higher sugar and starch content were obtained in seeds of plants grown in the activated CF air except for Jackson Wonder (4) and 79 Mildew Resistant Fordhook (8). Seed coat and cotyledon color do not appear to correlate with the compositional changes in lima bean seeds related to the O_3 treatments.

In summary, susceptibility to O_3 damage to leaves was determined for eight genotypes of lima beans. Greatest resistance to visible leaf injury from pollutants in ambient air was exhibited by genotypes with green cotyledons. Seed coat color was not related to leaf tissue injury. Seed yield was not related to the amount of visible plant tissue damage. The maximum Kjeldahl nitrogen and amino acid synthesis occurred in seeds from harvest one and two grown in CF air. Seeds from the first harvest grown in NF air and seeds from the second harvest grown in CF air had the highest carbohydrate and starch. Cotyledon or seed coat color was not related to O_3 effects on Kjeldhal nitrogen or starch. Treatment of ambient air O_3 on methionine, lysine, and fructose was not statistically significant. Additional studies are required to determine the effect of O_3 on the synthesis of biochemical components in bean seeds.

ACKNOWLEDGMENT

We thank Steve Hollander for the chemical analysis and Ruel Wilson for the statistical analysis.

Registry No. NH_3, 7664-41-7; starch, 9005-25-8; L-methionine, 63-68-3; L-leucine, 61-90-5; L-histidine, 71-00-1; L-lysine, 56-87-1; nitrogen, 7727-37-9; L-threonine, 72-19-5; L-valine, 72-18-4; L-isoleucine, 73-32-5; L-tyrosine, 60-18-4; L-phenylalanine, 63-91-2; L-aspartic acid, 56-84-8; L-serine, 56-45-1; L-glutamic acid, 56-86-0; L-proline, 147-85-3; glycine, 56-40-6; L-alanine, 56-41-7; L-arginine, 74-79-3; D-fructose, 57-48-7; sucrose, 57-50-1; D-raffinose, 512-69-6; stachyose, 470-55-3; ozone, 10028-15-6.

LITERATURE CITED

Adams, R. M.; Hamilton, S. A.; McCarl, B. A. "The Economic Effects of Ozone on Agriculture"; Environmental Protection Agency, Environmental Research Laboratory: Corvallis, OR, 1984; EPA-600/3-84-090, p 175.

Association Official Analytical Chemists "Official Methods of Analysis", 13th ed.; AOAC: Washington, DC, 1980.

Bennett, J. P.; Resh, H. M.; Runeckles, V. C. *Can. J. Bot.* **1974**, *52*, 35–42.

Clayberg, C. D. *HortScience* **1971**, *6*, 396–397.

Dugger, W. M., Jr.; Palmer, R. L. Proceedings, First International Citrus Symposium; University of California: Riverside, 1969; Vol 2, pp 711–715.

Dugger, W. M., Jr.; Taylor, O. C.; Cardiff, E.; Thompson, C. R. *Proc. Am. Soc. Hort. Sci.* **1962**, *81*, 304–315.

Durrum Instrument Corp. "Operation and Maintenance Manual, Durrum Amino Acid Analyzer Model D500"; Durrum Instrument Corp.: Palo Alto, CA, 1972.

Fleming, S. E. *J. Food Sci.* **1982**, *47*, 12–15.

Fleming, S. E. *J. Food Sci.* **1981**, *46*, 794–798, 803.

Heggestad, H. E.; Christiansen, M. N.; Craig, W. L.; Heartley, W.H. In "Air Pollution and Its Impact on Agriculture", Cottrell Centennial Symposium; California State College: Turlock, CA, 1977a; pp 101–127.

Heggestad, H. E.; Howell, R. K.; Bennett, J. H. *U.S. Environ. Prot. Agency, Res. Dev.* **1977b**, EPA-600/3-77-128, 37.

Heggestad, H. E.; Craig, W. L.; Meiner, J. P.; Saettler, A. W. *Proc. Am. Phytopath. Soc.* **1976**, *3*, 326.

