

## Functional Characterisation of the *Helicobacter pylori* Outer Membrane Protein BabC

Mohammed A. Alissa

University of Nottingham, United Kingdom

### Abstract:

*Helicobacter pylori* (*H. pylori*) is a pathogenic bacterium that colonises the gastric mucosa of more than 50% of all humans and a leading cause of peptic ulceration and gastric cancer worldwide. Despite the harsh conditions in the stomach, the bacterial adherence to the gastric mucosa and epithelium plays a significant role in initial colonisation and in long-term persistence by using multitude of adhesins that facilitate the adhesion to the host cells. Studies are greatly needed to understand the dynamic interplay between these adhesins, some of which have not been fully characterised. Furthermore, the need to develop alternative therapeutic strategies is important with the increased rate of antibiotic resistance associated with *H. pylori* eradication through targeting these adhesins.

This study is aimed at characterising the functional properties of the *H. pylori* outer membrane protein BabC, which has not been studied well previously with unknown structure and function. The first objectives of this study have been successfully completed through amplification of babC gene from the *H. pylori* strain 26695, cloning it into pOPE101, followed by transformation of the pOPE101\_babC construct into *E. coli* XL10 Gold, and periplasmic expression and purification of BabC. BabC protein did not show any binding to Lewis a (Lea), Leb, Ley, or sialyl-Lewis x (sLex) glycoconjugates which are considered as the main glycan epitopes in gastrointestinal mucins. Analysis of amino acid sequence of the crowns of BabA J99 and BabC 26695 shows that the residues D233 and S234 in BabA crown, partially responsible for BabA binding to Leb, are missing in the amino acid sequence of the predicted BabC crown in which three hydrogen bonds will not be mediated with Leb; two hydrogen bonds between D233 and Gal5/GlcNAc3 and



one hydrogen bond between S234 and Gal5. Based on this analysis, binding of BabC to Leb may not happen. Future work will reveal more about BabC and will bring us closer to achieve our aim.

### Biography:

Mohammed A. Alissa is a PhD student in School of Pharmacy at the University of Nottingham. He gained his MSc in Molecular Medical Microbiology from the University of Nottingham in 2014. He obtained his BSc in Clinical Laboratory Sciences from King Saud University (Riyadh, Saudi Arabia) in 2008. Mohammed is currently lecturer of molecular medical microbiology at Prince Sattam bin Abdulaziz University (Alkharj, Saudi Arabia) in 2015. He was appointed as Teaching Assistant at Prince Sattam bin Abdulaziz University in 2011. He was working as Medical Laboratory Scientific Officer at the Military Hospital (Alkharj, Saudi Arabia), where he worked for two years.

### Publication of speakers:

1. Hage, N. et. al. 2015. Protein Expr Purif 106: 25-30.
2. Barbosa-Jobim, G. S., et. al. 2020. Toxicol In Vitro 63, 104735.
3. IARC 1994. IARC Working Group, Lyon
4. Hage, N. et. al. 2015. Science advances 1, e1500315

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