sensitivity of young animals to the effects of this type of alkaloid[23,24]. The lack of reports of toxicity of this plant despite claims of dietary use over many years[2] is not necessarily an indicator of safety. The effects of such alkaloids are cumulative and overt damage may be long delayed, thus preventing association with the plant cause.

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Activity of precocene analogs on *Locusta migratoria migratorioides* (R. and F.)[1]

R. Chênevert, J.M. Perron, R. Paquin, M. Robitaille and Y.K. Wang

Départements de chimie et de biologie, Faculté des sciences et de génie, Université Laval, Québec G1K 7P4 (Canada),
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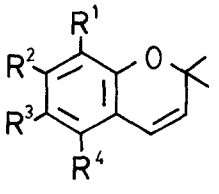
Summary. Several analogs of precocene have been synthesized and evaluated as potential inhibitors of juvenile hormone on *Locusta migratoria.* Only 5,7-dimethoxy-2,2-dimethylchromene was active; its ED_{50} and LD_{50} were measured and compared to those of precocene I and II.

Although considerable interest has been devoted to insect juvenile hormones, molting hormones (ecdysones) and synthetic juvenile hormone mimics, relatively little work has been done on inhibitors of hormones regulating metamorphosis in insects. This subject has been reviewed recently by Slama[2].

Bowers et al.[3] found that chromene derivatives extracted from the plant *Ageratum houstonianum* have anti-juvenile hormone like effects. Demethoxy-ageratochromene (7-methoxy-2,2-dimethylchromene) **1** and ageratochromene (6,7-dimethoxy-2,2-dimethylchromene) **2** possess anti-allatotropic properties and induce precocious metamorphosis in several insect orders. Because of their properties, they have been renamed precocene I and precocene II. Activity of precocenes has been already reported on 2 members of Orthoptera: *Locusta migratoria*[4] and *Schistocerca gregaria*[5]. We wish to report here the synthesis and evaluation of precocene analogs as potential inhibitors of juvenile hormone on *Locusta migratoria.*

Methods and materials. The substituted 2,2-dimethylchromenes (**3–8**) have been prepared following the general method of Hlubucek[6]: Substituted phenols (from Aldrich Co.) reacted with 3-chloro-3-methylbut-1-yne in refluxing acetone in the presence of anhydrous potassium carbonate and potassium iodide to produce in high yields the corresponding aryl α,α-dimethylpropargyl ethers. Thermal rearrangement of the ethers in boiling diethylaniline proceeded smoothly to give the chromenes in good yields. The 3-chloro-3-methylbut-1-yne was readily obtained from commercially available 3-hydroxy-3-methylbut-1-yne[7].

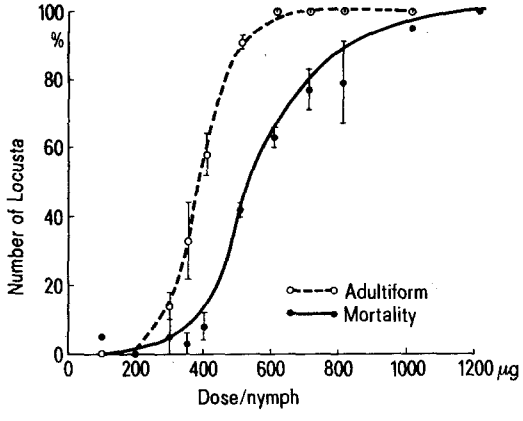
Crowded locusts were kept in a regime of 12 h light and 12 h darkness at 28 °C. Relative humidity was maintained at 45%. The compound to be tested was dissolved at different concentrations in spectral grade acetone so that each insect received 10 µl of the solution. This solution was applied topically on the ventral part of abdomen on 4th instar nymphs 1–24-h-old. Effects were recorded every day. Tests were performed on groups of 20 locust nymphs and repeated 2 times.



