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Title
Boron deficiency in the legume-rhizobia interaction:Symbiosis or pathogenesis?

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Authors
Reguera, María
Bolaños, Luis
Bonilla, Ildefonso

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Introduction

During growth of infection threads through which the rhizobia invade nodules, the host legume secretes glycoproteins (mainly a specific arabinogalactan protein extension, AGPE) (Rathbun et al., 2002) able to attach to the rhizobial cell surface (Bolaños et al., 1996). The bacterium seems to escape from this "glycoprotein trap" inducing AGPE peroxide-driven cross-linking which determines the polar growth of the infection thread (Brewin, 2004). This does not happen under B deficiency, and bacterial progress is arrested.

Moreover, cytological evidence for early necrotic events in B-deficient *Pisum sativum* root nodules suggests elicitation of host defense mechanisms similar to those in nodules induced by cell surface polysaccharide bacterial mutants (Perotto et al., 1994). Therefore, occurrence of pathogenesis-related (PR) proteins was studied in nodules developed in the presence (+B) or the absence (-B) of B. Following two-dimensional electrophoresis and MALDI TOF spectrometry analysis, two proteins of the PR10 family, highly induced in –B but not in +B nodules, were identified as *P. sativum* pathogenesis-related cytosolic proteins, PR10.1 and ABR17. The use of anti-ABR17 and analysis of gene expression confirmed that this protein was induced in B-deficient young nodules, being increased during nodule development. Moreover, independently of B, ABR17 protein increased in infected roots or in senescent tissues although at a level weaker than ABR17 occurrence in -B nodules.

Immunoassays in nodule sections indicated that ABR17 protein occurrence is restricted, at a low level, to the cortical tissues of B-sufficient nodules, but it increased during B-deficient development in all of nodule tissue, close to the cell surface or even inside necrotic bacteria from –B nodules, supporting the idea that both, PR10.1 and ABR17, could act as ribonuclease-like proteins (Srivastava et al., 2006, 2007).

A high induction of this PR protein was also observed in pseudonodules induced by non-invasive cell surface polysaccharide defective rhizobial mutants and, as in –B nodules, it was distributed throughout all nodule tissues.

These results suggest that rhizobia are able to switch-off ABR17 induction in nodules resulting from homologous interactions, but B deficiency or defective bacterial cell surface polysaccharides might alter the bacteria-legume dialogue and determine a pathogenic-like rather than a symbiotic interaction.

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