ENVIRONMENTAL TOBACCO SMOKE AND PARKINSON'S DISEASE IN THE CALIFORNIA TEACHERS STUDY

Nicole M. Gatto, Department of Epidemiology, School of Public Health, UCLA, USA
Dennis Deapen, Department of Preventive Medicine, USC Keck School of Medicine, USA
Beate Ritz, Departments of Epidemiology, Environmental Health Sciences and Neurology, UCLA, USA

Background and Aims: Epidemiologic studies have consistently demonstrated inverse associations between cigarette smoking and Parkinson's disease (PD) risk, leading to speculations about potential neuroprotective effects of smoking. However, it has been hypothesized that behavioral and genetic factors may influence propensity to initiate and maintain smoking in PD. **Methods:** We examined cigarette smoking behavior and environmental tobacco smoke (ETS) exposure using questionnaire data obtained at baseline from 403 self-reported female PD cases at a ten-year follow-up and 4,045 randomly selected age- and race-matched female controls in the California Teachers Study.

Results: The study population was an average 60.1 ± 12 years at baseline, predominantly white (91.5%) and largely non-smoking, with 63.3% reporting having never smoked; 46.4% had been exposed to ETS in their lifetime. Women who smoked did so for an average of 22.1 years, with cases smoking two years less than controls $(20.4 \pm 13.7 \text{ vs. } 22.2 \pm 14.6 \text{ years, respectively)}$. Women who had ever smoked were less likely to report PD at follow-up (OR = 0.87, 95% CI = 0.70, 1.08), an effect which was driven by former smokers (OR = 0.86, 95% CI = 0.69, 1.08). Any ETS exposure was not associated with PD (OR = 1.02, 95% CI = 0.76, 1.37) compared to no personal or environmental smoke exposure. Adulthood ETS exposure was not inversely associated with PD (OR = 1.15, 95% CI = 0.83, 1.60).

Conclusions: These results suggest that cigarette smoke and its components at least as inhaled as ETS may not be protective against PD, per se, although one cannot rule out a survival disadvantage for smokers with PD. Other explanations for the documented inverse associations between smoking and PD should continue to be explored, such as possible dysfunction in reward pathways resulting from disruptions in the dopaminergic system early in the course of PD.