



Risk factors associated with early smoking onset in two large birth cohorts

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HIGHLIGHTS

- Childhood smoking has been linked to later use and long-term health problems.
- We examined changes in childhood smoking and related risk factors in two cohorts.
- The risk of childhood smoking was 8 times higher among those born in 1970 vs 2001.
- Cohort decline mediated by changes in maternal education and parental/peer smoking.
- Early life disadvantages are more strongly linked to child smoking in 2001 cohort.

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ABSTRACT

We use prospective data from the ongoing British Cohort Study (BCS) and Millennium Cohort Study (MCS) to: 1) document changes in the prevalence of childhood smoking onset; 2) assess whether broad historic shifts in key risk factors, such as maternal education, parental smoking, and peer childhood smoking, explain observed cohort changes in childhood smoking; and 3) evaluate whether inequalities in onset have narrowed or widened during this period. The children in these two studies were born 31 years apart (i.e., BCS in 1970; MCS in 2001), and were followed from infancy through early adolescence ($n = 23,506$ children). Our outcome variable is child self-reports of smoking (ages 10, 11). Early life risk factors were assessed via parent reports in infancy and age 5. Findings reveal that the odds of childhood smoking were over 12 times greater among children born in 1970 versus 2001. The decline in childhood smoking by cohort was partly explained by increases in maternal education, decreases in mothers' and fathers' smoking, and declines in the number of children whose friends smoked. Results also show that childhood smoking is now more linked to early life disadvantages, as MCS children were especially likely to smoke if their mother had low education or used cigarettes, or if the child had a friend who smoked. Although the prevalence of child and adult smoking has dropped dramatically in the past three decades, policy efforts should focus on the increased social inequality resulting from the concentration of early life cigarette use among disadvantaged children.

1. Introduction

Of smokers, approximately 40% start by early to middle adolescence and early initiation is associated with heavier, chronic use and dependence in later adolescence and adulthood, as well as increased morbidity and mortality (Dunstan, 2012; GBD Tobacco Collaborators, 2015; U.S. Department of Health and Human Services [DHHS], 2012). The prevention of childhood experimentation with smoking is of urgent public health importance because so many individuals lose autonomy and develop dependence after smoking for very brief periods of time (Difranza, Savageau, Fletcher, et al., 2007). In the United Kingdom

(UK), smoking is estimated to have cost the health system £5.2 billion and the wider society £96 billion during 2005–2006 (Allender, Balakrishnan, Scarborough, et al., 2009; Nash & Featherstone, 2012). In the United States (US), the economic cost of smoking due to medical care and lost productivity was estimated to be between \$289 and \$332 billion annually from 2009 to 2012 (DHHS, 2014). Smoking among young people is subject to social stratification (Wellman et al., 2016) and is a major source of lifelong health inequalities (Dunstan, 2012).

Importantly, the number of youth who have smoked cigarettes has declined in recent years in the US, UK, and the vast majority of European countries (DHHS, 2012; Fuller, 2015; Johnston, O'Malley,

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Miech, et al., 2017; Kraus, Guttormsson, Leifman, et al., 2016; Scholes, Mindell, & Neave, 2016). Declining rates of youth and young adult cigarette use have been attributed to broad tobacco control efforts focused on making tobacco products less affordable (e.g., increased taxes), less desirable (e.g., marketing restrictions; graphic health warnings on tobacco products; school-based quit programs), and less accessible (e.g., increased smoke-free public spaces; prohibiting sales to minors; GBD Tobacco Collaborators, 2015; DHHS, 2012). Though numerous studies have examined the impact of tobacco control policy efforts on declines in youth smoking (DHHS, 2012; Brown, Platt, & Amos, 2014), less research has examined whether broad shifts in family and peer risk factors have also contributed to overall decreases in childhood cigarette use. Parents and peers feature prominently in theories of youth substance use (Chassin & Hussong, 2009) with empirical support for both sources of influence (Avenevoli & Merikangas, 2003; Leonardi-Bee, Jere, & Britton, 2011; Simons-Morton & Farhat, 2010). In addition to tobacco control efforts, changes over time in how parents and peer influences operate may provide additional explanations for why children currently are less likely to smoke cigarettes compared to children from past cohorts (Green, Leyland, Sweeting, et al., 2016).