	R^1	R^2	R^3	R^4
1	H	OMe	H	H
2	H	OMe	OMe	H
3	H	H	H	H
4	H	H	OMe	H
5	OMe	H	H	H
6	H	OMe	H	OMe
7	H	$O-CH_2-$	O	H
8	H	OMe	OMe	OMe

Results and discussion. Of the 6 new compounds (**3–8**) tested for anti-juvenile hormone activity, only the 5,7-dimethoxy-2,2-dimethylchromene **6** gave rise to symptoms characteristic of a disturbance in juvenile hormone function. All compounds were revealed to be more or less toxic but failed to produce precocious metamorphosis when applied in a single dose of up to 1500 µg per nymph.

The effects of analog **6** are illustrated in the figure; brackets represent standard error of the mean (SEM) and where no bracket is shown, SEM was smaller than the symbol used. The mortality curve includes all deaths occurring during the 4th instar and the subsequent molting. Mortality rate among control insects receiving only acetone was less than 5%. The adultiform curve describes the percent of adultiforms among the survivors. The effects of the 5,7-dimethoxy analog were very similar to those of precocene I and II published elsewhere[8]. The lethal dose evaluated graphically (LD_{50} = 520 µg) was comparable to those of precocene I (420 µg) and precocene II (295 µg). However, the effective dose (ED_{50} = 375 µg) was much higher than those of precocene I (85 µg) and precocene II (30 µg) indicating a loss of activity.



Dosage-mortality and dosage-adultiform production curves for *L. migratoria* exposed to 5,7-dimethoxy-2,2-dimethylchromene.

Besides the results illustrated in the figure, we have also noted the following effects:

- The compound greatly delayed the moult, and this delay increases with higher doses.
- The morphology of adultiforms varies from moderate adult characteristics to advanced adult characteristics. The effect has already been reported for precocene II by Pener et al.[4].
- The adultiforms are viable and survive at least 1 month after moult.
- The deaths occurred mostly within 24 h after the application. Only a few died during the moult.

Our results clearly indicate that any change in the chemical structure of precocene results in total or great loss of activity. The double bond is also an essential feature for the activity of precocene: we have prepared the saturated compounds corresponding to precocene I and II and found them inactive. It is also known that precocene II resistant insects species metabolise it via the epoxy-precocene[9,10]. These facts could be a severe limitation for the practical application of precocene analogs as insecticides. Search for compounds with similar biological action is under current investigation.

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Action spectra for bilirubin photodisappearance

D.A. Lightner[1], T.A. Wooldridge, S.L. Rodgers and R.D. Norris

Department of Chemistry, University of Nevada, Reno (Nevada 89557, USA), 15 September 1978

Summary. The excitation wavelength dependence of bilirubin photodestruction, as measured by quantum yields, has been determined in benzene, chloroform-1% ethanol, chloroform-1% hexane, methanol-1% concentrated ammonia, pH 8.5 aqueous buffer and pH 7.4 aqueous buffer with added albumin. The results show that in the visible spectrum the 370–490 nm excitation wavelength region is very effective in the photodestruction, but excitation in the UV-region ($\lambda < 320$ nm) is even more effective.

Although phototherapy has been used increasingly for over 20 years to treat physiologic jaundice in newly born infants[2,3], and the efficacy of the treatment as it pertains to the lamps used has been discussed from time to time[4–8], no action spectra have been reported for bilirubin, except an incomplete study of bilirubin (BR) in plasma[9]. In that work the jagged-looking action spectrum may have been an artifact of not correcting for the emission characteristics of the light source, i.e. the lamp did not have a constant emission (quanta/sec) over the region used. It is also not clear whether higher energy harmonic emission (UV) from the monochromator was filtered out – an important consideration since BR is highly UV-active. Since phototherapy owes its success, in part, to BR photodestruction[2–4,10] we judged it important to bring information to bear on the wavelength response of BR photodisappearance, especially since this crucial facet of BR photochemistry has been one of obvious interest for some time[4–9,11].

Materials and methods. Bilirubin IX*α* (BR IX*α*) was obtained from Sigma and purified by dissolving in chloro-