1. Introduction

Recent figures suggest that 207,000 children between the ages of 11 and 15 start smoking each year in the United Kingdom, with around 80% of adult smokers taking up smoking before the age of 20 (ASH, 2015). Therefore, preventing uptake of smoking in adolescence is of paramount importance. Smoking is associated with lower body weight (Chiolero et al., 2008) and there is good evidence that this link is causal (Freathy et al., 2011). However, the causal effect of body weight on smoking is largely unknown. There is some evidence that high body mass index (BMI) is a possible risk factor for smoking initiation because people may start smoking in order to control or lose weight. In one study, adolescents who reported trying to lose weight had increased rates of smoking initiation (Strauss and Mir, 2001). In another study, adolescents who reported trying to lose weight had increased rates of smoking initiation (Strauss and Mir, 2001). Therefore, preventing uptake of smoking in adolescence is of paramount importance.

Body dissatisfaction could be a mediator or confounder of the relationship between BMI and smoking; high BMI may cause body dissatisfaction which then leads to increased smoking behaviour (mediator) or, alternatively, body dissatisfaction could independently lead to both changes in BMI and smoking behaviour (confounder). In general, observational studies have found body dissatisfaction or weight concerns are risk factors for smoking in females but not males (Cawley et al., 2004; French et al., 1994; Tomeo et al., 1999; Winter et al., 2002). A review of studies investigating body weight concerns and tobacco use concluded that the evidence for a positive association depends largely on the dimension of the weight concern variable considered (e.g., dietary behaviour, disordered eating), and that the positive association between body dissatisfaction and smoking was more consistent amongst female adolescents than males (Potter et al., 2002). A review of studies investigating links between body weight and smoking are cross-sectional and therefore might be subject to reverse causation.
Unravelling the complex relationships between BMI, body dissatisfaction and adolescent smoking behaviour is of clinical importance as it may allow the identification of adolescents at greater risk of tobacco smoking and allow interventions to be targeted appropriately.

Using data from a large longitudinal study, the Avon Longitudinal Study of Parents and Children (ALSPAC), we examined the relationship between BMI, body dissatisfaction and the smoking habits of adolescents. We used latent classes of smoking initiation described previously (Heron et al., 2011), but extended to 18 years. First, we examined the relationships between BMI and body dissatisfaction at age 10.5 years with subsequent adolescent smoking initiation between the ages of 13 and 18. Second, we used a genetic risk score as a proxy for measured BMI in a Mendelian randomisation (MR) approach. We were primarily interested in evaluating the effect of BMI and body dissatisfaction on smoking while also interested in the nature of the relationship between BMI and body dissatisfaction (Fig. 1).

MR is an instrumental variable approach that uses genetic variant(s) associated with an exposure to examine the relationship between that exposure and an outcome. In principle it should be less susceptible to problems of confounding and reverse causation that can affect observational studies (Davey-Smith and Ebrahim, 2003) and is often used as a method of causal inference. MR relies on three assumptions: firstly that the instrument is robustly associated with the exposure, secondly that the only association between the instrument and the outcome is through the exposure and thirdly that there are no common determinants of the instrument and the outcome (e.g., population stratification) (Solovieff et al., 2013). Evidence for a causal role of smoking in reducing body weight has been found using MR methods (Åsvold et al., 2013). Evidence for a causal role of smoking in reducing body weight has been found using MR methods (Åsvold et al., 2013; Freathy et al., 2011). Higher genetically determined BMI (using a polygenic risk score based on 32 genetic variants) was found to be associated with smoking more cigarettes per day and increased risk of smoking initiation (Thorgerirsson et al., 2013). These results were interpreted by the authors as evidence for a shared genetic aetiology between smoking and BMI, but the results could also be interpreted as evidence for weight influencing smoking behaviour.

2. Material and methods

2.1. Study participants

We used data on children from the Avon Longitudinal Study of Parents and Children (ALSPAC), a longitudinal study that recruited pregnant women living in the former county of Avon (UK) with expected delivery dates between 1 April 1991 and 31 December 1992. Avon is a county in the southwest of the United Kingdom (UK), 120 miles west of London with a population of 1 million. Children living in Avon were surveyed and found to be broadly similar to the UK population in terms of socio-economic and ethnicity related demographics (Golding et al., 2001).

The initial number of enrolled pregnancies was 14,541, which resulted in 14,062 live births and 13,988 children alive at the age of 1. When the oldest children were approximately 7 years of age, the initial sample was boosted with eligible cases who had failed to join the study originally. Full details of the enrolment have been documented elsewhere (Boyd et al., 2013; Golding et al., 2001). Data have been gathered from the mother and her partner (during pregnancy and post birth) and the children from self-report questionnaires and clinical sessions. Ethics approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committee. The study website contains details of all available data through a searchable data dictionary (http://www.bristol.ac.uk/alspac/researchers/dataaccess/datadictionary/).

2.2. Measures

2.2.1. Smoking behaviour

The measures of smoking behaviour used in these analyses were collected at six time points. At 14, 16 and 18 years, data were collected using self-completed postal questionnaires, while data at 13, 15 and 17 years were collected during clinic visits. For the clinics at 15 and 17 years the smoking questions were answered using a computer-based questionnaire. The number of participants with smoking frequency data
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