Auxin and IAA oxidase activity related to the leaves gall formation in some forest trees

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ABSTRACT

Galls are the neoplastic outgrowths produced by the host organism usually in response to the presence of another living organism. The hypertrophy and hyperplasia of plant tissues result in the formation of galls in some forest trees. Leaves galls on Dalbergia sissoo Roxb. induced by Eriophyes sp. Tectona grandis L. induced by unknown gall midge, Salvadora oleoides dine induced by unknown itonididae and salvadora persica L. induced by Thomasiana salvadora are the forest trees of great economic importance. The result of the present investigation was to study the profile of auxin and IAA oxidase activity due to insect attack on leaves of these four trees and host - pathogen interaction. Bio-chemical analysis revealed hyperauxinxy and hypoindole 3 acetic acid oxidase activity in the gall tissues except Tectona grandis which have been correlated in relation to gall formation in trees.

Keywords: Auxin, IAA oxidase, gall formation, Gall tissues, normal counterparts, tree.

INTRODUCTION

This paper deals with leaves gall on Dalbergia sissoo Roxb. caused by Eriophyes Ap. (Acarina), Tectona grandis Linn. Caused by unknown itonididae (Deiptera), Salvadora oleoides Dine caused by unknown gall midge (Diptera) and Salvadora persica Linn. caused by Thomasiana salvadora Rao (Diptera) collected from Sitamata forest, Jaipur and adjoining areas. The tree species offers shade, firewood, timber, oil products, foods and forage for wildlife and domestic herbivores. The changes in auxin concentration and IAA oxidase activity after infection may thus cause abnormal growth response in the host, Dye et al. 1962, Sequeira and Kelman 1962, Srivastva and Shaw 1962 a, b, Ramani et al. 1989 and Santos and Varanda 2003). Singh et al. (2011) studies the auxin and IAA oxidase activity in diseased and healthy plant to correlate the changes in response due to infection in Brassica gall disease. An attempt has been made to study auxin contents (Total and free) and IAA oxidase activity of gall and normal tissues of leaves galls in four forest tree species.

RESULTS AND DISCUSSION

Results are presented in figures 1-3. Gall tissue of all the plants showed higher auxin (Free and total) contents as compared to the normal counterparts except Tectona grandis. Among the gall tissues 0.16 μg/g free and total auxin contents were recorded in Dalbergia sissoo. Gall tissue of sample tested, showed decreased IAA oxidase activity as compared to that of normal tissue except Tectona grandis. Tectona grandis gall tissue showed highest IAA oxidase activity while Dalbergia sissoo gall tissue showed 147 μg/h/g activity of enzyme. The increased auxin contents of gall tissues were related to the decreased IAA oxidase activity and vice-versa.

Generally, tumors are hyperauxinxy and produce auxin in more than regulatory amounts. Two elegant approaches to the problem concerning the role of auxin in gall formation have been reported. In one study mutants of the pathogen Pseudomonas savastanoi which were resistant to L-methyl tryptophane (MT) (an enzyme involved in IAA bio-synthesis) were shown to possess an altered pathway for IAA bio-synthesis. Isolates which were able to accumulate IAA caused gall formation, while MT sensitive isolates that could not synthesise and thus accumulate IAA in the presence of MT, did not produce gall formation. In another report a plasmid containing strain of Agrobacterium tumefaciens (C-50) was found to produce 5-10 times more IAA in presence of tyosine (A precursor in biosynthesis of IAA) than the strain devoid of Plasmid (Liu and Kado 1979).
**Fig. 1:** Free Auxin contents (μg/g dry weight of tissue).

- **Dalbergia sissoo**
- **Tectona grandis**
- **Salvadora oleoides**
- **Salvadora persica**

**Fig. 2:** Total Auxin contents (μg/g dry weight of tissue).

- **Series 1**
- **Series 2**

**Fig. 3:** μg IAA destroyed / h/g fresh weight of tissue.

- **Series 1**
- **Series 2**
High level of auxin in tumors resulted from reduced auxin destruction. Hyperauxiny has been reported for several insect and mite inducell gall tissues (Vyas, 1971, Byers et al., 1976, Chatterjee 1984, Kant & Ramani, 1986). Nakajima et al.; (1979) who analysed the crown gall cells of tobacco concluded that enhanced hormonal content is not the only factor associated with autonomous growth. It is not clear how the tumor inducing principle causes the tumor cells to become autonomous for auxin. Nor is it known whether there is any increased endogenous synthesis of auxin by the tumor cell. Is some subtle and as yet uncharacterized auxin-inactivating system, normally concerned with regulation for growth, destroyed as a result of the action of the tumor inducing principle? The imbalance of growth substance found in crown gall tumor cell appear not only to account for the continued abnormal growth of such cells but also plays an essential role in determining morphological, histological and cytological characteristics of the tumor. Although most investigations report hyperauxiny in galls, in a few cases reduced auxin levels has been reported (Ramani & Kant, 1989). The mechanism that culminate in reduced auxin level have been attributed to (i) auxin destruction by enzymes secreted by the parasite/insect (Krupasagar & Sequeira, 1969), (ii) degradation of auxin by enzyme of the infected plant (Daly & Devarall, 1963) and (iii) reduced concentration and conversion of auxin precursors (Scherphan 1950). Hyperauxiny may be attributed to the increased synthesis of auxin under the influence of cecidooza. Hypertrophy and hyperplasia in gall indicate that plant growth controlling substance such as auxin play an important role in gall formation. The evidences to support the findings are (i) the gall tissues showed lower IAA oxidase activity except Tectona grandis, (ii) the gall tissues showed more ortho-dihydroxyphenols than healthy ones (studied but not included in this paper). Which acts as IAA oxidase inhibitors and auxin protectors (Stonier, 1972, Tondon & Arya, 1982) and (iii) accumulation of ascobic acid (not included in this paper), the known factor which prevents IAA oxidase.

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