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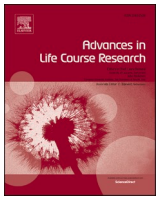

Sibling group size and BMI over the life course: Evidence from four British cohort studies

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ABSTRACT

Only children, here defined as individuals growing up without siblings, are a small but growing demographic subgroup. Existing research has consistently shown that, on average, only children have higher body mass index (BMI) than individuals who grow up with siblings. How this difference develops with age is unclear and existing evidence is inconclusive regarding the underlying mechanisms. We investigate BMI trajectories for only children and those with siblings up to late adolescence for four British birth cohorts and across adulthood for three cohorts. We use data on BMI from ages 2–63 years (cohort born 1946); 7–55 years (born 1958); 10–46 (born 1970) and 3–17 years (born 2000–2002). Using mixed effects regression separately for each cohort, we estimate the change in BMI by age comparing only children and those with siblings. The results show higher average BMI among only children in each cohort, yet the difference is substantively small and limited to school age and adolescence. The association between sibling status and BMI at age 10/11 is not explained by differential health behaviours (physical activity, inactivity and diet) or individual or family background characteristics in any of the cohorts. Although persistent across cohorts, and despite the underlying mechanism remaining unexplained, the substantively small magnitude of the observed difference and the convergence of the trajectories by early adulthood in all cohorts raises doubts about whether the difference in BMI between only children and siblings in the UK context should be of research or clinical concern. Future research could usefully be directed more at whether only children experience elevated rates of disease, for which high BMI is a risk factor, at different stages of the life course and across contexts.

1. Introduction

The association between family size and child outcomes is the subject of a rich research literature in the social sciences, suggesting children from smaller families generally fare better than children from large families on a range of outcomes such as education, occupational achievement and wealth (Lersch, 2019; Steelman et al., 2002). Yet only children, i.e. individuals growing up without siblings, stand out as having worse outcomes on certain health measures. In particular, only children have been found to have elevated Body Mass Index (BMI) and higher prevalence of BMI above the 'overweight' and 'obesity' thresholds in childhood, adolescence and/or adulthood in a range of contexts, including the USA, European countries, China, and Japan (A. Chen & Escarce, 2010; Haugaard et al., 2013; Hunsberger et al., 2012; Li et al., 2017; Lin & Falbo, 2022; Meller et al., 2018; Min et al., 2017; Mosli et al., 2015; Mosli et al., 2016; Wang et al., 2007). Despite being an indirect and inaccurate proxy for adiposity, BMI provides a cost-effective indicator of bodyweight associated with higher relative risk of clinical diagnoses at the population level and across population sub-groups. The recent steep increase globally in the prevalence of people with BMI conventionally classified as 'overweight' or 'obese' among both adults and children (Ng et al., 2014) has generated much

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research interest. Especially BMI classified as above the 'obesity' threshold among children is the frequent target of policy because of a tendency for high BMI to track into adulthood, with associated increased risk of cardiovascular disease (Araújo & Ramos, 2017). Given the associated health risks of elevated BMI, and only children being a growing population subgroup in the UK and elsewhere (Breton & Prioux, 2009; Frejka et al., 2010; Frejka, 2008; ONS, 2020), there is a need to better understand when and why the relationship between family size and BMI emerges and how it develops over the life course.

The consistent finding across studies of higher BMI among only children when compared with individuals with siblings notwithstanding, many important gaps in knowledge remain. Research to date has largely focused on the childhood and early adulthood stage and has not shown how this association develops as people age (Lin & Falbo, 2022, provide a recent exception). Moreover, we lack evidence of whether the association has changed in recent decades as BMI levels have increased substantially at the population level. To this end, the present paper first contributes to the literature on family size and child health outcomes by describing BMI trajectories over the life course for only children and individuals with siblings. Second, we compare these trajectories from childhood to late adolescence for four British cohorts and across adulthood for three of the cohorts to assess whether the association between sibling group size and BMI has changed or remained stable over the last several decades. Finally, research to date has been inconclusive on the potential mechanisms underlying the association between being an only child and having elevated BMI and these findings are not easily reconciled with dominant theoretical perspectives on the association between family size and child outcomes, warranting further investigation. The third contribution of the present study, therefore, is the investigation of possible mechanisms that might underlie the association between BMI and sibling status.

1.1. Change with age and over time

Interpretive frameworks of only children's differences from those with siblings (discussed further below) have typically focused on the childhood stage but life course epidemiology can help understand how childhood circumstances may affect not only concurrent but also later BMI. A range of different theories have been put forward to describe different health trajectories and associations between earlier life course circumstances and later health. The pathway model and the cumulative advantage/disadvantage model both highlight the role of social circumstances in early life and over the life course for the development of health (Corna, 2013). The former emphasises that the effects of early life experiences can be modifiable while the latter framework seeks to explain patterns of divergence in health trajectories between more and less privileged groups. In contrast, the age as leveller hypothesis has been proposed for patterns of convergence in health trajectories between groups whereby the effect of earlier circumstances or inequalities on health outcomes are attenuated with the passage of time (Corna, 2013). Finally, the critical period (or time associated vulnerability) focuses on the timing of exposure to risk for the development of (later) health problems (see e.g. Burton-Jeangros et al., 2015; Corna, 2013 for further discussion of these frameworks).

A study in the USA found that on average, compared to children with siblings, only children have higher BMI, a higher probability of having BMI classified as 'obese' at school entry, and experience a greater than average increase in BMI over the course of their years in primary education (A. Chen & Escarce, 2010). As a marker of relative risk of clinical outcomes in adulthood, it is important to look beyond the childhood phase to understand whether only children's elevated BMI in childhood persists in adulthood. For example, general studies of childhood weight development suggest that earlier occurrence of 'the adiposity rebound' (usually seen in children between ages 5–7 years) has been linked with worse later health outcomes (Burton-Jeangros et al., 2015; Parsons et al., 1999), possibly indicating a time-associated vulnerability that only children may be at greater risk of experiencing. Studies using Swedish register data have found persistent differences by sibling status among young adults, including higher BMI at age 17–20 (based on data for men; Keenan et al., 2022) and elevated mortality levels among adult only children (Baranowska-Rataj et al., 2017; Keenan et al., 2022). These results indicate that the disparity in BMI between only children and siblings could have implications for health outcomes later in life and mortality. However, most studies to date have either used cross-sectional data (Meller et al., 2018) or have been able to analyse BMI at a single time-point only (e.g. Keenan et al., 2022). We are aware of just one longitudinal study comparing repeated BMI measures of only children and those with sibling from childhood into adulthood (Lin & Falbo, 2022). That study, using US data on a cohort who were adolescents in the mid-1990s, found on that higher BMI among only children persisted into adulthood with the magnitude of the difference by sibling status remaining stable. More longitudinal analysis is required to better understand whether and how trajectories differ, which themselves may systematically relate to the risk of developing adverse health outcomes.

