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The redefinition of *Helicobacter pylori* lipopolysaccharide O-antigen and coreoligosaccharide domains

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Helicobacter pylori lipopolysaccharide promotes chronic gastric colonization through O-antigen host mimicry and resistance to mucosal antimicrobial peptides mediated primarily by modifications of the lipid A. The structural organization of the core and O-antigen domains of *H. pylori* lipopolysaccharide remains unclear, as the O-antigen attachment site has still to be identified experimentally. Here, structural investigations of lipopolysaccharides purified from two wild-type strains and the O-antigen ligase mutant revealed that the *H. pylori* core-oligosaccharide domain is a short conserved hexasaccharide (Glc-Gal-DD-Hep-LD-Hep-LD-Hep-KDO) decorated with the O-antigen domain encompassing a conserved trisaccharide (-DD-Hep-Fuc-GlcNAc-) and variable glucan, heptan and Lewis antigens. Furthermore, the putative heptosyltransferase HP1284 was found to be required for the transfer of the third heptose residue to the core-oligosaccharide. Interestingly, mutation of HP1284 did not affect the ligation of the O-antigen

and resulted in the attachment of the O-antigen onto an incomplete core-oligosaccharide missing the third heptose and the adjoining Glc-Gal residues. Mutants deficient in either HP1284 or O-antigen ligase displayed a moderate increase in susceptibility to polymyxin B but were unable to colonise the mouse gastric mucosa. Finally, mapping mutagenesis and colonisation data of previous studies onto the redefined organisation of *H. pylori* lipopolysaccharide revealed that only the conserved motifs were essential for colonization. In conclusion, *H. pylori* lipopolysaccharide is missing the canonical inner and outer core organization. Instead it displays a short core and longer O-antigen encompassing residues previously assigned as the outer core domain. The previously proposed LPS structure in strain 26695 wild-type (A), the redefined LPS structures of the G27 wild-type (B), G27 Δ HP1284 (C) and G27 Δ waaL (D). The nomination of different domains of the LPS is annotated.

Biography

Hong Li obtained his MD degree at North Sichuan Medical College, China in 2006. He then became a registered physician in West China Hospital in 2007. He obtained his Master degree in HBV infection at West China Hospital, Sichuan University in 2009. In October 2009, he was awarded the PhD Full-Scholarship from The University of Western Australia (UWA) under the supervision of Nobel laureate Professor Barry Marshall, Drs Mohammed Benghezal, Hans-Olof Nilsson and Keith Stubbs from UWA. His study in UWA led to the completion of his PhD thesis titled "Lipopolysaccharide biosynthesis and protein glycosylation in *Helicobacter pylori*" and awarded the PhD degree in Microbiology from UWA. In 2015, Hong Li returned back to West China Hospital of Sichuan University and actively promoted the academic collaboration between Sichuan University and UWA with the establishment of the collaboration center—"West China-Marshall Research Center for Infectious Diseases" in Chengdu.

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