J Plant Growth Regul (1985) 4:177–180

Journal of
Plant
Growth
Regulation

Short Communication

Germination Inhibition by 5,7-Dihydroxychromone, a Flavanoid Decomposition Product

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Received January 24, 1985; accepted May 8, 1985

Abstract. 5,7-Dihydroxychromone **1** isolated from seeds of *Polygonum lapathifolium* L. inhibits the germination of velvetleaf (*Abutilon theophrasti* Medic) seeds. Effective molar concentrations ranged from 1 mM to 6 mM. Flavanoids reported or suspected to cause inhibition were not active at concentrations above 10 mM. Compound **1** is a flavanoid moiety and can be formed during catabolic degradation, but its role in the allelopathic activity attributed to flavanoids remains to be determined.

Our program seeks to identify and evaluate natural products with herbicide potential (Wolf et al. 1984). A preliminary investigation of the acetone extract from seeds of *Polygonum lapathifolium* L. indicated antigermination activity against velvetleaf (*Abutilon theophrasti* Medic) seeds. Fractionation of the extract led to the identification of 5,7-dihydroxychromone **1** as the toxic agent. Compound **1** has been isolated from *Polygonum persicaria* L. seeds (Romussi and Ciarallo 1974) and from peanut (*Arachis hypogea* L.) hulls (Pendse et al. 1973), but, to our knowledge, its antigermination property has not been pointed out. An earlier example of chemical inhibition from a *Polygonum* species (*P. orientale* L.) was described by Datta and Chatterjee (1980), who implied that the ubiquitous flavone luteolin **2c** was an allelopathic principle in the leaves. This paper describes the elucidation of the active agent from *P. lapathifolium* and compares its activity against velvetleaf with those of certain flavanoids.

[1] The mention of firm names or trade products does not imply that they are endorsed or recommended by the U.S. Department of Agriculture over other firms or similar products not mentioned.

Table 1. Effects of chromone 1 and selected flavanoids on the germination of velvetleaf seeds.

	Concentration (mM)	% Germination[a]
5,7-Dihydroxychromone 1	1	77[b]
	2	30[b]
	4	15[b]
	6	0[b]
Chrysin **2a**	10	106
Apigenin **2b**	10	103
Luteolin **2c**	20	97
Kaempferol **3b**	10	102
Quercitin **3c**	10	98

[a] Germination of treated seeds ÷ germination of control × 100.
[b] Significantly different from controls at the 95% level or better as determined by the chi-square one-tailed test.

Materials and Methods

Finely ground *P. lapathifolium* seeds (150 g) were defatted with hexane and then extracted with acetone (Wolf et al. 1984). The concentrated acetone extract was partitioned between $CHCl_3$ and H_2O. Chromone **1** (50 mg) was isolated from the $CHCl_3$ layer by high-pressure liquid chromatography (HPLC) on a Zorbax C_{18} column (9.4 mm × 25 cm, Dupont) with H_2O/CH_3CN (7/3) as the mobile phase. Progress of the isolation was monitored by bioassay to determine which fraction(s) contained the active principle. Chromone **1** was identified by its mass spectrum (Kratos MS-30) and by its NMR (Brucher WH-90) spectrum, which was identical to that in literature (Pendse et al. 1973, Romussi and Ciarallo 1974).

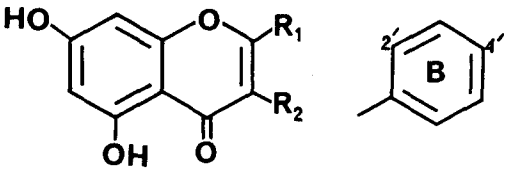
Dried, ground peanut shells (500 g) were percolated three times with acetone. After the solvent had been stripped on a rotatory evaporator, two layers formed. The lower, dark brown layer was drawn off and placed on a silica column (Silic AR®, 60–80 mesh, Mallinckrodt) which was eluted with benzene. HPLC of this benzene eluate afforded 35 mg of chromone **1.**

Standard materials were purchased from Aldrich (chrysin **2a,** quercitin **3c**), Sigma (apigenin **2b,** kaempferol **3b**), and Atomergic Chemetals, Plainview, NY (luteolin **2c**).

Bioassays for germination inhibition were carried out essentially as described by Wolf et al. (1984) except that 7 days elapsed before the germinated seeds were tallied. All bioassays were performed in duplicate.

Results and Discussion

The antigermination activity of chromone **1** is detailed in Table 1. A direct relationship between its toxicity and concentration is apparent. Also included in Table 1 are the results for selected flavanoids. Luteolin, **2c,** was suspected to inhibit mustardseed germination (Datta and Chatterjee 1980), although authenticated luteolin was not tested. Chrysin, **2a,** and apigenin, **2b,** are ana-



1 $R_1 = R_2 = H$

2 R_1 = B R_2 = H

3 R_1 = B R_2 = OH

OH at:

a none

b 4'

c 3', 4'

logues of luteolin with fewer hydroxyl groups on ring B. Kaempferol, **3b**, and quercitin, **3c**, are flavonols found to occur in *P. persicaria* (Romussi and Ciarallo 1974). The data in the table reveal that, in our bioassay system, these flavanoids have less than one-tenth the toxicity of chromone **1** despite the fact that it is a flavone moiety.

Flavanoids enjoy a reputation in allelopathy as inhibitors, but bioassay data on specific compounds are not entirely convincing (Rice 1974, Vickery and Vickery 1981). They are widespread in nature (Mabry et al. 1975), and it would seem that if they were indeed highly phytotoxic then their effects should be easily demonstrated. Work summarized by Barz (1980) and Barz and Hösel (1979) clearly demonstrates that dihydroxychromones can result from the enzymatic, peroxidative degradation of flavanoids. Although these reactions are very complex with many catabolites formed, one pathway involves elimination of ring B to form chromones. This information together with the data in Table 1 makes it tempting to attribute the activity reported by Datta and Chatterjee (1980) to chromone **1.** However, differences in the bioassay procedures and the specificity associated with allelopathic phenomena restrain such a speculation. Although beyond the scope of this report, the role of flavanoid degradation products is allelopathy is an intriguing subject for inquiry.

Acknowledgments. J. Lansden, National Peanut Laboratory, supplied peanut shells. E. P. Seest and T. S. Wilson assisted in the isolations and bioassays.

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