Finally, life course theory, with its emphasis on the importance of historical time and place (Elder, 1998), also serves as a reminder that patterns by sibling status are not necessarily stable across cohorts. Although there is clear evidence of increasing rates of 'overweight' and 'obesity' in the UK and globally in recent decades (e.g. Jebb et al., 2013; Moody, 2019; Ng et al., 2014), indicative that cohort effects are likely to be notable, evidence is limited on whether the relationship between sibling status and BMI has changed or persisted over time. One study that has compared cohorts found that the BMI difference by sibling status in children and adolescents in China increased between 2000 and 2011 (Min et al., 2017). However, it is difficult to assess longer terms trends based on existing evidence as different studies not only relate to different cohorts but also to different age groups and/or geographic contexts such as 17–20 year old men in Sweden (Keenan et al., 2022), adolescents and adults born in late 1970s-early 1980s in the US (Lin & Falbo, 2022) or kindergarten to elementary (primary) school aged children born in the 1990s also in the US (A. Chen & Escarce, 2010). It is thus also important to investigate whether and how the relationship between sibling status and BMI (trajectories) may have changed across cohorts.

1.2. Potential mechanisms

There is limited understanding of the mechanisms underlying the only child-BMI association, including whether they differ over time or across contexts. A logically plausible and popular explanation for the often negative association observed between sibship size and child outcomes is the resource dilution theory. This framework suggests that children in small families benefit from a greater share of parental resources - such as income, living space, time and attention – while the amount available to each individual child is much smaller when shared between a greater number of siblings (Blake, 1989; Downey, 1995). It has received much empirical support, especially for educational achievement, but also for other outcomes such as wealth in adulthood (Choi et al., 2020; Lersch, 2019; Steelman et al., 2002). Yet, as Steelman and colleagues point out, the theory does not successfully account for why only children do not consistently perform better than children in two-child families. Further, studies comparing only children to those with siblings suggest that patterns differ depending on the outcome and context investigated (see e.g. Choi & Monden, 2019; Falbo, 2012; Falbo & Polit, 1986; Mancillas, 2006; Polit & Falbo, 1987). In fact, for some health outcomes, including BMI as discussed above (Meller et al., 2018), the lack of siblings is associated with worse outcomes. Given that, in developed countries, greater parental (economic) resources are generally associated with healthier diet and lifestyle, and lower child BMI (Bann et al., 2018; Parsons et al., 1999), the resource dilution hypothesis does not appear to fit well. We therefore turn to two alternative explanations below in discussing potential mechanisms, which based on the

broader literature on BMI might be organised under three broad headings: behavioural, maternal/infant health and socio-economic differences between the groups.

First, the socialisation (or siblings as resources; Downey & Condron, 2004; Goetting, 1986) theory suggests that children benefit from the competition, negotiation and mutual support of growing up with siblings. Studies of BMI have often looked to diet, physical activity and sedentary activity as an explanatory mechanism (Burton-Jeangros et al., 2015; Parsons et al., 1999), and if children who grow up with siblings are more likely to have active lifestyles, for example playing outdoors with their siblings, this framework could help explain observed differences in BMI.

1.2.1. Health behaviours

Analysis of Australian time use data (among adults) has suggested that increased 'disorganization of eating', including a decline in dedicated and social mealtimes, has coincided with global increases in BMI and 'obesity' rates (Bittman et al., 2019). To the extent that larger families tend to have more organised mealtimes this could be a contributing factor in the association between sibling status and BMI. There are some indications that this may indeed be the case; Datar (2017) finds in a US study that children with siblings share mealtimes with their family and consume fruits and vegetables and healthier drinks (milk and 100% fruit juice) more frequently and have fast food and sweet drinks less frequently than only children. More directly, Mosli et al. (2015), found parental mealtime behaviour (use of verbal discouragement/ praise when their child was presented with familiar and unfamiliar foods) fully explained the higher rates of 'overweight' among only children aged 4–8 years in a small sample of low-income families from Michigan. Another suggested mechanism relates to childhood leisure activities, with research indicating some only children spend more time in sedentary activities and less time on physical activities in Australia (Bagley et al., 2006) and spend more time watching TV in the USA (Datar, 2017). Such findings might be interpreted as consistent with the socialisation hypothesis. However, these studies did not formally test whether such mealtime and screen time 'health behaviours' explained the association between sibling status and BMI. Meanwhile Hunsberger et al. (2012) found that although only children had less outdoor playtime and a higher sugar intake, these factors did not explain the higher prevalence in only children of having 'overweight' BMI. Further, although overall the increased prevalence of BMI classified 'overweight' among children generally has been widely attributed to the increase in sedentary leisure activities, a review of the literature found a consistent lack of association between only child status and television/video viewing time (Gorely et al., 2004). Thus, the evidence on the role of differential health behaviours in explaining the association between sibling status and BMI remains inconclusive.

Another explanation for the association between family size and child outcomes, relates to differential fertility according to parental characteristics and circumstances. Thus, one potential explanation for only children's higher BMI relates to health and socio-economic determinants of being an only child. Only child families can arise through parental separation, secondary infertility, parental or child ill-health, first birth experience (Elvander et al., 2015), or other factors that may also be relate to child outcomes (for example, the educational gradient to fertility in the UK is well-established, Sigle-Rushton, 2008).

1.2.2. Maternal/infant health

While some parents want one child only, for others reproductive health reasons result in having no further children. If there is a hereditary link, such underlying health reasons for the cohort member having no siblings could also be related to the cohort member's health and physical development. Parental smoking, birthweight and no or short breastfeeding have all been linked to elevated childhood BMI (Burton-Jeangros et al., 2015; Parsons et al., 1999). The BMI of children is also closely associated with the BMI of their parents, likely through a combination of both biomedical and social mechanisms such as shared family diet and lifestyle factors (e.g. C.Y. Lee, 2019). It is not clear from existing research to what extent these factors differ between only children and siblings. However, mean age at first birth tends to be higher among mothers of only children compared with women who go on to have more children (Jefferies, 2001; Parr, 2007), thus other factors may also vary in ways that might either mask or explain BMI patterns by sibling status.

