WOUND-INDUCED CHANGES IN ROOT AND SHOOT JASMONIC ACID POOLS CORRELATE WITH INDUCED NICOTINE SYNTHESIS IN Nicotiana sylvestris SPEGAZZINI AND COMES

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Abstract—Leaf damage by herbivores in Nicotiana sylvestris Spegazzini and Comes (Solanaceae) produces a damage signal that dramatically increases de novo nicotine synthesis in the roots. The increased synthesis leads to increases in whole-plant nicotine pools, which in turn make plants more resistant to further herbivore attack. Because signal production and the response to the signal occur in widely separated tissues, the speed with which different damage signals exit a damaged leaf can be studied. We propose that electrical damage signals should exit a leaf faster (less than 60 min) than chemical damage signals. Excision of a leaf induces a smaller increase in nicotine production than does puncture damage, so we examined our proposition by excising previously punctured leaves at 1, 60, and 960 rain after leaf puncture and quantifying the induced whole-plant nicotine pools six days later when the induced nicotine production had reached a maximum. Significant induced nicotine production occurred only if punctured leaves were excised more than 1 hr after puncture, which is consistent with the characteristics of a slow-moving chemical signal rather than a fast-moving electrical signal. We explore the nature of the chemical signal and demonstrate that additions of 90 μg or more of methyl jasmonate (MJ) in an aqueous solution to the roots of hydroponically grown plants induce de novo nicotine synthesis from 15NO3 in a manner similar to that induced by leaf damage. We examine the hypothesis that jasmonic acid (JA) functions in the transfer of the damage signal from shoot to root. Using GC-MS techniques to quantify whole-plant JA pools, we demonstrate that leaf damage rapidly (<0.5 hr) increases shoot JA pools and, more slowly (<2 hr), root JA pools. JA levels subsequently decay to levels

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found in undamaged plants within 24 hr and 10 hr for shoots and roots, respectively. The addition of sufficient quantities (186 μg) of MJ in a lanolin paste to leaves from hydroponically grown plants significantly increased endogenous root JA pools and increased de novo nicotine synthesis in these plants. However, the addition of 93 μg or less of MJ did not significantly increase endogenous root JA pools and did not significantly affect de novo nicotine synthesis. We propose that wounding increases shoot JA pools, which either directly through transport or indirectly through a systemin-like signal increase root JA pools, which, in turn, stimulate root nicotine synthesis and increase whole-plant nicotine pools.

Key Words—Induced defense, *Nicotiana sylvestris*, nicotine, damage signal, jasmonic acid, methyl jasmonate, electric signal.

INTRODUCTION

Leaf damage is known to increase rapidly the concentration of different types of secondary metabolites found in undamaged leaves. Many of these damage-induced increases have proven defense-related roles (Tallamy and Raupp, 1991; Baldwin, 1993). Much of the research on these systemic changes in plant chemistry induced by leaf damage has focused on identifying the chemical signals that activate the chemical changes (Enyedi et al., 1992; Staswick, 1992). For example, jasmonic acid and the polypeptide, systemin (Farmer and Ryan, 1990; Pearce et al., 1991), increase proteinase inhibitor (PI) proteins in tomato. However, chemical damage signals may not be the only type of signal involved. Wounding has long been known to result in electrical signals in plants (Davies, 1987), and a recent study (Wildon et al., 1992) has clearly implicated these electrical signals in the induction of PI proteins in young tomato plants after damage. Hence, in tomato, induced defense responses may be elicited by a combination of both electrical and chemical signals.

Electrical and chemical signals usually differ in the time required for them to exit damaged tissues; this difference may indicate which of the two is the primary damage signal. As reported in Davies (1987) and Wildon et al. (1992), electrical signals in pea and tomato plants travel at 3-5 cm/min and 6-24 cm/min, respectively. In tomato, the electrical signal implicated in the induction of PI proteins exited damaged cotyledons within 5 min (Wildon et al., 1992). Chemical signals, on the other hand, are likely to exit damaged leaves more slowly. Although the phloem transport rates of some chemical signals could reach speeds (Baker and Milburn, 1989) in the same range as the slowest electrical signals, chemical signals require either de novo or partial synthesis prior to the formation of the active signal molecule (Raskin, 1992; Farmer and Ryan, 1992; McGurl et al., 1992), which may result in additional delays in signal transduction.

The damage-induced increase in nicotine synthesis in *N. sylvestris* may be