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Family size effects on childhood obesity: Evidence on the quantity-quality trade-off using the NLSY

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ABSTRACT

In this study, we use matched mother-child data from the National Longitudinal Surveys to study the effects of family size on child health. Focusing on excess body weight indicators as children's health outcome of interest, we examine the effects of exogenous variations in family size generated by twin births and parental preference for mixed sex composition of their children. We find no significant empirical support in favor of the quantity-quality trade-off theory in instrumental variable regression analysis. This result is further substantiated when we make use of the panel aspects of the data to study child health outcomes of arrival of younger siblings at later parities. Specifically, when we employ child fixed effects analysis, results suggest that birth of a younger sibling is related to a decline in the likelihood of being overweight by 4 percentage points and a drop in the probability of illness by approximately 5 percentage points.

1. Introduction

The primary objective of this study is to investigate the effects of family size on child health. Family size effects on children's human capital outcomes have been studied extensively in the economic literature following the quantity-quality trade-off theory developed by Becker and his co-authors (Becker, 1960; Becker and Lewis, 1973; Willis, 1973; Becker and Tomes, 1976). The theory predicts a negative relationship between child quantity and quality as individual allocation of fixed parental resources declines with additional children. In this study, we present a comprehensive empirical analysis of how a change in family size affects child health, as measured by excess body weight indicators (overweight and obesity). To the best of our knowledge, this is the first paper to utilize a US-based sample to evaluate child health outcomes of variations in family size.

Empirical literature on family size effects tends to specifically focus on children's educational outcomes, future labor market performance, and parental investments in childhood development (Rosenzweig and Wolpin, 1980; Blake, 1981; Hanushek, 1992; Black et al., 2005, 2010; Cáceres-Delpiano, 2006; Lee, 2008; Li et al., 2008; Angrist et al., 2010; De Haan, 2010; Ponczek and Souza, 2012; Juhn et al., 2015). Despite the extensive empirical support provided by the majority of existing studies, validity of the quantity-quality trade-off argument is likely to vary by social structure and extent of economic development of the regions studied (Sudha, 1997; Buchmann and Hannum, 2001; Li et al., 2008; Maralani, 2008). For example, generous welfare and institutional support systems, commonly observed in economically advanced countries, can mitigate parents' financial burden imposed by an expansion in family size. On the other hand, families in economically disadvantaged societies are more likely to observe a trade-off between child quantity and quality (Li et al., 2008; Maralani, 2008).

Compared to children's cognitive abilities, child health, an important indicator of child quality, has not received much research attention in the family size literature. Furthermore, the limited number of studies that have explored a causal link between family size and child health are primarily based on developing regions (Rosenzweig and Zhang, 2009; Millimet and Wang, 2011; Peters et al., 2014; Zhong, 2017). Our analysis therefore fills a substantial gap in the existing literature by empirically testing the

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quantity-quality trade-off theory with respect to child health in the context of a developed economy.

Child body weight is an important health indicator. In the US in particular, childhood obesity is a large health-related concern, as excess body weight during childhood increases the risk of being exposed to serious long-term health conditions (Lobstein et al., 2004; Leonard et al., 2008; Reilly and Kelly, 2011; Fryar and Ogden, 2014; Ogden et al., 2016). This has prompted researchers across various academic disciplines to identify social and family-level determinants of childhood obesity and to analyze effectiveness of social policies aimed at addressing the health concern (Dietz, 1998; Cawley et al., 2007a, 2007b; Clark et al., 2007; Cawley, 2010). Based on the quantity-quality trade-off hypothesis, an increase in family size could potentially elevate the risk of having excess weight among children. Given the numerous health-related problems associated with childhood obesity, it is important to understand the validity of the quantity-quality hypothesis in the context of a developed country like the US.

For our analysis, we match mothers' information from the original cohort of the National Longitudinal Survey of Youth (NLSY, 1979) to their children's data in the NLSY Child and Young Adult surveys (NLSY, CYA). We estimate children's body mass index (BMI) using objective measurements of children's height and weight. Using these estimates of BMI, we construct excess weight indicators. In particular, we refer to the age- and sex-specific body weight standards (BMI-for-age) of Centers for Disease Control's (CDC) growth charts[1] to construct binary indicators for being overweight and obese. Further, we estimate the effects of family size on binary health indicators of underweight and incidence of child illness (that requires medical attention).

The identification of family size effects relies on exogenous variation in quantity of children triggered by twin births and parental preference for a mixed sex composition of their children. Regression estimates from our instrumental variable (IV) regressions do not provide evidence of a trade-off between child quantity and health quality. Our cross-sectional regression estimates are robust to multiple sensitivity checks.

In addition, we make use of the longitudinal format of the NLSY mother-child data to study how birth of a younger sibling affects older children's health outcomes. Controlling for unobserved child-specific time-invariant characteristics, we find that birth of a younger sibling leads to a decrease in children's probability of being overweight by 4 percentage points and a decrease in the likelihood of being ill by 5 percentage points. In general, our fixed effects regressions provide additional support to the findings obtained from the IV regression analyses.

The paper is organized as follows: in Section 2, we review existing theories and empirical findings in related literature to explore the underlying mechanisms that can explain a causal link between family size and child health. In Section 3, we describe the NLSY mother-child data used for our empirical analysis. In Section 4, we discuss the empirical strategies employed in our analysis. In Section 5, we present our findings from cross-sectional and longitudinal regression analyses. Finally, in Section 6, we provide brief concluding remarks.

2. Literature review

2.1. Child quantity and health quality trade-off – a theoretical framework

The quantity-quality trade-off theory postulates that given a budget constraint, an increase in the number of children increases the marginal cost of family investments, which influence child quality (Becker, 1960; Becker and Lewis, 1973; Becker and Tomes, 1976). This is because having additional children requires parental resources to be allocated across a larger number of siblings. In this context, an increase in the quantity of children is expected to adversely affect each child's physical well-being, assuming child health depends on limited parental resources (e.g. market-based goods including food, nutrition, and medical inputs and parental time).

Millimet and Wang (2011) present a simplified extension of Becker & Tomes's (1976) quantity-quality trade-off model by considering health-related resources and health endowment as inputs of child quality. Households' objective is to maximize their utility U given by the function $U = U(n, q, c)$, where n represents child quantity, q represents child quality, and c is consumption. Further, child quality q is a function of market-based health inputs w and child's health endowment θ. In particular, child quality is represented by the production function: $q = q(w, \theta)$, where q is positively related to both w and θ ($q_w > 0$ and $q_\theta > 0$).

Households maximize their utility subject to a budget constraint given by[2]:

$$c.p_c + n.p_n + wn.p_w = I \tag{1}$$

where I denotes household income, p_c is the unit price of consumption, p_n is the cost per child, and p_w represents price of market-purchased health inputs.

The equilibrium condition is:

$$\frac{\partial U}{\partial c} = \lambda p_c = \lambda \pi_c$$

$$\frac{\partial U}{\partial q} = \lambda \frac{p_w}{\partial q / \partial w} n = \lambda \pi_q \tag{2}$$

$$\frac{\partial U}{\partial n} = \lambda (w p_w + p_n) = \lambda \pi_n$$

In the above equations, π_c, π_q, and π_n are the shadow prices of consumption, child quality, and child quantity, respectively.[3] The equilibrium condition suggests that an unplanned or exogenous increase in the number of children increases the shadow price of child quality, and a rise in child quality increases the shadow price of child quantity. In other words, it is costlier for parents to improve child health quality if there are a large number of children in the household, and it is costlier to have additional children if child quality is high.

Additionally, effects of family size on child health outcomes may depend on children's birth spacing and birth order. In particular, closely-spaced younger siblings tend to involve higher parental engagement, thereby subjecting older children to reduced

[1] See https://www.cdc.gov/growthcharts/html_charts/bmiagerev.htm; Retrieved on November, 12, 2016.