1.2.3. Socio-economic differences

BMI tends to be higher among the more socio-economically disadvantaged in many developed countries and, in the UK at least, these social inequalities in BMI among children have grown over time (Bann et al., 2018; Parsons et al., 1999). In a life-course perspective, socio-economic disadvantage in early life is central to the cumulative advantage/disadvantage model (Burton-Jeangros et al., 2015; Corna, 2013). It is noteworthy that the prevalence of only children differs cross-nationally and that the prevalence is positively associated with the socio-economic profile of one-child families. In countries where small families are more prevalent (East European countries as well as Italy and Portugal), one-child families tend to be socio-economically more advantaged, whereas they tend to be less advantaged than other families in countries where one-child families are rarer (e.g. Iceland, Sweden and Ireland). This variation in the characteristics of families with one child explains why on some measures the outcomes of only children vary cross-nationally (Choi & Monden, 2019). In the UK, based on the data used in this paper, the proportion of only children has fluctuated over time from a high of about 14% among those born in the mid-1940s, falling to about 7% among individuals born in the late 1950s before rising to about 9% among those born at the turn of the millennium.

There is a lack of existing UK research on only children's BMI, but we know that in other contexts only children tend to have higher BMI. However, to the extent that this may at least in part be linked to only child families being more disadvantaged, it is unclear whether the same will apply in the UK. In terms of socio-economic profile, one child families in the UK tend to be less advantaged than two-child families, but more advantaged than large families with five or more children (Choi & Monden, 2019). For other socially patterned measures such as cognitive and educational outcomes, only children generally perform similarly to children with one sibling and better than children from large families (Choi & Monden, 2019; Laybourn, 1990). Although one study found disparities in parental education by family size were less pronounced and more stable over time among one-child families than among sibling families (Präg et al., 2020), the study did not account for parental separation which is also related to socio-economic disadvantage and associated child outcomes. Only children are more likely to experience parental separation and grow up in a lone parent household (e.g. Datar, 2017; Jefferies, 2001; Laybourn, 1990), and increasingly so over time in the UK context (Goisis et al., 2021). Through its association with socio-economic disadvantage, parental separation may thus be an important factor in the association between sibling status and BMI. Indeed, one US study found that controlling for lone parenthood and family socio-economic status attenuated (but did not fully explain) the association between sibship size and BMI (Datar, 2017). On the other hand, studies from Denmark and Sweden have found differences in 'overweight' and 'obesity' levels by sibling status persist after robust adjustment for potential selection (Haugaard et al., 2013; Keenan et al., 2022). Interestingly, some studies report that differences in BMI by sibling status increase slightly when adjusting for covariates (A. Chen & Escarce, 2010; Haugaard et al., 2013), suggesting that to some extent differences in the characteristics of families of different sizes may mask or statistically compensate for, rather than explain, underlying differences in BMI by sibling status. In the UK context, there are also indications that selection into being an only child has changed over time so that only children have become a more diverse group (Goisis et al., 2021), suggesting a need for cross-cohort investigation of potential

mechanisms. As the outcomes of only children for other measures have been shown to vary cross-nationally and both average BMI and its relationship with socio-economic circumstances have changed over time in the UK, it is neither necessarily the case that the patterns in BMI by sibling status found in other countries hold for the UK nor that the association is necessarily stable over time.

1.3. Study aims and contribution

We focus on the UK context, where the link between BMI and sibling status has thus far not been explored. Given the existing evidence on BMI by sibling status and the tendency for BMI to track into adulthood, we might expect to find higher BMI levels among only children that persist into adulthood. However, considering mixed indications regarding the (changing) socio-economic profile of only child families in the UK context, it is unclear whether we should expect trajectories to diverge over the life course, as would be predicted by the cumulative advantage/disadvantage framework, or for sibling status differences to have changed across cohorts.

The primary aim of this paper is to describe BMI trajectories over the life course for only children and individuals with siblings. We do this drawing on repeated BMI measures through childhood to late adolescence for four large-scale British birth cohorts born over a 50-year period, and across adulthood to mid-40s, mid-50s and early-60s, respectively, for three of the cohorts. The second aim is to test possible mechanisms that might underlie the association between BMI and sibling status. Focusing on middle childhood (10/11 years), we analyse whether differences in health behaviours (physical activity, inactive leisure, and diet) and/or parental and early childhood characteristics and circumstances help better understand BMI patterns by sibling status in three cohorts. Placing the results in conversation with theoretical frameworks from life course epidemiology, this study contributes to developing the literature on only children's outcomes which has hitherto predominantly focused on the childhood period.

2. Data & methods

2.1. Data

We analyse data from four British cohort studies, born in 1946, 1958, 1970 and 2000–2002 respectively. The National Survey of Health and Development, has followed a subsample of the individuals born in a given week in 1946 (5,362 of the initially surveyed 13,687 births). The 1958 National Child Development Study and the 1970 British Cohort Study follow cohorts of initially approximately 17,000 people born in a particular week in 1958 and 1970, respectively. The Millennium Cohort Study has surveyed a representative sample of nearly 19,000 individuals born between September 2000 and January 2002 since the cohort members were age 9 months to the latest sweep at age 17 years. For brevity we refer to this as the 2001 cohort. The studies are ideally suited for investigating both life course and secular change (see e.g. Bann et al., 2018; Lacey et al., 2017; McMunn et al., 2021) and across these different cohorts we observe BMI from age 2–63 (1946 cohort); 7–55 (1958 cohort); 10–46 (1970 cohort) and 3–17 (2001 cohort), separately for only children and for siblings.

2.2. Measures

2.2.1. BMI

Weight and height have been recorded in all four studies at most sweeps, either through interviewer measurements or self-reports. The harmonisation of these height, weight and resultant body mass index variables across sweeps and studies has been documented elsewhere (Hardy et al., 2019), and we use the harmonised longitudinal datasets for each study, which we have updated to include the most recent data collection sweep, where necessary. On average, weight and BMI are lower when self-reported rather than measured, where relevant we therefore control for whether recorded weight was measured or self-reported. We primarily analyse and report on BMI as a continuous measure but where we report on BMI as a categorical measure, we have used the conventional cut-off at BMI > 25 for ages 18 and over, and the equivalent sex and age-adjusted International Obesity Task Force (IOTF) cut-offs for ages below 18 years. Preferring to categorise the BMI rather than the individual, we refer to this as indicating BMI classified as 'overweight'.

All of our models control for cohort member's sex, and because BMI differs by ethnic group (which is also related to family size), we include the cohort member's ethnic group as recorded at age 11 (white; mixed; Indian; Pakistani and Bangladeshi; Black; and other ethnicity) in all analyses of the 2001 cohort. Ethnicity information is not available for the 1946 cohort and very few respondents in the 1958 and 1970 cohorts were not white.

