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Examining the temporal relationships between childhood obesity and asthma

Tiffany L. Green*

Department of Healthcare Policy and Research, Virginia Commonwealth University, P.O. Box 980430, Richmond, VA, 23298, United States

Abstract

Childhood obesity has become an issue of increasing concern to health researchers and policymakers in the United States. One important chronic health condition linked to obesity is pediatric asthma. Although researchers have speculated that both conditions may have common origins, the majority of research in this area has focused on a unidirectional relationship between obesity and later asthma. However, much of the literature is limited by its reliance on cross-sectional data and its failure to examine the possibility that asthma may influence weight fluctuations through changes in physical and sedentary activity. Using data from the Early Childhood Longitudinal Study-Kindergarten Cohort (ECLS-K), I explore the bidirectional relationships between childhood obesity and asthma. The results in this paper suggest that past asthma levels are positively correlated with changes in BMI and the onset of obesity. However, only new onset asthma is positively correlated with subsequent changes in BMI. The potential mechanisms are unclear, as I find little evidence that asthma is structurally related to changes in physical or sedentary activity over time. When testing the prevailing hypothesis that obesity is related to subsequent asthma, I find that lagged weight status is strongly related to asthma prevalence levels but that the onset of overweight or obesity is not associated with the subsequent onset of asthma. These results suggest that the onset of asthma may be related to subsequent weight gain over time.

Keywords

Economics of child health; Asthma; Obesity

1. Introduction

Childhood obesity has become an issue of increasing concern to public health researchers and policymakers in the United States. The proportion of overweight and obese children under 18 has tripled over the last few decades. In 2004, 17% of U.S. children were obese and 35% of children were overweight (Ogden et al., 2006). An emerging economics literature has linked obesity to decreased skill attainment in childhood (Cawley and Spiess, 2008; Datar et al., 2004), a potential mechanism for the poor labor market outcomes

reported among obese adults (Baum and Ford, 2004). Obesity is also associated with a number of detrimental health conditions, including cardiovascular problems, metabolic disorders such as diabetes, and poor mental health (Daniels, 2006).

One important health condition linked to obesity is asthma. While the rates of asthma among U.S. children have plateaued in recent years, pediatric asthma is the most common chronic childhood disease in the United States, with 9.6 million U.S. children (13.1%) having received a diagnosis in their lifetimes as of 2007 (Akinbami et al., 2009). Childhood asthma entails a number of financial and non-financial costs, including expenditures associated with medication, hospitalizations and outpatient care (Wang et al., 2005), and missed school/work days for children and their parents (Akinbami, 2006).

Researchers have long recognized that children with asthma are more likely to be obese (Epstein et al., 2000; Gennuso et al., 1998). Although there has been speculation that both asthma and obesity might have common origins (Tantisira and Weiss, 2001), the vast majority of epidemiological and clinical research on pediatric asthma and obesity in children has focused on a unidirectional relationship between obesity and asthma, with obesity posited as a causal factor in the development of asthma. Researchers also report that obese children with asthma have more frequent use of inhaled and oral asthma medication, higher rates of emergency room utilization and greater care-associated costs compared to non-obese children with asthma (Black et al., 2012; Trasande et al., 2009). However, much of this literature yields mixed results and employs cross-sectional data, making it challenging to ascertain the causal directions between asthma diagnosis and weight gain (Epstein et al., 2000; Sithole et al., 2008; von Mutius et al., 2001). The few longitudinal studies on pediatric asthma and obesity report some relationships between obesity and subsequent asthma in children, but do not empirically account for the possibility of alternative causal pathways (Gilliland et al., 2003; Zhang et al., 2010).

On one hand, research from animal models suggests that obesity might drive subsequent asthma through increased airway hyperresponsiveness, common genetic susceptibility to both conditions, and the potential for excess weight to trigger modifications in the immune system (Lu et al., 2006; Tantisira and Weiss, 2001). On the other hand, there are also plausible medical and behavioral mechanisms through which asthma can influence subsequent weight outcomes in children. The steroids in asthma inhalers can increase the risk of weight gain, even over short periods (Pianosi and Davis, 2004). Also, parents might change their child health-seeking behaviors in response to their children's health outcomes (Liu et al., 2009). For example, some parents of asthmatic children, concerned about exercise-induced wheezing, might change the ways in which their children allocate their time to physical and sedentary activity (Gannotti et al., 2007; Lang et al., 2004; Pianosi and Davis, 2004). Thus, as Fletcher et al. (2010) suggest, the fact that adults with asthma are less likely to get regular exercise than their non-asthmatic counterparts implies that asthma-induced changes in physical activity in childhood might have a more permanent effect on weight outcomes.