[2] Millimet and Wang (2011) also include children's sex ratio in their model assuming that having more children belonging to the same sex can provide certain cost advantages to households (discussed later). However, to provide a basic understanding of the quantity-quality trade-off, it is not required to account for sex ratio in the main model.

[3] λ in Eq. (2) represents the Lagrange multiplier of the optimization problem.

parental attention (Zajonc, 1976; Black et al., 2005; Angrist et al., 2010; De Haan, 2010). Moreover, smaller childbearing intervals can have adverse impacts on maternal health and pregnancy outcomes (Dewey and Cohen, 2007; Conde-Agudelo et al., 2012a, 2012b). Not surprisingly, empirical evidence provided in related literature indicates that siblings' birth interval is positively related to child health and cognitive abilities (Huttly et al., 1992; Dewey and Cohen, 2007; Buckles and Munnich, 2012; Conde-Agudelo et al., 2012a, 2012b).

In addition, many previous studies stress the importance of birth order and its role in influencing child outcomes (Hanushek, 1992; Black et al., 2005; Price, 2008). However, there is some level of ambiguity in the existing literature regarding the impact of birth order on children's well-being and development. While a few studies hypothesize that later-born children might benefit from parents' prior child-rearing experiences and increased life cycle earnings, the majority of empirical studies argue that children born earlier benefit more from a greater share of parental attention (Hanushek, 1992; Price, 2008; Booth and Kee, 2009; De Haan, 2010).

2.2. Role of family in childhood obesity

Extreme body weight conditions during childhood can have major health implications. Excess body weight increases the risk of long-term health conditions including diabetes, heart disorders, hypertension, and cancer (Must et al., 1992; Power et al., 1997; Lobstein et al., 2004; Leonard et al., 2008; Reilly and Kelly, 2011). Conversely, stunted growth during childhood, primarily resulting from malnourishment and undernutrition, can elevate the risk of mortality (by weakening immunity) and affect children's cognitive development (Walker et al., 2000; De Onis et al., 2004; Victora et al., 2008).

It is important to recognize that the prevalence of children being underweight or obese varies markedly across countries. In general, the likelihood of children being underweight is relatively more common in the developing economies of Asia and Africa, whereas the proportions of children with excess weight are much higher in industrialized regions (Wang et al., 2002a, 2002b; Bloss et al., 2004; De Onis et al., 2004, Janssen et al., 2005; Wang and Lobstein, 2006; Wang and Lim, 2012; De Onis et al., 2010). Consistent with the international evidence, recent US-based estimates show that American children are more likely to suffer from obesity than they are from being underweight.[4]

In developed economies, one of the important factors to consider is family's socio-economic status, which appears to have a strong inverse relationship with risk of childhood obesity (Baum and Ruhm, 2009; Murasko, 2009; Wang and Lim, 2012; Alviola et al., 2014; Walsh and Cullinan, 2015; Antelo et al., 2017). In particular, incidence of childhood obesity is found to be greater in families that are characterized by lower levels of household income and parental education (Baum and Ruhm, 2009; Antelo et al., 2017). Such high prevalence of obesity in economically disadvantaged families is likely due to food insecurity, sedentary lifestyle, and unhealthy dietary practices, e.g. consumption of high-caloric fast foods and beverages (Smoyer-Tomic et al., 2008; Drewnowski, 2009; Murasko, 2009; Walsh and Cullinan, 2015; Antelo et al., 2017). In addition, maternal employment may also increase child risk of having excess weight (Anderson et al., 2003; Bauer et al., 2012). A number of studies have found a positive association between mother's labor market involvement and childhood obesity, postulating that working longer hours reduces parental involvement in health-promoting childcare activities such as preparing homemade meals and performing physical activities with children (Bauer et al., 2012; Cawley and Liu, 2012).

2.3. Existing evidence on child health outcomes of increase in family size

As noted earlier, the few empirical studies on effects of family size on child health outcomes have primarily focused on developing regions (such as Bangladesh, China, and Indonesia). Peters et al. (2014) examine family size effects on child health in rural Bangladesh. Using multiple health outcomes (including diarrhoea, respiratory infections, eye infections, and colds), the authors do not find any empirical evidence in support of the quantity-quality trade-off argument. On the other hand, using data on Chinese children, Rosenzweig and Zhang (2009); Zhong (2017) find a significant negative relationship between family size and child health.[5] However, in a cross-sectional survey across middle-school children in eastern China, Hesketh et al. (2003) show that children in one-child families are more likely to be overweight and tend to visit doctors and dentists more frequently due to poor health. Millimet and Wang (2011) employ data from the Indonesian Family Life Survey to study the relationship between family size and child health. The authors find mixed results for family size effects on height-for-age and BMI-for-age indicators. In particular, their results support the quantity-quality trade-off theory, but only at select quantiles of BMI distribution of the child sample used.

The only empirical evidence on a potential causal link between family size and child health in a developed economy's context is offered by Lundborg et al. (2015). Using Swedish data on the male population from 1965 to 1978 birth cohorts, the analysis indicates that an exogenous increase in family size is positively related to children's height.

Research from an alternative strand of child health literature suggests that children in larger families tend to have better health outcomes. The positive association between family size and child health is in line with the hygiene hypothesis proposed by Strachan (1989). The hygiene hypothesis argues that compared to smaller families, children in large families develop stronger immune system as they are more likely to be exposed to various diseases during the early years of their childhood. A number of studies in child health research have corroborated the hygiene hypothesis, showing that children in large families are less likely to have excess body weight (Edwards and Grossman, 1979; Kruger et al., 2006). Moreover, some studies indicate that children in large families are also less prone to suffer from diseases and medical conditions, such as diarrhoea, respiratory infection, hay fever, asthma, and allergy (Strachan, 1989, 1997; Rona et al., 1997; Ponsonby et al., 1999; Jensen and Ahlburg, 2002).

The above empirical evidence indicates that it is possible that the combination of health benefits of a large sibship size (such as development of immunity) and the presence of a generous social safety net could offset the negative health impacts of resource constraints imposed by a rise in family size. Furthermore, a higher level of sibling interactions in large families may promote children's physical activities, and thereby reduce the risk of childhood obesity (Hallal et al., 2006; Hohepa et al., 2007).

Finally, studies by Angrist and Evans (1998); Cáceres-Delpiano

[4] National Health and Nutrition Survey's recent estimates suggest that prevalence of childhood obesity in the US was 17% in 2011–2012 and prevalence of childhood underweight was 3.5% in 2011–2012 (Fryar & Ogden 2014; Ogden et al., 2016)

[5] Both studies utilize China's one-child policy to construct an IV for exogenous variations in family size. Health outcomes in the studies are measured by children's self-assessed health, BMI measures, and height-for-age Z-scores.

(2006) predict that exogenous increases in family size reduce mothers' labor market activities (labor supply and labor force participation). Although their results tend to provide empirical support in favor of the quantity-quality trade-off theory, a mother's decision to reduce her labor market participation may result from her intention to take an active role in childcare. Therefore, the existing research evidence (discussed in the previous section) of a positive association between maternal employment and childhood obesity may implicitly point towards the presence of an inverse relationship between family size and the risk of childhood obesity.

2.4. Exogenous variation in family size

Family size is endogenously determined by parents. Therefore, family size is likely to be related to unobserved parental characteristics (such as parental ability) that may affect child outcomes. In order to isolate the true causal effect of family size, empirical analyses have extensively relied on twin births and parental preference for a mixed sex composition of their children as sources of exogenous variation in family size (some important studies include Angrist et al., 2010; Black et al., 2005; Black et al., 2010; De Haan, 2010).