Heggestad, H. E. *Am. Potato J.* **1973**, *50*, 315–328.

Heggestad, H. E.; Heck, W. W. *Adv. Agron.* **1971**, *23*, 111–145.

Heggestad, H. E.; Menser, H. A.; Bailey, W. A. Preprint 67-38h, Air Pollution Control Association Annual Meeting, Pittsburgh, PA, 1967; pp 1–6.

Howell, R. K.; Devine, T. E.; Hanson, C. H. *Crop. Sci.* **1971**, *11*, 114–115.

Lee, E. H.; Bennett, J. H.; Heggestad, H. E. *Plant Physiol.* **1981**, *67*, 347–350.

Lefohn, A. S.; Tingey, D. T. *Atmos. Environ.* **1984**, *18*, 2521–2526.

Little, T. M.; Hill, F. J. "Agricultural Experimentation Design and Analysis"; Wiley: New York, 1978.

Menser, H. A. *J. Am. Soc. Sugar Beet Technol.* **1974**, 19, 81–86.

Moore, S.; Stein, W. H. *Methods Enzymol.* **1963**, 820–923.

Ray, A. A. "SAS Users Guide: Statistics"; SAS Institute, Inc.: Cary, NC, 1982.

Reinert, R. A.; Dunning, J. A.; Heck, W. W.; Benepal, P. S.; Rangappa, M. *HortScience* **1984**, *19*, 86–88.

Reinert, R. A.; Heggestad, H. E.; Heck, W. W. In "Breeding Plants for Less Favorable Environments"; Christiansen, M. N., Lewis, C. F., Eds.; Wiley: New York, 1982; pp 260–267.

Received for review June 28, 1985. Accepted November 25, 1985. Reference to brand or firm names does not constitute endorsement by the U.S. Department of Agriculture over others of a similar nature not mentioned.

Analogues of Phytoalexins. Synthesis of Some 3-Phenylcoumarins and Their Fungicidal Activity

Anna Arnoldi,* Gandolfina Farina, Remo Galli, Lucio Merlini, and Maria Grazia Parrino

Little is known about the relationship between fungicidal activity of isoflavonoid phytoalexins and their physicochemical properties, in particular partition properties. As a first part of an investigation on the influence of partition coefficients on the fungicidal activity of analogues of isoflavonoid phytoalexins, a series of 3-phenylcoumarins, including ethers and side-chain derivatives of high lipophilicity, were synthesized and tested in vitro and in vivo for antifungal activity. The results seem to indicate that an increase of lipophilicity has a negative effect and a free OH is indispensable for activity.

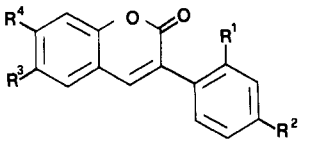
INTRODUCTION

Phytoalexins are chemical compounds involved in the resistance of plants to fungal infection (Bailey and Mansfield, 1982). They exhibit fungistatic, fungicidal, and, in some cases, also antibacterial activity (Grisebach and Ebel, 1978). They have been studied especially by plant pathologists interested in understanding the mechanism of resistance of plants to diseases. Some suggestions on the possibility of using them as models for the synthesis of new fungicides have also been made (Polter, 1974; Ward et al., 1975). The work of Rathmell and Smith (1980)

Istituto di Biochimica e Chimica (A.A., R.G., L.M.) and Istituto di Patologia Vegetale (G.F., M.G.P.), Facoltà di Agraria, Università, I-20133 Milano, Italy.