In contrast to the prevailing hypothesis, recent findings from the economics literature suggest substantial impacts of childhood asthma on adult obesity. Fletcher et al. (2010) use

longitudinal data to explore the causal impact of childhood asthma on adult health, including self-reported health status, missed days at school and work, and obesity. They find that childhood asthma is associated with both large increases in the probability of obesity and the number of missed school and work days and a large decrease in the probability of reporting excellent self-rated health. In other work from the clinical literature, Beckett et al. (2001) test whether asthma is associated with weight gain in a 10-year study of African-Americans and whites aged 18–30 years. The authors find evidence that a new diagnosis of asthma is associated with decreased physical activity and weight gain over time among women. Finally, in a 20-year longitudinal study of Swiss young adults, Hasler et al. (2006) find strong associations between asthma and subsequent weight gain, even after controlling for a wide range of confounding weight- and physical activity-related variables. The authors fail to find any associations between weight gain and subsequent asthma. However, none of the papers above address the dynamic relationships between obesity and asthma during childhood or preadolescence.

Understanding the causal directions between asthma and obesity has important implications for health policy and spending. If obesity is a proximate cause of subsequent asthma, then it may be prudent to invest more resources in effective programs designed to prevent and mitigate the effects of childhood obesity through better nutrition and increased physical activity levels (Cawley, 2008). Conversely, if asthma is causally related to the onset of obesity, then targeting pre- and postnatal child health investment behaviors such as maternal smoking and asthma treatment adherence may be a more optimal strategy (Currie, 2009; Sabia, 2008). This paper extends the work of Fletcher et al. (2010), Beckett et al. (2001) and Hasler et al. (2006) to examine whether pediatric asthma is associated with subsequent weight gain and physical/sedentary activity in preadolescents. I also evaluate the claims of previous literature that posit obesity as a causal factor in the subsequent development of asthma (Gilliland et al., 2003; Zhang et al., 2010). To accomplish these aims, I use the Granger causal framework extended to panel data (Adams et al., 2003) to test for the absence of statistical causality running from asthma to changes in weight status (and *visa versa*). The detection of Granger causality does not automatically denote true causality. However, finding a lack of evidence for Granger causality may imply the absence of true structural causality (Adams et al., 2003; Stowasser et al., 2011).

The results in this paper suggest that past asthma levels are Granger causal for changes in BMI and the onset of obesity. However, only new onset asthma is positively correlated with subsequent changes in BMI. The potential mechanisms are unclear, as I find little evidence that asthma is structurally related to changes in physical or sedentary activity over time. When testing the alternative hypothesis, I find that while BMI and obesity are Granger causal for new onset asthma, neither the onset of overweight nor obesity is associated with the subsequent development of asthma. These results suggest that the onset of asthma may be related to subsequent weight gain over time.

This paper will proceed as follows: Section 2 provides a description of the data and estimation sample. Section 3 describes the empirical approach and estimation methods employed in this paper, and Section 4 contains a discussion of the estimation results. Section

5 concludes with further discussion of the implications of the empirical findings, limitations, and plans for future work.

2. Description of data and sample derivation

The Early Childhood Longitudinal Study-Kindergarten Class of 1998–1999 (ECLS-K) is a nationally representative, longitudinal study that follows a cohort of children from kindergarten to middle school. The ECLS-K focuses on children's early school experiences and allows researchers to study how individual, household, educational, and community factors are related to school performance. The ECLS-K also contains information on child health, including asthma and weight outcomes.¹ The public data consist of interviews conducted in the fall and the spring of kindergarten (1998–1999), the fall and spring of 1st grade (1999–2000), and the spring of 3rd (2002), 5th (2004), and 8th (2007) grades, for a total of seven waves. Interviewers surveyed children, parents, teachers, and school administrators. The survey data also contain information derived from both student records and a school facilities checklist.

Table 1 contains summary statistics for all dependent and independent variables. All of the outcome variables of interest come from parent and child interviews and assessments at 3rd, 5th, and 8th grades, although I derive data on birth weight and baseline income from the fall kindergarten survey. Interviewers gathered information on asthma outcomes during the 3rd, 5th, and 8th grades and asked parents whether their child had ever been diagnosed with asthma and if their child was receiving treatment for asthma. Using this information, I construct variables that indicate whether a child has ever been diagnosed with asthma and whether a previously undiagnosed child became asthmatic during the course of the survey period.² One important consideration is that the degree of asthma severity may affect weight outcomes. To address this, I also use an alternative definition of asthma that only includes children who have received medical treatment for asthma.³ In this case, I allow asthma treatment, conditional upon an asthma diagnosis, to vary from period to period to period. Additionally, I create a variable indicating whether a child received asthma treatment for the first time between either the 3rd and 5th grades or 5th and 8th grades.