2.2.2. Sibling status

We identify only children in each of the studies based on information about co-residence of siblings at age 10/11. As the studies do not enable us to identify siblings living in other households (including step or half siblings), we focus on co-residence at age 10/11 which we deemed late enough in the cohort member's childhood to capture the existence of younger siblings in the vast majority of cases, and also early enough that older siblings would still be co-resident. (Age gaps of more than 10 years are rare; in the 2001 cohort, among firstborns with at least one co-resident sibling at age 14, 2.9% had an age gap of 11 + years and 1.9% an age gap of 12 + years.) All our models also control for birth order (prior research indicating that among siblings last-borns are also at risk of elevated BMI; Haugaard et al., 2013; Mosli et al., 2015; 2016), combining third and later order in one group. Restricting the sample to first-borns for sensitivity analysis we distinguish those with siblings by number of (younger) siblings (1, 2 or 3 +). As the overall conclusions remained substantively unchanged, we report on the results using the binary indicator of sibling status.

We group a range of individual, early life and parental covariates included in our analyses according to the three potential mechanisms they may capture: maternal/infant health, socio-economic selection and health behaviours.

2.2.3. Maternal/infant health

We include maternal age at the time of the cohort member's birth (5-year age bands). We lack data on secondary infertility, but the 1958 and 1970 studies asked about miscarriages and stillbirths that mothers had experienced prior to the birth of the cohort member, and we include a binary indicator of any such occurrence. We also include a number of covariates potentially more directly related to weight in childhood and later life, including an indicator of whether the mother smoked at any point during the pregnancy, whether the birth was by caesarean section (each available in 1970, 1958 and 2001 cohorts only), an indicator of low birthweight ($<$2500 g) and an indicator of whether the cohort member was breastfed (any length of time).

The studies collected (self-reported) parental height and weight when the cohort member was a child (age 6 in the 1946 cohort, age 10/11 in the 1970 and 1958 cohorts, age 7 in the 2001 cohort). For cases with only one parent's BMI available we include the measure for that parent and where both parents' measures are available to us we choose the higher BMI of the two. Where BMI is available for both parents, the father has the higher BMI in 58–65% of cases (depending on the cohort). Including both parents' measures separately would exclude children of lone parents from the analysis and the magnitude of the coefficient is similar whether we use the higher parental BMI or maternal BMI in our models. Our preference is therefore for the (higher) parental BMI to not implicitly reinforce gendered social notions of maternal responsibility for child outcomes (Milliken-Smith & Potter, 2021). This variable is not part of our core set of covariates entered in all analyses, instead we

include parental BMI in additional models as a potential moderator of the relationship between sibling status and BMI.

2.2.4. Socio-economic

We include maternal education and parental social class for all cohorts. Although the exact measures differ, we aim for both conceptual comparability and measures that are socially meaningful within the historical context of the cohort analysed. For the 1946, 1958 and 1970 cohorts we use an indicator of the mother remaining in education beyond compulsory schooling age and the father's social class using the General Register Office occupational categorisation. For the 2001 cohort the educational measure is an indicator of whether the mother had a degree-level qualification, and the social class categorisation is based on the standard occupational classification, taking the higher category of either parent. As an indicator of parental separation during childhood, we include whether the father was recorded as living in the household (1946, 1958 and 1970 cohorts) or parental relationship status (2001 cohort), at the age 10/11 interview.

2.2.5. Health behaviours

We included items that related to health behaviours in childhood/ adolescence under three broad headings: physical activity, inactivity (predominantly related to watching/ playing on screens), and diet (consumption of specific foods/drinks such as sweet drinks, white bread, fruits, vegetables). None of the items capture actual time use, and since the wording of the questions and the answer categories differed across the cohorts the items cannot be fully harmonised, but where possible we have selected items that relate to behavioural frequency and comparable answer categories. See the descriptive analysis of the health behaviour variables and answer categories for more details (Online Table 8).

2.3. Methods

We ran two sets of analyses. In the first, we use mixed effects regression, or growth curve modelling, to estimate the change in BMI by age and compare these between only children and those with siblings. Mixed models are ideally suited to our analysis because the approach explicitly models the shape of individual trajectories over time for repeated measures of a continuous outcome variable, allows for irregularly spaced measurements across time (a salient feature of the data we use), and incorporates a robust approach to missing data through maximum likelihood estimation (Hedeker & Gibbons, 2006; Rabe-Hesketh & Skrondal, 2012). For the long-running cohorts in particular, attrition and intermittent non-participation means that restricting analysis of BMI trajectories to complete cases would substantially reduce the size of the analysis sample (see Online Table 1). As a robustness check we also used multiple imputation to account for the missing data prior to fitting the mixed models for the 1946, 1958 and 1970 cohorts. The results were substantively unchanged so we report results from the mixed models run on unimputed data.

We fit the models separately for each cohort, using Stata mixed command for the 1946, 1958 and 1970 cohorts, and using meglm (compatible with the complex survey design specifications and weights) for the 2001 cohort. The relationship between age and BMI is non-linear and exploratory analyses revealed that for some of the datasets the growth was more complex than adequately captured by a quadratic, cubic or quartic transformations of age. We therefore fit age as a piecewise linear growth function, with knot points at the timepoints (ages) when BMI was collected in each of the cohort studies (see Online Table 2). We allow for individual variation on the intercept and effect of time, with age entered as a linear term in the random part of the model.

For ease of interpretation, we show time as age in years on the x-axis in all graphs. However, since the placement of the knots is determined by the timing of the sweeps, and differs across the datasets, we use general life stage terminology (e.g. early childhood, primary school age, adolescence) as recommended in existing work (Crozier et al., 2019) to avoid attaching undue importance to the specific timing of changes in the slope of the curves. We include the maternal/infant health and socio-economic sets of covariates listed above (see Online Table 3). In each cohort, we tested for the effect of sibling status both on the intercept and interacted with age and since the interaction is significant in each cohort this is the model we report in the results below.

