# Researchers investigate cellular mechanisms leading to immune response in airway epithelium

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Researchers from the Boston University School of Medicine (BUSM) have demonstrated that commensal species of the genus *Neisseriae* are low inducers of human airway epithelial cell responses as compared to the pathogenic species. Specifically, the study indicates that a Neisserial outer membrane component appears to play a differential role in the host inflammatory responses via interaction with a receptor on the surface of human airway epithelial cells.

Paola Massari, an assistant professor in the section of infectious diseases at BUSM, is lead author of this study, which is published in the Dec. 2010 issue of the journal *Infection and Immunity*.

The team focused their research on *Neisseria lactamica*, a gram negative organism comprising both commensal and pathogenic bacteria. *Neisseria meningitidis*, the causative agent of meningococcal meningitis, as well as *Neisseria lactamica*, colonize the human nasopharynx, but only *Neisseria meningitidis* is pathogenic.

"We set out to understand the relationship between commensal *Neisseriae* organisms and the human hosts," said Massari. "Although *Neisseriae* organisms express mostly identical surface antigens and structures, they appear to induce different responses when they interact with the host."

To examine how the bacteria interact with human nasopharyngeal cells, Massari and her research team honed in on a bacterial surface component, the PorB porin, present in all *Neisseriae* organisms. After purifying the PorB, they found that the protein from the commensal bacteria induced lower levels of human airway epithelial cell activation compared to PorB purified from the pathogenic organisms.

Next, the team demonstrated that PorB from *N. lactamica* and PorB from *N. meningitidis* appear to interact with the host cell surface receptor, Toll-like receptor 2 (TLR2), in a differential manner, thus leading to different inflammatory responses in human airway epithelial cells.

"This study confirms that TLR2 signaling is essential for the activation of human airway epithelial cells," said Massari. "This is likely one of the mechanisms by which the body limits inflammation in response to colonization with harmless commensal bacteria, thus avoiding exacerbation of inflammatory responses and local chronic local inflammation.

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