Twelve percent of parents report that their child was diagnosed with asthma by 3rd grade, and the proportion of affected children rises steadily to 16% in 5th grade and 20% by 8th grade. This number is higher than other national estimates (around 13%) of the lifetime prevalence of pediatric asthma (Dey and Bloom, 2006). However, a small percentage of children in the study have inconsistent parental reports of asthma ($n = 566$). For example, some parents reported at 3rd grade that their children had been diagnosed with asthma, but then reported never having received an asthma diagnosis at 5th grade. However, cross-sectional reports of asthma “ever” at 5th and 8th grades yield asthma prevalence figures (14 and 16%, respectively, not shown) that are closer to other national estimates.⁴

¹See <http://www.nces.ed.gov/ecls/kindergarten.asp> for more information about the survey.

²Once parents report an asthma diagnosis, the focal child “always” has asthma. A change in asthma diagnosis occurs when a child whose parent has never reported an asthma diagnosis in previous waves reports a diagnosis of asthma between either the 3rd and 5th or 5th and 8th grades.

³The survey does not include information on what kinds of treatments the child receives (e.g. steroid inhalers). It could be that this category captures children with more well-controlled asthma and/or children that required emergency room treatment for asthma.

Trained assessors measure children's height and weight during each round of the ECLS-K; this study only uses relevant data from the 3rd, 5th, and 8th grades. I use the collected height and weight data to calculate body mass index-related (BMI) statistics. I calculate *z*-scores using the zanthro routine in STATA (see Kuczmarski et al. (2002) for the corresponding growth charts). Those children whose BMI-for-age falls between the 5th percentile to less than the 85th percentile are considered normal weight. I treat the few children who are underweight (BMI-for-age is less than the 5th percentile) as normal weight. Children whose BMI-for-age measurements are between the 85th to less than the 95th percentile are considered overweight; those with a BMI equal to or greater than the 95th percentile are considered obese. About one-third of the sample is overweight or obese by 3rd grade; this percentage rises in 5th grade (39%) and drops again by 8th grade (36%).⁵ The appropriateness of the BMI-for-age measure as an instrument for pediatric overweight/obesity has been a source of previous controversy. However, Mei et al. (2002) perform a validation study on BMI-for-age for children aged 2–19 years. The authors find that the instrument performs better than the Rohrer Index in detecting overweight and underweight and performs similarly to weight-for-height measures. The International Task Force on Obesity also considers BMI to be a reasonable index of adiposity in children and adolescents (Dietz and Robinson, 1998).

Lastly, each round of parental surveys collects data on physical and sedentary activity. I measure physical activity as the number of days a child engaged in at least 20 min of vigorous aerobic activity and measure sedentary activity using the number of minutes of television viewing per week. For each outcome, I also measure the change in days of both aerobic activity and television viewing between waves.

Table 2 contains descriptive data on the relationships among the outcome variables of interest – asthma, weight status and physical activity – summed over grades 5 and 8 only.⁶ As expected, average BMI-for-age is slightly higher among asthmatics. While there is generally little difference in overweight between asthmatic and non-asthmatic children, asthmatic children have much higher rates of obesity (at least 10%). However, there do not appear to be large differences in days of aerobic activity. In fact, for nearly all measures, asthmatic children have slightly *higher* levels of reported aerobic activity compared to their non-asthmatic counterparts. In contrast, with the exception of newly diagnosed asthmatics, parents of asthmatic children report that their children watch more television – than their non-asthmatic counterparts (approximately 19 to 42 min of television per week).

2.1. Sample derivation

I employ the following exclusion criteria when deriving the final estimation sample (see Table A.1 in Appendix A). First, I exclude every observation in the sample without valid measures for birth weight ($n = 871$). I also excluded all observations without information on race and gender ($n = 19$). Next, I exclude every observation without parent ($n = 11,825$) and

⁴I estimate models excluding those children with inconsistent asthma reports, but the substantive results remain unchanged.

⁵I define “became overweight” as going from normal weight to overweight or from obese to overweight. Children who became obese went from overweight to obese or from normal to obese, and children who became normal weight went from either overweight or obese to normal.

