Early Elicitor-Induced Events in Chickpea Cells: Functional Links between Oxidative Burst, Sequential Occurrence of Extracellular Alkalinisation and Acidification, K^+/H^+ Exchange and Defence-Related Gene Activation

Oliver Otte, Andreas Pachten, Frauke Hein, and Wolfgang Barz*

Institut für Biochemie und Biotechnologie der Pflanzen, Hindenburgplatz 55, Westfälische Wilhelms-Universität, 48143 Münster, Germany.
Fax: ++49–251–8328371. E-mail: pachten@uni-muenster.de

* Author for correspondence and reprint request

Z. Naturforsch. **56 c,** 65–76 (2001); received August 29, 2000

Cicer arietinum, Defence Related Gene Activation, Extracellular Acidification, Oxidative Burst

Elicitation of cultured chickpea (*Cicer arietinum* L.) cells stimulates a signal transduction pathway leading to several rapid responses: (1) oxidative burst, (2) extracellular alkalinisation, (3) extracellular acidification, (4) transient K^+ efflux, and (5) activation of defence related genes all within 2 hours. Induced genes are encoding acidic and basic chitinases, a thaumatin-like protein and isoflavone reductase. All these elicitor-induced responses are inhibited by the Ser/Thr protein kinase inhibitor staurosporine and the anion channel blocker anthracene-9-carboxylic acid but stimulated by the Ser/Thr protein phosphatase 2A inhibitor cantharidin. The oxidative burst leads to a transient extracellular H_2O_2 accumulation which seems to be preceded by $\cdot O_2^-$ production, indicating dismutation of $\cdot O_2^-$ to H_2O_2. The oxidative burst is accompanied by transient alkalinisation of the culture medium which is followed by long-lasting extracellular acidification. An 80 percent inhibition of the alkalinisation after complete inhibition of the H_2O_2 burst with diphenylene iodonium indicates that the elicitor induced increase of extracellular pH is mainly based on a proton consumption for $\cdot O_2^-$ dismutation. A simultaneous deactivation of the plasma membrane H^+-ATPase during oxidative burst and extracellular alkalinisation is also suggested. The elicitor-stimulated extracellular acidification is inhibited by the plasma membrane H^+-ATPase inhibitor N, N′-dicyclohexylcarbodiimide assuming a reactivation of the H^+-ATPase 25 min after elicitation. Extracellular acidification seems not to be necessary for elicitor-induced activation of defence related genes. Opposite modulation of K^+ and proton fluxes after elicitation and/or treatment with the H^+-ATPase effectors fusicoccin or N, N′-dicyclohexylcarbodiimide indicate that the elicitor induced transient K^+ efflux is regulated by a K^+/H^+ exchange reaction.