

Prenatal tobacco use and risk of stillbirth: A case-control and bidirectional case-crossover study

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We sought to estimate the association between prenatal smoking and stillbirth in a longitudinal cohort using two study designs: a case-control study and a bidirectional case-crossover study. The analysis was conducted using the Missouri maternally linked cohort dataset from 1978 through 1997. In the case-control study, each mother contributed only one birth to the analysis. For the bidirectional crossover design, analysis was restricted to women who gave birth to at least one stillbirth, and the controls comprised all live births before and after the stillbirth. The independent association between prenatal smoking and stillbirth was computed using nonconditional (case-control design) and conditional (bidirectional case-crossover design) logistic regression. Prenatal smoking decreased from 29.7% in 1978 to 21.2% by 1997 ($p < .001$). The absolute risk of stillbirth was greater among smokers (7.7/1000) than nonsmokers (5.3/1000), $p < .001$. In the case-control design, the risk of stillbirth was 34% greater among smokers than nonsmokers ($OR = 1.34$, 95% CI 1.26-1.43). For each 10-unit increase in the number of cigarettes consumed per day prenatally, the likelihood of stillbirth rose by about 14% ($p < .001$). In the bidirectional case-crossover design, the association between stillbirth and smoking during pregnancy was confirmed, although the magnitude of the relationship was smaller ($OR = 1.20$, 95% CI 1.03-1.39). In conclusion, we found prenatal smoking to be a risk factor for stillbirth even after minimizing the influence of known and unknown sources of confounding as well as changes in temporal trend in prenatal smoking.

Introduction

Prenatal smoking is an important cause of adverse birth outcomes. Fetal exposure to tobacco smoke has been consistently found to be linked to adverse pregnancy outcomes including low birth weight (Berkowitz & Papiernik, 1993; Cnattingius, Granath, Petersson, & Harlow, 1999; Hellerstedt, Himes, Story, Alton, & Edwards, 1997; Meis et al., 1997; Pollack, Lantz, & Frohna, 2000; Salihu, Aliyu, Bosny, & Alexander, 2003; Wang, Tager, Van

Vunakis, Speizer, & Hanrahan, 1997), preterm delivery (Cnattingius et al., 1999; Hadley, Main, & Gabbe, 1990; Kyrklund-Blomberg & Cnattingius, 1998; M. Meyer & Tonascia, 1976), premature rupture of the membranes (Hadley et al., 1990; Kyrklund-Blomberg & Cnattingius, 1998), placenta previa (M. Meyer & Tonascia, 1976; Naeye, 1980; Williams et al., 1991), abruptio placenta (Ananth, Smulian, & Vintzileos, 1999; Cnattingius, Mills, Yuen, Eriksson, & Salonen, 1997; M. Meyer & Tonascia, 1976; Naeye, 1980), perinatal mortality (Ananth et al., 1999; Centers for Disease Control and Prevention, 2002; National Center for Health Statistics, 1995-1997), and sudden infant death syndrome (Blair et al., 1996; Hoo, Henschen, Dezateux, Costeloe, & Stocks, 1998; Tuthill, Stewart, Coles, Andrews, & Cartledge, 1999). Recent studies have confirmed that maternal smoking during pregnancy also is associated with infant mortality (Salihu et al., 2003). The causal relationship between prenatal smoking and adverse birth outcomes is frequently confounded by other

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