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**Keywords: lipid II, moenomycin, transglycosylase**

## MS36.P09

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**Structural analysis of Toll-like receptor 2-activating lipoprotein**

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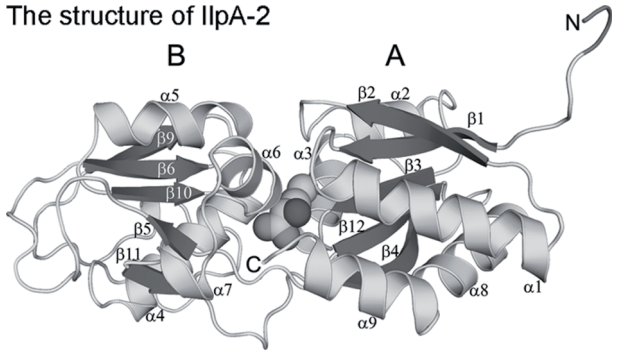
IlpA, a surface protein of the human pathogen *Vibrio vulnificus*, is the first lipoprotein to be characterized in *Vibrio* spp. as a major immunostimulant. Previously, it was characterized that IlpA was subject to lipidation at its N-terminal cysteine residue. The resulting IlpA then activates Toll-like receptor 2 in human cells, and induces overproduction of proinflammatory cytokines closely associated with septic shock in infected individuals. To identify structural features of IlpA, we determined the crystal structure of IlpA at 2.6 Å resolution. Specifically, IlpA consists of two homologous domains, each with α/β topology, similar to the structure of substrate-binding protein which is a component of ATP-binding cassette transporter. In fact, binding of L-methionine was observed in the pocket between the two domains, suggesting that IlpA is an L-methionine-binding protein. The structural features of IlpA in this study, along with the immunological properties of IlpA identified previously and other substrate-binding proteins, suggest that substrate-binding lipoproteins of ATP-binding cassette transporter present at the bacterial cell surface could serve as pathogen-associated molecular patterns to Toll-like receptor 2, causing host immune responses against infection.

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**Keywords: lipoprotein, structure, substrate-binding**

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**Structure of the catalytic domain of *H. pylori* cholesterol-α-glucosyltransferase**

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α-Glucosyl cholesterol and its derivatives are the major cell wall components of *Helicobacter pylori*, also playing an important role in immune evasion and survival. *H. pylori* makes α-glucosyl cholesterol by glucosylating cholesterol extracted from the plasma membranes of human gastric mucosa cells, using the enzyme cholesterol-α-glucosyltransferase. Here we present the crystal structure of the catalytic domain of cholesterol-α-glucosyltransferase from *H. pylori* at 1.50 Å resolution, providing a platform for discovering specific inhibitors of *H. pylori* cholesterol-α-glucosyltransferase that could be developed as novel antibiotics. This work was funded by Korea Ministry of Education, Science, and Technology, National Research Foundation of Korea; Basic Science Outstanding Scholars Program, World-Class University Program, and Innovative Drug Research Center for Metabolic and Inflammatory Disease; Korea Ministry of Health, Welfare & Family Affairs (Korea Healthcare Technology R&D Project, Grant no. A092006).

**Keywords: cholesterol-α-glucosyltransferase, HP0421, helicobacter pylori**

## MS36.P11

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**Structure of the *Streptococcus pyogenes* β-NAD+ glycohydrolase-inhibitor complex**

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*Streptococcus pyogenes* (group A streptococcus; GAS) secretes several extracellular proteins which contribute to pathogenesis. Among them, β-$NAD^+$ glycohydrolase (SPN) is an important virulence factor. The mechanism for pathogenesis is intracellular β-$NAD^+$ depletion within the host cell due to the β-$NAD^+$ hydrolytic activity of SPN. SPN is also toxic to the bacterium itself; therefore, GAS encodes the *ifs* gene, whose product (IFS) is an endogenous inhibitor of the $NAD^+$ glycohydrolase. In order to understand the inhibition mechanism of SPN by IFS, we have determined the crystal structure of the SPN-IFS complex at 1.8 Å resolution. SPN is an atypical member of the ADP-ribosyltransferase superfamily, lacking a canonical binding site for the protein substrate. The SPN-IFS complex is stabilized by numerous hydrogen bonds and electrostatic interactions. In the complex structure, IFS covers the active site of SPN and blocks the binding of β-$NAD^+$.

**Keywords: β-NAD+ glycohydrolase, IFS, streptococcus pyogenes**

## MS36.P12

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**Crystal structures of Eis proteins from *M. tuberculosis* and *M. smegmatis***

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