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Fatal outcome of pandemic H1N1 2009 influenza virus infection is associated with immunopathology and impaired lung repair, not enhanced viral burden in pregnant mice

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Abstract

Pandemic A (H1N1) 2009 influenza virus (pH1N1) infection in pregnant women can be severe. The mechanisms that affect infection outcome in this population are not well understood. To address this, pregnant and non-pregnant Balb/c mice were inoculated with the wild-type pH1N1 strain A/California/04/09. To determine whether innate immune responses are associated with severe infection, we measured the innate cells trafficking into the lung of pregnant versus non pregnant animals. Increased infiltration of pulmonary neutrophils and macrophages strongly correlated with an elevated mortality in pregnant mice. In agreement with this, the product of nitric oxide (nitrite) and several cytokines associated with recruitment and/or function of these cells was increased in the lungs of pregnant animals. Surprisingly, increased mortality in pregnant mice was not associated with higher virus load because equivalent virus titers and immune-histochemical staining were observed in the nasal cavities or lungs of all mice. To determine whether exacerbated inflammatory responses and elevated cellularity resulted in lung injury, epithelial regeneration was measured. The lungs of pregnant mice exhibited reduced epithelial regeneration suggesting an impaired lung repair. Despite these immunologic alterations, pregnant animals demonstrated equivalent percentages of pulmonary influenza-specific $CD8^+$ T lymphocytes although they displayed elevated T-regulator lymphocytes (Tregs) in the lung. Also, pregnant mice mounted equal antibody titers in response to virus or immunization with a monovalent inactivated pH1N1 A/California/07/09 vaccine. Therefore, immunopathology likely caused by elevated cellular recruitment is an implicated mechanism of severe pH1N1 infection in pregnant mice.

Biography

Glendie Marcelin has received her doctoral training in Molecular Virology at Baylor College of Medicine and completed her postdoctoral studies at St. Jude Children's Research Hospital. Her area of focus is viral pathogenesis: understanding the immunological mechanisms of disease severity in animal models. She currently serves as a Virology Collection Scientist with the non-profit biological resource company, American Type Culture Collection (ATCC).