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Substance abuse in parents and subsequent risk of offspring psychiatric morbidity in late adolescence and early adulthood: A longitudinal analysis of siblings and their parents

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ABSTRACT

The effects of substance abuse on other family members are not fully established. We estimate the contribution of parental substance abuse on offspring psychiatric morbidity in late adolescence and early adulthood, with emphasis on the timing and persistency of exposure. We used a nationally representative 20% sample of Finnish families with children born in 1986–1996 (n = 136,604) followed up in 1986–2011. We identified parental substance abuse and offspring psychiatric morbidity from hospital discharge records, death records and medication registers. The effects of parental substance abuse at ages 0–4, 5–9 and 10–14 on psychiatric morbidity after age 15 were estimated using population averaged and sibling fixed effects models; the latter controlling for unobserved factors shared by siblings. Parental substance abuse at ages 0–14 was associated with almost 2-fold increase in offspring psychiatric morbidity (HR = 1.86, 95% CI 1.78–1.95). Adjustment for childhood parental education, income, social class and family type reduced these effects by about 50%, with some further attenuation after adjustment for time-varying offspring characteristics. In the sibling fixed effects models those exposed at 0–4 or 5–9 years had 20% (HR = 1.20, 95% CI 0.90–1.60) and 33% (HR = 1.33, 95% CI 1.01–1.74) excess morbidity respectively. Also in sibling models those with early exposure at ages 0–4 combined with repeated exposure in later childhood had about 80–90% higher psychiatric morbidity as compared to never exposed siblings (e.g. for those exposed throughout childhood HR = 1.81, 95% CI 1.01–3.25). Childhood exposure to parental substance abuse is strongly associated with subsequent psychiatric morbidity. Although these effects are to a large extent due to other characteristics shared within the parental home, repeated exposure to parental substance abuse is independently associated with later psychiatric morbidity.

1. Introduction

It is well established by prior studies that excessive alcohol consumption and other substance abuse are associated with social disadvantage, poor health and higher mortality for the user (Rehm et al., 2010). However, these studies do not adequately acknowledge that substance abuse may also pose harm to others – sometimes referred to as collateral damage or spill-over effects of substance abuse (Rogers et al., 2016; Gell et al., 2015). This study assesses the impact of substance abuse on others by studying its effects on the psychiatric morbidity of a particularly vulnerable group, the children of substance users (Solis et al., 2012).

Heavy maternal drinking and other substance abuse are known to be associated with poorer birth outcomes and early life health conditions including preterm birth, low birth weight and foetal alcohol syndrome (Henderson et al., 2007; Rehm et al., 2010; Behnke et al., 2013; Huizink, 2015). Prenatal exposure to alcohol and other drugs has also been shown to associate with childhood behavioural problems and cognitive development (Behnke et al., 2013), and children of substance-abusing mothers are more likely to be hospitalized for injuries and infectious diseases (Raitasalo et al., 2015). A less healthy start in life may entail consequences for offspring also in the long run. Prior evidence indicates that parental substance use disorders associate with offspring psychopathology in adolescence and early adulthood, with a

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particularly strong intergenerational link in alcohol and other substance use disorders (Marmorstein et al., 2009; Hill et al., 2011; Sørensen et al., 2011; Mellentin et al., 2016). Studies using linked population registration data from Denmark and Sweden show that parental substance use has an impact on a broad range of mental health outcomes in adolescence and early adulthood including psychiatric disorders, self-harm, and substance misuse (Christoffersen and Soothill, 2003; Björkenstam et al., 2016). However, several commentators have stressed the need for more studies using population-based family data, as most of the existing evidence is based on student, clinical, and high-risk community samples of lesser generalizability (Park and Schepp, 2015; Mellentin et al., 2016; Christoffersen and Soothill, 2003). It has also been pointed out that many of the previous studies assessing health consequences of parental substance abuse still focus either on short-term effects or more general measures of lifetime exposures, and much less is known about timing or accumulation of exposure to parental substance abuse at different stages of childhood (Björkenstam et al., 2016; Park and Schepp, 2015; Mellentin et al., 2016).

Life-course theory posits that the effect of childhood experiences on later health may depend on the timing of events (Ben-Shlomo and Kuh, 2002). During sensitive periods adverse exposures have stronger effects on later disease risk than exposures at other times. Sensitive period 'denotes the time in which the developing child is particularly responsive to certain forms of experience or particularly hindered by their absence' (Sylva, 1997). In addition to sensitive periods, life course models also stress the importance of duration and accumulation of exposures for later health outcomes. However, few studies have assessed the timing and persistency of exposure to parental substance abuse on offspring health, although cross-sectional studies have reported older children of alcoholic parents to be more resilient (Park and Schepp, 2015). Two longitudinal US studies based on community samples of children of alcoholics and their controls found a strong effect of having ever experienced parental alcohol abuse, as well as time-varying effects of exposures to parental alcohol abuse on offspring externalizing behaviour (Hussong et al., 2010), and maternal alcohol abuse on internalizing behaviour (Hussong et al., 2008). In a Swedish register study, parental substance abuse in childhood was consistently associated with psychiatric disorder in late adolescence and early adulthood with no evidence of particularly sensitive periods, but excess risk among those with repeated exposure (Björkenstam et al., 2016). Similar results were found in another Swedish study on young adult alcohol use disorders (Edwards et al., 2017).

