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The intergenerational transmission of smoking in adulthood: A 25-year study of maternal and offspring maladaptive attributes

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HIGHLIGHTS

- Structural equation modeling tested pathways to adult smoking, across 25 years.
- Maternal and child factors were assessed from (offspring) adolescence to adulthood.
- Continuity of psychological maladjustment predicted offspring smoking in adulthood.
- Maternal smoking and less offspring education also predicted adult offspring smoking.
- Mother–adolescent attachment had an enduring effect on predictors of smoking.

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ABSTRACT

While smoking is a major cause of mortality and morbidity, and maternal smoking is a risk factor for smoking among their offspring, the mechanisms involved in the intergenerational transmission of smoking are not well understood. This study examines the pathways from maternal and adolescent child factors, and the parent–child relationship, to smoking among the adult offspring, approximately 25 years later. Data for the present analysis were based on time waves 2 (T2; 1983) and 7 (T7; 2007–2009) of an on-going study of a community sample of mothers and their children. Offspring and mother $\overline{X}$ ages were 14.1 and 40.0 years, respectively, at T2, and 36.6 and 65.0 years, respectively, at T7. At T2, trained interviewers administered individual structured interviews. Psychosocial questionnaires were self-administered at T7. Structural equation modeling (SEM) was used to analyze the interrelationships among maternal and offspring attributes (T2 and T7). SEM results indicated a satisfactory model fit (RMSEA = 0.052; CFI = 0.91; SRMR = 0.057), and confirmed hypothesized pathways. One pathway linked maternal maladaptive attributes (T2) to the mother–adolescent child attachment relationship (T2), which was associated with the offspring's maladaptive attributes over time (T2 to T7), which then predicted the adult offspring's smoking (T7). Other pathways highlighted the stability of maternal smoking, the continuity of maladaptive attributes, and less offspring educational attainment as predictors of offspring smoking at T7. Findings suggest the importance of early interventions to treat maternal smoking, maternal and offspring maladaptive attributes, and the mother–child relationship in order to reduce risk factors for the intergenerational transmission of smoking behavior. Interventions which enhance educational success should also prove effective in reducing smoking.

1. Introduction

Cigarette smoking accounted for over 6 million deaths globally in 2010, and is the second leading cause of disability worldwide (Lim et al., 2012). In the United States, approximately one in five deaths is attributable to cigarette smoking (CDC, 2008). Although maternal smoking is a risk factor for smoking among their offspring, the mechanisms involved in the intergenerational transmission of smoking are not well understood. Numerous studies have identified associations between maternal attributes (e.g., smoking), adolescent psychological problems, aspects of the mother–child relationship, and adolescent smoking (Chassin, Presson, Todd, Rose, & Sherman, 1998; Ennett et al., 2010; Lawlor et al., 2005; SAMHSA, 2010a). There is a dearth of research, however, on the longitudinal impact of these earlier influences, or the effects of contemporaneous factors, on smoking among adults. A better understanding of the impact of these maternal factors on smoking among their offspring may contribute to the development of prevention and intervention programs which reduce the intergenerational transmission of risk factors

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for smoking. The present study, therefore, uses data spanning approximately 25 years to examine the pathways from earlier maternal attributes and the mother–adolescent relationship to the offspring's smoking in adulthood.

1.1. Maternal and offspring maladaptive attributes and smoking

Prior research has found that maternal smoking during pregnancy is a risk factor for numerous adverse outcomes among their offspring, such as externalizing behaviors and disorders (Brook, Zhang, Rosenberg, & Brook, 2006; Fitzpatrick, Barnett, & Pagani, in press), diminished academic achievement (Agrawal et al., 2010), and substance use, including smoking (Agrawal et al., 2010; D'Onofrio et al., 2012). The association of maternal gestational smoking and problems among their offspring may be both causal (e.g., specific to the in utero effects of nicotine on neurodevelopment) as well as derived from environmental factors shared by mother and child (e.g., Agrawal et al., 2010; D'Onofrio et al., 2008; Lieb, Schreier, Pfister, & Wittchen, 2003; O'Callaghan et al., 2006).

A substantial literature has also found that maternal smoking during the offspring's childhood or adolescence is related to the offspring's smoking during adolescence or young adulthood (e.g., Gilman et al., 2009; Kelly et al., 2011; Leonardi-Bee, Jere, & Britton, 2011; Osler, Clausen, Ibsen, & Jensen, 1995; SAMHSA, 2010a). In one of few investigations to examine the link between earlier maternal smoking and the offspring's smoking in adulthood, Vink, Willemsen, and Boomsma (2003) showed that maternal smoking during the offspring's adolescence predicted offspring smoking during adolescence, young adulthood, and adulthood. To our knowledge, there are no studies on the relationship of concurrent smoking among older women and their adult offspring. Some factors which have been shown to operate between maternal smoking and smoking among the offspring include role modeling of maternal smoking behavior (Weden & Miles, 2012), fewer anti-smoking rules (Sargent & Dalton, 2001), and common genetic vulnerabilities shared by mother and child (Weden & Miles, 2012).

