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CHARAKTERYSTYKA LIPOPOLISACHARYDOW *RHIZOBIUM* I ICH ZNACZENIE W PROCESIE SYMBIOZY

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Artykuł dedykowany prof., dr hab. Krystynie Kotełko, jest zmodyfikowaną formą wykładu wygłoszonego 26 października 2004 na Sesji Naukowej pt. „Badania immunochemiczne endotoksyn (LPS) bakterii", podczas uroczystości poświęconej pamięci prof., dr hab. Krystyny Kotełko, na Wydziale Biologii i Ochrony Środowiska Uniwersytetu Łódzkiego

The characterization of the *Rhizobium* lipopolysaccharides and their significance in symbiosis

Abstract: Four types of rhizobial polysaccharides i.e. exopolysaccharides (EPSs), cyclic -glucans, K antigens, and Lipopolysaccharides (LPSs), have been shown by mutation studies to be required in the development of various root nodule symbioses. This chapter deals with LPS, a major constituent of the bacterial outer membrane which plays a crucial, albeit poorly understood role in nodule invasion in the symbiotic process.

Mutations that affect LPS structure indicate that normal abundance and certain structural features of the LPS O-specific polysaccharide are required for successful infection of host leguminous plants. Consequences of abnormal O antigen deficiency include the induction of host defence responses, from early blocks in infection thread formation to defects in infection thread release, severely reduced proliferation of bacteroids and finally stunted nodule development. The LPS also plays an important role in adapting bacteria to the plant and other environments. Its structure is altered during biosynthesis in response to factors such as low pH, low oxygen, and host anthocyanins. The more hydrophobic character of LPS occurred during the differentiation from bacterium to bacteroid and is due to higher proportion of long chain fatty acids attached to the lipid A moiety, shortening of PS O-chain and alterations in the composition of 0 antigen including an increase in acetylation and methylation pattern of the saccharidic domains. Interestingly, these changes are similar to those exhibited by some pathogenic bacteria on infection of their hosts.

Currently, no information is available on the molecular mechanisms in the symbiont triggering these alterations.

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