Correspondence

The α-Enolase of Streptococcus suis: A Previously Well-Known and Well-Characterized Protein

To the Editor—We read with interest the article by Feng et al [1] that describes the α-enolase of Streptococcus suis (SsEno). S. suis is an important swine pathogen and is considered an emerging zoonotic agent, mainly in Asian countries, and severe human outbreaks with clinical manifestations of streptococcal toxic shock–like syndrome have been reported [2]. As correctly mentioned by the authors, the strain responsible for this episode presents some atypical features, such as a pathogenicity island of 89 kb [3]. Also, in collaboration with the Center for Disease Control in Beijing, we performed multilocus sequence typing and showed that this strain belongs to a different sequence type (ST) that was classified as ST7 [4]. Besides the unique 89-kb pathogenicity island, other putative virulence factors were proposed to explain the higher virulence of this strain [5]. However, more studies are needed to achieve a definitive conclusion.

This letter raises concerns about the novelty of results presented by Feng et al [1] and the hypothetical association of SsEno with the highly virulent Chinese S. suis strain. Three previous articles, specifically on SsEno, were published before the reception date of the manuscript by Feng et al [1]: Esgleas et al (2008) [6], Esgleas et al (2009) [7], and Zhang et al (2009) [8]. Although 2 of these publications were cited [6, 8] by Feng et al [1], the authors failed to unambiguously explain previous findings. We would like to also take into consideration the following facts.

Publication by Esgleas et al (2008) [6]. These authors had previously cloned SsEno, expressed it as a His-tagged fusion protein, and purified it. Similarities with other bacterial enolases were also discussed. The authors demonstrated the biochemical enolase activity of the purified protein. It was shown that SsEno was present in S. suis supernatant, cell wall, and cytoplasmic fraction. Even more, these authors demonstrated that SsEno is expressed on the cell surface, by means of electron microscopy. Unfortunately, in the study of Feng et al [1] these results were partially confirmed by indirect methods, because electron microscopy results were presented as “preliminary” and not shown. Esgleas et al [6] also clearly demonstrated the role of SsEno on adhesion and invasion of host cells.

Publication by Zhang et al (2009) [8]. Similar to Feng et al [1], Zhang et al [8] had previously demonstrated protection with SsEno in a mouse model, by using a method almost identical to that reported by Feng et al [1], including animals of the same age, the same adjuvant, and booster vaccination after 14 days. Similar to Feng et al [1], Zhang et al [8] had previously used immunofluorescence to verify the attachment of SsEno to Hep-2 cells, the same cell line used by Feng et al [1], and had previously reported protection of S. suis adherence to Hep-2 cells by SsEno.

Publication by Esgleas et al (2009) [7]. Similar to what was reported by Feng et al [1], Esgleas et al (2009) [7] had previously used a very similar enzyme-linked immunosorbent assay and demonstrated that serum from convalescent pigs (different from control pigs) strongly recognize SsEno. Unfortunately, reference to this previous work was not included in the article by Feng et al [1].

It would be extremely hazardous to speculate that “the presence of [SsEno] on the cell surface could be correlated with high invasiveness of Chinese [S. suis type 2] strains” [1]. In fact, as shown by Esgleas et al [6], all reference strains from the 35 serotypes of S. suis tested so far express SsEno at the bacterial surface. SsEno is by far not restricted to the Chinese strain. We believe that Feng et al [1] made a premature and rather incorrect statement regarding the importance of SsEno for the ST7 strain.

In conclusion, the information indicating that SsEno is a surface-exposed important antigen that may elicit protection against S. suis infection is not new, and most data had already been published by other research groups.

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