Three key demographic changes in particular may help directly explain the decline in childhood smoking or operate as mechanisms of influence between increased tobacco control in later cohorts and youth smoking. 1) *Increases in Maternal Education*. It is well known that female educational accessibility has increased in recent decades in the UK and internationally (Gakidou, Cowling, Lozano, et al., 2010), and cigarette use is inversely correlated with years of education in adults (Giskes et al., 2005). Furthermore, parental education is a consistent predictor of child smoking initiation (Chassin, Presson, Sherman, et al., 1992; Conrad, Flay, & Hill, 1992; Green et al., 2016; Hiscock, Bauld, Amos, et al., 2012; Kandel, Griesler, & Hu, 2015; Staff J, Maggs, Cundiff, and Evans-Polce, 2016; Taylor-Robinson, Wickham, Campbell, et al., 2017). Thus, the risk of youth smoking may have declined partly due to historic increases in maternal education. 2) *Decreases in Parental Smoking*. Children are significantly more likely to smoke if one or more of their parents is a current or even prior smoker (Jackson & Henriksen, 1997; Kandel et al., 2015; O'Loughlin, Paradis, Renaud, et al., 1998; Sylvestre, Wellman, O'Loughlin, et al., 2017; Vuolo and Staff, 2013). Given the substantial drop in rates of adult smoking due to tobacco control efforts, mothers and fathers may be less likely to smoke when they have a young child than in prior generations, reducing the well-established intergenerational transmission risk for offspring use. 3) *Decreases in Peer Smoking*. Children who smoke often report that friends had given them cigarettes (Fuller, 2015), peer influences may be especially important earlier in adolescence (Fuemeller, Lee, Ranby, et al., 2013), and some studies show that friends have a stronger effect on childhood smoking initiation than do parents (Jackson, 1997; Kelly, O'Flaherty, Connor, et al., 2011). Given tobacco control efforts to make tobacco products less affordable, accessible, and desirable to youth in recent years, children today may be less likely to smoke because they do not have any friends who smoke.

When documenting cohort changes in childhood smoking, it is also important to assess whether inequalities in smoking have narrowed or widened in recent cohorts of children. Among UK 11–15 year olds born roughly 1979 to 1997, Green et al. (2016) found that social inequalities indexed by parents' education were maintained across the years 1994 to 2008, though some fluctuations were observed. Wellman et al. (2018), using a 2005 longitudinal sample of 10 year old children in Montréal, Canada, found a higher risk of cigarette initiation among children whose mothers have low education.

In this article, we use nationally representative data from two national birth cohorts in the UK (born in 1970 and 2001) to: 1) replicate prior research showing the substantial decline in childhood cigarette use over the past three decades; 2) assess whether the inclusion of a series of variables capturing cohort changes in parental and peer risk factors (i.e., increases in maternal education coupled with decreases in

parental and peer smoking) mediates cohort differences in the risk of childhood smoking; and 3) evaluate whether inequalities in childhood smoking initiation have changed. These multigenerational, longitudinal datasets are particularly advantageous for addressing these questions for three reasons. First, early life confounders (e.g., sociodemographic background) and mediators (e.g., maternal education, parental cigarette use) were assessed prospectively, which provides appropriate temporal ordering to control for spurious influences with respect to the direction of association, as well as conduct tests of mediation using Karlson, Holm, and Breen's (KHB) method for testing indirect effects in logit models (Breen, Karlson, & Holm, 2013; Karlson, Holm, & Breen, 2012). Second, data were collected directly from mothers, fathers, and children, which reduces potential biases due to children incorrectly reporting their parents' use or older youth misremembering their age of first use. Finally, few prospective studies linking parent and child cigarette use are based on nationally-representative samples (Avenevoli & Merikangas, 2003; Wellman et al., 2016). The large birth cohorts we use here allow us to assess changes in the prevalence of early initiation as well as whether smoking prevalence has become more common or less in certain population subgroups in recent years (Chassin, Presson, Seo, Sherman, et al., 2008; Kandel et al., 2015).

