a pattern of Hx accumulation different from wheat and maize.

Furthermore, there exists evidence that higher levels of Hx may be induced by insect feeding in maize (*25*) and in wheat (*26*) and by artificial damage in maize (*27*), and that some abiotic factors such as light (*28*), temperature (*29*), water stress (*30*) and minerals (*6,7*) can modify the levels of Hx in maize. The mechanims involved in the regulation of Hx accumulation and degradation will be a major area of future research in this field.

Role of Hx in Insect Resistance

One of the first examples of the deliberate use of Hx in the control of a cereal pest involves the European corn borer, *Ostrinia nubilalis* Hübner, an important pest of maize in temperate regions. In the late 1960s, Hx were identified as a resistance factor against leaf feeding first generation larvae of this insect. Efforts were directed towards the production of hybrid maize with increased concentrations of Hx, which indeed showed increased resistance to the insect (*31,32*). The effect of DIMBOA on the life cycle of the insect has been studied in artificial diets (*33*). Studies on the mechanism of toxicity of DIMBOA towards the insect showed that DIMBOA acts primarily as a digestive toxin (*34*) and that the activity of detoxifying enzymes in the insect's midgut was modified by DIMBOA (*35*). Maize germplasm has been analyzed extensively for sources of resistance to the borer (*36*).

Artificial feeding of the borer *Sesamia nonagrioides* Lef., a pest of maize in the Mediterranean area, with diets supplemented with maize leaves or stems containing high levels of Hx, decreased the performance of the insect relative to diets without additive or diets supplemented with maize tissue with low Hx levels (*37*), suggesting a possible role for Hx in the resistance of maize to this borer.

The presence of Hx in the roots of maize has been related to resistance to the rootworm *Diabrotica virgifera virgifera* Le Conte. Performance of rootworm larvae was lower on maize accumulating high concentrations of Hx in its roots. Conversely, a high-Hx maize showed less damage due to rootworm than did a low Hx line (*38*). In choice tests, rootworm larvae 'preferred' to burrow into control maize roots rather than into roots treated with Hx, and Hx treatment of corn roots produced feeding deterrency in the larvae (*39*). Another report claimed that 6-methoxy-benzoxazolinone (MBOA), the main decomposition product of DIMBOA, acted as a volatile cue to the rootworm to find its host (*40*).

Recently, the presence of N-O-methyl-DIMBOA was demonstrated in maize whorl wax (*41*). The compound was toxic to the southwestern corn borer, *Diatraea grandiosella* Dyar, and was suggested as a possible resistance factor in maize to the insect (*41*).

Hx have been shown to be a major biochemical mechanism of resistance of wheat to aphids, acting through antibiosis and feeding deterrency. Thus, negative correlations have been described between Hx levels in the plants and growth rate and intrinsic rate of natural increase of cereal aphid populations (*18,20,42-44*), and mean relative growth rate of aphids (*45*). Population increase of aphids feeding on excised barley leaves (originally lacking Hx) into which different levels of DIMBOA had been incorporated, lead to similar negative correlations (*18*). Survival of cereal aphids in artificial diets decreased with increasing DIMBOA concentrations in them (*43,46*).

In choice tests, both winged and wingless *Rhopalosiphum padi* (L.) preferentially settled on seedlings with lower Hx levels (*47,48*). Electronic monitoring of aphid feeding behavior showed that in seedlings with higher Hx levels, fewer aphids reached the phloem within a given time, and they required longer times to contact a phloem vessel (*48*). This feeding deterrency decreased transmission of barley yellow dwarf virus to wheat seedlings in the laboratory (*48*).

The developmental time of the predatory ladybird, *Eriopis connexa* Germar., was shorter and the number of aphids ingested higher when the beetle fed on aphids from a high Hx wheat cultivar as compared with an intermediate Hx cultivar (*49*), suggesting that higher levels in wheat could potentiate the beneficial effects of the predator.

Sub-lethal doses of an insecticide were more effective on aphids feeding on a high-Hx wheat cultivar than on a low one (*50*).

Although these are strong arguments for the inclusion of Hx in strategies for the integrated control of aphid pests in cereals, field experiments are needed to validate the laboratory results.

