Phaeomoniella chlamydospora-induced Oxidative Burst in *Vitis vinifera* Cell Suspensions: Role of NADPH Oxidase and Ca^{2+}

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Abstract

The biphasic oxidative burst induced by *Phaeomoniella chlamydospora* extract (Pce) in *Vitis vinifera* (Vv) cell suspensions was investigated. Treatment of cell suspensions with diphenyleneiodonium chloride, an inhibitor of NADPH oxidase, prevented the Pce-induced biphasic reactive oxygen species (ROS) accumulation, suggesting that NADPH oxidase is the primary ROS source in the oxidative burst induced by Pce elicitation of Vv cells. The role of Ca^{2+} in the oxidative burst was also investigated using a Ca^{2+} chelator and several Ca^{2+} channel blockers. The treatment of Vv cell suspensions with the Ca^{2+} chelator ethylene glycol-bis(2-aminoethylether)-N, N, N'; N'-tetraacetic acid (EGTA) completely inhibited Pce-induced ROS accumulation, suggesting that Ca^{2+} availability is necessary for occurrence of the induced oxidative burst. However, only the Ca^{2+} channel blocker ruthenium red strongly inhibited the Pce-induced ROS accumulation, suggesting that the specific Ca^{2+} channel types from which Ca^{2+} influx is originated also play an important role in the Pce-induced oxidative burst. Furthermore, Ca^{2+} availability seems to be necessary for the Pce-induced activity of NADPH oxidase.

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