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The Role of Community in Immunity Against SARS-CoV-2

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Introduction

Natural immunity weakens through lack of exposure to various types of bacteria and viruses in the environment. The immune system can "remember" and detect the viral and bacterial antigenic markers which then enable the production of antibodies to eradicate them. Research shows that immunity in individuals is suppressed when they feel lonely. For example, researchers have found that people who are more socially connected are 50% less likely to die over a given period (Holt-Lunstad et al., 2010). Therefore, it can be said that social interactions can help to build immunity. Several genes have been found to contribute to the anti-viral immune response and have the ability to be activated through social interactions. Two examples of genes involved in immunity that are triggered by social interactions are interleukin-6 (IL-6) and toll-like receptor 4 (TLR4). The IL-6 gene is an immunoregulatory cytokine that plays a role in managing inflammation (Zilberstein et al., 1986). The TLR4 gene activates inflammatory cytokine production, therefore genes such as IL-6 and TNF-α are products of TLR4 (Medzhitov et al., 1997). Cytokines are cell signaling molecules that guide the movement of cells towards sites of inflammation or infection. Cytokines can be both pro-inflammatory (an abundance in cytokines can stimulate an adverse response) and anti-inflammatory for viral infections and inflammation. This research aims to investigate the roles that the IL6 and TLR4 genes have in helping to build immunity against COVID-19.

Results

Examples of the genes activated by social interactions are interleukin-6 (IL-6) and toll-like receptor 4 (TLR4). A study conducted by Hennessy et al. (2014) explored the relationship between social isolation and sickness. An injection of lipopolysaccharide (LPS) was given to healthy young men and women resulted in elevated levels of IL-6 and TNF-α. The findings revealed that the injection increased feelings of social disconnection in healthy young humans. It was concluded that not only can social isolation induce sickness responses, but induction of sickness can stimulate feelings of isolation (Hennessy et al., 2014). Research has shown how the loss of the IL-6 gene has stimulated the progression of the influenza virus which has resulted in severe lung damage and death in mice (Dienz et al., 2012). The IL-6 gene provides protection against influenza affecting the lungs by promoting the survival of neutrophils. Additionally, IL-6 has contributed to infection susceptibility in the herpes simplex virus-1 (HSV-1) (Magro, 2020). However, it has been discovered that high levels of IL-6 was present in patients with COVID-19 that correlated with immune dysregulation as well as lung damage and pulmonary inflammation (Vatansever & Becer, 2020). From past studies, it has been observed that the TLR4 gene is a key receptor for LPS present on the gram-negative bacteria that cause diseases such as typhoid fever and meningitis (Molteni et al., 2016). Although the TLR4 gene is effective against many viruses, the SARS-CoV-2 spike protein interacts with the TLR4 gene and stimulates high levels of cytokine production that leads to severe dysfunction in the immune system (Brandão et al., 2021).

Discussion and Conclusions

The results do not support the initial hypothesis that genes activated by social interactions, specifically IL-6 and TLR4, help to build immunity against COVID-19. The research conducted by Hennessy et al. (2014) involved cytokines being injected into healthy young men and women and concluded that social isolation can induce sickness responses due to the lack of social interactions to increase immunity. However, the genes associated with cytokines such as the

TLR4 and IL-6 genes have been determined to have an adverse role in COVID-19 immunity. Cytokine genes such as IL6 are known to cause an extensive secretion of cytokines called cytokine storm in patients that are severely ill as a result of COVID-19 (Magro, 2020). During cytokine storm, elevated levels of cytokines cause the body to attack its own cells and tissues rather than fighting off the virus, similar to an autoimmune disease. Nevertheless, the TLR4 and IL-6 genes are instrumental in diseases such as HSV-1, typhoid fever, meningitis, and influenza. Further research can be conducted to determine how the cytokine storm against COVID-19 can be suppressed.

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