Role of Hx in Disease Resistance

Resistance of maize to Northern corn leaf blight caused by *Helminthosporium turcicum* Pass., was associated with the presence of Hx (*51,52*). DIMBOA inhibited the germination of spores of *H. turcicum* (*53*). Maize mutants lacking the Bx allele coding for the accumulation of Hx were more susceptible to infection than maize possessing it. Diffusates from young maize plants were more active in inhibiting spore germination and germ tube elongation of *H. turcicum* than diffusates from older plants (*54*). Differences in mycelial growth were noticeable when the mycelium reached the vascular bundles (*55*). Interestingly, the glucoside of DIMBOA has been found in the phloem sap of wheat (Givovich, A., Sandström, J, Niemeyer, H.M. and Pettersson, J., *J. Chem. Ecol.*, in press)

Correlations have also been reported between Hx concentration in a cereal plant and resistance of the plant to various fungal infections, such as those produced in maize by *Diplodia maydis* (Schw.) Lev. (*56*) and *Cephalosporium maydis* (*57*), and in wheat by *Puccinia graminis* var. *tritici* Erikss. and Henn. (*58*). It was recently reported that infection by *P. graminis* produced a substantial increase in the synthesis of the glucoside of the DIMBOA derivative methylated at the hydroxamic acid function, which may thus function as a phytoalexin inhibiting mycelial growth (*59*). However, evidence has been presented that Hx concentrations are not related to resistance of maize to *Colletotrichum graminicola* (Ces.) Wils. (*60*).

The presence of Hx in maize plants was associated with the inability of certain species of soft rotting bacteria of the genus *Erwinia* to attack them, the lag phase of bacterial growth being prolonged by DIMBOA (*61*). Further experiments indicated that Hx were not the sole factor responsible for bacterial resistance in maize (*62*).

The role of Hx in the interaction of cereals with fungi remains an important area where much research is needed.

Role of Hx in Allelopathy

Biochemical interactions among plants appears to be a fairly ubiquitous phenomenon, occurring in most natural and agricultural ecosystems (*63,64*). However, the occurrence of this phenomenon is not easy to demonstrate due to the complexity of the sequence of events involved. Host plants must produce allelochemicals which must, directly or indirectly, interfere with the target plants. The allelochemicals must be released to the environment by means such as volatilization from the living plant, leaching of water-soluble compounds by the action of rain, fog or dew, root exudation, incorporation of plant parts (*65*), or decomposition of plant residues (*64*). Finally, the allelochemicals must be available to the target plant in sufficient amount to produce the allelopathic effects. Additionally, allelochemicals can be transformed chemically or microbially in the soil (*59,66,67*), and soil itself can influence the transfer, transformation and retention of allelochemicals (*68*).

Phytotoxicity of Hx. Bioassays carried out with Hx indicate that they inhibit seedling growth and the emergence of several mono- and dicotyledoneous species (*23,69*). Bioassays based on a cress (*Lepidium sativum* L.) root growth assay were used to assess the phytotoxicity of residues and extracts of rye, most active fractions having been found in the ethereal extract. Further purification identified DIBOA, the main Hx found in rye and its breakdown product benzoxazolin-2-one (BOA), as the main compounds of the extracts (*70*). An assessment of Hx toxicity towards weeds from different families normally associated to Chilean cereal crops, showed in most cases that cotyledons and root growth were inhibited significantly by 1 mM DIMBOA (Pérez, F.J. and Ormeño, J., unpublished data). On the other hand, Hx showed no autotoxic effects on cereals producing them, such as maize, wheat and rye (*7*; Pérez, F.J. and Gonzáles, L., unpublished data). These observations make Hx interesting as natural herbicides, since many weeds are susceptible to their inhibitory effects, while cereals producing them are not affected.

Root Exudation of Hx. Hx are non-volatile compounds. They are not leached from leaves (*21*), but can be released to the soil by root exudation. Pérez and Ormeño-Núñez (*71*), using a continuous root exudate trapping system, reported that two cultivars of rye exudate DIBOA through their roots while three different wheat cultivars that accumulate Hx in their roots do not. In a recent report, Pethós (*7*), using hydroponic cultures and placing the plants in distilled water for 2 to 6 hours after removal from the nutrient solution, found that wheat and maize also exudate Hx through their roots. Both research groups found that in the root exudates Hx occur as aglucones, while in root extracts they occur as glucosides, suggesting their transformation previous to release. A reexamination of rye root exudates during the first 24 days after emergence, showed that the pattern of compounds exudated changes with the age of the plant and confirm that DIBOA was one of the main compounds exudated. However, HDIBOA-glucoside, the reduced form of DIBOA, was the main compound identified in the exudates (Pérez, F.J., unpublished data).