Rosenzweig and Wolpin (1980) are the first authors to use twin births as a natural experiment for an exogenous increase in family size. Angrist and Evans (1998) considered sex composition of children born earlier in a family as an important determining factor for sibling size. They argued that parents with children of the same sex are more likely to have an additional child in order to have a mixed sex-composition of their children. An important consideration for the validity of their instrumental variable is that children's sex is randomly determined.

However, it is important to note, that the local average treatment effects (LATE) of family size may vary by the nature of interventions generated by the two IV's (Black et al., 2010). In families where the first two children are of the same sex, the decision to have an additional child varies by parental preference for variety in the sex composition of their children. On the other hand, twin births are usually unplanned in nature and involve almost perfect compliance with respect to increase in family size (Angrist et al., 2010; Black et al., 2010). However, estimation of family size effects using multiple sources of exogenous variation allows researchers to compare and ensure robustness of their findings across various model specifications (Angrist et al., 2010; Black et al., 2010; De Haan, 2010).

Recent empirical literature has discussed some possibilities that demonstrate how both twin births and sibling sex compositions are likely to be correlated with unobserved heterogeneities that can affect child outcomes (Rosenzweig and Zhang, 2009; Black et al., 2010; Angrist et al., 2010; De Haan, 2010). For example, following from the evidence on potential impacts of children's birth spacing, twin births can adversely affect older siblings' health outcomes (see Angrist et al., 2010; Black et al., 2010). On the other hand, children's sex composition may involve economies of scale in child-rearing process such as sharing of room, clothes, and other resources among same sex siblings (Rosenzweig and Zhang, 2009; Angrist et al., 2010). To test the legitimacy of these concerns in our analysis, we perform additional robustness checks similar to methods adopted by Black et al. (2010); De Haan (2010).

3. Data: the NLSY sample

We match mothers' data from the original NLSY 79 cohort with their children from the NLSY CYA. Children in NLSY CYA are surveyed biennially from 1986 (ongoing). We apply a few conditions to the matched mother-child data to obtain a relevant sample for the purpose of our empirical analyses. First, we restrict the NLSY data to families with at least two children (Black et al., 2005; Angrist et al., 2010). Second, since our objective is to analyze effects of family size on children's health, we limit our sample to children aged 2–17.

Further, children who grow up in households with both biological parents tend to exhibit better cognitive, behavioral, and health outcomes than children in non-traditional households, e.g. single parent or step-parent households (Amato Paul and Keith, 1991; Brown, 2004; Brown et al., 2015). In this regard, variations in family structure may have a confounding influence on the estimated relationship between family size and child health. To address this empirical concern, our study focuses on children who reside with both their parents.[6,7] This results in a sample in which, for approximately 95% of the observations, mothers are reported to be married. Although, we do not control for mothers' marital status (due to lack of variation), inclusion of their marital information does not affect our regression analysis.

Finally, height and weight information reported by children and their mothers in NLSY CYA may suffer from reporting errors (Gunnell et al., 2000; Cawley et al., 2015). For example, while overweight people may understate their true weight, smaller people are likely to over-report their actual height and/or weight. Therefore, in order to ensure precision of our child health indicators, we restrict our regression sample to objective measurements (scale and tape-measured information) rather than relying on self-reported data.

The above steps result in a sample of 7342 observations, which includes 3052 children from 1540 families (full sample). For our IV regression analyses, the full sample is subjected to further modifications depending on the nature of the IV employed in our estimated regressions. We discuss these modifications in the next section.

We provide descriptive information of all the variables used in our analysis in Table 1. Using estimated BMI (computed from NLSY CYA height and weight information), we construct children's body weight indicators. Based on the age- and sex-specific BMI-for-age distribution from 2000 CDC Growth Charts for the United States (Kuczmarski et al., 2000), we create binary indicators for being overweight, obese, and underweight (see Cawley et al., 2010, 2013). In particular, BMI values greater than or equal to 85th and 95th percentile of the age- and sex-specific BMI distribution are classified as overweight and obese, respectively. BMI values less than or equal to 5th percentile of the distribution are classified as underweight.

In the full sample, 28% of the sample is overweight and almost half of the overweight observations can be classified as obese. Focusing on additional child health measures, while 11% of the full sample is underweight, 35% of the sample is reported (by mothers) to have suffered from illness that required medical attention in the year prior to the survey. On average, households are comprised of approximately three non-adult members in the full sample (explanatory variable of interest).

Our cross-sectional regression analyses incorporate a number of relevant child-, mother-, and family-specific characteristics.

[6] At least 73% of US children resided with their parents up to year 2000. In the NLSY CYA sample used in our analysis, 97.3% of children were born prior to the year 2000. See http://www.pewsocialtrends.org/2015/12/17/1-the-american-family-today/; Retrieved on February 22, 2017.

[7] NLSY CYA includes information on child's usual residence and whether child's father lives in her/his household. We also perform regressions using an unrestricted child sample that includes all children who reside with their mothers, while their fathers' presence in households may vary (due to various reasons such as mother's marital status or parents' occupational choices). Controlling for mother's marital status in regressions using unrestricted child sample, results are qualitatively consistent with our main findings.

Table 1
Descriptive information of regression sample from NLSY's (1986–2012).

Variables	Mean/Proportion (SD)
Child characteristics	
Body Mass Index (BMI)	18.083 (5.675)
Overweight	0.277 (0.448)
Obese	0.140 (0.347)
Underweight	0.114 (0.318)
Incidence of child illness (Illness)	0.346 (0.476)
Household Size (under 18 years)	2.712 (1.100)
Hispanic	0.199 (0.399)
White	0.613 (0.487)
Females	0.491 (0.500)
Age	8.460 (3.366)
Weight at birth (in ounces)	120.085 (23.962)
Mother Characteristics	
Highest grade completed (HGCM)	13.364 (2.641)
AFQT Scores	48466.200 (28772.720)
Rotter Scale	8.632 (2.361)
Body Mass Index	26.666 (6.036)
Age at child birth	28.057 (4.705)
Age	36.544 (4.723)
Family and birth-order characteristics	
Poverty status	0.117 (0.321)
First child	0.323 (0.467)
Second child	0.382 (0.486)
Third child	0.192 (0.394)
Fourth child	0.066 (0.248)
Fifth (or more) child	0.038 (0.191)
IV information	
Family with twins (later births)	0.004 (0.062)
First two children of same sex	0.470 (0.499)
First two children are girls	0.222 (0.416)
First two children are boys	0.247 (0.432)
Number of families (full sample)	1540
Number of children (full sample)	3052
Full sample size	7342
Twin IV regression sample size	7057
Same-sex IV sample size (first two children only)	5041

Note: The mean (standard deviation) of the variables used in regression analyses are based on mother-child sample used in full sample regression analyses. The summary statistics of the variables in our IV regression samples are similar. The IV information in the above table are with respect to respective samples used. The proportions of same-sex children (same sex, both girls, and both boys) are based on child sample of first two children of families with at least two children. The mean value of HGCM represents that on average, mothers have at least one year of college education. The IV information are based on respective IV samples used in regression analyses.

Child-specific controls include information on birth weight (in pounds), age, sex, race, and ethnicity. With respect to children's demographic characteristics, 20% belong to the Hispanic ethnicity, while 61% of our sample is White.

Following from our earlier discussion, we also control for children's birth order in our models using separate dummies for the second child, the third child, the fourth child, and for the fifth (or more) child (see Table 1). The first child is the excluded category.

We also include mothers' information on schooling (highest grade completed), Armed Forces Qualification Test raw scores (AFQT scores), and scores on Rotter's scale (locus of control measure) to control for mothers' academic achievement, cognitive ability, and non-cognitive characteristics, respectively. Additional maternal controls include mothers' age (as of survey years) and mothers' body mass index (as a measure of maternal health). On average, in the full sample, mothers tend to have childbirth at the age of 28.