2. Method

2.1. Participants

We rely on two nationally representative birth cohorts: The British Cohort Study (BCS) focuses on all those living in Britain who were born in one week in April 1970. After the initial assessment of 16,571 infants (96% of births), follow-ups were conducted at ages 5, 10, 16, 26, 30, 34, 38, 42, and 46 (Brown & Hancock, 2014). The Millennium Cohort Study (MCS) targeted infants born between September 2000 and January 2002 who were alive and residing in the UK at 9 months of age (Hansen, 2014; Plewis, 2007). Cohort members were selected from a random sample of electoral wards, and wards were oversampled to achieve representation from the four UK countries, economically deprived areas, and areas with high concentrations of racial/ethnic minority families. In total, 18,552 nine-month old children participated (91% of targeted sample). Follow-up surveys occurred at ages 3, 5, 7, 11, and 14, with age 17 data collection in progress in 2018. In both cohorts, multiple sources of data were collected from multiple informants (e.g., parents, teachers, etc.).

In the current study, we rely on self-report surveys from the BCS and MCS children at ages 10–11 years, as well as interview data from their mothers and fathers regarding their socioeconomic background and smoking behavior when the child was an infant and age 5. Since families were not followed past the age of 5 in Northern Ireland in the BCS, for comparability across cohorts we included only children who were born in England, Wales and Scotland in our analyses. In the BCS (MCS) 89% (72%) of children completed the survey at age 10 (11). Prior research in both samples has shown that boys and children from disadvantaged backgrounds were less likely to be retained than girls and more advantaged children (Mostafa, 2014; Mostafa & Wiggins, 2014). Approximately 52% of BCS children are male and 4% were ethnic minority, compared to 50% and 11% of MCS children, respectively. These differences in minority/majority group representation reflect increased immigration to the UK and the sampling strategies used in the two studies.

2.2. Measures

2.2.1. Outcome variable: childhood cigarette use

BCS and MCS children completed confidential self-report surveys in 1980 and 2012, respectively (mean age: BCS = 10.16 years; MCS = 11.16 years), indicating whether they had *ever tried a cigarette*. For MCS children, lifetime cigarette users included even those children

who reported only having one “puff.” Self-report surveys have been shown to be a reliable indicator of cigarette use, even when completed by children (Henriksen & Jackson, 1999). Only a small percentage of children did not report their smoking at age 10/11 (< 1% in BCS; < 4% in MCS), and thus were excluded from analyses.

2.2.2. Key predictor variable and mediators

Our analyses included *cohort* as a predictor (coded 1 = BCS; 0 = MCS), as well as three potential mediators that were measured when the child was age 5 (i.e., in 1975 and 2006). Key mediators included: 1) *mother's self-reported educational level* was assessed with five dummy variables indicating her highest national vocational qualification (NVQ), coded as the attainment of: postsecondary academic or vocational qualifications (NVQ4+); two or more A levels (NVQ3); qualifications equivalent to the General Certificate of Secondary Education (GCSE) or O levels of grades A-C (NVQ2); lower grades of GCSE, O levels, or vocational certificates (NVQ1); and no qualifications (reference category); 2) *Mother's self-reported smoking behavior* was captured with three dummy variables indicating whether the mother did not smoke (reference category), was a light smoker (i.e., averaged < 1 pack per day), or was a heavier smoker (i.e., pack or more per day). We also included a measure of *father's self-reported smoking behavior*, distinguishing fathers who smoked lightly or heavily versus those who did not smoke (reference category), as well as fathers who smoked pipes and/or cigars. (No separate dummy variable was included for mothers because very few reported smoking pipes and/or cigars). We also included a dummy variable indicating whether the father was surveyed; 3) At ages 10/11, children in both cohorts were also asked “how many of your friends smoke cigarettes?” We distinguished children who reported “none of them” (coded 0) versus those who said at least some of their friends smoked (coded 1).

2.2.3. Sociodemographic background variables

All analyses adjust for child gender (1 = male; 0 = female); ethnic majority status (1 = white; 0 = non-white); child age at the time of survey completion (coded in months); and mother married (1 = yes), age 19 or less (1 = yes), and parity (ranging from 0 to 5 or more older siblings) at the time of the child's birth. We also include dummy variables indicating whether the child was born in England (reference category), Wales, or Scotland.