Spring-sown living rye reduced weed biomass by 93 % over plots without rye (*72,73*). Field experiments using a rye variety exuding Hx through their roots reduced total weed biomass by 83 and 76 % compared with wheat and forage oats. Moreover, the specific reduction of *Avena fatua* L. biomass, observed with the rye cultivar and not with the wheat and forage oat cultivar, correlated well with the phytotoxicity of Hx on *A. fatua* observed in bioassays (*24*). These results suggest that simply identifying roots with high contents of Hx is not adequate for the selection of varieties with allelopathic potential. Root exudate analysis will be also required.

Reduced Tillage and Cover Crops. The use of allelopathic cover crops in reduced tillage cropping systems may provide an ecologically and environmentally safe management strategy for weed control. In this regard rye has been extensively studied, and numerous reports show the phytotoxicity of rye and its residues (*72-81*). Rye residues reduced total weed biomass by 63 % when *Populus excelsior* was used as a control for the mulch effect (72). In a four-year experiment carried out to evaluate the effect of rye cover crop on weed control, soybean (*Glycine max* L.) yield and soil moisture, and control of giant foxtail (*Setaria faberi*, Herrm.), velvetleaf (*Abutilon theophrasti*, Medik.), smooth pigweed (*Amaranthus hibridus* L.) and common lambsquarters (*Chenopodium album* L.), the effect recorded was generally greater than 90 % in the rye mulch plots and better than the corn residue treatments. No differences in soybean yield were found between conventional tillage using herbicides, compared with a no-tillage system using rye mulch without herbicides. Herbicides improved weed control in the corn residue plots but did not do so in the no- tillage rye treatment, due to the excellent control by rye mulch. Weed control by rye residue treatments, regardless of herbicide treatment, was explained by the allelopathic effect of rye and the physical presence of the mulch on the soil surface (*80*). Since Hx are the main allelochemicals involved in allelopathic effects of rye (*24,73*), rye germplasm leading to high accumulation of Hx to be used as mulch in weed control seems desirable. On the other hand, the assessment of rye mulch in weed control of other crops such as wheat and maize represents an interesting possibility since Hx are not toxic towards these cereals (*7*; Pérez, F.J. and Gonzáles, L., unpublished).

Transformation of Hx in the Soil. It was reported that BOA, the breakdown product of DIBOA, was transformed by the action of soil microorganisms to 2,2'-oxo-1,1'-azobenzene (AZOB) a compound with herbicidal activity stronger than DIBOA or BOA (*82,83*). The same authors identified *Acinetobacter calcoaceticus*, a Gram-negative bacteria isolated from field soil as the factor responsible for the biotransformation of BOA to AZOB (*84*). However, Gagliardo and Chilton (*85*) did not find AZOB in incubations of soil with BOA, o-aminophenol or o-azophenol, and claimed that the red pigment obtained by Chase *et al.* (*83*) corresponded to 2-amino-3H-phenoxazin-3-one which can be formed by microbial hydrolysis of BOA to o-aminophenol which is oxidized to the corresponding aminophenoxazinone by air. Further supporting this conclusion, the microbial transformation in the soil of two other naturally-occurring hydroxamic acids gave the corresponding aminophenoxazinones (*86*).

Clearly, more work will be needed to define precisely the nature of the compounds involved in field allelopathy by hydroxamic acids.

