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Editorial

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The Road Not Taken: Commensal or Virulent Pathogen

Despite the ongoing COVID-19 pandemic, it is important to remember that bacterial pathogens remain a constant threat, not least in the light of resistance to antibiotics and an increasing number of immunocompromised individuals [1]. It is fascinating to observe that bacteria have evolved such different strategies, paving their way to success. Bacterial species being part of the human microbiota often promotes the well-being of the host through symbiosis, thereby securing their own longstanding ecological niche [2]. However, a disturbed composition of the microbiota may have a deleterious impact on, for example, energy metabolism, remote organ functions, and inflammatory diseases [3–6]. How could the strategies exerted by virulent bacterial pathogens result in evolutionary success, even though they harm and may even kill their host? The question is likely to have several complex answers, and opportunistic pathogens may hide in the shades in between. In this issue of the *Journal of Innate Immunity,* Siemens et al. [7] characterize a novel virulence factor of group B streptococci, i.e., a pigment toxin that caused the release of proinflammatory cytokines and induced blood clotting on the bacterial cell surface. In a clinical setting, the cytotoxic properties indeed play important roles in the clinical picture [8]. In another interesting article, Tsai et al. [9] show that when exposing macrophages to group B streptococci, sialic acid-binding immunoglobulin-type lectin receptors (Siglecs) modulate the response, where Siglec-14 had an enhancing effect whereas Siglec-5 reduced inflammasome activation and macrophage IL-1β release. The findings may have a bearing on modulation of the inflammatory response during severe infections. Liu et al. [10] found that activation of TLR3 results in IL-1 receptor antagonist expression through interferon regulatory factor 3, demonstrating yet another intrinsic anti-inflammatory pathway. However, bacteria are also able to corrupt inflammasome activation as exemplified by the ubiquitination and degradation of pro-IL-1β by streptolysin O released by group A streptococci (*Streptococcus pyogenes*) [11]. These bacteria are highly pathogenic, causing a broad range of severe clinical conditions [12]. One important virulence factor of group A streptococci is the surface-associated M protein. Using cold atmospheric plasma, Persson et al. [13] investigated how these bacteria can be disarmed with regard to this virulence factor using this potentially important therapeutic approach.

The delicate balance between the virulence of bacterial pathogens and the degree of inflammatory response mounted by the host to clear the infection continues to challenge scientists and clinicians. As always, we hope that this collection of articles will be of great interest to the readership of the *Journal of Innate Immunity*.

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