2.3. Strategy of analysis

Our first goal is to document changes in the prevalence of childhood smoking onset and then assess whether maternal education, parental smoking, and peer smoking explain observed cohort changes in childhood smoking. We first combine the BCS and MCS datasets and then estimate weighted logistic regression models predicting childhood smoking, with and without each of the mediators. We compare estimates from these models using Karlson et al.'s (2012) test for indirect effects to determine whether mother's education, parents' smoking, and friends' smoking mediate the effect of cohort on child smoking (Breen et al., 2013; Karlson et al., 2012). Using the KHB command in STATA 15, this method applies decomposition properties of linear models to the logit model, allowing us to test for indirect effects of our key risk factors. The rarity of childhood smoking in these models helps avoid potential underestimates of mediation due to the non-collapsibility of odds ratios, and provides odds ratios that are similar to risk ratios (VanderWeele, 2016). Our second goal is to assess whether the risk and protective factors associated with child smoking have changed over the past 30 years. Because differences in indirect effects between the cohorts cannot be assessed with confidence in non-linear probability models (Breen et al., 2013), we instead estimate the models separately by cohort and then use z-tests to compare the equality of the estimates (Clogg, Petkova, & Haritou, 1995).

Background variables shown to predict survey non-response in both

Table 1
Weighted descriptive statistics.

	BCS	MCS	% imputed
Child has smoked	14.3%	2.4%	0%
Mother education when child age 5			20%
No qualifications	57.0%	8.2%	
NVQ1	14.5%	7.2%	
NVQ2	17.7%	28.0%	
NVQ3	3.4%	15.3%	
NVQ4+	7.4%	41.3%	
Mother smoked when child age 5			15%
No	57.7%	77.1%	
Light	24.9%	18.3%	
Heavy	17.4%	4.6%	
Father smoked when child age 5			15%
No	42.6%	61.4%	
Light	18.3%	12.0%	
Heavy	25.9%	5.2%	
Pipe/cigar	8.0%	1.1%	
Not surveyed	5.2%	20.2%	
Child has a friend who smokes	16.1%	4.9%	5%
Background measures			
Male	51.7%	50.0%	0%
White	95.9%	88.4%	11%
Age at survey completion (mean)	10.16	11.16	4%
Child born in England	83.9%	85.8%	4%
Child born in Wales	5.7%	5.3%	
Child born in Scotland	10.4%	8.9%	
Parity (mean)	1.18	0.87	4%
Child born to unmarried mother	6.1%	36.9%	4%
Child born to teenage mother	9.4%	6.1%	4%
Sample size	12,597	10,909	

cohorts (Mostafa, 2014; Mostafa & Wiggins, 2014), such as child gender, ethnic majority status, age, and region, were included in all regressions. Furthermore, to reduce potential bias from item-missing data, we used multiple imputation procedures (Johnson & Young, 2011) in STATA 15 to create 20 complete datasets using chained regressions to impute values for missing data on the predictor variables. These imputed datasets were created separately for each cohort and then combined. As shown in Table 1, the proportion of imputed responses ranged from 0 (for gender and cohort) to 20% (for mother's education). Finally, following Rubin (1987) the estimates were combined across the 20 imputed datasets, and all estimates were weighted to account for the oversampling of ethnically diverse and economically disadvantaged areas in the MCS.

3. Results

Table 1 provides weighted descriptive statistics for all variables separately for BCS and MCS children. Approximately 14% of children in the BCS had previously smoked, compared to 2.4% of MCS children.

Table 1 reveals dramatic changes in the risk factors associated with childhood smoking between the BCS and MCS children. For instance, 57% of BCS mothers reported no qualifications compared to just over 8% of MCS mothers, and only 7.4% of BCS mothers had achieved at least a post-secondary diploma or qualification (i.e., NVQ 4+), in comparison to over 41% of MCS mothers. In addition, a higher percentage of mothers abstained from smoking in the MCS (77%) versus the BCS (58%). A similar pattern was also shown for fathers (abstaining fathers in MCS = 61%; BCS = 43%). The percentage of mothers and fathers who smoked heavily when the child was young also decreased. Finally, 16.1% of BCS children had a friend who smoked, compared to 5% of MCS children.