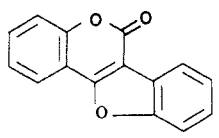
0021-8561/86/1434-0185$01.50/0

Table I. Compounds Tested and Their Activities

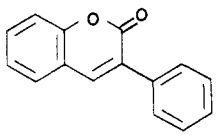


compd	R^1	R^2	R^3	R^4	log P^a	in vitro tests[b]			in vivo tests[b]			tests on hypocotyls[e] (1000 mg L^{-1})	
						C_5[c]	B	P	U/P[d]	S/C	C_5/P	C_{12}/P[f]	C_5/P
1a	OH	H	H	H	2.63	21	>100	>100	>500	>500		0.5	2.0
1b	$OCH_2C{\equiv}CH$	H	H	H	3.23	>100	>100	>100	>500	>500		0.5	1.6
1c	OCH_2Ph	H	H	H	4.00	96	>100	>100	>500	>500		0.5	3.1
1d	*O*-geranyl	H	H	H	5.22	>100	>100	>100	>500	>500		0.6	4.2
2a	H	OH	H	H	2.63	>100	>100	25	>500	>500	400	0.7	0.8
2b	H	OCH_2Ph	H	H	4.41	>100	>100	>100	>500	>500		0.7	4.6
2c	H	*O*-geranyl	H	H	5.46	>100	>100	100	>500	>500		0.5	3.8
2d	H	*O*-$C_{16}H_{33}$	H	H	>6	>100	>100	>100	>500	>500			
2e	H	$OCOCH_3$	H	H	3.73	>100	>100	>100	>500	>500	700	0.6	0.2
3a	H	H	OH	H	2.39	50	>100	50	>500	>500		0.6	4.6
3b	H	H	$OCH_2C{\equiv}CH$	H	3.65	>100	>100	>100	>500	>500		0.6	2.9
3c	H	H	OCH_2Ph	H	4.72	>100	>100	>100	>500	>500		0.5	1.6
3d	H	H	*O*-geranyl	H	5.56	>100	>100	100	>500	>500	Ph[g]	0.6	0.8
4a	H	OH	H	OCH_2Ph	3.44	>100	>100	>100	>500	>500		0.6	4.0

[a] Calculated from experimental R_M. [b] Activity expressed as ED_{50} (mg L^{-1}). [c] C_5 = *Colletotrichum lindemuthianum* (strain C_5); B = *Botrytis cinerea*; P = *Phytophthora cactorum*. [d] U/P = *Uromyces appendiculatus* on *Phaseolus vulgaris*; S/C = *Sphaerotheca fuliginea* on *Cucumis sativus*; C_5/P = *C. lindemuthianum* (strain C_5) on *P. vulgaris*. [e] Activity expressed as disease symptoms by a 6-point scale; 0, no symptoms; 0.5–1, hypersensitive reaction; 2–4, necrotic lesions of increasing dimensions; 5, anthracnose. [f] C_{12}/P and C_5/P = strain C_{12} or C_5 of *C. lindemuthianum* on *P. vulgaris*. [g] Ph = phytotoxic (at 1000 and 500 mg L^{-1}), 0 at 250 mg L^{-1}.



Coumestan



3-Phenylcoumarin

Figure 1. Structure of the coumestans and of the 3-phenylcoumarins.

showed that the most important isoflavonoid phytoalexins lack activity in the tests used for selection of synthetic fungicides.

A drawback of these phytoalexins is that they are not easily translocated in the plant tissues; in fact the resistance to disease of plants inoculated with hypovirulent fungi is limited to the immediately near tissues (Bailey and Mansfield, 1982). Although some structure–activity investigations have been reported (Van Etten, 1976; Krämer et al., 1984) no systematic study of the possible correlation between the partition or other physicochemical parameter of phenolic phytoalexins with antifungal activity has been undertaken. The only exception is the work on 2-phenylbenzofurans by Carter et al. (1978), who however report only relative lipophilicity and not absolute partition coefficients.

We have observed from the data on in vitro activity (Bailey and Mansfield, 1982) that, in the class of isoflavonoid phytoalexins, compounds containing one or more prenyl chains, therefore more lipophilic, are generally more active than nonprenylated ones. As a first part of an investigation on the influence of these parameters on the fungicidal activity of isoflavonoid phytoalexins, we have undertaken the synthesis of some 3-phenylcoumarins (compounds that can be considered coumestans lacking the C-ring (Figure 1)), containing lipophilic chains.

We have assayed them in vitro and in vivo against some important phytopathogenic fungi of different taxonomic classes and have verified whether the activity was correlated with their partition coefficients. The structures of the compounds prepared are shown in Table I.

