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[> J Virol.](#) 2011 Oct;85(20):10582-97. doi: 10.1128/JVI.00671-11. Epub 2011 Jul 20.

Anti-severe acute respiratory syndrome coronavirus spike antibodies trigger infection of human immune cells via a pH- and cysteine protease-independent

FcγR pathway

Martial Jaume ¹, Ming S Yip, Chung Y Cheung, Hiu L Leung, Ping H Li, Francois Kien, Isabelle Dutry, Benoit Callendret, Nicolas Eschnou, Ralf Altmeyer, Beatrice Nal, Marc Daeron, Roberto Bruzzone, J S Malik Peiris

Affiliations

PMID: 21775467 PMCID: [PMC3187504](#) DOI: [10.1128/JVI.00671-11](#)[Free PMC article](#)**Abstract**

Public health measures successfully contained outbreaks of the severe acute respiratory syndrome coronavirus (SARS-CoV) infection. However, the precursor of the SARS-CoV remains in its natural bat reservoir, and reemergence of a human-adapted SARS-like coronavirus remains a plausible public health concern. Vaccination is a major strategy for containing resurgence of SARS in humans, and a number of vaccine candidates have been tested in experimental animal models. We previously reported that antibody elicited by a SARS-CoV vaccine candidate based on recombinant full-length Spike-protein trimers potentiated infection of human B cell lines despite eliciting in vivo a neutralizing and protective immune response in rodents. These observations prompted us to investigate the mechanisms underlying antibody-dependent enhancement (ADE) of SARS-CoV infection in vitro. We demonstrate here that anti-Spike immune serum, while inhibiting viral entry in a permissive cell line, potentiated infection of immune cells by SARS-CoV Spike-pseudotyped lentiviral particles, as well as replication-competent SARS coronavirus. Antibody-mediated infection was dependent on Fcγ receptor II but did not use the endosomal/lysosomal pathway utilized by angiotensin I converting enzyme 2 (ACE2), the accepted receptor for SARS-CoV. This suggests that ADE of SARS-CoV utilizes a novel cell entry mechanism into immune cells. Different SARS vaccine candidates elicit sera that differ in their capacity to induce ADE in immune cells despite their comparable potency to neutralize infection in ACE2-bearing cells. Our results suggest a novel mechanism by which SARS-CoV can enter target cells and illustrate the potential pitfalls associated with immunization against it. These findings should prompt further investigations into SARS pathogenesis.

Figures

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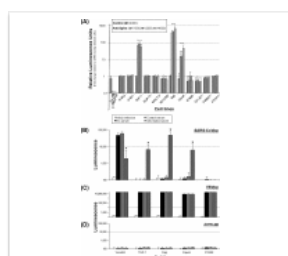


Fig. 1. Susceptibility of hematopoietic cell lines...

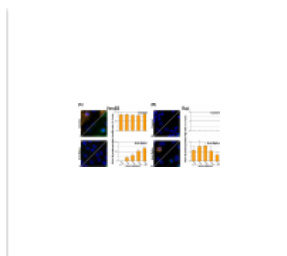


Fig. 2. Anti-Spike antibodies trigger SARS-CoV infection...

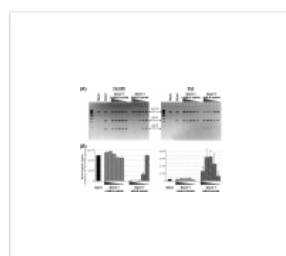


Fig. 3. Endpoint and real-time PCR detection...

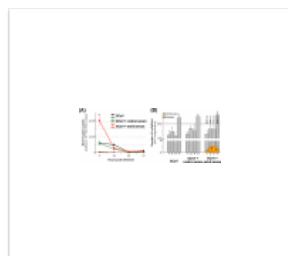


Fig. 4. Abortive replication of SARS-CoV in...

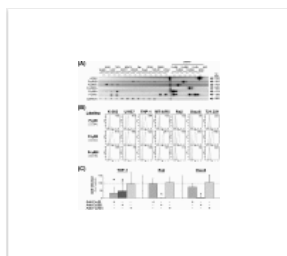


Fig. 5. Blockade of FcγRII abrogates antibody-mediated...

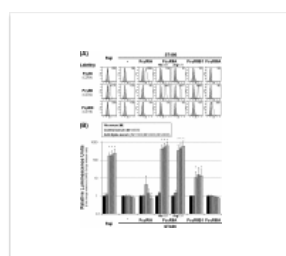


Fig. 6. Human FcγRIIA and B1, but...

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