In the second set of analyses, we investigate potential mechanisms underlying the association between BMI and sibling status. Given that health behaviour is an often-cited mechanism in BMI/ 'obesity' research that has not been systematically tested in the existing research on only children's BMI, this part of our analysis was guided by the availability of information on cohort member's health behaviours. Although such information has been collected at various data collection sweeps in these studies differences in question wording means it is not possible to include these as time-variant variables in the growth curve models. We therefore focus on ages that both included health behaviour questions and when the BMI trajectory analysis has revealed a difference between only children and siblings for additional analysis investigating mechanisms. We ran a series of cross-sectional linear regression models at age 10/11 and 16/17, in the 1958, 1970 and 2001 cohorts respectively (the 1946 cohort did not include health behaviour questions at these ages). Item missingness on the analysis variables reduced the sample available for analysis (Online Table 4), and descriptive analysis of the profile of the complete case analysis sample suggested some introduction of bias, in the absence of adjustment for missing data. This was particularly the case for the 1970 age 16 and 2001 age 17 sweeps. The complete-case samples at these sweeps had an overrepresentation of girls, white children (2001 cohort), children who had been breastfed, had higher educated or older mothers, and children who lived with both parents at age 11 (2001 cohort; see Online Table 5). We used multiple imputation to adjust for missing data on the covariates and health behaviour variables, conditional on sweep participation and non-missing BMI at the age analysed. We created 20 imputed data sets separately for each cohort and age analysed using chained equations imputations and including both parents' BMI as auxiliary variables in the imputation model. Due to the 1970 age 16 data collection having coincided with teacher strikes which affected response rates and higher missingness on BMI than for the other cohorts, we conditioned the imputation only on sweep participation and included BMI in the imputation model, along with the cohort member's earlier BMI measure (age 10) as an additional auxiliary variable.

For each cohort, we ran four models. In the baseline model (M1), we included sibling status, birth order, sex and ethnicity (2001 cohort only). In the second model (M2) we added the health behaviour variables, followed by further adding the maternal/infant health and socio-economic covariates outlined above (M3) and finally adding parental BMI (M4). The focus of the analysis is to examine whether, and if so how, the regression coefficient for being an only child changes when each set of variables is added to the previous model. We include the maternal/ infant health and socio-economic covariates as a single step because the maternal/infant health indicators are socially patterned, and maternal age reflects both biomedical/health and socio-economic processes (Goisis et al., 2018). Other research using these cohort studies has shown that the association between maternal age and child cognitive outcomes has reversed over time (Goisis et al., 2017), indicating that in the most recent cohort older maternal age is an indicator of social advantage. Since the relationship may differ at the tails of the distribution compared to the average, we also ran the final model specification (M4) using binary logistic regression for an indicator of BMI classified as 'overweight' as the outcome (sex and age adjusted equivalent to a BMI>25 in adults).

Finally, as an additional robustness check, focusing on the three cohorts with adult BMI data we explore whether the highest recorded BMI differs by only child status. Prior research has identified that maximum or peak BMI is a more accurate predictor of mortality than contemporary BMI among older adults (C. Chen et al., 2019; Stokes,

2014). All analyses were run using Stata 16.

3. Results

3.1. Change with age and over time

We begin with the growth curve analysis to address the first aim of describing how the BMI of only children and siblings develop with age, comparing whether these patterns have changed over time. Fig. 1 shows BMI trajectories for only children and those with siblings for the four cohorts, adjusting for the cohort member's sex and whether weight was measured or self-reported (for full model results see Online Table 6).

As Fig. 1 shows, the solid curve for only children lies fairly consistently above the dashed curve of cohort members with siblings across most of the ages in all cohorts. There are also notable similarities across the cohorts in the shape of the curves and the relationship between sibling status and BMI by age. The magnitude of the difference between only children and those with siblings fluctuates with age and confidence intervals overlap both in early childhood and throughout adulthood. However, a small gap is apparent during school age (95% confidence intervals do not overlap in any of the cohorts).

In the two studies with pre-school aged measures of BMI (1946 age 2; 2001 age 3), average BMI is similar among only children and children with siblings and confidence intervals overlap. In both cohorts the BMI for both groups declines until early primary school age when BMI starts to rise again and a small gap emerges between only children and siblings, persisting through adolescence. Consistent with this pattern, in the 1958 and 1970 cohort studies, which recorded the first measure of BMI during primary school (7 and 10 years), we find a difference in BMI by sibling status at the initial measure. In all four cohorts the gap shrinks with age as the BMI of cohort members with siblings rises more steeply in late adolescence. Thus, while only children are slightly heavier on average for their height at earlier ages, cohort members with siblings 'catch up' by early adulthood.

The gap in BMI by sibling status in childhood (during school age) thus persists across the four cohorts but the magnitude of the difference appears to be small. Model predictions can assist with substantive interpretation. For example, from these models the differences between only children and those with siblings in the mean predicted BMI at age 10/11 are 0.73, 0.69, 0.40, and 0.79 points for people born in 1946, 1958, 1970 and 2001, respectively. At median height at this age, this translates to only children in the respective cohorts being 1.43 kg, 1.35 kg, 787 g and 1.54 kg heavier than their peers with siblings. Later in adolescence the differences in mean predicted BMI are equivalent to a weight difference at (sex and cohort-specific) median height of 957 g at age 15 among those born 1946, 592 g and 641 g at age 16 among those born in 1958 and 1970, and 1.22 kg at age 17 among those born in 2001.

Among those born in 1946, 1958 and 1970, the BMI curve for only children appears slightly above siblings at various ages. However, confidence intervals overlap throughout adulthood for all three cohorts.