MATERIALS AND METHODS

Chemistry. Two routes of synthesis of the coumarin nucleus were used. The first one (Walker, 1958) is the Knoevenagel condensation between a salicylic aldehyde with the proper substituents and a phenylacetic acid in the presence of acetic anhydride and potassium acetate. In this way **2e** (Walker, 1958) and **4a** acetate were obtained. Subsequent hydrolysis with KOH in ethanol gave respectively **2a** (Walker, 1958) and **4a**.

Compound **1a** (Walter et al., 1966) was obtained via a similar procedure from benzofuran-2-one and salicylic aldehyde in the presence of triethylamine.

The second route (Buu-Hoi et al., 1969) consists of the condensation of 2-methoxybenzaldehyde and a phenylacetonitrile to give a substituted acrylonitrile and its subsequent demethylation and cyclization to 3-phenylcoumarin in the presence of pyridine hydrochloride. This sequence gave **3a** (Buu-Hoi et al., 1969) but failed when applied to the synthesis of 7-hydroxy-3-phenylcoumarin and 6-hydroxy-3-(3-pyridyl)coumarin.

Etherification of the phenolic group with the suitable halide and K_2CO_3 in acetone (or methyl ethyl ketone) gave the ethers reported in Table I. The compounds were purified by crystallization whenever possible or by column chromatography. Thin-layer chromatography on silica gel was used as the criterion of purity for all compounds.

Synthesis of Compound 4a. A mixture of (4-hydroxyphenyl)acetic acid (2.21 g, 14.5 mmol), 2-hydroxy-4-(phenylmethoxy)benzaldehyde (3.32 g, 14.5 mmol), potassium acetate (4.28 g, 43.7 mmol), and acetic anhydride (15 mL) was refluxed with stirring for 4 h. The hot solution was then poured in 50 mL of warm water, and the resulting precipitate was collected, washed with ethanol, and crystallized from toluene. Compound **4a** acetate was obtained as a white solid: 2.76 g (49%); mp 165–167 °C (toluene). It was hydrolyzed by refluxing for 2 h with 15% NaOH. After neutralization with HCl, the mixture was extracted with ethyl acetate, and the organic layer was dried and concentrated. The residue was crystallized from ethanol to give **4a** as a white solid, 85%: mp 200–201 °C

Table II. Physical Data of New Compounds

compd	method	yield, %	mp, °C	mol form.[a]	$M^{+\cdot}$ m/z	IR: ν, cm^{-1}	^1H NMR ($CDCl_3$): δ (J, Hz)
1b	A	95	135–138	$C_{18}H_{12}O_3$	276	2200, 1725	2.5 (1 H, t, J = 1.5, C≡CH), 4.70 (2 H, d, J = 1.5, CH_2), 7.0–7.6 (8 H), 7.8 (1 H, s, H-4)
1c	A	76	173	$C_{22}H_{16}O_3$	328	1725	5.10 (2 H, s, CH_2O), 6.9–7.5 (13 H), 7.73 (1 H, s, H-4)
1d	B	66	63	$C_{25}H_{26}O_3$	374	1725, 1630	1.56 (3 H, s, CH_3), 1.63 (3 H, s, CH_3), 1.68 (3 H, s, CH_3), 2.0–2.2 (4 H), 4.56 (2 H, d, J = 6, CH_2O), 5.03 (1 H, m, H-6′), 5.40 (1 H, t, J = 6, H-2′), 6.9–7.6 (6 H), 7.75 (1 H, s, H-4)
2b	B	54	162–163	$C_{22}H_{16}O_3$	b	1725	5.11 (2 H, s, OCH_2), 6.9–7.8 (14 H)
2c	A	42	114–116	$C_{25}H_{26}O_3$	b	1725, 1630	1.61 (3 H, s, CH_3), 1.66 (3 H, s, CH_3), 1.78 (3 H, s, CH_3), 2.0–2.2 (4 H), 4.65 (2 H, d, J = 6, CH_2O), 5.13 (1 H, m, H-6′), 5.50 (1 H, t, J = 6, H-2′), 6.9–7.8 (8 H)
2d	A	26	96	$C_{31}H_{42}O_3$	462	1725	1.2–1.5 (31 H), 4.03 (2 H, d, J = 6, OCH_2), 6.9–8.1 (9 H)
3b	A	86	149–153	$C_{18}H_{12}O_3$	276	2200, 1725	2.50 (1 H, t, J = 2, C≡CH), 3.76 (2 H, d, J = 2, OCH_2), 7.0–7.8 (8 H), 7.80 (1 H, s, H-4)
3c	A	91	165	$C_{22}H_{16}O_3$	328	1725	5.15 (2 H, s, CH_2Ph), 7.0–7.8 (13 H)
3d	B	85	94	$C_{25}H_{26}O_3$	374	1725, 1630	1.58 (3 H, s, CH_3), 1.63 (3 H, s, CH_3), 1.73 (3 H, s, CH_3), 2.0–2.2 (4 H), 4.5 (2 H, t, J = 6, CH_2O), 5.03 (1 H, m, H-6′), 5.4 (1 H, t, J = 6, H-2′), 6.9–7.6 (6 H), 7.7 (1 H, s, H-4)

