Salivary immunoglobulin A antibodies to gp41 in human immunodeficiency virus—seropositive patients: lack of correlation with disease progression


Mucous membranes are the main route of transmission of human immunodeficiency virus (HIV). Interestingly, some viral inhibitory activities have been found in saliva. The purpose of this study was to determine the level of salivary immunoglobulin A (IgA) antibodies to gp41 in HIV+ patients at various disease stages to identify whether gp41 was able to induce vigorous humoral responses. Unstimulated saliva samples were obtained from three groups of subjects (n=37): group A (HIV+), group B (HIV+, CD4+ <200/mm³), and group C (HIV+, CD4+ >200/mm³). IgA antibody levels to purified gp41 were determined by enzyme-linked immunosorbent assay (ELISA). Western blot analyses were performed using HIV+ saliva to confirm IgA reactivity to gp41. ELISA demonstrated that HIV+ subjects had higher IgA antibody to gp41 than HIV- individuals. No significant differences were noted between HIV+, CD4+ <200/mm³ and CD4+ >200/mm³ subjects. High (81.25%) IgA reactivity to gp41 was demonstrated by Western blotting of saliva from all HIV+ individuals. In conclusion, gp41 responses are important in the HIV disease process, as indicated by the high IgA levels and gp41 reactivity in saliva of HIV+ patients.

Acquired immunodeficiency syndrome (AIDS) is an important cause of mortality and morbidity. AIDS is responsible for almost 1.4 million deaths worldwide. It has been estimated that 33.4 million people are affected with HIV/AIDS (27). Human immunodeficiency virus (HIV), the causative agent of AIDS, is transmitted through contact with body fluids (27). Despite detectable virus in saliva and oral tissues of infected patients, the risk of infection with HIV from sexual, occupational, or casual contact with saliva is low, suggesting that some inhibitory activity can be found in saliva (3-5). Whole saliva from seropositive individuals contains antibodies directed to viral proteins (2, 3). Salivary secretory leukocyte protease inhibitor (SLPI) has been implicated as an antiviral factor by protecting peripheral blood mononuclear cells against HIV infection (29). Saliva may destroy infected mononuclear leukocytes by hypotonic disruption and block their attachment to mucosal epithelial cells, hence preventing transmission (5).

Soon after the onset of the HIV infection, the host mounts a variety of cellular and humoral responses against the virus. Humoral responses include production of neutralizing antibodies, polyclonal activation of B lymphocytes, and binding of immune complexes to follicular dendritic cells (17, 27). Con-