**Supplementary results**

**Effect of selenium on serum immunoglobulins and specific antibodies**

Supplementary Figure 2 shows that serum levels of IgM and IgG were higher in infected animals from seven until twenty eight days post-infection compared to non-infected animals, independently of selenium supplementation. Nevertheless, the difference between them was not significant (for all comparisons, *p* > 0.05). Additionally, circulating levels of *S. aureus*-specific IgM (SpIgM) and IgG (SpIgG) antibodies were increased from 7 until 28 days after infection in selenium-supplemented animals as compared with non-selenium-supplemented animals; whereas, the significant level was reached only for SpIgG at day 7 after infection (*p* = 0.010).

**Discussion**

In addition to its role in the inflammatory response, nutritional supplementation of selenium can also play a role in boosting immunity, especially in the regulation of cytokines expression and enhancing Th1 immunity, which is required for the differentiation of activated T cells into cytotoxic T cells [1], but also in the humoral response by stimulating the production of antibodies by auxiliary activated B cells [2,3].

The immunoglobulin production based on selenium intakes have been evaluated by different authors. Therefore, the intramuscular injection of vitamin E and selenium, or the single oral administration of selenium, increases the production of immunoglobulins in calves after injection of egg lysozyme [4]. These data are fully consistent with ours. However, there are heterogeneous results in terms of the production of immunoglobulins, depending on the animal species, the specificity of the antibody and the nature of the infectious agent. Therefore, similarly to our results about specific antibodies aginst *S. aureus*, it has been observed in sheep and lambs that the selenium supplementation in feed led to an increased antibody production against *Clostridium tetani* [5] or PI3 virus and *Corynebacterium ovis* [6]. In contrast, in vitamin E and selenium doubly deficient mice infected with *Citrobacter rodentium*, the antigen-specific IgG or IgM production is not affected by vitamin E and selenium administration [7].

We observed that at day 7 post-infection, the IgM and IgG titers reach their maximum. So, during the normal course of infection, the pathogen proliferates and reaches the threshold level of induction of the adaptive immune response. Over time, most of the effector T cells die, and antibody levels gradually decline, because the antigens that induced the immune response are no longer present at the level needed to sustain it. This mechanism would be considered as a feedback loop regulating immune response [8].

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