Comment on "Highly Pathogenic Influenza Virus Infection of the Thymus Interferes with T Lymphocyte Development"

Nirmal Singh Panesar

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I read with much interest the article by Vogel et al. (1), who state that the cause for lymphopenia in severe infections has been hitherto unclear and, based on their findings, propose that highly pathogenic infectious agents such as influenza virus directly impede the development of lymphocytes, and hence account for lymphopenia. However, the authors have overlooked a well-studied and very important mechanism for lymphopenia, one involving glucocorticoids, which have been used as chemotherapeutic agents against hematological malignancies for decades.

The authors cite several studies involving lymphopenia during viral infections, including the 2003 outbreak of severe acute respiratory syndrome (SARS), for which they have cited one of my papers (2), which specifically asked the question, “What caused lymphopenia in SARS?” The answer, of course, was glucocorticoids (3). This answer was based on their well-established role in the treatment of leukemia (4). Furthermore, in SARS patients with lymphopenia, the cortisol levels were higher (5).

It must be emphasized that any severe infection should activate the hypothalamic–pituitary–adrenal (HPA) axis, with the copious release of glucocorticoids (6). Thus, during the 6 days of infection with various strains of influenza virus, the experimental mice in the authors’ study must have been stressed and their HPA axis activated. Corticosterone released into the blood circulation would have impacted the lymphocytes, causing migration of T lymphocytes out of the peripheral circulation (7) and apoptosis (8), thus resulting in lymphopenia. Glucocorticoids are well known for causing regression of the thymus gland. Unfortunately, the authors did not report on the health status of the animals during the 6 days of infection, with regard to whether animals infected with the more virulent strain of influenza virus were sicker than those infected with the less virulent strain; had they measured corticosterone levels, they may have found the former to have higher levels. The release of corticosterone would have influenced the fate of lymphocytes.

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