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SHORT COMMUNICATION

REGULATION OF LATERAL BUD ACTIVATION IN ALASKA PEA

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The concept of breaking dormancy as a process of derepressing genetic information was first proposed by Tuan and Bonner (1964), from experiments which showed an increased template activity of isolated chromatin of potato tuber upon emergence from dormancy. This concept has since received general support (Amens, 1966; Wareing, 1969; Rappaport and Wold, 1969). When apical dominance is destroyed, the bud undergoes abrupt metabolic changes in DNA, RNA, protein and carbohydrates as demonstrated histochemically in pea (Chang, 1970) and biochemically in tobacco (Schaefer and Sharpe, 1970). It seems, therefore, lateral bud under correlative inhibition may also be condidered functionally repressed. A number of inhibitor test have supported this concept. Azauracil and related nucleic acid analogs showed effective inhibition on axillary bud growth in tobacco (Schaefer and Steffens, 1965). Schaeffer and Sharpe (1969) showed that chlormycetin and 6-azauracil inhibited bud growth and development but do not interfere with benzyladenine (BA) initiated DNA synthesis. This communication reports the inhibitory effect of chloramphenicol on bud growth, and that delayed application of chloramphenicol to the lateral bud does not completely inhibit bud growth for 3 to 4 days after decapitation.

The secondary lowest lateral buds of 12-days old Alaska pea (*Pisum sativum* L.) were used since this bud always becomes dominant over other bud and showed a remarked stimulation in active growth within 24 hr after release from apical dominance. Seedlings were grown in a growth chamber providing a day-night alternation of 25°C/20°C in a 12 hr photoperiod at a light intensity of about 2000 ft-c. Chloramphenicol in lanolin paste (about 0.1 ml) at 1 mg/mg was carefully applied to one side of the 2nd lateral bud of 12-day-old seedlings at different time following decapitation. Before de-

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capitation at the 5th internode bud in the other leaf axils were removed. When applied at the time of decapitation, the inhibitor was effective in retarding lateral bud growth (Fig. 1 and Table 1). But application at 12 or 24 hr after decapitation showed less response to the applied inhibitor. However, the buds treated with inhibitor never grow as normal shoots. Eventually the bud growth was completely suppressed. The leaves of treated buds showed some abnormal expansion and chlorosis. However, within the first 3 days following decapitation, the response to the inhibitor were significantly different at intervals between decapitation of inhibitors. These results suggest that some substances which are necessary for bud growth and development are synthesized before inhibition by chloramphenicol. The synthesized substances probably are protein, since chloramphenicol is an inhibitor of protein synthesis (Margulies, 1962). It may also be new polysomes, since the chloramphenicol inhibits the process which requires the formation of new polysomes (Haber, *et al.*, 1968).

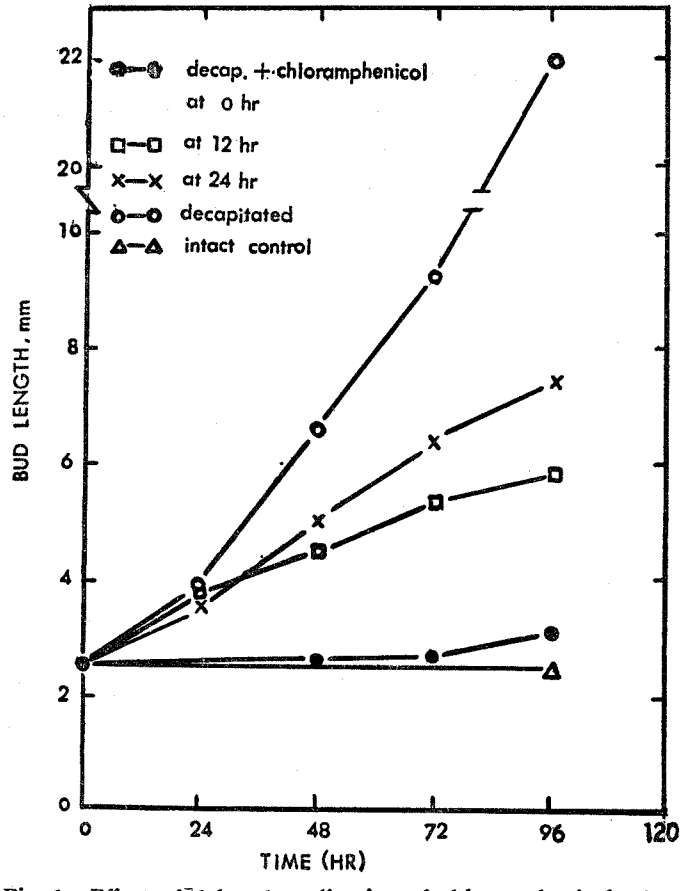

Fig. 1. Effect of delayed application of chloramphenicol, after decapitation, on 2nd lateral bud growth. Inhibitor concentration was 1 mg/ml in lanolin.

Table 1. *Effect of chloramphenicol on the growth of the 2nd lateral bud of* ***Pisum sativum*** *L. cv. Alaska after release from apical dominance. Inhibitor coconcentration was 1 mg/ml in hydrous lanolin.*

Treatments	3 days		4 days	
	bud length	% inhibition	bud length	% inhibition
Intact control	2.5±0.2 mm	—	2.5±0.3	—
Decapitated + chloramphenicol				
applied at 0 hr	2.7±0.9	96.5	3.2±0.7	96.4
12 hr	5.3±1.1	50.2	6.8±1.2	77.4
24 hr	6.3±1.5	11.2	7.3±1.6	75.4
Decapitated control	9.1±1.6	—	22.0±3.5	—

During the period between decapitation and chloramphenicol application there may have been some synthesis of these substances which caused the temporary growth of the bud. However, after this period these substances graduately disappear and the chloramphenicol inhibited the formation of new ones. Eventually, the bud stops growing. The effects of delayed chloramphenicol application on chlorophyll accumulation has been studied by Margulies (1967). Esashi and Leopold (1969) observed that delayed application of chloramphenicol during tuber maturation of *Begonia* caused little inhibition of the entry into dormancy. While application at the beginning of the period of onset of dormancy completely prevented the tuber from going into dormancy. Results of histochemical studies on pea also support this theory (Chang, 1970). Within 24 hr or even 12 hr after decapitation, the lateral bud had already accumulated and synthesized considerable RNA and protein. This RNA and protein would have supported a temporary growth for a while. However, the chloramphenicol applied at 12 hr or 24 hr eventually stopped the supply of RNA and protein; thus, the bud never established as a normal shoot.

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碗豆側芽活化的控制

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用 chloramphenicol 延遲處理經去頂的碗豆的側芽，僅部份抑制側芽的生長，表示除去頂芽優勢的控制後及用 chloramphenicol 處理前已有部份供側芽生長的必要物質產生，也表示側芽的活化可能是一種 derepression 現象。