

Smoking May Trigger Autoantibodies in Lupus

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Smokers with systemic lupus erythematosus are more likely to be seropositive for double-stranded DNA autoantibodies, compared with those patients who never smoked or quit, according to the findings of a recent study.

Epidemiologic data suggests that smoking is associated with autoimmune disease as well as with autoantibody formation.

The cohort analysis included 410 white patients who were participants in the University of California, San Francisco, Lupus Genetics Project. Patients were included if they provided a complete self-report of their smoking history and had adequate medical records of autoantibody status.

Among these patients, 140 were current smokers, 89 former smokers, and 181 never smokers. Current smokers had a significantly higher risk of double-stranded DNA (dsDNA) seropositivity, compared

with never smokers (OR 4.0). Current smokers also were more likely than former smokers to be dsDNA seropositive (OR 3.0), in multivariate analyses, (*Ann. Rheum. Dis.* 2005 Sept. 8; [Epub doi:10.1136/ard.2005.039438]). The association between current smoking and dsDNA seropositivity remained significant after adjustment for gender, age at lupus diagnosis, amount smoked, age when smoking began, and the duration of smoking cessation for former smokers.

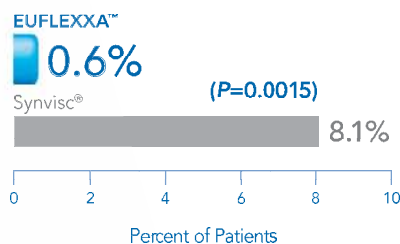
“The association of current smoking with dsDNA seropositivity provides evidence supporting a potential pathogenetic mechanism for the formation of such antibodies,” reported Dr. Michelle M. Freemer of San Francisco General Hospital. In some lupus patients, DNA adducts may serve as the antigen for the formation of dsDNA autoantibodies, or antibodies to DNA adducts could act as anti-idiotypes for the formation of dsDNA autoantibodies. ■

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