Prospects for Increasing Hx Levels in Cereals and their Use in the Control of Pests, Disease and Weeds

The decline in the concentration of Hx as the plant matures, as has been shown for maize and wheat, limits their action as a chemical defence, especially in the cases of organisms that invade the plant at its later stages of development. Little is known about the genetic expression and biosynthesis of Hx. This knowledge is highly desirable if Hx are to be used to their full potential in the protection of cereal plants. The first attempts to elucidate the biosynthesis of Hx were limited to feeding experiments using exogenously radiolabelled compounds (*87*). Anthranilic acid was identified as one of the precursors of Hx. On the other hand, the interconversion of the lactams and Hx was demonstrated *in vivo*, but not in a cell-free extract, suggesting that the substrates for the interconversion are the glucosides. Two Hx UDP-glucosyltransferases isolated from etiolated maize coleoptiles have been partially purified and characterized (*88*). Later, this activity was also identified in rye, wheat and *Hordeum lechleri* (Stend.) Schenk., a wild barley containing Hx (*12*). The enzyme in rye was partially purified and characterized, presenting a molecular mass of 43 Kda, a pI of 4.4 and K_m for DIBOA and DIMBOA of 73 and 82 μM respectively, while the corresponding lactams were not substrates for the enzyme (Leighton, V., Niemeyer, H.M. and Jonsson, M.V.L., *Phytochemistry*, in press). A cytochrome P-450 dependent N-monooxygenase which catalyzes the N-hydroxylation of HBOA, the lactamic derivative of DIBOA, to form DIBOA has also been identified in maize microsomal fractions. The enzyme required NADPH and was inhibited by sulfhydryl reagents, NADP, cytochrome c, cations, carbon monoxide and nitrogen (*89*) and did not recognize the 7-methoxylated analogue HMBOA nor the corresponding 2-O-ß-glucosides as substrates (Leighton, V., Niemeyer, H.M. and Jonsson, M.V.L., *Phytochemistry*, in press).

A single gene homozygous recessive mutant (bxbx) of maize resulted in an 8- to 10-fold reduction in Hx content of the seedlings (*3*). This bx locus was localized in the short arm of chromosome 4 by monosomic and B-A translocation analyses (*90*). Bailey and Larson found that the N-hydroxylase activity in homozygous (bxbx) maize seedlings was reduced in one half respect to heterozygous (Bxbx) ones, indicating that the Bx gene does not represent the structural gene for the N-hydroxylase activity, and suggesting that Bx could be a regulatory gene (*89*).

Hx could provide substantial benefits in the control of pests, diseases and weeds of cereals. The economic losses associated with the possible yield penalty for producing higher levels of defence chemicals in an annual crop may be compensated for by a reduction in the input of agrochemicals (*91,92*).

The establishment of breeding programs aimed at increasing Hx levels in wheat seems justified. Recent research has focussed on screening a wide genetic range of cultivars and species of the genus *Triticum*, in a search for germplasm useful for breeding programs aimed at increasing Hx levels. These studies have shown that there is more than an order of magnitude of variation in concentrations of Hx and suggests that there is scope to enhance the level of resistance to fungi and arthropod pests in modern wheat cultivars (*9,13,14,47,93*).

Alternatively, if techniques of genetic engineering are chosen which involve the use of *Agrobacterium tumefaciens* as a vector, care should be exercised since DIMBOA shows adverse effects on populations of *A. tumefaciens* and on the induction of virulence genes in the presence of acetosyringone (*94*).

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Literature Cited

1. Virtanen, A.I.; Hietala, P.K.*Acta Chem. Scand.* **1960**, *14*, 499-502.
2. Niemeyer, H.M.*Phytochemistry* **1988**, *27*, 3349-3358.
3. Hamilton, R.H.*Weeds* **1964**, *12*, 27-30.
4. Shimabukuro, R.H.; Swanson, H.R.; Walsch, W.C.*Plant Physiol.* **1970**, *46*, 103-107.
5. Malan, C.; Visser, J.H.; Van de Venter, H.A.*S. Afr. J. Plant Soil* **1987**, *4*, 7-11.
6. Tipton, C.L.; Buell, E.L.*Phytochemistry* **1970**, *9*, 1215-1217.
7. Pethó, M.*Acta Agron. Hung.* **1992**, *41*, 57-64.
8. Pethó, M.*Acta Agron. Hung.* **1992**, *41*, 49-56.
9. Niemeyer, H.M.*Euphytica* **1988**, *37*, 289-293.
10. Zúñiga, G.E.; Argandoña; V.H.; Niemeyer, H.M.; Corcuera, L.J. *Phytochemistry* **1983**, *22*, 2665-2668.
11. Copaja, S.V.; Barría, B.N.; Niemeyer, H.M.*Phytochemistry* **1991**, *30*, 1531-1534.
12. Barría, B.N.; Copaja, S.V.; Niemeyer, H.M.*Phytochemistry* **1991**, *31*, 89-91.
13. Copaja, S.V.; Niemeyer, H.M.; Wratten, S.D.*Ann. Appl. Biol.* **1991**, *118*, 223-237.
14. Niemeyer, H.M.; Copaja, S.V.; Barría, B.N.*Hereditas* **1992**, *116*, 295-299.
15. Hofman, J.; Hofmanova, O.*Eur. J. Biochem.* **1969**, *8*, 109-112.