Table 2 presents weighted prevalence estimates of childhood smoking for each risk factor (i.e., mother's education, parental cigarette use, and peer smoking), separately by cohort. In both cohorts, the prevalence of childhood smoking was lowest among mothers with high levels of education and both mothers and fathers who did not smoke

Table 2
Prevalence of child smoking for each risk factor by cohort.

	% of children who have smoked	
	BCS	MCS
Mother's education when child age 5		
No qualifications	14.4	5.3
NVQ1	15.2	4.8
NVQ2	14.1	3.2
NVQ3	13.8	1.5
NVQ4+	12.6	1.3
Mother smoked when child age 5		
No	12.6	1.5
Light	15.6	5.0
Heavy	18.3	8.2
Father smoked when child age 5		
No	12.1	1.3
Light	15.0	2.9
Heavy	16.1	5.5
Pipe/cigar	15.2	1.6
Child does not have a friend who smokes	10.0	1.7
Child has a friend who smokes	36.9	16.9
Sample size	12,597	10,909

cigarettes, whereas the prevalence was highest among children whose mothers had low levels of education, whose parents smoked heavily, and who had a friend who had smoked.

Table 3 presents odds ratios and 95% confidence intervals from five logistic regression models predicting childhood smoking in the BCS and MCS. In Model 1, we first estimated a model that includes cohort as a predictor, as well as background variables that distinguish the two cohorts. Children in the BCS were over 12 times as likely to have smoked compared to children born 30 years later in the MCS. Boys were more than twice as likely to have smoked as girls, and the risk of smoking was significantly higher among children who were older when surveyed, white, and born to unmarried or teenage mothers. The risk of

early smoking was also higher among children with older siblings and those born in England (compared to Wales and Scotland).

Including mothers' educational level (Model 2), mothers' and fathers' smoking (Model 3), and peer smoking (Model 4) as mediators significantly reduced the effect of cohort on childhood smoking, compared to Model 1 (based on KHB tests). The parental risk factors predicted childhood smoking in expected ways. For instance, children were significantly less likely to smoke if their mothers had a college degree (Model 2), if their mothers or fathers did not smoke (Model 3), and if they did not have a friend who had smoked (Model 4). In Model 5, we included all the mediators, which together reduced the cohort estimate from Model 5 (log odds = 2.08) compared to Model 1 (log odds = 2.49) by approximately 16.5%.

Finally, we estimated Model 5 separately by cohort and then tested the equality of the estimates. Table 4 provides odds ratios and 95% confidence intervals for these logistic regression models. The results show statistically significant differences (i.e., $p < 0.05$) by cohort in the effects of the mediators. For instance, the risks posed by no maternal education (relative to A-level and higher qualifications), mothers' smoking, fathers' heavy smoking, and children's friends' smoking were significantly stronger in the MCS sample compared to the BCS sample. The effects of all but one background variable on the likelihood of child smoking did not vary between the cohorts. That is, teenage parenthood increased the risk of child smoking in the BCS cohort but not the MCS.

To highlight differences in risk between the cohorts in their probability of childhood smoking, in Fig. 1 we present a series of predicted probabilities for each cohort based on the estimates shown in Table 4. Across the two cohorts, the columns reveal differences in predicted probabilities among children whose mothers had low versus high levels of education (first two sets of columns), whose parents smoked heavily versus not at all (second sets), and whose friend(s) had smoked versus those who had non-smoking friends (third sets).

Table 3
Logistic regression models predicting childhood smoking.