[a] Satisfactory microanalyses were obtained: C, ±0.5; H, ±0.3. [b] $M^{+\cdot}$ lacking in mass spectra.

(ethanol); NMR ($CDCl_3$) δ 4.88 (OH), 5.14 (2 H, s, OCH_2Ph), 6.8–7.8 (14 H, aromatic + H-4). Anal. Calcd for $C_{22}H_{16}O_4$: C, 76.73; H, 4.68. Found: C, 76.61; H, 4.75.

General Procedure for the Synthesis of Ethers 1b–d, 2b–d, and 3b–d. A mixture of phenol (3 mmol), alkyl bromide (3.3 mmol), and K_2CO_3 (6 mmol) in 50 mL of acetone (method A) or butan-2-one (method B) was refluxed for 3–5 h, the reaction being monitored by TLC. The mixture was filtered, and the solid was washed repeatedly with acetone. The solvent was concentrated. The residue was diluted with water (25 mL) and extracted with ethyl acetate (3 × 25 mL). After drying and concentration, the pure ethers were obtained by crystallization. Physicochemical data of new ethers are reported in Table II.

Physicochemical Characterization of the Compounds. Infrared spectra were recorded in liquid film or in Nujol mull on a Perkin-Elmer Model 21 infrared spectrometer. NMR spectra were recorded using tetramethylsilane as internal standard on a Bruker WP-80SY spectrometer at 80 MHz. Mass spectra were obtained on a Finnigan 4021 gas chromatograph/mass spectrometer equipped with an INCOS data system.

Partition Coefficients. The log P (where P is the octanol–water partition coefficient) was calculated by comparison with those of five reference compounds whose log P values are known: 4-nitrophenol, log P = 1.91; 4-chlorophenol, log P = 2.39; 2-naphthol, log P = 2.98; biphenyl, log P = 4.04; anthracene, log P = 4.45 (Nys and Rekker, 1974). The R_F were calculated as mean of three independent determinations (σ_{max} 4%) on Merck RP-18 (0.25-mm-thick) plates using acetonitrile–water (80:20) as eluent. The R_M were obtained from R_F by the equation (Tomlinson, 1975)

$$R_M = \log [(1/R_F) - 1]$$

The dependence of log P from R_M was calculated by regression analysis on the five reference compounds as

$$\log P = 3.57 + 2.40R_M,\ n = 5,\ r = 0.993$$

This equation was used to calculate the log P of our compounds.

Biological Assays. The fungicidal activity of the 14 compounds was tested in vitro and in vivo against five phytopathogenic fungi of different taxonomic classes.

Fungi. *Botrytis cinerea* was grown on malt agar, *Colletotrichum lindemuthianum* on neopeptone–yeast extract–glucose agar, and *Phytophthora cactorum* on corn meal agar. *Sphaerotheca fuliginea* and *Uromyces appendiculatus* were maintained on stock plants.