16. Cuevas, L.; Niemeyer, H.M.; Jonsson, L.M.V.*Phytochemistry* **1992**, *31*, 2609-2612.
17. Argandoña, V.H.; Niemeyer, H.M.; Corcuera, L.J.*Phytochemistry* **1981**, *20*, 673-676.
18. Argandoña, V.H.; Luza, J.G.; Niemeyer, H.M.; Corcuera, L.J. *Phytochemistry* **1980**, *19*, 1665-1668.
19. Leszczynski, B.; Wright, L.C.; Bakowski, T.*Entomol. exp. apl.* **1989**, *52*, 135-139.
20. Thackray, D.J.; Wratten, S.D.; Edwards, P.J.; Niemeyer, H.M.*Ann. Appl. Biol.* **1990**, *116*, 573-582.
21. Argandoña, V.H.; Zuñiga, G.E.; Corcuera, L.J.*Phytochemistry* **1987**, *26*; 1917-1918.
22. Argandoña, V.H.; Corcuera, L.J.*Phytochemistry* **1985**, *24*, 177-178.
23. Barnes, J.P.; Putnam, A.R.*J. Chem. Ecol.* **1987**, *13*, 889-905.
24. Pérez, F.J.; Ormeño, J.*Weed Res.* **1993**, *33*, 115-119.
25. Gutiérrez, C.; Castañera, P.; Torres, V.*Ann. Appl. Biol.* **1988**, *113*, 447-454.
26. Niemeyer, H.M.; Pesel, E.; Copaja, S.V.; Bravo, H.R.; Franke, S.; Francke, W.*Phytochemistry* **1989**, *28*, 447-449.
27. Morse, S.; Wratten, S.D.; Edwards, P.J.; Niemeyer, H.M.*Ann. Appl. Biol.* **1991**, *119*, 239-249.
28. Manuwoto, S.; Scriber, J.M.*Agric. Ecosys. Environ.* **1985**, *14*, 221-236.
29. Epstein, W.W.; Rowsemitt, C.N.; Berger, P.J.; Negus, N.C.*J. Chem. Ecol.* **1986**, *12*, 2011-2020.
30. Richardson, M.D.; Bacon, C.W.*J. Chem. Ecol.* **1993**, *19*, 1613-1624.
31. Grombacher, A.W.; Russell, W.A.; Guthrie, W.D.*J. Kansas Ent. Soc.* **1986**, *62*, 103-107.
32. Guthrie, W.D.; Tseng, C.T.; Russell, W.A.; Coats, J.R.; Robbins, J.C.; Tollefson, J.J.*J. Kansas Ent. Soc.* ***1986***, *59*, 356-360.
33. Campos, F.; Atkinson, J.; Arnason, J.T.; Philogène, B.J.R.; Morand, P.; Werstiuk, N.H.; Timmins, G.*J. Chem. Ecol.* **1989**, *15*, 1989-2001.
34. Houseman, J.G.; Campos, F.; Thie, N.M.R.; Philogène, B.J.R.; Atkinson, J.; Morand, P.; Arnason, J.T.*J. Econ. Entomol.* **1992**, *85*, 669-674.
35. Feng, R.; Houseman, J.G.; Downe, A.E.R.; Atkinson, J.; Arnason, J.T. *Pest. Biochem. Physiol.* **1992**, *44*, 147-154.
36. Reid, L.; Arnason, J.T.; Nozzolillo, C.; Hamilton, R.*Can. J. Bot.* **1990**, *68*, 311-316.
37. Gutiérrez, C.; Castañera, P.*Inv. Agrar.: Prod. Prot. Veg.* **1986**, *1*, 109-119.
38. Xie, Y.S.; Arnason, J.T.; Philogène, B.J.R.; Lambert, J.D.H.; Atkinson, J.; Morand, P.*Can. Ent.* **1990**, *122*, 1177-1186.
39. Xie, Y.S.; Arnason, J.T.; Philogène, B.J.R.; Atkinson, J.; Morand, P. *J. Chem. Ecol.* **1992**, *18*, 945-957.
30. Bjostad, L.B.; Hibbard, B.E.*J. Chem. Ecol.* **1992**, *18*, 931-944.
41. Hedin, P.A.; Davis, F.M.; Williams, W.P.*J. Chem. Ecol.* **1993**, *19*, 531-542.