Finally, since family's socio-economic status is an important determinant of children's health outcomes (see Section 2.2), we control for family's poverty status in our regression models as an indicator of socio-economic conditions.[8] Approximately 11% of the full sample is classified as poor.[9]

4. Empirical strategy

4.1. Cross-sectional analysis using instrumental variables

Our cross-sectional analyses employ IV regressions to isolate causal effects of household size[10] on child health. The IV regression model is-

$$Y_i = \beta_0 + \beta_1 \hat{HHSIZE}_i + X_i'\beta_2 + \varepsilon_i \quad (3)$$

$$HHSIZE_i = \alpha_0 + \alpha_1 Z_i + X_i'\alpha_2 + v_i \quad (4)$$

where Y_i represents child i's health outcome (BMI and binary health indicators). $HHSIZE_i$ denotes the number of non-adult members in i's household. X_i is a vector of child-, mother-, and family-specific controls related to child i. β_1 captures child health effects of changes in household size.

Eq. (4) represents first stage of our IV regression analysis, where Z_i is treated as an instrumental variable for exogenous variation in household size. As mentioned earlier, we use two sources of exogenous variation in household size: twin births and parental preference for mixed sex composition of their children.

First, utilizing the full sample, we allow for household size to be endogenously determined by unobserved family characteristics and estimate regression models similar to Eq. (3). We perform ordinary least squares regression (OLS) for BMI (continuous measure) and probit regressions for our binary health indicators.

In the regression analysis using twin births as an instrumental variable, Z_i is a binary indicator for the presence of a twin in child i's family (twin IV). The indicator is constructed by comparing survey years with twin birth years. In the twin IV regression sample, we select only non-twin children from our full sample. Further, in families with twins, we restrict our sample to children who were born prior to twin births (Black et al., 2010; Juhn et al., 2015).[11] This further limits the analysis sample to 7057 observations.

In regression models where we use sibling sex composition of a family's first two children as a source of exogenous variation in household size, Z_i is a binary indicator that equals 1 when the first two siblings in i's family are of the same sex (same-sex IV). The same-sex IV is likely to generate exogenous changes in household

[8] NLSY 79 also provides information on annual net family income. However, since the income measures are likely to be in nominal terms, we use family's poverty status as an indicator of family's economic well-being. However, further estimation of regression models by controlling for family income (or real measures of family income by using annual average CPI values in the US) provides consistent findings with our main analysis.

[9] See https://www.nlsinfo.org/content/cohorts/nlsy79/topical-guide/income/poverty-status-public-assistance-support-sources; Retrieved on August 23, 2017.

[10] For our cross-sectional regression analysis, based on the specific NLSY CYA information, we are able to look at the effects of number of household members who may not be child's immediate family. Hence, to describe our results in Section 5.1, we use the term 'household size' instead of 'family size'. The effects of family size variations are more precisely captured in section 5.3.

[11] Likelihood of multiple births may increase with number of deliveries (Rosenzweig & Wolpin 2000; Li et al. 2008). In this context, Li et al. (2008) employ binary twin indicators for having a twin birth at the nth delivery (as IV) and perform separate IV regressions on samples of families with at least n births (n = 1,2, and 3). However, the limited number of observations and lack of sufficient number of twins in NLSY CYA (only 1% of the overall child sample) restrict us from adopting similar empirical strategy as the regression estimates may not be reliable. However, using Black et al.'s (2010) exact approach (which uses similar strategy as Li et al. (2008) but corrects for sample selection by excluding children born after 'n' births), we find that the family size effects are qualitatively similar to relationships estimated in our primary analysis. Results are available upon request.

size for the first two siblings. Therefore, we limit our analysis to a sample of the first two children from non-twin families. Causal effects of family size are estimated by the difference between health outcomes of the first two children in same-sex siblings' families and families where the first two children are of different sex (Black et al., 2005; Angrist et al., 2010; De Haan, 2010). The same-sex IV sample includes 5041 observations.

With respect to our IV estimation, the binary indicator of sibling sex composition identifies a LATE from increasing the number of children from two to three. In comparison, the binary indicator for twin births is constructed in a way such that the LATE identified by the IV depends on the birth parity of the twins.

Finally, we employ both the twin and same-sex IV's (combined IV) together in our regression models to perform Sargan-Hansen test of overidentifying restrictions to empirically evaluate the validity of our instruments using the same-sex IV regression sample. A statistically insignificant χ^2 value (Sargan-Hansen J statistic) shall support the statistical validity of the exclusion restriction assumption of the IV's. We also employ additional robustness tests to verify whether younger siblings' birth intervals (test for twin-IV's validity) and siblings' sex composition (test for same-sex IV's validity) have any potential effect on the child health outcomes of interest.

For our IV regression analyses, we estimate a two-stage least squares (2-SLS) model where child BMI is the dependent variable and probit regressions in the second stage for our binary health indicators. The standard errors in all our regressions are corrected for clustering at the family-level.

4.2. Longitudinal regression analysis

An additional empirical concern may arise from the possibility that the effects of an increase in family size for large families may differ from the effects of having an additional member in smaller families. However, the cross-sectional analyses discussed in the previous section do not address this potential selection issue. Furthermore, the cross-sectional study incorporates NLSY's information on number of (non-adult) household members as an explanatory variable, which may include individuals who are not part of the children's immediate family. To address these issues, we take advantage of the longitudinal format of the NLSY data to perform child fixed effects regression analysis. The fixed effects regressions capture how child health outcomes vary with the arrival of younger siblings by controlling for child-specific time-invariant characteristics that may be correlated with family size and also affect child health outcomes (Juhn et al., 2015). In this context, it is important to note that the average treatment effects estimated in the longitudinal analysis is not comparable to the IV regression estimates due to variation in the treatments assessed across regression model specifications. Nonetheless, the purpose of this additional empirical exercise is to further understand the relevance of the quantity-quality trade-off theory with respect to child health. Our baseline fixed effects regression model is-

$$Y_{ijt} = \gamma_0 + \gamma_1 1.\{after\}_{ijt} + H_{it}^{'}\gamma_2 + a_i + u_{ijt} \quad (5)$$

where Y_{ijt} is the health outcome of child i with a younger sibling j (i's immediate next sibling) at time t. H_{it} is a vector of time-varying controls. a_i represents child fixed effects. The sample for estimation of Eq. (5) includes children whose mothers gave birth to at least one younger sibling during the study period. $1.\{after\}_{ijt}$ is a binary indicator constructed by comparing birth year of younger sibling j with NLSY CYA survey years such that it equals 1 for survey years greater than or equal to j's birth year. γ_1 captures the impact of having an additional sibling on the children's health outcome.

Further, the NLSY child sample allows us to capture changes in child health outcomes across successive births of younger siblings. In particular, we modify Eq. (5) by introducing multiple indicators for successive births of younger siblings. The estimated child fixed

Table 2
Relationship between household size and child body mass index (BMI).