Plants. Bean plants cv. Borlotto nano and cucumber plants cv. Marketer were grown from seed in 10- and 7-cm plots, respectively, and maintained in a greenhouse (16–21 days, temperature 22 ± 3 °C, RH 75 ± 10%). To obtain hypocotyls, bean plants were also maintained in growth chambers under the same conditions of T and RH, but in the dark.

In Vitro Assays: Inhibition of Mycelial Growth. Solutions of each compound were prepared by dissolving the appropriate amounts of compound in 2 mL of Me_2SO plus Tween 20 (0.01%). Equal volumes of Me_2SO-containing diluted compounds were added to sterile cool (50 °C) agar media to give concentrations of 100, 50, 25, and 10 mg L^{-1} for each compound. A zero concentration treatment was prepared for each fungus and contained the same percent of Me_2SO and Tween to ensure equivalent concentrations of these components in all treatments. Compound-amended agar media were dispersed aseptically into 9-cm-diameter plastic Petri dishes (10 mL/dish). Inocula consisted of plugs (o.d. 7 mm) taken from the edges of colonies actively growing and inverted on the agar (two per dish) with two replicate plates for each fungus–compound combination. Colony diameter (mm) were measured after 2, 4, and 7 days of incubation at 22 °C, in the dark. The data reported in Table I refer to the 7 day results. Activity was expressed as the percent inhibition of mycelial growth compared with the control colony. ED_{50} values were estimated by plotting the percentage inhibition of the mycelial growth against the compound concentration on logarithmic probability paper, constructing the best straight line and recording the concentration at which 50% inhibition of growth occurred (Martin, 1978).

Excised Bean Hypocotyls. Ten-day-old etiolated bean hypocotyls were cut, paraffined at the extremities, surface sterilized with 1% NaClO and laid horizontally in a Petri dish at 22 °C (100% RH in the dark). Five-microliter drops of suspensions of each compound, prepared as described above at the concentration of 1000, 100, and 10 mg L^{-1}, were applied on each hypocotyl at a 2-cm distance (four drops for hypocotyl and five hypocotyls for compound and for concentration). After 48 h the nonadsorbed solution was removed, and a 5-μL drop of 10^6 conidia/mL suspension of *C. lindemuthianum* strain C_{12} (hypovirulent) or strain C_5 (virulent) was deposited on each treated site. As controls, water- (plus 0.01% Tween 20) treated hypocotyls were inoculated with the strain C_{12} or C_5 of *C. lindemuthianum*. The disease symptoms were measured by

a 6-point scale, as follows: 0, no symptoms; 0.5–1, hypersensitive reaction; 2–4, necrotic lesions of increasing dimensions; 5, anthracnose.

In Vivo Assays: Direct Protectant Activity. The compounds suspended with a Potter homogenizer in the minimum of water containing 0.01% of Tween 20 and then diluted to the concentration of 1000, 500, 250, and 125 mg L^{-1} were applied to both surfaces of the plant leaves to run off. Inoculation was performed 24 h after treatment (Arnoldi et al., 1982). The area of inoculated leaves covered by disease symptoms were assessed on a six-point scale from 0 to 5 on which 0 corresponded to no visible symptoms and 5 to 100% of the leaves covered. The compound activity was calculated on the basis of the percentage inhibition of the disease in comparison with the inoculated untreated plants. The ED_{50} values were estimated by plotting the percentage inhibition of the disease against the spray concentration on logarithmic probability paper, constructing the best straight line, and recording the concentration at which 50% inhibition of disease occurred (Martin, 1978).

RESULTS AND DISCUSSION

The results of the tests of activity of the compounds are reported in Table I.

None of the compounds exhibited an appreciable antifungal activity in vitro in the mycelial growth inhibition tests, only few being moderately inhibitory, in particular toward *P. cactorum* and *C. lindemuthianum*.