42. Long, B.J.; Dunn, G.M.; Bowman, J.S.; Routley, D.G.*Crop Sci.* **1977**, *17*, 55-58.
43. Corcuera, L.J.; Argandoña, V.H.; Niemeyer, H.M. In *Chemistry and Biology of Hydroxamic Acids*; Kehl, H., Ed.; Karger AG, Basel, **1982**; pp 111-118.
44. Bohidar, K.; Wratten, S.D.; Niemeyer, H.M.*Ann. Appl. Biol.* **1986**, *109*, 193-198.
45. Thackray, D.J.; Wratten, S.D., Edwards, P.J.; Niemeyer, H.M. In *Proceedings of the 1990 Brighton Crop Protection Conference, Pests and Diseases*, **1990**; pp 215-220.
46. Argandoña, V.H.; Corcuera, L.J.; Niemeyer, H.M.; Campbell, B.C. *Entomol. exp. appl.* **1983**, *34*, 134-138.
47. Nicol, D.; Copaja, S.V.; Wratten, S.D.; Niemeyer, H.M.*Ann. Appl. Biol.* **1992**, *121*, 11-18.
48. Givovich, A.; Niemeyer, H.M.*Entomol. exp. apl.* **1991**, *59*, 79-85.
49. Martos, A.; Givovich, A.; Niemeyer, H.M.*J. Chem. Ecol.* **1992**, *18*, 469-479.
50. Nicol, D.; Eaton, N.; Wratten, S.D.; Copaja, S.V.*Ann. Appl. Biol.*, **1992**, *121*, 11-18.
51. Long, B.J.; Dunn, G.M.; Routley, G.M.*Crop Sci.* **1978**, *18*, 573-575.
52. Toth Toldi, E.*Novenytermeles* **1984**, *33*, 213-218.
53. Couture, R.M.; Routley, D.G.; Dunn, G.M.*Physiol. Plant Pathol.* **1971**, *1*, 515-521.
54. Calub, A.G.; Dunn, G.M.; Routley, D.G.; Couture, R.M.*Crop Sci.* **1974**, *14*, 359-361.
55. Mace, M.E.*Phytopathology* **1973**, *63*, 243-245.
56. Dabler, J.M.; Pappelis, A.J.; BeMiller, J.N.*Phytopathology* **1969**, *59*, 1098-1101.
57. Kostandi, S.F.; Koraiem, Y.S.; Kamara, A.; Omar, M.A.*Agrochimica* **1981**, *25*, 367-375.
58. El-Naghy, M.A.; Shaw, M.*Nature* **1966**, *210*, 417-418.
59. Bücker, C.; Grambow, H.J.*Z. Naturforsch.* **1990**, *45c*, 1151-1155.
60. Lyons, P.C.; Nicholson, R.L.*Can. J. Plant Path.* **1989**, *11*, 215-220.
61. Corcuera, L.J.; Woodward, M.D.; Helgeson, J.P.; Kelan, A.; Upper, C.D. *Plant Physiol.* **1978**, *61*, 791-795.
62. Lacy, G.H.; Hirano, S.S.; Victoria, J.I.; Kelman, A.; Upper, C.D. *Phytopathology* **1979**, *69*, 757-763.
63. Einhellig, F.A.; Leather, G.R. *J. Chem. Ecol.* **1988**, *14*, 1829-1843.
64. Rice, E.L.*Allelopathy*, 2nd. ed. Academic Press, Orlando, **1984**.
65. Inderjit; Dakshini, K.M.M.*Amer. J. Bot.* **1992**, *79*, 977-981.
66. Da Hon, B.R.; Blum,U. Weed, S.B.*Soil Sci. Soc. Amer. J.* **1989**, *53*, 757-762.
67. Blum, U.; Shafer, S.R.*Soil Biol. Biochem.* **1988**, *20*, 793-800.
68. Cheng, H.H. In *Allelopathy: Basic and Applied Aspects*; Riavi, S.J.H.; Riavi, V., Eds.; Chapman and Hall, **1992**, pp 21-29.
69. Pérez, F.J.*Phytochemistry* **1990**, *29*, 773-776.