	Full sample	Twin IV sample		Same-sex IV sample		
	OLS (1)	OLS (2)	IV (3)	OLS (4)	Same-sex IV (5)	Combined IV (6)
BMI	S.M. = 18.083	S.M. = 18.110		S.M. = 18.024		
Household size	−0.289***	−0.289***	−0.305	−0.237**	0.818	0.184
	(0.100)	(0.103)	(0.769)	(0.099)	(1.534)	(0.699)
Birth weight	0.013***	0.013***	0.013***	0.010**	0.008*	0.009**
	(0.004)	(0.004)	(0.004)	(0.005)	(0.005)	(0.004)
2nd child	0.059	0.069	0.073	0.109	−0.109	0.022
	(0.161)	(0.165)	(0.234)	(0.167)	(0.350)	(0.203)
3rd child	0.515**	0.520**	0.534			
	(0.253)	(0.263)	(0.702)			
4th child	1.001**	1.223**	1.247			
	(0.469)	(0.510)	(1.266)			
5th (or more) child	0.823*	0.829*	0.867			
	(0.476)	(0.505)	(1.909)			
First stage- twin IV			1.067***			1.135***
			(0.259)			(0.175)
First stage- Same-sex					0.125***	0.124***
					(0.045)	(0.022)
Partial F-statistic 1st stage			49.15		16.04	37.10
Sargan statistic (p-value)						0.47
Sample size	7342	7057	7057	5041	5041	5041

Note: Linear regression coefficients are reported in the above table. We estimate two-stage least squares models for our IV regression analysis. Standard errors are corrected for clustering at the family-level and are provided in parentheses. Child controls include information on birth weight (in ounce), age, sex, race, and birth order. Mother controls include BMI, AFQT scores, Rotter's scale, highest grade completed, and current age. Family control includes family's poverty status. The mother-child matched regression sample includes children who reside with both their parents. Full sample (column 1) includes children in families with at least two children (discussed in Section 3). Twin IV sample (columns (2) and (3)) includes non-twin children in families with at least two children. Same-sex IV sample (columns (4), (5) and (6)) includes only first two non-twin children in families with at least two children. P-value of Sargan-Hansen statistic (overidentification test) indicates the empirical validity of the IV's used in our cross-sectional analysis.
S.M. – Sample mean. *** $p < .01$, ** $p < .05$, * $p < .1$.

effects regression model is-

$$Y_{ikt} = \rho_0 + \sum_{k=1}^{N} \lambda_j 1.\{after\}_{ikt} + H_{it}'\rho_1 + a_i + e_{ikt} \quad (6)$$

In Eq. (6), for each child i we capture variation in i's health outcomes across births of younger siblings from the first, up to the N^{th} parity (N being the total number of i's younger siblings captured in the study period for each family). For example, for a firstborn child, Eq. (6) will estimate the effects of birth of the second child up to the N^{th} younger sibling on the firstborn child's health outcome. Each $1.\{after\}_{ikt}$ is a binary indicator that equals 1 from the time of arrival of a younger sibling until the next sibling is born. λ_j's are the estimates of the effects of the birth of younger siblings. Since Eq. (6) is a modified version of Eq. (5), variables $H_{it,}$ and a_i have similar interpretations. Given our limited sample size, especially at higher birth parities, we restrict our analysis to estimating the effects of younger siblings' arrival at the first, up to the third (or more) parity.

5. Results

5.1. IV regression analysis

In Table 2, we report OLS and IV (2-SLS) regression estimates of the impact of household size on children's BMI. Our OLS regression result (using the full sample) suggests that an increase in household size has a strongly significant and negative association with child BMI. In particular, an increase in household size by an additional member is related to a decrease in child's BMI by 0.29 units (column 1), which is equivalent to 1.7% of the relevant sample mean. This finding contrasts with our IV results, which shows that exogenous variations in household size do not have any significant impact on child BMI (columns 3 and 5). Household size and BMI are negatively related when increase in household size is triggered by twin births (column 3). However, in regressions using the same-sex sibling IV, the regression coefficient on household size is positive (column 5). We also estimate the OLS specification for the respective IV regression samples (see columns 2 and 4).

The first stage regression coefficients in Table 2 are strongly significant for both the IV's. Twin birth leads to an increase in

Table 3
Relationship between household size and likelihood of having excess body weight.

	Full sample	Twin IV sample		Same-sex IV sample		
	Probit	Probit	IV	Probit	Same-sex IV	Combined IV
	(1)	(2)	(3)	(4)	(5)	(6)
Overweight	S.M. = 0.277	S.M. = 0.280		S.M. = 0.264		
Household size	−0.015*	−0.015*	−0.004	−0.014	−0.095	−0.048
	(0.008)	(0.008)	(0.109)	(0.009)	(0.134)	(0.064)
Birth weight	0.001**	0.001**	0.001**	0.001	0.001	0.001*
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
2nd child	−0.004	−0.002	−0.004	−0.005	0.012	0.003
	(0.016)	(0.016)	(0.029)	(0.017)	(0.032)	(0.019)
3rd child	0.025	0.025	0.015			
	(0.022)	(0.023)	(0.096)			
4th child	0.042	0.058	0.041			
	(0.032)	(0.034)	(0.172)			
5th (or more) child	0.047	0.052	0.025			
	(0.042)	(0.044)	(0.267)			
Sargan statistic (p-value)						0.48
Obesity	S.M. = 0.140	S.M. = 0.142		S.M. = 0.127		
Household size	−0.016**	−0.017**	−0.046	−0.016**	−0.035	−0.021
	(0.006)	(0.007)	(0.051)	(0.007)	(0.107)	(0.048)
Birth weight	0.001**	0.001***	0.001**	0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
2nd child	0.001	0.004	0.010	0.001	0.006	0.004
	(0.013)	(0.013)	(0.017)	(0.013)	(0.026)	(0.014)
3rd child	0.032*	0.036**	0.061			
	(0.018	(0.018)	(0.047)			
4th child	0.036	0.051**	0.096			
	(0.024)	(0.025)	(0.082)			
5th (or more) child	0.059*	0.066	0.137			
	(0.031)	(0.033)	(0.127)			
First stage- twin IV			1.067***			1.135***
			(0.259)			(0.175)
First stage- Same-sex					0.125***	0.124***
					(0.045)	(0.022)
Partial F-statistic first stage			49.15		16.04	37.10
Sargan statistic (p-value)						0.88
Sample size	7342	7057	7057	5041	5041	5037

Note: We report marginal effects from our probit regressions (second-stage in IV regressions). Standard errors are corrected for clustering at the family-level and are provided in parentheses. Child controls include information on birth weight (in ounce), age, sex, race, and birth order. Mother controls include BMI, AFQT scores, Rotter's scale, highest grade completed, and current age. Family control includes family's poverty status. The mother-child matched regression sample includes children who reside with both their parents. Full sample (column 1) includes children in families with at least two children (discussed in Section 3). Twin IV sample (columns (2) and (3)) includes non-twin children in families with at least two children. Same-sex IV sample (columns (4), (5) and (6)) includes only first two non-twin children in families with at least two children. P-value of Sargan-Hansen statistic (overidentification test) indicates the empirical validity of the IV's used in our cross-sectional analysis.
S.M. – Sample mean. *** $p < .01$, ** $p < .05$, * $p < .1$.

household size by an additional member (regression coefficient equals 1.1; see column 3). The effects of twin births on household size are similar to estimates found in recent US-based literature (De Haan, 2010 (0.9); Juhn et al., 2015 (1.8)). On the other hand, the first stage regression coefficient on same-sex IV (0.13, see column 5) is marginally higher than estimates found in recent studies (coefficients in studies by Angrist et al., 2010, Black et al., 2005, and De Haan, 2010 vary between 0.07 and 0.11). It is useful to note, however, that the 95% confidence intervals around our point estimate ([0.06, 0.19]) are within the range of coefficient values obtained in the aforementioned international literature. Empirical validity of both the IV's is partially supported by large F-values in the first stage regressions.

The BMI threshold for having a healthy body weight varies by child's age. Therefore, the regression estimates in Table 2 do not directly indicate whether changes in household size affect children's likelihood of having unhealthy body weight. For more explicit evidence on child health outcomes of family size variations, we focus on excess body weight indicators. In Table 3, we report the estimated marginal effects from probit and IV regressions with respect to the binary indicators for being overweight and obese. Probit regression results (using the full sample) suggest that an increase in household size by an additional member is associated with a decrease in the probability of being overweight and the probability of being obese by approximately 2 percentage points each (column 1). The effects are statistically significant at the 10% level for the overweight indicator and at the 5% level for the obesity indicator. In our IV regressions, although household size appears to be negatively related to the likelihood of having excess body weight (columns 3 and 5), the marginal effects are not statistically significant.

