Smoking and the Risk of Incident Type 2 Diabetes Mellitus

Year: 2004

Abstract Number: 283-OR

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Results:

Previous studies suggest smoking as a risk factor for incident diabetes, but most lacked the physiologic data required to explore the potential mechanisms. We hypothesize that two emerging diabetes risk factors -- inflammation and reduced lung function -- might explain the association. To test this hypothesis, we analyzed longitudinal data from the Atherosclerosis Risk in Communities (ARIC) Study, a population-based study of adults aged 45-64 years. Smoking history was obtained at baseline in 8,747 adults with complete data (laboratory, anthropometric, and spirometry) but without prevalent diabetes or heart disease or chronic lung disease. At baseline, after adjustment for age, sex, race, body mass index, waist-to-hip ratio, physical activity, and ARIC center, more pack-years of smoking were associated with greater white cell count (beta = 0.3 (x $10\omega9o/L$) per 10 pack-years) and lower forced expiratory volume (FEVµ1v) (beta = -73 ml per 10 pack-years). During 9 years of follow-up. 1,017developed incident diabetes as defined by physician diagnosis, use of antidiabetic medications, fasting glucose ≥ 126 mg/dL, or non-fasting glucose≥ 200 mg/dL. In prospective analyses using Cox proportional hazard models adjusted for age, sex, race, body mass index, waist-to-hip ratio, physical activity, and ARIC center, there was a graded association between cumulative exposure to smoking and diabetes risk: compared to never smokers, the relative hazards (95% confidence interval) rose from 1.1 (0.9 \Box 1.3) in those with ≤ 13 pack-years to 1.3 (1.1 \Box 1.6) in those with 14 to 30 pack-years and 1.5 (1.2 \square 1.7) in those with > 30 pack-years (p for trend < 0.001). The associations were significantly attenuated after additional adjustment for white cell count and FEVµ1v: compared to never smokers, the relative hazard dropped to 1.2 $(0.99 \Box 1.4)$ in those with >30 pack-years (p for trend = 0.03). As in prior studies, smoking predicts incident diabetes in the ARIC cohort. Whether this effect may be partially medicated by smokings relationship with inflammation and impaired lung function deserves further attention. Category:

Epidemiology