	Model 1		Model 2		Model 3		Model 4		Model 5	
	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
BCS cohort (vs. MCS cohort)	12.10	[9.40–15.57]	10.74	[8.17–14.12]	11.07	[8.52–14.39]	8.59	[6.63–11.14]	8.01	[6.01–10.68]
Mother's education (ref = no qualifications)										
NVQ1			1.19	[1.02–1.40]					1.29	[1.09–1.52]
NVQ2			1.08	[0.91–1.26]					1.20	[1.01–1.43]
NVQ3			0.83	[0.63–1.09]					0.95	[0.71–1.26]
NVQ4+			0.73	[0.59–0.91]					0.94	[0.75–1.19]
Mother smoked when child age 5 (ref = no)										
Light					1.31	[1.14–1.51]			1.25	[1.08–1.44]
Heavy					1.45	[1.24–1.69]			1.30	[1.11–1.53]
Father smoked when child age 5 (ref = no)										
Light					1.24	[1.04–1.47]			1.21	[1.01–1.44]
Heavy					1.26	[1.08–1.47]			1.24	[1.06–1.45]
Pipe/cigar					1.33	[1.07–1.65]			1.22	[0.98–1.53]
Father not surveyed					1.82	[1.47–2.25]			1.66	[1.33–2.07]
Child has a friend who smokes							5.22	[4.67–5.85]	5.02	[4.49–5.62]
Background measures										
Male	2.05	[1.86–2.27]	2.06	[1.86–2.28]	2.06	[1.86–2.28]	1.78	[1.61–1.98]	1.79	[1.61–1.99]
White	1.67	[1.26–2.21]	1.64	[1.24–2.17]	1.50	[1.13–1.99]	1.68	[1.26–2.25]	1.52	[1.13–2.05]
Age at survey completion	1.61	[1.33–1.95]	1.60	[1.32–1.94]	1.59	[1.31–1.93]	1.46	[1.20–1.79]	1.44	[1.18–1.77]
Child born in Wales (ref = England)	0.84	[0.68–1.04]	0.84	[0.68–1.04]	0.82	[0.67–1.02]	0.90	[0.73–1.11]	0.88	[0.71–1.09]
Child born in Scotland (ref = England)	0.72	[0.60–0.85]	0.73	[0.62–0.87]	0.70	[0.59–0.83]	0.72	[0.61–0.87]	0.72	[0.60–0.87]
Parity	1.18	[1.14–1.22]	1.17	[1.13–1.21]	1.14	[1.11–1.18]	1.14	[1.10–1.18]	1.13	[1.09–1.17]
Child born to unmarried mother	1.69	[1.44–1.99]	1.64	[1.39–1.94]	1.34	[1.12–1.61]	1.55	[1.31–1.85]	1.27	[1.05–1.54]
Child born to teenage mother	1.38	[1.16–1.63]	1.34	[1.12–1.59]	1.25	[1.05–1.48]	1.20	[1.00–1.44]	1.12	[0.93–1.34]
Significant ($p < 0.01$) change in cohort estimate from Model 1 (based on KHB test)?			Yes		Yes		Yes		Yes	

Note. Sample size = 23,506 respondents. OR = Odds ratio, CI = Confidence interval.

Table 4
Logistic regression models predicting childhood smoking in the BCS and MCS.

	BCS sample only		MCS sample only		BCS and MCS estimates significantly different?
	OR	95% CI	OR	95% CI	
Mother's education (ref = no qualifications)					
NVQ1	1.26	[1.06–1.49]	0.93	[0.51–1.71]	No
NVQ2	1.18	[0.98–1.42]	0.78	[0.48–1.29]	No
NVQ3	1.15	[0.81–1.62]	0.50	[0.26–0.97]	Yes
NVQ4+	1.14	[0.88–1.49]	0.55	[0.32–0.95]	Yes
Mother smoked when child age 5 (ref = no)					
Light	1.13	[0.97–1.33]	1.94	[1.34–2.80]	Yes
Heavy	1.20	[1.01–1.43]	2.32	[1.43–3.76]	Yes
Father smoked when child age 5 (ref = no)					
Light	1.17	[0.96–1.42]	1.45	[0.90–2.36]	No
Heavy	1.19	[1.01–1.40]	2.35	[1.37–4.03]	Yes
Pipe/cigar	1.17	[0.94–1.47]	1.23	[0.20–7.52]	No
Not surveyed	1.46	[1.11–1.93]	1.98	[1.32–2.98]	No
Child has a friend who smokes (1 = yes)	4.64	[4.14–5.21]	7.66	[5.36–10.95]	Yes
Background measures					
Male	1.81	[1.63–2.02]	1.66	[1.23–2.24]	No
White	1.79	[1.20–2.66]	1.20	[0.75–1.90]	No
Age at survey completion	1.45	[1.16–1.82]	1.40	[0.89–2.19]	No
Child born in Wales (ref = England)	0.88	[0.69–1.13]	0.81	[0.55–1.21]	No
Child born in Scotland (ref = England)	0.74	[0.61–0.90]	0.64	[0.39–1.04]	No
Parity	1.11	[1.07–1.16]	1.22	[1.09–1.38]	No
Child born to unmarried mother	1.20	[0.94–1.52]	1.15	[0.81–1.62]	No
Child born to teenage mother	1.22	[1.01–1.49]	0.69	[0.41–1.17]	Yes
Sample size	12,597		10,909		

