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# Response to Letter to the Editor, “The Role of Membrane Attack Complex Formation against Gram-positive Bacteria”

C. Jeanneau<sup>1</sup> and I. About<sup>1</sup>

It has long been accepted that Gram-positive bacteria resist the membrane attack complex (MAC) because of a protective, thick peptidoglycan layer (Joiner et al. 1983). However, this is contradicted by reports showing that MAC components are assembled on Gram-positive bacteria in specific localizations and that this assembly differs among bacterial strains (Berends et al. 2013). Also, MAC components' deficiency increases patients' susceptibility to infections not only with Gram-negative but also with some Gram-positive bacteria (Skattum et al. 2011). Moreover, many Gram-positive bacteria not only recruit the host inhibitors of MAC but also express proteins that inhibit MAC formation (Laursen et al. 2010). For example, *Streptococcus pyogenes* secretes streptococcal inhibitor of complement, which blocks the C5b-7 membrane insertion site and thereby prevents MAC formation (Akeson et al. 1996). This shows clearly that the peptidoglycan wall is not enough to protect all Gram-positive bacteria from MAC lysis.

During carious decay, the pulp is subject to invasion mainly by Gram-positive bacteria. This specialized tissue has a terminal circulation, and it has been shown that, in addition to the systemic regulation, the pulp plays a role in its own local regulation of innervation, vascularization, and regeneration (About 2013). Also, it appears unique with regard to complement synthesis and function (Chmilewsky et al. 2014).

Thus, we share the enthusiasm of Alves and Freires (2016) in their letter to the editor regarding our recently published work (Jeanneau et al. 2015). Indeed, these new data pave the way for future research on pulp synthesis of MAC co-stimulators/inducers and the way that this affects Gram-positive bacteria.

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Scientifique. The authors declare no potential conflicts of interest with respect to the authorship and/or publication of this article.

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