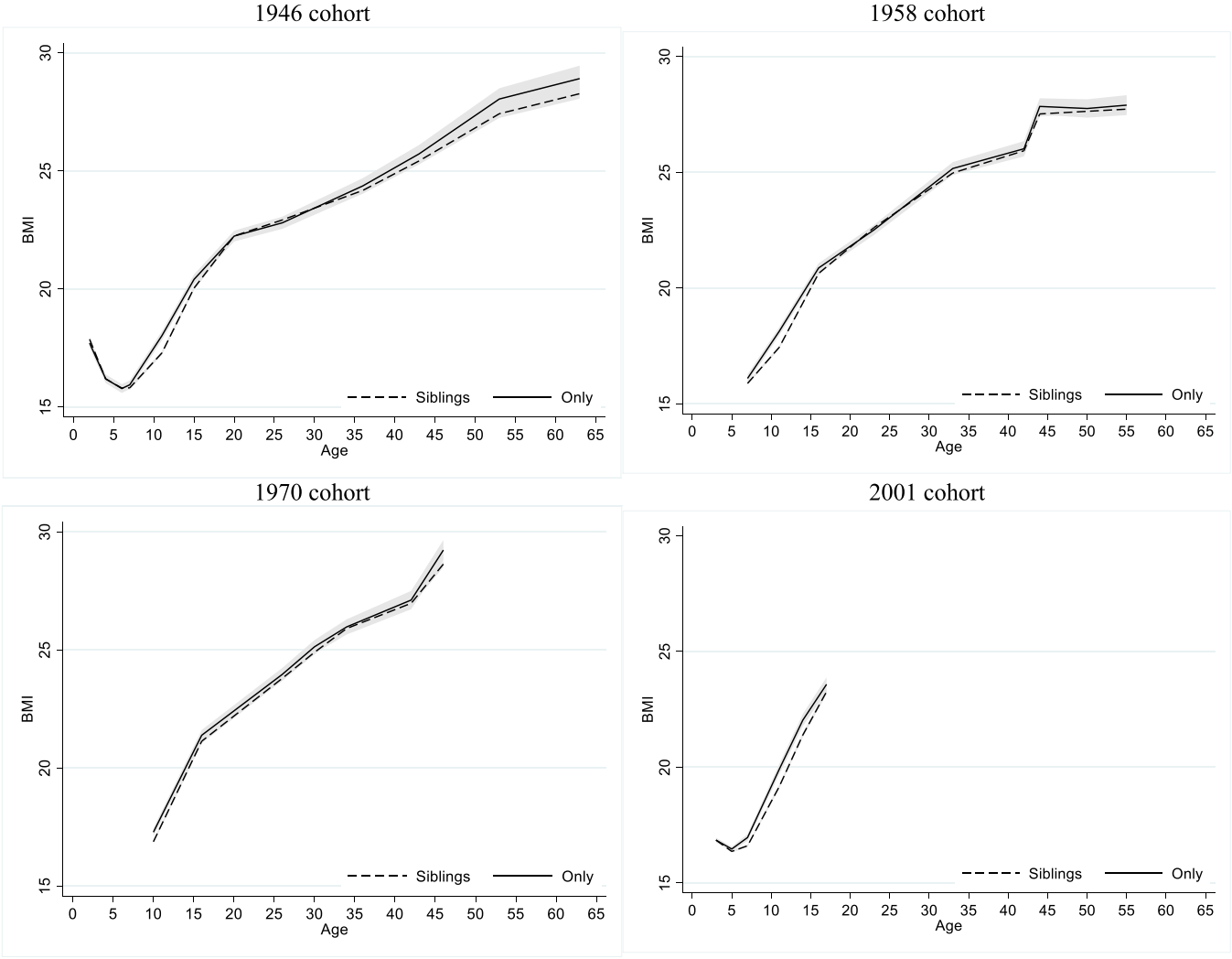

Fig. 1. Predicted mean BMI trajectories by cohort. Note: Models control for whether weight was self-reported or measured and cohort member's sex.

Towards the end of the age range observed in the 1970 and the 1946 cohorts the gap between only children and siblings appears to be growing, but the wide confidence intervals at these ages suggest this may not be reliable evidence of an apparent re-emergence of a gap by sibling status.

Including birth order and covariates in the model did not affect the shape of the trajectories nor fully explain the gap between only children and siblings during school age in any of the cohorts (Online Table 6). Second-borns in the 1946 and 1970 cohorts had slightly higher BMI than first-borns but those born third or later did not differ significantly from firstborns in these cohorts, and birth order was unrelated to BMI in the other cohorts. Including parental BMI (measured during the cohort member's childhood) also did not explain the difference by sibling status (see Online Fig. 1). As a robustness check, we repeated the models discussed above on multiply imputed data for the longer running cohorts (1946, 1958 and 1970); the results did not differ substantively (see Online Table 7).

Additional analyses suggested that the BMI differences observed in childhood remained in all four cohorts when restricting the sample to firstborns. In these analyses we compared only children with cohort members who were firstborns who subsequently had younger siblings, disaggregating the latter by the number of younger siblings (1, 2 or 3 or more). In all cohorts, the only child group remained distinct from firstborns with younger siblings regardless of the number of siblings, although in the 1958 cohort firstborns with one younger sibling also had slightly higher BMI in childhood than those with more younger siblings (but lower than only children). In these models although the gap between the lines appears similar confidence intervals overlapped in childhood for all but the youngest cohort (2001) when including covariates (see Online Fig. 2). We reiterate the point made above that even when confidence intervals do not overlap the differences observed are substantively small.

Overlaying the curves (Fig. 2) illustrates the average increase in BMI across the cohorts, with later-born cohorts reaching higher average BMI at earlier ages. While the leftward shifts between the 1946, 1958 and 1970 cohorts are noticeable in adulthood, in the 2001 birth cohort BMI was noticeably higher already in middle childhood and adolescence. This pattern is consistent with the secular trend of increasing weight in recent decades (Moody, 2019; Ng et al., 2014). Yet the association between sibling status and BMI does not appear to have changed with this general increase in BMI over time.

In sum, we found that the difference in BMI, although substantively small, is evident during primary school age in all four cohorts. However, as the BMI of individuals with siblings grows more steeply through adolescence the difference in average BMI is no longer evident during adulthood, in any of the cohorts. The pattern may thus be described as consistent with the 'age as leveller' hypothesis.

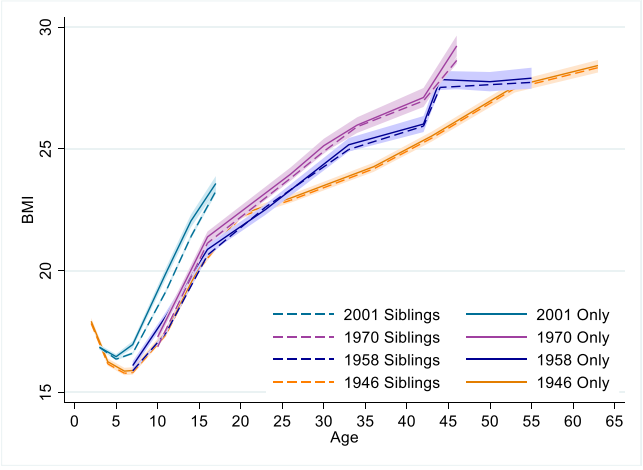

Fig. 2. Cohort differences in predicted mean BMI trajectories. Note: Models control for whether weight was self-reported or measured and cohort member's sex.

3.2. Potential mechanisms

The consistency of the finding of only children's higher BMI, across cohorts as shown above and across contexts as reported in the wider literature, suggests a need to better understand the potential mechanisms underlying the differences observed during childhood (albeit substantively small at least in the UK context). To address the second aim, we focus on age 10/11 for the three latest cohorts which allow us to analyse whether differential health behaviours and/or background characteristics may either contribute to explaining the difference observed or function as compensating factors that partly mask the association of only child status and BMI. The health behaviours available differed across the studies and ages, but can be categorised as physical activity, inactivity (such as watching TV or playing video games), and diet (such as drinking soft drinks or eating fruit). Descriptive analyses indicate that some of these health behaviours differ by sibling status and/or are associated with BMI, although differences are small (Online Table 8). The maternal/infant health and socio-economic covariates are the same as used in the longitudinal analysis (listed in Online Table 3).