4. Discussion

Consistent with recent trends showing substantial historical declines in adolescent smoking in the US, UK, and most European countries (DHHS, 2012; Johnston et al., 2017; Scholes et al., 2016), childhood cigarette use declined substantially in these two representative samples of British children born 30 years apart. Children born in 2001 are eight

times less likely to have tried a cigarette by age 10–11 than children born in 1970, even after controlling for a high number of early life risk factors. Importantly, the historic reductions in childhood smoking shown in this study would likely have been even larger if our BCS smoking measure included those who only had one “puff” of a cigarette. The relatively low number of childhood smoking initiators in the MCS is consistent with a 2014 survey of 6173 secondary school pupils in England showing that only 4% of 11-year olds reported that they had previously smoked a cigarette (Hawkins, 2015).

Our findings based on nationally representative data also highlight sizeable cohort changes in the early life risk factors associated with childhood cigarette use. For instance, mothers in the more recent cohort are more educated and were less likely to smoke when the child was young, both of which predicted reduced risk of offspring cigarette use in late childhood. Smoking among fathers has also declined, and the percentage of children reporting having a friend who smoked declined by almost two-thirds.

However, it is important to note that though the key parental and peer risk factors decreased in prevalence, their links with child smoking have increased in magnitude. For instance, children in the MCS cohort were nearly 2 times as likely to have tried smoking if their mother was a light smoker, compared to mothers who did not smoke, whereas in the earlier BCS sample mother light smoking did not predict child smoking at this young age. Furthermore, MCS children were 7 times more likely to have smoked if they had a friend who smoked. The results also revealed that the protective effect of mothers' education on offspring smoking has strengthened over the 30-year period. Whereas mothers' education did not predict children's use in the BCS, children in the MCS were 40% less likely to smoke if their mothers had a post-secondary qualification, in comparison to those who did not. Overall, our findings suggest that childhood smoking in today's young people in the UK is now more strongly linked to early life disadvantages compared to a generation ago, consistent with prior research on youth smoking (DHHS, 2012) and a broader move to a concentration of adverse outcomes among a core group of multiply disadvantaged youth (Kneale, Fletcher, Wiggins, et al., 2013; Wellman et al., 2018).

4.1. Limitations

First, our analyses focus on cigarette use at only ages 10/11 due to comparability of ages in measurement and data availability at the time of writing. Future research should compare BCS/MCS cohort differences in age 16/17 smoking as additional MCS cohort data become available. Second, although our analyses account for both mothers' and

