MECHANISM OF PACLOBUTRAZOL REGULATION OF SHOOT GROWTH IN STURT'S DESERT PEA, *Swainsona formosa* (G. Don) J. Thompson (syn. *Clianthus formosus*)

by

Mohammad Monjur Hamid

B. Sc. Ag. Honours; M. Sc. Ag. (Bangladesh Agricultural University);

Dip. in Vegetable Production (Kasetsart University, Thailand)

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Dedicated

to my dear parents

Hazera Khatoon and Late Mohammad Shamsul Islam

Certificate of Originality

I certify that the substance of this thesis has not already been submitted for any degree and is not currently being submitted for any other degree or qualification.

I certify that any help received in preparing this thesis, and all sources used, have been acknowledged in this thesis.

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Abstract

Sturt's Desert Pea (SDP) (*Swainsona formosa*) is an Australian native plant species with considerable potential as an ornamental crop, including as a flowering pot plant, provided compact forms can be produced. Plant growth regulators (PGR's) are widely used to control plant growth. A range of PGR's were screened for use on SDP and paclobutrazol (PBZ), which has previously been used with variable results on SDP, was found to be the least toxic and most effective. Little previous research has been done to determine its mode of action in SDP. Understanding the mode of action of PBZ may enable more precise and more efficient use, and minimise concern regarding the impact of PGR's on safety standards and environmental pollution.

PBZ has been shown to inhibit GA biosynthesis in a range of plant species and may also affect the levels of other endogenous hormones. It is also thought to inhibit sterol synthesis. To understand its mode of action, particularly its interaction with the plant hormones, GA_3, IAA and BAP were applied alone and in various combinations with PBZ, to intact or decapitated plants and to shoot cuttings obtained from main or lateral shoots of SDP. The anatomical responses following PBZ and PGR applications were also examined.

In intact plants, PBZ reduced main shoot elongation, increased lateral shoot number but reduced lateral shoot elongation. Simultaneous or delayed application of GA_3 reversed the inhibitory effect of PBZ on main shoot growth. However, the reversal of PBZ inhibition of growth in lateral shoots was only achieved if the application of GA_3 was delayed until after the release of the lateral buds from apical dominance or the commencement of lateral shoot growth. Simultaneously applied GA_3 increased apical dominance in intact plants and correlative inhibition in decapitated plants thereby inhibited the growth of further lateral shoots. That was why GA_3 still did not reverse the PBZ inhibition of lateral shoot growth in intact or decapitated plants.

The inability of GA_3 in PBZ-treated lateral shoots of intact or decapitated plants to reverse PBZ inhibition, in contrast to the main shoot, was attributed to a lower auxin content and, or a higher GA content in the laterals. Delayed GA_3 application was effective, presumably due to a delay in PBZ-induced auxin activity.

In lateral shoot cuttings, exogenous supply of auxin as IAA did not enable GA_3 reversal of the PBZ inhibition but GA_3 alone or with L-tryptophan was effective; this was attributed to a difference in the location or active form of auxin derived from L-tryptophan in the presence of GA_3 and also due to a balanced supply of auxin : GA.

At the anatomical level, differential effects on cell division and cell elongation were observed. PBZ reduced internode length by reducing both cell number and mean cell length. GA_3 reversed the inhibition of cell division and auxin reversed the inhibition of cell elongation, but either response appeared dependant on a commensurate supply of the other hormone in the shoots.

Overall, the evidence is consistent with PBZ action via both inhibition of GA synthesis and promotion of auxin activity. The net result depends on the time of treatment, the stage of

lateral shoot development and the influence of other parts of the shoot system, particularly the occurrence of apical dominance or correlative inhibition.

This thesis clearly established an inherent difference between the responsiveness of the main shoots and lateral shoots to PBZ. This appears to be due to differences in the levels of endogenous hormones.

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Abbreviations

2,4-D	2,4-dichlorophenoxyacetic acid
a.i.	Active ingredients
ABA	Abscisic acid
ATP	Adenosine triphosphate
ATPase	Adenosine triphosphatase
BAP	Benzylaminopurine
CCC	Chlormequat
DD	Deionised-distilled
DMRT	Duncan multiple range test
DNA	Deoxyribonucleic acid
GA	Gibberellin
GA_X	Gibberellin A_X (x = 1, 2, 3 .....n)
GA_3	Gibberellic acid
mg/L	Milligram/litre
IAA	Indoleacetic acid
IBA	Indolebutyric acid
kDa	Kilodalton
LS	Longitudinal section
MCPA	2-methyl-4-chlorophenoxyacetic acid
mRNA	Messenger ribonucleic acid
NAA	α-Napthaleneacetic acid
NPA	Naphthyphthalmic acid
PAA	Phenylacetic acid
PBZ	Paclobutrazol
PGR	Plant growth regulator
PP333	Paclobutrazol
ppm	Parts per million
RCBD	Randomised complete block design
RNA	Ribonucleic acid
SDP	Sturt's desert pea
STS	Silver thiosulphate
TS	Transverse section

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