Table 1 summarises the coefficients for only children (reference category: siblings) from the separate models for each cohort and age (full model results in Online Table 9). The headline finding is that the association between sibling status and BMI is not explained by differential health behaviours or early life and family characteristics or circumstances. Comparing the coefficient for only child in Model 1 with Model 2 shows that in every cohort the magnitude of the coefficient is minimally or not at all affected when health behaviours are added to the model.

When including the maternal/infant health and socio-economic covariates (Model 3) the difference in average BMI between only children and children with siblings remains but is partially attenuated in every cohort at age 10/11. Comparing Models 2 and 3, the coefficient for only child is reduced from 0.83 to 0.62 in the 1958 cohort, from 0.45 to 0.40 in the 1970 cohort and from 0.97 to 0.82 in the 2001 cohort. In percentage terms, the reduction in the only child coefficients is 25%, 11% and 19% in the 1958, 1970 and 2001 cohorts respectively, when adjusting for maternal/infant health and socio-economic differences between only children and those with siblings. Additional analysis suggested that in the 1958 and 1970 cohorts the attenuation observed is driven by the inclusion of maternal age, while in the most recent cohort the attenuation is most notable when both maternal age and family social class are included. Adding only maternal age to Model 1 reduced the only child coefficient to 0.66 in the 1958 cohort and to 0.42 in the 1970 cohort but increased the only child coefficient slightly to 1.04 in the 2001 cohort, indicating that in 2001 the maternal age profile of only children somewhat compensates for whatever the underlying reason is for their higher observed BMI. This increase in the only child coefficient is of a similar magnitude to the reduction in the coefficient when instead adding to Model 1, each individually, maternal smoking, caesarean birth or maternal education, and smaller than the reduction observed when adding parental social class, parental separation or parental BMI. Thus, the overall effect is to attenuate the association between only child status and BMI in the 2001 cohort when maternal/infant health and/or socio-economic variables are included. No variable included in the modelling emerged as a consistent potential compensating factor across all three cohorts.

Including parental BMI (Model 4) further attenuated the difference between the two groups at age 10/11 only slightly in the 2001 cohort and did not fully explain it in any cohort. These results are consistent with the growth curve analysis and indicates that the small difference in average BMI by only child status cannot be fully explained by differences in the observable characteristics of only child families and those with

Table 1
Linear Regression summary table: BMI at age 10/11.

	1958	Age 11		1970	Age 10		2001	Age 11	
	Coeff.	se	p	Coeff.	se	p	Coeff.	se	p
M1: Ind. chars	0.83	0.10	0.00	0.45	0.08	0.00	1.01	0.17	0.00
M2: + Health	0.83	0.10	0.00	0.45	0.08	0.00	0.97	0.17	0.00
M3: + Covariates	0.62	0.11	0.00	0.40	0.08	0.00	0.82	0.17	0.00
M4: + Par. BMI	0.61	0.10	0.00	0.41	0.08	0.00	0.77	0.17	0.00
n	10,651			11,181			12,781		

Notes: Analysis run on 20 imputed datasets. Model 1 (M1) includes cohort member's sex, birth order and ethnic group (2001). Model 2 (M2) adds health behaviours. Model 3 (M3) adds all maternal/infant health and socio-economic covariates previously listed. Model 4 (M4) additionally includes parental BMI measured when the cohort member was aged 10/11 (1970 and 1958) or age 7 (2001).

two or more children.

We repeated the age 10/11 fully adjusted model restricting the sample to firstborns (Online Table 10). The results showed more clearly and consistently than the growth curve analysis that the difference in BMI between only children and firstborns with younger siblings increased with sibship size in all three cohorts. Since the association may differ at the tails of the distribution compared with the average, we also ran the final specifications of the models above (Model 4) using logistic regression on a binary indicator of BMI categorised as 'overweight'. In each of the cohorts only children had higher odds of having BMI classified above this threshold at age 10/11, controlling for health behaviours, individual and family characteristics and parental BMI (Online Table 11). In line with the findings from the growth curve analysis showing the convergence of trajectories by early adulthood, analysis for age 16/17 showed less consistent evidence across the cohorts of a difference by sibling status, both in average BMI and in terms of probability of having BMI classified as 'overweight' (Online Tables 9 and 11).

3.3. Additional analyses

As an additional robustness check and to explore whether there may be any ongoing knock-on effects for only children's BMI in adulthood, as the critical period framework might predict, we analyse the maximum recorded BMI in the three cohorts for which we have adult measures. The maximum BMI (on average around 29–30 in each cohort) was recorded during adulthood in about 98% of cases in each cohort. There was no evidence of a difference by only child status in maximum recorded BMI in the 1958 cohort, while on average it was about half a point higher among only children in the 1946 and 1970 cohorts (Online Table 12). Although these differences are statistically significant at the 10% and 5% levels respectively, and fully explained by childhood BMI recorded at age 10/11 which could indicate possible longer-term implications of the observed childhood BMI gap, a half-point difference seems unlikely to be clinically important.

Finally, we conducted additional analysis directed at providing further evidence for the above conclusion that the small and age-specific difference we found in BMI, even if unexplained, is unlikely to be of concern. Higher childhood BMI can be associated with an earlier onset of puberty and menstruation, which has been linked to poorer mental health in adolescent girls and higher risk of adverse later physical health outcomes including cancer, diabetes and cardiovascular disease (Gong et al., 2015; J.J. Lee et al., 2019; Lienet al., 2010; Mendle et al., 2018; Pandeya et al., 2018; Parsons et al., 1999; Peters & Woodward, 2018). Focusing on age at menarche, which is consistently reported across the datasets, we found that girls who were only children experienced menarche on average 2–4 months earlier than those with siblings, depending on the cohort (although the association is not statistically significant in 2001 cohort; see Online Table 13). 'Early' menarche is often operationalised as before age 10 (i.e. 2 + *years* earlier than average) in research finding an association with adverse outcomes, and the difference we see in mean age at menarche by sibling status is therefore reassuringly small. Thus, consistent with the small difference observed in BMI, in the UK context this does not then appear to be a pathway by which only children might experience worse health outcomes.