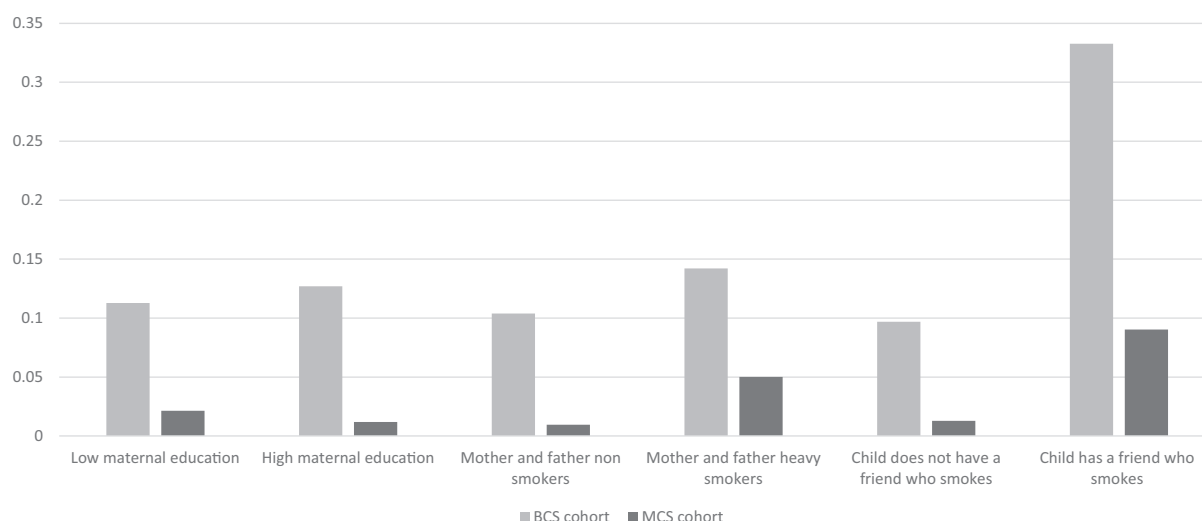


Fig. 1. Predicted probabilities of childhood smoking based on maternal education and parental/peer smoking in the BCS and MCS.

fathers' smoking (via self-reports from surveys of parents), we do not measure other family influences, in particular the smoking behavior of older siblings and other adults in the household or close social network. The sizeable effect of birth parity suggests that older sibling smoking may be increasing the risk of early onset cigarette use in both cohorts (Slomkowski, Rende, Novak, et al., 2005; Vuolo and Staff, 2013). In addition, parents in the BCS and MCS unfortunately were not asked about smoking-specific parenting practices, such as household bans on indoor or any smoking, which also likely account for cohort changes in childhood smoking (Hiemstra, de Leeuw, Engels, et al., 2017). Third, it is also plausible that children are now beginning to replace combustible cigarettes with electronic cigarettes. Although the prevalence of e-cigarette use would have been minimal before 2011 (DHHS, 2016), especially among children, e-cigarette use is increasing. For instance, in the 2015 National Youth Tobacco Survey, about 14% of US middle school students had ever tried an e-cigarette. In a 2014 survey of 6173 secondary school pupils in England, 80% of 11 year old children were aware of e-cigarettes and 5% reported previous e-cigarette use (Hawkins, 2015). Fourth, it is important to note that MCS children reported on their peers' smoking, and thus the influence may be circular due to overestimation of similarity of peer behaviors and selection processes in which children prone to smoking may attract older youth or other smoking children as friends (Schaefer, Haas, & Bishop, 2012). However, our goal was to assess whether changes in peer smoking accounted for the cohort decline in childhood smoking onset, and not necessarily the causal effects of peer influences. Finally, in some instances our goal of making the control measures compatible between the BCS and MCS cohorts led to a loss of precision, particularly in the MCS when we collapsed ethnicity into white versus non-white status and mother's marital status at birth into married versus unmarried.

5. Conclusions

Our findings offer cause for both celebration and worry. Given rapid transitions to dependence (DiFranza et al., 2007) and substantial long-term health risks associated with childhood smoking onset (DHHS, 2012), the impressive drop in the percentage of childhood smokers across the last generation is a cause for celebration. It is striking that only 3% of MCS children reported smoking by age 10–11, compared to 14% of BCS children born just one generation earlier. However, our findings also raise two key public health concerns. First, although the prevalence of childhood smoking has declined, health inequalities have increased as childhood smoking is increasingly overrepresented in disadvantaged households. Across the life course, it is plausible that early smoking onset may exacerbate socioeconomic disparities in health (Maynou & Saez, 2016). Second, although cigarette use in childhood has declined, e-cigarette use has become more prevalent and e-cigarette use has been shown to increase the risk of combustible cigarette use in later adolescence (National Academies of Sciences, Engineering, and Medicine, 2018). To address these issues, implementation of population-level interventions known to be effective particularly for disadvantaged smokers or potential smokers (e.g., increased price via taxation; Hill, Amos, Clifford, et al., 2014) as well as increasing access to targeted individual-level interventions for smokers/potential smokers (such as school-based quit programs) are needed.

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Contributors

Drs. Staff and Maggs designed the study. Dr. Staff conducted the analyses and drafted the initial version, and Drs. Maggs, Ploubidis, and Bonell contributed significantly to the revision of the manuscript. All authors approved the final manuscript as submitted.

Competing interests

The authors have no conflicts of interest to declare.

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