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3450 - Category: IMMUNOLOGY OF INFECTIOUS AND PARASITIC DISEASES (ID)
Hyperimmune bovine colostrum as a source of anti-SARS-CoV-2 neutralizing antibodies

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Introduction: The global need for affordable and scalable immunoprophylaxis strategies against SARS-CoV-2 has renewed interest in bovine colostrum as a source of neutralizing antibodies. This study aimed to evaluate the immunogenicity and neutralizing capacity of colostrum from cows immunized with SARS-CoV-2 recombinant receptor-binding domain (RBD) proteins.
Methods: Pregnant cows received three intramuscular immunizations at 45, 30, and 15 days before calving with recombinant SARS-CoV-2 RBD produced in HEK293F cells and formulated with either Quil-A saponin or aluminum hydroxide (Alum) as adjuvants. Antibody responses in serum and colostrum were analyzed by indirect ELISA for total IgG, IgG1, IgG2, and avidity index. Neutralizing activity was assessed by competitive ELISA. Statistical analyses were performed using two-way ANOVA and Tukey's test ($P < 0.05$).
Results: Both adjuvanted formulations induced robust systemic and mucosal humoral responses. Vaccinated animals showed significantly higher anti-RBD IgG titers in serum and colostrum compared to controls ($P < 0.001$; Mann-Whitney test). Quil-A induced higher serum IgG1 and IgG2 levels than Alum ($P < 0.01$). The competitive ELISA confirmed neutralizing activity of colostrum antibodies, with inhibition of RBD-ACE2 binding exceeding 80% in the Alum group and 96% in the QuilA group.
Conclusions: Immunization of pregnant cows with recombinant RBD and adjuvants efficiently induced RBD-specific antibodies in colostrum with demonstrated neutralizing capacity. These findings support the use of hyperimmune bovine colostrum as a scalable, safe and cost-effective source of passive immune protection.
Keywords: Bovine colostrum ;Covid-19;neutralizing activity.

3452 - Category: IMMUNOLOGY OF INFECTIOUS AND PARASITIC DISEASES (ID)
Investigating the impact of skin colonization by Staphylococcus spp. on Leishmania amazonensis infection and inflammasome activation in a murine model

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Cutaneous leishmaniasis (CL), caused by *Leishmania* parasites, is characterized by chronic skin lesions whose severity varies depending on multiple factors, including host–microbe interactions. Growing evidence indicates that the skin microbiota plays a key role in modulating disease progression. In murine models, germ-free mice infected with *Leishmania major* develop smaller lesions than conventional mice, and recolonization with *Staphylococcus epidermidis* restores lesion development. Similarly, co-infection with *S. aureus* exacerbates skin pathology and increases the production of pro-inflammatory cytokines such as IL-1β and IL-17. In clinical settings, CL lesions from patients infected with *L. braziliensis* have shown a predominance of *Staphylococcus* spp., which correlates with elevated expression of IL-1α/β, suggesting skin microbiota regulates inflammasome activation. To investigate the contribution of different skin-colonizing *Staphylococcus* species to *Leishmania amazonensis* infection, we developed a murine model of sequential colonization followed by parasitic challenge. C57BL/6J mice were topically colonized on the ear five times at 4-day intervals with isolates from human skin: *S. aureus* (AD04.E17), *S. epidermidis* (NIHLM087), or *S. xylosus* (42C08). Colonization was confirmed via ear swabs cultured on Mannitol Salt Agar. Subsequently, mice were infected intradermally in the ear with 10^6 *L. amazonensis*-PH8. A control group received parasite infection only. We are currently optimizing the colonization protocol and monitoring lesion development across groups. This model aims to elucidate whether and how different *Staphylococcus* species modulate the inflammatory response and inflammasome activation during *Leishmania* infection.
Keywords: Staphylococcus sp;Leishmania amazonensis;inflammasome.