70. Barnes, J.P.; Putnam, A.R.; Burke, B.A.; Aasen, A.J.*Phytochemistry* **1987**, *26*, 1385-1390.
71. Pérez, F.J.; Ormeño-Núñez, J.*J. Chem. Ecol.* **1991**, *17*, 1037-1043.
72. Barnes, J.P.; Putnam, A.R.*J. Chem.Ecol.* **1983**, *9*, 1045-1057.
73. Barnes, J.P.; Putnam, A.R.*Weed Sci.* **1986**, *34*, 384-390.
74. Hill, H.H.*J. Agric. Res.* **1926**, *33*, 77-99.
75. Osvald, H. In *Proceedings of the 7th International Botany Congress.* Osvald, H.; Eberg, E., Eds.; Stockholm; **1953**.
76. Patrick, Z.A.; Koch, L.W.*Can. J. Bot.* **1958**, *36*, 621-647.
77. Patrick, Z.A.*Soil Sci.* **1971**, *11*, 13-18.
78. Kimber, R.W. *Plant Soil* **1973**, *38*, 347-361.
79. Chou, C.H.; Patrick, Z.A.*J. Chem. Ecol.* **1976**, *2*, 369-387.
80. Putnam, A.R.; DeFrank, J.*Crop Prot.* **1982**, *2*, 621-647.
81. Liebl, R.; Simmons, W.; Wax, L.M.; Stoller, E.W.*Weed Tech.* **1992**, *6*, 838-846.
82. Nair, M.; Whitenack, C.J.; Putnam, A.R.*J. Chem. Ecol.* **1990**, *16*, 353-364.
83. Chase, W.R.; Nair, M.G.; Putnam, A.R.*J. Chem. Ecol.* **1991**, *17*, 9-19.
84. Chase, W.R.; Nair, M.G.; Putnam, A.R.; Mishra, S.K..*J. Chem. Ecol.* **1991**, *17*, 1575-1583.
85. Gagliardo, R.W.; Chilton, W.*J. Chem. Ecol.* **1992**, *18*, 1683-1691.
86. Kumar, P.; Gagliardo, R.W.; Chilton, W.S.*J. Chem. Ecol.* **1993**, *19*, 2453-2461.
87. Tipton, C.L.; Wang, M.C.; Tsao, F.H.; Tu, C.L.; Husted, R.L. *Phytochemistry* **1973**, *12*, 347-352.
88. Bailey, B.A.; Larson, R.L.*Plant Physiol.* **1989**, *90*, 1071-1076.
89. Bailey, B.A.; Larson, R.L.*Plant Physiol.* **1991**, *95*, 792-796.
90. Simcox, K.D.; Weber, D.F.*Crop Sci.* **1985**, *25*, 827-830.
91. Lovett, J.V. In *Soil Management, Energy Use and Crop Production*; Lovett, J.V.; So, H.B., Eds.; University of New England Printing Unit, Sydney, **1980**; pp 181-208.
92. Lovett, J.V. In *Perspectives in World Agriculture*, compiled by Commonwealth Agricultural Bureaux, Farnham Royal, **1980**; pp 91-122.
93. Escobar. C.A.; Niemeyer, H.M.*Acta Agric. Scand. Sect. B. Soil and Plant Sci.*, **1993**, *43*, 163-167.
94. Sahi, S.V.; Chilton, M.-D.; Chilton, W.S.*Proc. Nat. Acad. Sci.* **1990**, *87*, 3879-3883.

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