Phytochemistry, Vol. 20, No. 5, pp. 989–991, 1981.
Printed in Great Britain.

0031-9422/81/050989-03 $02.00/0
Pergamon Press Ltd.

STEROID HORMONE EFFECTS ON GROWTH AND APICAL DOMINANCE OF SUNFLOWER

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(*Revised received* 14 *July* 1980)

Key Word Index—*Helianthus annuus*; Compositae; steroid hormones; root growth; shoot growth; apical dominance; estradiol-17β; progesterone; testosterone.

Abstract—External application of estradiol-17β increased shoot growth but decreased root growth of sunflower seedlings. It completely inhibited cotyledonary axillary bud development in decapitated plants at the concentration of 1 μg/plant. Concentrations lower than this promoted cotyledonary axillary bud formation. Testosterone on the other hand inhibited both shoot and root growth and promoted cotyledonary axillary bud formation at all the concentrations used. Progesterone at high (0.25 μg/plant) concentration promoted shoot growth but inhibited root growth. A low concentration (0.1 μg/plant) of progesterone produced the opposite effect.

INTRODUCTION

Free sterols, steryl glycosides and other forms of steroids are widely reported in plants [1, 2]. Though several physiological roles for plant sterols have been proposed [3–5], the role played by steroid hormones such as estradiol, progesterone and testosterone are yet to be clearly established. Steroids are reported [6–10] from plant species and Geuns [11] has mentioned the involvement of hormonal steroids in plant growth and development. However, estrogens could not be identified in plant materials [12] from which these were isolated earlier by other workers [6, 9, 10]. Fiedler [13] observed the stimulative effect of estrone on the growth of isolated corn root tips. Singh and Kapoor [14] reported testosterone propionate inhibition of shoot growth. Kopcewicz [15] showed that estradiol-17β and sitosterol increased the growth of dwarf pea plants. Estradiol and estrone induced increases of gibberellin and auxin are also reported [16, 17].

An attempt has now been made to observe the effect of some steroid hormones such as estradiol-17β, progesterone and testosterone in controlling shoot growth and root growth of a dwarf variety of sunflower (cv Modern). In addition their role in controlling apical dominance was also taken into consideration. Sunflower possesses a strong apical dominance and apical dominance is in general auxin mediated [18–20]. If a hormonal steroid acts through auxin production [17] it should show controlling ability so far as the apical dominance is concerned.

RESULTS AND DISCUSSION

Estradiol-17β was found to increase the stem growth at the concentrations of 0.1 and 0.25 μg/plant (Table 1). Progesterone on the other hand caused an increased stem growth at the concentration 0.25 μg/plant but at the lower concentration of 0.1 μg/plant it showed inhibition (Table 1). Estradiol induced growth at 0.1 μg/plant has been reported previously [15]. Increased growth by estradiol might be due to increased production of endogenous auxin content through initial increase of GA [16]. Increased growth by a higher concentration (0.25 μg/plant) of progesterone might also be due to increased

Table 1. Effect of estradiol-17β, progesterone and testosterone on shoot growth of sunflower seedlings

	Percentage elongation after 7 days	
Treatment	0.1 μg/Plant	0.25 μg/Plant
Estradiol-17β	167	160
Progesterone	93	188
Testosterone	114	118
Control	135	

CD at 5% in between treatments—3.04.

Table 2. Effect of estradiol-17β, progesterone and testosterone on root growth of sunflower seedlings

	Percentage elongation after 7 days	
Treatment	0.1 μg/Plant	0.25 μg/Plant
Estradiol-17β	19.1	3.1
Progesterone	42.6	5.6
Testosterone	1	15.3
Control	24	

CD at 5% in between treatments—1.90.

Table 3. Effect of estradiol-17β and testosterone application at decapitated shoot tip of sunflower plants on axillary branch formation

Treatment	Cotyledonary axillary branch in percentage of axils		
	0.1 μg/Plant	0.25 μg/Plant	1 μg/Plant
Estradiol-17β	70	25	0
Testosterone	70	72	89
Control	16.6		

CD at 5% in between treatments—2.24.

production of endogenous GA. Testosterone inhibited shoot growth at both the concentrations (Table 1) and this is in conformity with the observations of other workers [14]. Whether the inhibitory effect of testosterone is due to its direct effect on inhibition of plant growth processes or indirectly due to production of antiauxin-like compounds is, however, open to question.

Estradiol inhibited root growth (Table 2) at both concentrations and this inhibition was marked when the concentration was higher. Testosterone in this case showed inhibition but inhibition was greater at the lower concentration. Progesterone on the other hand caused inhibition (Table 2) at the higher concentration but considerable promotion was noted at the lower concentration. Thus it showed a dual effect so far as shoot and root growth are concerned.

Auxin control of apical dominance has been widely studied [1, 7]. Estradiol completely inhibited the production of cotyledonary axillary buds (Table 3) at the highest concentration (1 μg/plant). This inhibitory effect was completely lost at the concentrations 0.25 and 0.1 μg/plant. In fact at the lowest concentration (0.1 μg/plant) considerable promotion of cotyledonary axillary bud formation was noted. The maintenance of strong apical dominance by a high concentration of estradiol strengthens the view that it acts probably through induction of auxin production. However, from the concentration effect a dual nature of action is speculated. Testosterone in all the concentrations used promoted cotyledonary axillary bud formation. This suggested its antiauxin-like property and disruption of apical dominance by antiauxin is reported in the literature [15].

The steroid hormones used showed differential responses both in growth studies and studies relating to apical dominance. However, as their presence within the plant was not studied we have some reservations in assigning them as natural hormones in relation to plant growth and development.

EXPERIMENTAL

Seeds of a dwarf variety of sunflower were germinated in sterile sand in diffused light at 24 ± 2°. After 6 days abnormally grown seedlings were uprooted leaving only the normal seedlings. From such normal seedlings 50 seedlings were taken out at random with precautions so that the tap roots were not damaged. Average hypocotyl length and tap root length were measured. The remaining seedlings were grown in a temperature and light controlled room and then fed with steroid hormone solns through the shoot apex at the rate of 10 μl/plant. For each of the treatments 30 plants were selected at random from the growing seedlings and tagged. Steroids included estradiol-17β, progesterone, and testosterone. Steroids were dissolved in EtOH. To this soln 0.05% Teepol in H_2O was added to yield a final EtOH concn of 30%. Control plants were treated with a 10 μl drop of 30% EtOH–Teepol mixture. The elongation growth of shoot and root was measured after 7 days.

In another set of experiments 7-day-old uniformly grown seedlings were selected at random from sand culture and transferred to 100 ml Erlenmeyer flasks containing Hoagland soln. Each flask contained one seedling only. When the epicotyl was 30 mm long, the plants were decapitated 15 mm above the axils of the cotyledons. Outgrowths of visible buds were noted 3–4 days later. This was taken as indicative of loss of apical dominance in the decapitated seedlings. Just after decapitation tips were fed with 1 mg lanolin paste containing 1 μg, 0.25 μg and 0.1 μg estradiol and testosterone separately. For each treatment 30 seedlings were selected at random from treated growing seedlings and tagged. Control plants were fed with 1 mg lanolin EtOH paste in the same way. Lanolin steroid paste was prepared by initially dissolving the steroid in 1 ml EtOH and then homogenizing with lanolin. Data on branch formation were taken 7 days after application and expressed in terms of percentage of cotyledonary axils. Results were analysed for critical difference.

Acknowledgements—The authors thankfully acknowledge the financial assistance of Burdwan University for supporting the project and also the Sigma Chemical Company, U.S.A. for supplying the chemicals.

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