Letter to Editor

**Genetic Background as a Missing Factor in COVID-19 Severity**

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**Abstract**

The clinical outcome of coronavirus disease 2019 (COVID-19) varies considerably from one individual to another and severe illness occurs only in a minority of cases. Evidence supports the role of genetic background alongside host, viral and environmental factors for COVID-19 severity. It seems that host genetic background affects the balance of immune responses during SARS-CoV-2 infection, leading to different clinical courses of the disease.

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The coronavirus disease 2019 (COVID-19) is caused by the novel coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). As a new emerging infectious agent, everyone are susceptible to this viral infection, but the nature and severity of the disease vary significantly among individuals and populations (1). While several predisposing risk factors have been reported to be associated with disease severity and mortality rate, life-threatening infections may also occur in previously healthy people (2). Due to the lack of enough information, it is hard to determine decisive factors in this variation. Since we have to live with SARS-CoV-2 for a long time, it is important to find specific factors associated with complicated disease outcomes. Although our understanding of the virus behavior is still in its infancy, recent reports have provided valuable information regarding the relative importance of multiple components of the host, virus and environment in the COVID-19 clinical phenotype.

Host differences could dictate the clinical response to any viral infection (3). Evidence supports the role of genetic predisposition alongside other factors for COVID-19 severity. Results of a recent modeling study indicated that genetic factors are responsible for 50% of the variance in the 'predicted COVID-19' phenotype (4). Host genetic background affects the balance of immune responses during SARS-CoV-2 infection. If the SARS-CoV-2-induced immune response is dysregulated, the balance between viral control and tissue damage is lost, resulting in pathology and severe illness. Identification of stimulating genes and determining the relationship between host genetics and clinical outcomes of SARS-CoV-2 infection are essential for identifying high-risk individuals. Such studies certainly will provide valuable information and basis for personalized therapy.

REFERENCES


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