Similary none of them, when applied to bean excised hypocotyls, modified the process of infection of the hypovirulent strain (C_{12}) of *C. lindemuthianum*, which gave the typical hypersensitive reaction. On the contrary compounds **2a**, **2e**, and **3d** and in a lower degree **1a**, **1b**, and **3c** reduced the virulence of the C_5 strain. The compounds **2a**, **2e**, and **3d** were tested in vivo toward *C. lindemuthianum* (strain C_5) on *P. vulgaris*. The results of Table I show that compounds **2a** and **2e** provided moderate protectant activity, but only at the highest concentrations, while compound **3d** was phytotoxic.

None of the compounds in protectant tests against *U. appendiculatus* on *P. vulgaris* and *S. fuliginea* on *C. sativus* showed any activity.

The 3-phenylcoumarins showed low activity both in vitro and in vivo against different phytopathogenic fungi. They do not modify, too, the susceptibility of bean tissues to two strains of *C. lindemuthianum* with different degree of virulence on *P. vulgaris*. In fact, the reduction of infection determined by **2a**, **2c**, and **3d** applications on excised bean hypocotyls to the virulent strain does not depend on an effect on the host, but as shown by the results in vivo on *C. lindemuthianum* (C_5 strain)/*P. vulgaris*, on a direct fungitoxic activity on the pathogen.

As regards the structure–activity relationship in the classes **1** and **2**, an increase of lipophilicity determines a decrease of biological activity both in vitro and on hypocotyls tests, while only in class **3** the highly lipophilic compound **3d** reduces the virulence of the C_5 strain better than **3a**; this activity decreases, however, at lower concentration. Therefore, a modification of partition properties of these compounds toward lipophilicity did not give the desired results. This could indicate that a free OH group is indispensable in binding the molecule to the receptor responsible of their activity.

ACKNOWLEDGMENT

Research work supported by the CNR, Italy. Special Grant IPRA, Subproject 1, Paper No. 441.

LITERATURE CITED

Arnoldi, A.; Betto, E.; Farina, G.; Formigoni, A.; Galli, R.; Griffini, A. *Pestic. Sci.* **1982**, *13*, 670–678.

Bailey, J. A.; Mansfield, J. W. "Phytoalexins"; Blackie: Glasgow and London, 1982; Chapter 2.

Buu-Hoi, N. P.; Saint-Ruf, G.; Lobert, B. *J. Chem. Soc. C* **1969**, 2069–2070.

Carter, G. A.; Chamberlain, K.; Wain, R. L. *Ann. Appl. Biol.* **1978**, *88*, 57–64.

Grisebach, H.; Ebel, J. *Angew. Chem., Int. Ed. Engl.* **1978**, *17*, 635–647.

Krämer, P. R.; Hindorf, H.; Jha, H.; Kallage, J.; Zilliken, F. *Phytochemistry* **1984**, *23*, 2203–2205.

Martin, Y. V. "Quantitative Drug Design: A Critical Introduction"; Marcel Dekker: New York and Basel, 1978; p 62.

Nys, G. G.; Rekker, R. F. *Eur. J. Med. Chem.–Chim. Ther.* **1974**, *9*, 361–375.

Polter, C. "Systemische Fungizide", International Symposium, 1974; Lgr, H.; Polter, C., Eds.; Springer-Verlag: Berlin, 1975; pp 27–48.

Rathmell, W. G.; Smith, D. A. *Pestic. Sci.* **1980**, *11*, 568–572.

Tomlinson, E. *J. Chromatogr.* **1975**, *113*, 1–54.

Van Etten, H. D. *Phytochemistry* **1976**, *15*, 655–659.

Walker, G. N. *J. Am. Chem. Soc.* **1958**, *80*, 645–652.

Walter, R.; Zimmer, H.; Purcell, T. C. *J. Org. Chem.* **1966**, *31*, 3854–3857.

Ward, E. W. B.; Unwin, C. H.; Stoessl, A. *Phytopathology* **1975**, *65*, 168–169.

Received for review August 8, 1985. Accepted November 18, 1985.