Additionally, we examine the relationship between household size and binary child health indicators of underweight and illness that requires medical attention. Estimating household size's effects on incidence of child illness shall provide useful insights into the validity of the hygiene theory. Prior to running any regressions, we utilize the full sample data to observe age-specific trends in incidence of child illness across households with different sibling size. In particular, we compare children with no siblings to firstborn children in a multiple-sibling household. In Fig. 1, we find that in both the household types, children are more likely to fall ill during the early childhood years followed by a declining trend in the incidence of illness. Interestingly, we find that after reaching a maximum (at age one), the trend in incidence of illness for children with at least one sibling consistently lies below the line representing trend for children with no sibling, until the age of 17. The observed trends in incidence of illness by sibling size appears to support the hygiene theory.

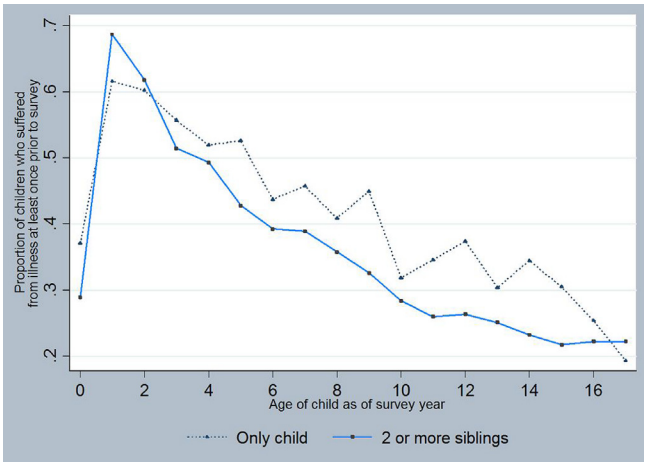

Fig. 1. Age-specific trends in incidence of child illness by sibling size.
Note: The above graph compares proportion of firstborn children falling ill (that requires medical attention) in the year prior to survey (as reported by mothers) in households with two or more siblings with proportion of children falling ill in the year prior to survey in one-child households.

In Table 4, we report the marginal effects from the regression analysis using binary indicators of underweight and child illness. In our full-sample probit regression, we find no evidence of a significant association between household size and probability of being underweight. However, household size is associated with a 2-percentage point decrease (6.6% relative to sample mean) in the probability of illness (see column 1). Turning to the IV regression estimates, we find that variation in household size does not have any significant effect on the likelihood of being underweight or suffering from illness (columns 3 and 5).

To summarize, the full sample probit regression analyses show that an increase in household size is associated with better child health outcomes. However, controlling for endogeneity in household size, we do not find any significant relationship between a change in the number of children in a household and child health outcomes. Simply put, the IV regression results do not provide any empirical support in favor of the quantity-quality trade-off theory. Further, the statistically insignificant IV regression estimates support the assumption that potential health benefits associated with a large sibship size (as discussed earlier) are likely to weaken the negative impact on child health quality due to resource constraints, as highlighted by the quantity-quality trade-off theory.

5.2. Additional robustness analysis

We perform additional tests to verify the statistical validity of our IV's. First, we use the same-sex IV regression sample to estimate IV regressions using both twin and same-sex IV's together (combined analysis). In the sample used for the combined analysis, twin births generate exogenous household size variations from the third delivery onwards. Using both the IV's allows us to test for overidentifying restrictions (Sargan, 1958). The joint null hypothesis in the Sargan-Hansen overidentification test considers the possibility that the excluded instruments are uncorrelated with the error term in the estimated model. We report the p-values of the Sargan-Hansen statistic estimated from the combined IV regression analysis in column 6 of Tables 2–4. We find that the χ^2-value is statistically insignificant across all child health measures.

Second, we use Black et al.'s (2005) methodology to study if birth spacing of younger siblings is related to older siblings' probability of having excess weight. For our analysis, we consider a sample of firstborn children from non-twin families with three or more siblings. Next, we construct a binary indicator for the first child that equals 1 if the time between the birth of the second and the third sibling is less than eighteen months.[12] Regressing excess body weight indicators on the binary indicator of birth space between the second and the third sibling indicate that closely-spaced younger siblings are not related to the older children's probability of having excess body weight (see Table 5; Panel A).

Finally, we follow De Haan's (2010) approach to verify if children's sex composition has any significant relationship with the child health outcomes of interest (excess body weight indicators). In particular, we regress the excess body weight indicators (of being overweight and obese) on two dummy variables representing whether all the siblings in a family are boys ('all boys') and whether all the siblings are girls ('all girls') by

[12] Black et al. (2005) also look at younger siblings (second and third siblings) whose births are less than a year apart. However, the lack of sufficient data in our analysis sample restricts us from performing further analysis.

Table 4
Relationship between household size and likelihood of being underweight and ill.

	Full sample	Twin IV sample		Same-sex IV sample		
	Probit (1)	Probit (2)	IV (3)	Probit (4)	Same-sex IV (5)	Combined IV (6)
Underweight	S.M. = 0.114	S.M. = 0.113		S.M. = 0.112		
Household size	0.003	0.004	0.037	0.001	0.002	0.004
	(0.005)	(0.005)	(0.065)	(0.006)	(0.085)	(0.046)
Birth weight	−0.001***	−0.001***	−0.001***	−0.001***	−0.001***	0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
2nd child	−0.006	−0.005	−0.012	−0.005	−0.005	−0.004
	(0.010)	(0.011)	(0.018)	(0.011)	(0.020)	(0.013)
3rd child	0.000	0.001	−0.026			
	(0.014)	(0.014)	(0.057)			
4th child	−0.014	−0.027	−0.078			
	(0.019)	(0.021)	(0.103)			
5th (or more) child	−0.019	−0.018	−0.096			
	(0.028)	(0.028)	(0.158)			
First stage- twin IV			1.067***			1.135***
			(0.259)			(0.175)
First stage- Same-sex					0.125***	0.124***
					(0.045)	(0.022)
Partial F-statistic first stage			49.15		16.04	37.10
Sargan statistic (*p*-value)						0.97
Sample size	7342	7057	7057	5041	5041	5037
Illness	S.M. = 0.346	S.M. = 0.345		S.M. = 0.359		
Household size	−0.023***	−0.021***	0.004	−0.018	0.134	0.040
	(0.009)	(0.009)	(0.146)	(0.012)	(0.124)	(0.070)
Birth weight	−0.000	−0.000	−0.000	−0.000	−0.000	−0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
2nd child	−0.002	−0.000	−0.005	−0.007	−0.037	−0.020
	(0.013)	(0.013)	(0.035)	(0.014)	(0.027)	(0.020)
3rd child	−0.014	−0.010	−0.031			
	(0.020)	(0.021)	(0.126)			
4th child	0.024	0.016	−0.023			
	(0.029)	(0.031)	(0.228)			
5th (or more) child	0.032	0.027	−0.033			
	(0.042)	(0.044)	(0.354)			
First stage- twin IV			1.066***			
			(0.259)			
First stage- Same-sex					0.125***	
					(0.045)	
Partial F-statistic first stage			49.02		16.20	37.10
Sargan statistic (*p*-value)						0.17
Sample size	7348	7063	7063	5048	5048	5044

Note: We report marginal effects from our probit regressions (second-stage in IV regressions). Standard errors are corrected for clustering at the family-level and are provided in parentheses. Notes related to controls and methods utilised, as described in Table 3, apply here. *** $p < .01$, ** $p < .05$, * $p < .1$.

Table 5
Testing importance of birth spacing and sex composition in risks of having excess weight.

