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LETTER TO THE EDITOR

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Defining protective epitopes for COVID-19 vaccination models

To the Editor,

Recent papers in the Journal provide tangible avenues for COVID-19 vaccine production as immunoreactive epitopes are brought to the forefront in these and many other emerging studies.[1,2] The development of a consistent predictable animal model of COVID-19 infection is evidently also a welcome event for preliminary antiviral and vaccine assessments and surely brings us to another level of progression.[3]

The hamster model is not new to coronavirology but has the potential to provide a more stable and predictable model of infection in contrast to the murine models.[4] Pulmonary infection, whether in the context of chemotherapy or vaccine trials, can be easily graded with a histopathological scoring method previously defined in another context and shown to be useful for small experimental animal groups.[5] The latter has been applied to experimental endeavor with severe acute respiratory syndrome coronavirus (SARS-CoV).[6]

Initial enthusiasm to assess whole virus vaccines prepared in a variety of options have historically been followed by focused work on component vaccines. Regardless of the vaccine format, however, one major concern is that vaccination for some viruses and bacteria can be associated with adverse early recall responses after subsequent infections.[7,8] Such a phenomenon was also postulated in early human vaccine trials after parenteral vaccination with *Mycoplasma pneumoniae* and respiratory syncytial virus.[9,10] Hyperaccentuated immune responses after vaccination with SARS-CoV was previously recognized in murine models.[6,11] Although antibody-dependent enhancement as an explanation of such post-vaccine pathology has been postulated by some for several vaccines, a confirmation of the latter and a workable solution have at times been elusive.[12-14] Nevertheless, the critical lesson in vaccine assessment in animal models for COVID-19 is that the review of post-vaccine disease and prevention should therefore include an assessment of both the early and late lung in whichever model so adopted.[6-8,11]

The current yet preliminary understanding of COVID-19 genome and structure offers several candidates for vaccination.[1,2,15] In any such assessments, the examination of systemic humoral or cell-mediated responses to the vaccine are often sought, and thereafter, their association with vaccination outcomes is determined. One lesser sought method for looking at protective antibody at least at the entry-level is to examine the mucosal immune response postinfection that develops in lactating females.[16] Immunoblotting for secretory Immunoglobulin A (IgA) (rather than IgA generally) with breast milk samples from those previously documented to have had COVID-19 infection has the potential to identify immunogens as a surrogate to the finding of protective secretory IgA in the respiratory tract. This would not preclude other research that may focus on systemic protection rather than mucosal or on protection simultaneously from both aspects.

CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

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