4. Discussion

In this paper, we investigated BMI of only children and those with siblings across four British birth cohorts. The difference we found during school age is consistent with evidence from other contexts finding higher BMI among only children (Meller et al., 2018), and our analysis extends existing research by showing that this is also consistent across cohorts. This persistence across cohorts suggests that the association between sibling status and BMI has not changed in the context of secular increase in BMI (e.g. Jebb et al., 2013; Moody, 2019; Ng et al., 2014) and possible changes in the characteristics of only children in the UK. Further, our analysis also extends the existing literature by showing how the difference in BMI by sibling status develops with age. We observed that in the UK context the difference in average BMI that emerges during primary school age disappears again in late adolescence and early adulthood, consistent with an 'age as leveller' pattern. This finding contrasts with evidence from the US suggesting a persistence in only children's higher BMI into adulthood (Lin & Falbo, 2022).

As for the mechanism underlying this association, our analyses have not been able to settle this issue. The difference by sibling status in BMI during school age, although somewhat attenuated by inclusion of covariates, cannot be fully explained by health behaviours or early life individual and family characteristics. Our findings are similar to those from the USA, also indicative that controlling for lone parenthood and family socio-economic status attenuates but does not fully explain the association between sibship size and BMI (Datar, 2017), but differ from the Danish study where the inclusion of covariates (including maternal age at birth, maternal BMI and paternal social class) did not attenuate only children's higher odds of having BMI classified as 'obese' (not using IOTF thresholds; Haugaard et al., 2013). Given the detailed information available in our data, including aspects relating to maternal pregnancy history, antenatal and neonatal measures and parental BMI, our results may suggest that the remaining difference between only children and siblings may be more behavioural. Although we also included children's own physical activity, inactivity and diet, these health behaviours were measured quite crudely which may mean that this potential mechanism cannot be ruled out as an explanation.

Taking the results from both sets of analyses together, we can conclude that similar to studies conducted in other contexts only children in the UK have higher BMI than their peers with siblings during childhood/adolescence, that this pattern is consistent across cohorts and it is not explained by differential family background characteristics or childhood health behaviours. On the one hand, to the extent that higher BMI is linked to adverse health outcomes, we may conclude that, similar to other geographic contexts, only children in Britain may be at a health disadvantage relative to siblings (whereas on other outcomes only children in the UK tend to do as well as, or better than, individuals with siblings; e.g. Choi & Monden, 2019; Laybourn, 1990). There is in fact

evidence from Sweden showing that individuals without siblings have higher all-cause mortality from middle adulthood (Keenan et al., 2022), as well as elevated mortality attributable to diseases of the circulatory system (Baranowska-Rataj et al., 2017). On the other hand, our analysis also showed that the BMI trajectories of only children and siblings converge by early adulthood, that the gap in childhood is reassuringly small, and that there may not be any ongoing knock-on effects for only children in adulthood in the UK context. Since past research shows that the characteristics of one-child families differ cross-nationally, with implications for child outcomes observed for the group, our findings may be context-specific. Moreover, given that much of the research from different contexts has analysed the odds of having BMI classified as 'overweight' or 'obese' with the inclusion of different set of covariates, it is difficult to get a sense of the substantive magnitude of the statistically significant difference reported elsewhere. Nevertheless, we can compare our results to a few studies which have analysed BMI as a continuous measure. Our results showed that at age 11 only children's BMI was on average 0.83 points higher among those born in 1958, controlling for sex and birth order, which was reduced to 0.61 when including all covariates and parental BMI. In the 1970 and 2001 cohorts at age 10/11 the BMI of only children was on average 0.45 and 1.01 points higher in the first model, reducing to 0.41 and 0.77 respectively when controlling for all other variables. One US study found on average approximately of 0.8 points higher BMI among only children in fifth grade (also corresponding to approximately age 10–11), compared with children from large families (3 siblings or more; A. Chen & Escarce, 2010, adjusted for family characteristics). A repeated cross-sectional study of school-aged children in China (6–18 year olds) found only children's BMI was 0.43 points higher in 2000 and 1.32 points higher in 2011 (Min et al., 2017, adjusted for parental weight status and other family covariates). Our findings are thus broadly in line with these previous studies. As BMI is not itself an adverse outcome but a marker of potential increased risk of health problems, it is important to convey the substantive magnitude of statistically significant differences between groups, which also helps when comparing findings across contexts and time periods.

Findings on only children's BMI thus need to be viewed in perspective also in relation to the limitations of the measure itself and reflecting on the social context within which BMI research is carried out. BMI is an imperfect measure of adiposity, with documented issues relating to the conventional use of universal thresholds despite individual health and morbidity risk at a given BMI being affected by age, sex, ethnic group, relative lean muscle and body fat levels, type of diet and level of physical activity (e.g. Jackson et al., 2002; Rahman & Berenson, 2010; Rey-López et al., 2014; W.H.O, 1998). The scientific, medical and media discourse of 'obesity' as an inherently adverse outcome that is increasingly individualised in terms of cause and responsibility can be stigmatizing and harmful to people's mental health and self-image (Baker et al., 2020; Gage & Patalay, 2021; Greenhalgh, 2012; Solmi et al., 2021). Analysis of BMI as the outcome variable is thus not neutral but risks feeding into this contemporary culture of 'fat talk' (Greenhalgh, 2012). Although we have throughout focused on BMI as an indicator of potential health risks, establishing whether the BMI trajectories observed are in turn associated with differential health outcomes for only children and those with siblings is beyond the scope of this paper. This would be a useful direction for future research because there is limited direct evidence of only children's health outcomes, at different stages in the life course, across geographical contexts.

Our findings suggesting that the difference in BMI between only children and siblings are substantively small and that, consistent with an 'age as leveller' pattern, trajectories converge by early adulthood may raise the question of whether this difference and as yet unexplained underlying mechanism continue to be of substantive research or clinical concern in the UK context. The difference observed during childhood and the underlying mechanism should not matter unless only children are at higher risk of disease attributable to their childhood BMI (which is only slightly higher, at least in the UK). Yet viewed alongside evidence from Sweden of higher mortality for only children, our findings draw attention to the relevance of cross-national variation in the selection into being an only child. Nevertheless, given the limitations of the BMI measure, our suggestion to shift attention to more direct measures of only children's health is arguably also applicable beyond the UK context.

Ethics approval and consent to participate

Ethics approval was not required for this project as the data used had been anonymised by the survey teams prior to being made available for analysis.

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Conflict of interest

The authors declare no conflicts of interest.

Data Availability

The data that support the findings of this study are available to registered users from the UK Data Service (https://ukdataservice.ac.uk/) with End User Licence, and from MRC Unit for Lifelong Health and Ageing at UCL (LHA; https://www.nshd.mrc.ac.uk/) with the permission of LHA.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.alcr.2022.100493.

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