	Overweight		Obese	
	(1)	(2)	(3)	(4)
	S.M. = 0.235	S.M. = 0.277	S.M. = 0.105	S.M. = 0.140
Panel A				
Birth spacing < = 18 months (between 2nd and 3rd child)	0.037		−0.008	
	(0.064)		(0.040)	
Sample size	901		901	
Panel B				
All girls		0.001		0.007
		(0.020)		(0.015)
All boys		−0.013		0.003
		(0.021)		(0.015)
Sample size		7362		7362

Note: Marginal effects from probit regressions are reported in the above Table. Standard errors are corrected for clustering at the family-level and are provided in parentheses. Child controls include information on birth weight (in ounce), age, sex, race, and birth order (for all girls and all boys regressions only). Mother controls include BMI, AFQT scores, Rotter's scale, highest grade completed, and current age. Family control includes family's poverty status. Panel A results are based on all firstborn children in non-twin families with at least three children. Panel B results are based on full sample. All marginal effects are statistically insignificant.

Table 6
Effects of birth of a younger sibling on child health using child fixed effects.

	BMI		Overweight		Obese		Underweight		Illness	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
	S.M. = 17.841		S.M. = 0.271		S.M. = 0.156		S.M. = 0.091		S.M. = 0.330	
After birth of younger sibling	−0.343		−0.044*		−0.017		0.001		−0.049*	
	(0.307)		(0.024)		(0.019)		(0.021)		(0.027)	
Short-term (0–3 years)		−0.337		−0.043*		−0.016		0.000		−0.044
		(0.307)		(0.024)		(0.019)		(0.021)		(0.027)
Long-term (3+ years)		−0.357		−0.048*		−0.021		−0.003		−0.063**
		(0.341)		(0.027)		(0.022)		(0.023)		(0.032)

Note: Linear child fixed effect regression coefficients are reported in the above Table. Standard errors are corrected for clustering at the family-level and reported in parentheses. The models control for time-varying characteristics including child age, mother's age, mother's BMI, and family's poverty status. The above regression analyses include 10611 observations.
* $p < .1$.
** $p < .05$.

including controls used in our full-sample regressions (Eq. (3)). The estimated marginal effects of 'all boys' and 'all girls' for both the health outcomes of interest are statistically insignificant, suggesting that children's sex composition does not have a direct association with the health outcomes of interest in our sample (see Table 5; Panel B). However, it is important to note that the regression estimates (reported in Table 5) obtained from the additional empirical tests do not represent causal relations.

5.3. Health effects of arrival of younger siblings using fixed effects regressions

Table 6 presents our linear child fixed effects regression estimates of the impact of the birth of a younger sibling on the older sibling's health outcomes. Controlling for time-invariant child fixed effects, we estimate regression models represented by Eq. (5). We find that the arrival of a younger sibling is related to a 4-percentage point decline in the probability of being overweight and a 5-percentage point drop in the likelihood of suffering from illness (columns 3 and 9). The effects are statistically significant at the 10% level.

Further, we estimate the short-term and long-term impacts of the birth of a younger sibling on children's health measures. To perform our analysis, we incorporate two binary indicators in Eq. (5) to capture a short-term effect (for 0 to 3 years after younger sibling *j* is born in child *i*'s family) and a long-term effect (for 3+ years). Our results suggest that the negative relationship between the arrival of a younger sibling and the probability of being overweight and falling ill is greater in the long-term (columns 4 and 10).

Additionally, we estimate fixed effects regression by controlling for family fixed effects as childbirth and child health quality depend on parental decisions. We report our family fixed effects linear regression results in Table 7. We do not find any significant relationship between birth of a younger sibling and older children's health outcomes.

Finally, we study how birth of younger siblings at successive parities affect older siblings' health outcomes (see Eq. (6)). The

Table 7
Effects of birth of a younger sibling on child health using family fixed effects.

	BMI		Overweight		Obese		Underweight		Illness	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
After birth of younger sibling	0.032		0.002		−0.004		−0.011		−0.007	
	(0.169)		(0.013)		(0.011)		(0.010)		(0.012)	
Short-term (0–3 years)		−0.043		0.003		−0.003		−0.006		−0.008
		(0.181)		(0.014)		(0.011)		(0.013)		(0.015)
Long-term (3+ years)		0.077		0.001		−0.005		−0.013		−0.006
		(0.186)		(0.014)		(0.013)		(0.010)		(0.013)

Note: Linear family fixed effect regression coefficients are reported in the above Table. Standard errors are corrected for clustering at the family-level and reported in parentheses. The models control for time-varying characteristics including child age, mother's age, mother's BMI, and family's poverty status. The above regression analyses include 10611 observations. All regression coefficients are statistically insignificant.

Table 8
Effects of birth of additional siblings on older children's health outcomes (using child fixed effects).

Arrival of younger sibling at:	BMI (1)	Overweight (2)	Obese (3)	Underweight (4)	Illness (5)
First parity	−0.321	−0.043*	−0.015	0.001	−0.043
	(0.306)	(0.023)	(0.017)	(0.021)	(0.027)
Second parity	−0.748	−0.044	−0.042	0.020	−0.094**
	(0.541)	(0.035)	(0.027)	(0.031)	(0.043)
Third (or more) parity	−0.677	−0.025	−0.030	0.028	−0.009
	(0.557)	(0.052)	(0.043)	(0.040)	(0.064)

Note: Linear child fixed effect regression coefficients are reported in the above Table. Standard errors are corrected for clustering at the family-level and reported in parentheses. The models control for time-varying characteristics including child age, mother's age, mother's BMI, and family's poverty status. The results are consistent when we perform regressions using sample of first-born children only. The above regression analyses include 10611 observations.
* $p < .1$.
** $p < .05$.

Table 9
Effects of birth of additional siblings on older children's health outcomes (using family fixed effects).

Arrival of younger sibling at:	BMI (1)	Overweight (2)	Obese (3)	Underweight (4)	Illness (5)
First parity	0.044	0.003	−0.002	−0.010	−0.005
	(0.167)	(0.013)	(0.010)	(0.010)	(0.012)
Second parity	−0.368	−0.016	−0.029	−0.010	−0.007
	(0.313)	(0.022)	(0.017)	(0.015)	(0.019)
Third (or more) parity	−0.134	0.011	0.010	−0.010	−0.011
	(0.347)	(0.035)	(0.027)	(0.021)	(0.030)

Note: Linear family fixed effect regression coefficients are reported in the above Table. Standard errors are corrected for clustering at the family-level and reported in parentheses. The models control for time-varying characteristics including child age, mother's age, mother's BMI, and family's poverty status. The results are consistent when we perform regressions using sample of first-born children only. The above regression analyses include 10611 observations. All regression coefficients are statistically insignificant.

estimated regression coefficients reported in Table 8 suggest that the birth of a younger sibling at the first parity is related to a 4-percentage point decrease in the probability of being overweight (column 2). Further, the birth of a younger sibling at the second parity is related to a 9 percentage-point drop in the likelihood of falling ill (column 5). In general, when we control for child fixed effects, the negative effect between increase in family size and children's health problems are relatively large when a younger sibling is born at the second parity. The results from a similar analysis of family fixed effects are reported in Table 9, where we find that all of the estimated coefficients are statistically insignificant.

The fixed effects analyses show that the arrival of a younger sibling is unrelated to a decline in child health quality. Moreover, when controlling for child fixed effects, we find that the arrival of a younger sibling at the first parity is related to better health outcomes of the older child. It is important to note here that depending on the model specifications, the IV estimation and fixed effects regressions identify the effects of a change in family size at different margins. The discrepancies between our cross-sectional and panel-data analyses can likely be attributed to the way the models are classified. Nonetheless, the additional longitudinal analysis corroborates our cross-sectional findings. Overall, we do not find any evidence of a quantity-quality trade-off between family size and child health.

