

# Bacterial Lipopolysaccharide-Dependent Complement Evasion

Bożena Futoma-Koloch\*

Department of Microbiology, University of Wrocław, Przybyszewskiego Wrocław, Poland

\*Corresponding author: Bożena Futoma-Koloch, Department of Microbiology, University of Wrocław, Przybyszewskiego Wrocław, Poland, Tel: 48 71 375 62 22; Fax: 48 71 325 21 51; E-mail: [bozena.futoma-koloch@uw.edu.pl](mailto:bozena.futoma-koloch@uw.edu.pl)

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## Editorial

Finding factors that control complement (C) components deposition on bacterial cells is the way to explain the sophisticated mechanism of bacteraemia. Lipopolysaccharide (LPS, endotoxin) is a surface antigen found in the cell walls of Gram-negative bacteria, such as *Salmonella* [1]. LPS action on the human body is often considered in the context of sepsis, which is characterized by an excessive inflammatory response in the presence of endotoxin. At low concentrations, LPS stimulates innate immune system, which helps the host to eliminate the invader; but at high levels, it causes shock and even death [2]. Moreover, recent LPS characterization supports the development of a new vaccine against non-typhoidal *Salmonellae* for Africa [3]. From the point of view of pathogens, LPS-dependent resistance to C-mediated killing is an essential virulence property. In this paper, I want to highlight the main findings on the role of LPS in bacterial C-dependent serum susceptibility. To introduce, it is worth to bring forward a general structure of endotoxin. LPS of smooth strains (S) consists of three structural domains: lipid A, which anchors the whole molecule in the bacterial cell wall, core oligosaccharide, and O-specific polysaccharide chains (O antigen, O-Ag). Lipooligosaccharide (LOS) is analogous to the LPS, but it lacks O-Ag. When the LPS is connected to the bacterium, the outermost fragment - O antigen determines the response of the inflammatory system. In case of killing or disruption of bacterial cells with drugs (i.e. antibiotics) results in a lipid A release, the most toxic domain of endotoxin [1]. The role of LPS and its chemotypes in the resistance of bacteria to C has not been investigated thoroughly. Since the 80s, it has been known that long-chain LPS with complete O-specific chains confers bacterial resistance to serum by promoting the deposition of C components at a distance from the cell wall, thus preventing its disruption with the C5b-9 complexes. It was demonstrated that the amount of LPS O-Ag and its chain length distribution are important factors that protect f

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