SMOKING DELAYS CIA ONSET IN MICE

Cigarette smoking and nicotine exposure have been found to delay the development of collagen-induced arthritis (CIA) in mice, according to a paper now published in *Arthritis Research & Therapy*. “Our data [show] no [evidence] of [a] detrimental influence of cigarette smoke on the development and cause of arthritis,” says the lead researcher Maria Bokarewa from the University of Göteborg, Sweden.

Previous studies have implicated cigarette smoking as a risk factor in the development of rheumatoid arthritis; however, other findings seem to contradict this, such as smoking conferring a beneficial effect in some autoimmune disorders, and nicotine displaying an anti-inflammatory effect in ulcerative colitis. Thus, the researchers evaluated the impact of cigarette smoke and nicotine on arthritis development in mice.

Adult DBA/1 mice ($n=25$) were exposed to cigarette smoke (4 non-filtered cigarettes per day) for 16 weeks before immunization with collagen type II, and for a further 6 weeks following immunization. Bokarewa explains that “this duration of smoking corresponded to a regimen of 1.5 packs per day for 15 years in human settings.” A further 10 DBA/1 mice were given nicotine in drinking water from the day of immunization through to the end of the experiment, and 35 immunized mice served as controls.

The investigators observed less-severe inflammation in mice exposed to cigarette smoke compared with control animals. “Smoke exposure changed [the] immune response in mice [and alleviated] the development of [autoantibodies and collagen-specific arthritis],” Bokarewa comments. Owing to similar observations in the mice supplemented with nicotine, Bokarewa adds that “the results of our study suggest nicotine to be one of the components of cigarette smoke [that regulates the] development of antigen-specific inflammation, thereby delaying the onset of arthritis.”

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