5.4. Event study – test for endogenous changes in family size

Children's health outcomes may affect parental decisions to have an additional childbirth. For example, healthy children may incentivize parents to conceive more children. This may lead to reverse causality concerns that could bias our fixed effects regression coefficients. Hence, using children's birth year information, we perform an event study to see if there is any evidence of endogenous birth timing (Juhn et al., 2015). We estimate-

$$Y_{ijt} = \theta_0 + \sum_{r=-4}^{-1} \delta_r T^r_{ijt} + \sum_{r=1}^{5} \delta_r T^r_{ijt} + X'_{it}\theta_2 + a_i + \mu_{it} \quad (7)$$

where T^r_{ijt} is a dummy variable that equals 1 for year t when t is r years relative to the year of birth of the next younger sibling j. To account for anticipatory effects, we create dummies for up to 4 years prior to a younger sibling's birth year. For post-treatment effects (where childbirth is the treatment), we create dummies for up to 5 years after the younger sibling's birth keeping the birth year as the excluded category.

Controlling for child fixed effects, the results of our event study are reported in Table 10. The point estimates for body weight indicators for years prior to younger siblings' birth are statistically insignificant. However, we find a statistically significant increase in the probability of suffering from illness during the year prior to birth of a younger sibling. Nonetheless, the F-values associated

Table 10
Event study to explore trends in children's body weight before and after birth of an additional sibling birth (using child fixed effects).

Time relative to younger sibling's birth	BMI (1)	Overweight (2)	Obese (3)	Underweight (4)	Illness (5)
4+ years before	0.755	−0.029	0.059	−0.006	0.004
	(0.622)	(0.057)	(0.051)	(0.049)	(0.063)
3 years before	0. 573	0.010	0.034	0.002	0.080
	(0.692)	(0.061)	(0.051)	(0.051)	(0.084)
2 years before	−0.255	−0.063	−0.027	−0.061*	0.063
	(0.482)	(0.048)	(0.043)	(0.033)	(0.053)
1 year before	−0.070	−0.025	−0.020	−0.001	0.115**
	(0.611)	(0.052)	(0.041)	(0.045)	(0.057)
1 year after	−0.715	−0.043	−0.002	−0.001	0.024
	(0.545)	(0.040)	(0.030)	(0.037)	(0.045)
2 years after	−1.153**	−0.071**	−0.017	−0.005	−0.022
	(0.566)	(0.032)	(0.024)	(0.031)	(0.036)
3 years after	−1.361**	−0.061*	−0.020	−0.009	0.002
	(0.536)	(0.037)	(0.027)	(0.033)	(0.044)
4 years after	−1.617***	−0.049	−0.025	−0.014	−0.047
	(0.589)	(0.033)	(0.025)	(0.030)	(0.039)
5+ years after	−1.416***	−0.071**	−0.022	−0.008	−0.020
	(0.534)	(0.035)	(0.026)	(0.029)	(0.040)
F of $\delta_{lead1} + \delta_{lead2} + \delta_{lead3} + \delta_{lead4} = 0$	F = 0.24	F = 0.48	F = 0.26	F = 0.24	F = 2.10
	p = 0.63	p = 0.49	p = 0.61	p = 0.62	p = 0.15

Note: Linear child fixed effect regression coefficients are reported in the above Table Standard errors are corrected for clustering at the family-level and reported in parentheses. The models control for time-varying characteristics including child age, mother's age, mother's BMI, and family's poverty status. The sample contains 11131 observations. *** $p < .01$, ** $p < .05$, * $p < .1$.

Table 11
Event study to explore trends in children's body weight before and after birth of an additional sibling birth (using family fixed effects).

Time relative to younger sibling's birth	BMI (1)	Overweight (3)	Obese (4)	Underweight (2)	Illness (5)
4+ years before	−0.072	−0.006	0.006	−0.015	−0.002
	(0.183)	(0.016)	(0.013)	(0.011)	(0.013)
3 years before	0.303	0.038	0.052	−0.022	0.038
	(0.504)	(0.050)	(0.040)	(0.044)	(0.062)
2 years before	0.043	−0.044	−0.026	−0.064	0.059
	(0.694)	(0.041)	(0.035)	(0.029)	(0.043)
1 year before	−0.215	0.006	−0.005	−0.013	0.058
	(0.448)	(0.043)	(0.035)	(0.036)	(0.045)
1 year after	−0.692*	0.007	−0.002	−0.015	0.009
	(0.413)	(0.032)	(0.026)	(0.028)	(0.033)
2 years after	−1.018**	−0.042	−0.025	−0.016	−0.028
	(0.453)	(0.028)	(0.022)	(0.025)	(0.029)
3 years after	−1.236***	−0.004	−0.009	−0.022	−0.022
	(0.381)	(0.030)	(0.024)	(0.024)	(0.029)
4 years after	−1.317***	−0.006	−0.015	−0.024	−0.031
	(0.421)	(0.028)	(0.022)	(0.023)	(0.029)
5+ years after	−0.984***	−0.024	−0.020	−0.027	−0.018
	(0.365)	(0.027)	(0.022)	(0.020)	(0.026)
F of $\delta_{lead1} + \delta_{lead2} + \delta_{lead3} + \delta_{lead4} = 0$	F = 0.00	F = 0.00	F = 0.10	F = 1.16	F = 2.05
	p = 0.96	p = 0.95	p = 0.75	p = 0.28	p = 0.15

Note: Linear family fixed effect regression coefficients are reported in the above Table Standard errors are corrected for clustering at the family-level and reported in parentheses. The models control for time-varying characteristics including child age, mother's age, mother's BMI, and family's poverty status. The sample contains 11131 observations. *** $p < .01$, ** $p < .05$, * $p < .1$.

with overall significance of our lead dummies do not provide any empirical evidence of a reverse causality between child outcomes and parental decisions to have an additional child. Finally, considering childbirth as an outcome of parental choices, we estimate a variant of Eq. (7) by controlling for family fixed effects. The estimated family fixed effects regression results, reported in Table 11, indicate that younger siblings' births are not significantly related to older siblings' prior health outcomes.

6. Conclusions

This study is among the first to conduct a comprehensive empirical analysis on the effects of

family size on child health (in terms of body weight) in developed economies. Previous studies have often focused on other outcomes such as educational attainment; and the few related studies in the child health space have focused on developing countries, where population density is a large social concern. Given the socio-economic and cultural differences across countries at varying stages of development, the current analysis evaluates the relevance of family planning policies in the context of developed countries.[13] In particular, the absence of an inverse relationship between child quantity and health quality in traditional US families provides empirical support in favor of studies that discuss the importance of a well-organized social welfare system commonly attributed to advanced societies. However, as the proportion of American children born in disadvantaged families continues to rise, our study opens further scope for future research that can focus on more contemporaneous and detailed evaluations of the child health outcomes of family size.[14]

Furthermore, the absence of a quantity-quality trade-off with respect to excess body weight indicators would encourage policymakers to streamline their attention to other social and family-level determinants of childhood obesity in the US. Finally, US-based studies that provide empirical evidence of the quantity-quality trade-off theory find a negative relationship between family size and children's cognitive abilities or educational achievements (Blake, 1981; Cáceres-Delpiano, 2006; Juhn et al., 2015). In this context, the analysis in this paper suggests that parental investments in the child-rearing process are not only affected by changes in family size, but may also vary by parental priorities assigned to different aspects of child development. It is plausible that in traditional US households, child health receives higher degrees of parental importance than cognitive development during the early stages of childhood.

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[13] A few past studies that have explored the relevance of family planning policies in developing world include Hossain (1989; Philippines); Rosenzweig & Wolpin (1986; Philippines); Rosenzweig & Zhang (2009; China).

[14] Recent US-based statistical estimates on American families suggest that while 39% of all children reside in single-parent or no-parent families, around 21% of all children live in poverty (Livingston, 2014). Also see http://www.nccp.org/topics/childpoverty.html; Retrieved on September 15, 2017.

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