Disentangling causal pathways has also remained difficult. Families with parental substance abuse are typically also characterised by poor parental mental health and social disadvantage (Christoffersen and Soothill, 2003; Sørensen et al., 2011). Some of the children of substance abusing parents are thus likely to face additional concurrent risk factors for poorer health outcomes. In addition to parental health problems besides substance use, these include adverse socioeconomic characteristics, strain on family relationships, unstable home environment, disrupted parenting and child maltreatment (Harter, 2000; Staton-Tindall et al., 2013). Although many studies have been able to control for some of these factors, such as parental socioeconomic status, the cross-sectional and observational nature of most studies hampers the identification of confounding factors and mediating mechanisms. Many studies are also based on retrospective self-reports of childhood adversity. Significant residual confounding may thus bias the results.

This study adds to the literature in three ways. First, we focus on the timing of exposure to parental substance abuse in three different stages of childhood (ages 0–4, 5–9 and 10–14 years) in order to establish sensitive periods of exposure. Second, we estimate the effects of repeated exposure to parental substance abuse. Third, to obtain a more accurate understanding of the mechanisms and causal effects of parental substance abuse on offspring mental health we estimate both population averaged models controlling for observed parental characteristics and time-varying offspring characteristics, as well as sibling fixed effects models that control for all unobserved characteristics shared by siblings. Finally, the analyses are based on high quality register data on a large population-representative sample of Finnish families with children followed for exposures to parental substance abuse from birth to age 14, and for psychiatric morbidity from age 15 over the years 2001–2011. These administrative data are unique as they do not suffer from reporting bias, selective loss to follow-up or small sample size.

2. Data and methods

2.1. Data and variables

This study was based on annually updated individual-level register data maintained by Statistics Finland. We used data that consist of a 20% random sample of Finnish households with at least one child aged 0–14 at the end of 2000, a 20% sample of 0–14-year-olds not living in private households at the end of 2000, and non-coresident biological parents of all 0–14-year-olds in the two samples. The data were linked with individual-level sociodemographic information for both offspring and their parents for years 1987–2011, hospital discharge records (maintained by the National Institute for Health and Welfare) for 1986–2011, and the national prescription register on all purchases of prescription medication (maintained by the Social Insurance Institution of Finland) for 2001–2011.

In the current study, we included individuals born in years 1986–1996 (n = 136,604) and followed them from the beginning of the year of their 15th birthday until first incidence of psychiatric morbidity, the end of the year of their 25th birthday, emigration, death, or the end of year 2011, whichever came first. Offspring psychiatric morbidity was defined on the basis of indicators available in administrative register data: psychotropic medication purchases (including the Anatomical Therapeutic Chemical (ATC) codes N05 and N06 but not N06D) or admission to inpatient hospital care with a psychiatric diagnosis (International Classification of Diseases (ICD-10) codes F10–69, F80–98) (for more detail see Supplementary Table 1). Defined in this way about 20% of all offspring psychiatric cases were based on hospital data.

Exposure to parental substance abuse in each calendar year at ages 0–14 was assessed using information of hospital diagnoses and cause of death of the biological parents in years 1986–2010. We used the tenth revision of ICD for years 1996–2010 to identify mental and behavioural disorders due to alcohol (F10) and substance use (F11–16, F18–19), alcohol-related diseases (E24.4, E52, G31.2, G40.51, G62.1, G72.1, I42.6, K29.2, K70, K85.2, K86, Y90–91), toxic effects and poisoning by alcohol (T51, X45) and other substances (T40, T42.3–42.4, T42.6–42.7, T43.0–43.5, T43.8–43.9, T50.7, T36, X44) and other contact with health services due to alcohol (R78.0, Z50.2, Z71.4, Z72.0) or substance use (R78.1–78.5, Z50.3, Z71.5, Z72.2). Corresponding ICD-8 codes were used for 1986 and ICD-9 codes for 1987–1995 (for more detail see Supplementary Table 1). Substance abuse was identified if any of the codes were reported as primary or additional hospital diagnosis, or as the underlying or contributory cause of death. Deaths accounted for 6% of all annual substance abuse cases. 80% of all cases were related to alcohol, of which most common were mental and behavioural disorders due to use of alcohol. We classified exposure to parental substance abuse according to the age of the child at exposure and frequency of exposures.

2.2. Covariates

We used parental education, household income, occupational social class and family type, measured at ages 0–14, to adjust for the socioeconomic characteristics of the childhood family. Parental education at ages 0–14 was based on the highest achieved educational level of either parent in the household, and categorized as tertiary, secondary and