Salicylic Acid Glucoside Acts as a Slow Inducer of Oxidative Burst in Tobacco Suspension Culture

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Salicylic acid β-glucoside (SAG) is a storage form of a defense signal against pathogens, releasing free salicylic acid (SA), to meet the requirements in plants. Since excess SA induces locally restricted cell death following oxidative burst and Ca^{2+} influx in plants, the effects of SAG on cell viability, Ca^{2+} influx, and generation of superoxide ($O_2^{\bullet-}$) were examined in suspension-cultured tobacco BY-2 cells expressing aequorin. Among SA-related chemicals tested, only SAG induced the slow and long-lasting $O_2^{\bullet-}$ generation, although SAG was less active in acute $O_2^{\bullet-}$ generation, Ca^{2+} influx and induction of cell death. The prolonging action of SAG is likely due to gradual release of SA and the data suggested that a peroxidase-dependent reaction is involved. Notably, pretreatment with low-dose SA (50 μM) enhanced the response to SAG by 2.5-fold. There are four possible secondary messengers in early SA signaling detectable in the BY-2 culture, namely $O_2^{\bullet-}$, H_2O_2, Ca^{2+} and protein kinase (PK). If these messengers are involved in the low-dose SA-dependent priming for SAG response, they should be inducible by low-dose SA. Among the four SA-inducible signaling events, PK activation was excluded from the low-dose SA action since a much higher SA dose (> 0.4 mM) was required for PK activation.

Key words: Priming, Salicylic Acid, Salicylic Acid β-Glucoside