Although a review of the genetics of smoking is beyond the scope of this paper, both genes and environment have been shown to contribute to smoking behavior, with genetic influences exerting greater effects on later stages of smoking and nicotine dependence (Shenassa et al., 2003; White, Hopper, Wearing, & Hill, 2003). Human and animal studies have identified variants of cholinergic nicotinic receptor subunit gene clusters (e.g., CHRNA5-CHRNA3-CHRNB4) and nicotine-metabolizing enzymes (e.g., CYP2A6 and CYP2B6) which have been implicated in smoking (Saccone et al., 2009). These variants have also been linked to heavy smoking and nicotine dependence (Berrettini et al., 2008; Saccone et al., 2009; Thorgeirsson et al., 2008), the age of smoking cessation (Chen et al., 2012), severity of withdrawal symptoms (Baker et al., 2009), and smoking-related morbidity, i.e., lung cancer and peripheral arterial disease (Saccone et al., 2009; Thorgeirsson et al., 2008).

In addition, mothers who smoke tend to have elevated rates of psychological symptoms and psychiatric disorders (Degenhardt & Hall, 2001), which have been found to be linked with smoking among their offspring (SAMHSA, 2010a). However, as noted, the precise mechanisms involved in the intergenerational transmission of smoking behavior are not entirely understood.

Smoking and psychological problems (e.g., depression) are highly related among both adolescent and adult smokers (Hu, Davies, & Kandel, 2006; Klungsøyr, Nygård, Sørensen, & Sandanger, 2006), although it is unclear whether this relationship is causal, reciprocal, or stems from a shared underlying etiology (e.g., Boden, Fergusson, & Horwood, 2010; Orlando, Ellickson, & Jinnett, 2001). Furthermore, the results of some studies suggest that psychological maladjustment during adolescence may predict smoking and nicotine dependence during later developmental stages (McKenzie, Olsson, Jorm, Romaniuk, & Patton, 2010).

There is considerable evidence (especially in the literature on adolescence) of the intergenerational transmission of psychological difficulties, such as depression, anxiety, and low self-esteem (Campbell, Morgan-Lopez, Cox, & McLoyd, 2009; Papp, Cummings, & Goeke-Morey, 2005). The concordance of psychological problems among mothers and their offspring may stem from maternal role modeling of maladaptive affect and behaviors (Brook, Brook, Gordon, Whiteman, & Cohen, 1990; Campbell et al., 2009), shared contextual influences (such as exposure to stressful neighborhoods; Patterson, Eberly, Ding, & Hargreaves, 2004), a genetic vulnerability (Kendler, Myers, Maes, & Keyes, 2011), and/or the adverse effects of maladaptive maternal attributes on the mutual attachment relationship with the child (e.g., Hammen, Shih, & Brennan, 2004; National Research Council and Institute of Medicine [IOM], 2009).

1.2. Family interactional theory and the mother–child mutual attachment relationship

The theoretical framework for the present study is based on Family Interactional Theory (FIT; Brook et al., 1990). FIT posits that there is a developmental progression from maternal to child maladaptive psychological attributes, which may be mediated by the effect of maternal attributes on the mother–child relationship (Papp et al., 2005). According to FIT, maternal substance use and/or psychopathology may undermine the development of a close, non-conflictual mutual attachment relationship with the child. Children with a weaker and more distant maternal bond, in turn, are less likely to identify with and model parental pro-social behaviors and, therefore, may be at risk for maladaptive personal attributes, behaviors, and functioning, e.g., Brook et al., 1990; Papp et al., 2005. Furthermore, the FIT paradigm may be extended to later developmental stages. That is, the mother–adolescent child attachment relationship may have enduring effects on the offspring. Flouri (2005), for instance, reported that maternal involvement with her adolescent child predicted less distress among the offspring in adulthood. Bell and Bell (2005) also demonstrated a pathway from parental resources (e.g., ego development) to positive family dynamics (e.g., connectedness) during the offspring's adolescence to the offspring's well-being in the 30s and 40s.

Here, we extend FIT to an adult sample to test the longitudinal impact of a weak and conflictual mother–adolescent attachment relationship on the adult offspring's maladaptive attributes, educational attainment, and smoking in the late 30s.

1.3. Continuity of smoking, stability of maladaptive attributes, and the adult offspring's educational attainment

With a few important exceptions, there is limited research on intra-individual continuity of smoking across adulthood and into late midlife (Yong, Borland, Thrasher, & Thompson, 2012). However, Frosch, Dierker, Rose, and Waldinger (2009) identified several developmental patterns of smoking throughout adulthood, which demonstrated both continuity and discontinuity (i.e., smoking cessation at different stages) in smoking behavior.

With respect to maladaptive attributes, several investigations (e.g., Colman, Wadsworth, Croudace, & Jones, 2007; Roza, Hofstra, van der Ende, & Verhulst, 2003) have reported considerable stability of psychopathology over time, although most of this research has focused on the period from adolescence to young adulthood. For example, Dekker et al. (2007) demonstrated that trajectories of depressive symptoms (beginning in childhood or adolescence) predicted more symptoms of depression and other mental health problems in young adulthood. Clark, Rodgers, Caldwell, Power, and Stansfeld (2007) found that internalizing and externalizing disorders in