⁶The sample size, $N = 12,904$ reflects summary statistics for two waves of data.

child ($n = 356$) assessments at 8th grade. I also exclude children without valid values for the dependent variables: asthma at 3rd, 5th, and 8th grades ($n = 544$) and weight and height information at 3rd, 5th and 8th grades ($n = 1329$). The final sample ($N = 6452$) is substantially smaller than the original sample at baseline ($N = 21,396$). The vast majority of excluded cases ($n = 14,944$) are attributable to sample attrition. At 8th grade, there were 8809 completed parental interviews and 9358 child assessments (Tourangeau et al., 2009) and 68.9% of participating children are eligible for inclusion in the sample. To account for the possibility of sample selection due to attrition, I include the appropriate longitudinal sampling weights in regression analyses. Black and Latino children are less likely to be included in the final estimation sample, but children from rural areas and those with mothers married at the time of the baseline survey are more likely to be included in the final estimation sample.

3. Empirical approach

I begin with a simple empirical model describing the proposed relationships between asthma and obesity:

$$W_{it} = \beta_{0j} + \beta_{1j}H_{i0} + \beta_{2j}A_{it-l} + \beta_{3j}X_{it} + \beta_{4j}N_{it} + \varepsilon_{it}^w, \quad (1)$$

where weight status W_{it} is the dependent variable for child i at time t . Weight outcomes include BMI, overweight, and obesity. A child's weight status at time t is a function of birth weight (initial health), H_{i0} ; individual and family level factors (X_{it}), including gender, race/ethnicity, maternal education, and maternal marital status; having Medicaid-only insurance (to partially control for undiagnosed asthma); food insecurity status; and household income at kindergarten (baseline). In addition, I include an indicator for whether a child received a diagnosis of non-asthmatic hay fever at 3rd grade to control for propensity for allergy. I also include indicators for whether a child lived in a city or suburb, N_{it} . Weight is also a function of asthma, A_{it-l} , where A_{it-l} is permitted to vary in order to ascertain the associations between contemporaneous asthma, A_{it} ; lagged asthma, A_{it-l} ; and weight. Due to the structure of the data, in the main models, I only estimate a 1-period asthma lag A_{it-l} . I define asthma in two ways: "ever" having asthma, and, alternatively, whether a child received treatment for asthma during the period. I estimate continuous weight (BMI) using OLS regression and dichotomous weight outcomes (overweight/obese, overweight-only, obese-only) using logit regression. To test for Granger causality, I estimate an adaptation of Adams et al. (2003):

$$f(\Delta W_{it} | H_{i0}, A_{it-l}, X_{it}, N_{it}) = f(\Delta W_{it} | H_{i0}, X_{it}, N_{it}), \quad (2)$$

where W_{it} represents innovations (changes) in weight status. Asthma is Granger noncausal for weight outcomes, W_t , if lagged asthma fails to contribute predictive information on changes in weight outcomes. Changes in weight outcomes include change in BMI, becoming overweight, and becoming obese.

In supplementary analyses, I estimate the relationships between asthma and physical/sedentary health behaviors, P_{it} (including days of aerobic activity and hours of television, both estimated using OLS regression):

$$P_{it} = \beta_{0j} + \beta_{1j}H_{i0} + \beta_{2j}A_{it-l} + \beta_{3j}X_{it} + \beta_{4j}N_{it} + \varepsilon_{it}^p \quad (3)$$

I also test for Granger causality between physical/sedentary health behaviors and asthma:

$$f(\Delta P_{it}|H_{i0}, A_{it-l}, X_{it}, N_{it}) = f(\Delta P_{it}|H_{i0}, X_{it}, N_{it}). \quad (4)$$

Lastly, I also estimate the associations between asthma and weight status, where asthma is the outcome of interest:

$$A_{it} = \alpha_{i0} + \alpha_1 B_{i0} + \alpha_{it} W_{it-l} + \alpha_2 X_{it} + \alpha_3 N_{it} + \varepsilon_{it}^a \quad (5)$$

and test for Granger causality running from weight status to asthma:

$$f(\Delta A_{it}|H_{i0}, W_{it-l}, X_{it}, N_{it}) = f(\Delta A_{it}|H_{i0}, X_{it}, N_{it}). \quad (6)$$

4. Estimation results

4.1. Associations between asthma and prevalence of weight outcomes

In this section, I discuss the estimated associations between asthma and weight outcomes, where weight outcomes are the dependent variable. I estimate each model utilizing the panel form of the data. Rather than showing logit coefficients or odds ratios, I report average partial effects (APE), or marginal effects, for ease of interpretation and understanding (see Wooldridge (2002)). All results tables within the body of the paper report the APEs associated with asthma and weight status only. Complete models for BMI status regressed on asthma with the APEs for all of the covariates are located in Table A.3 in Appendix A; models for overweight and obesity regressed on asthma are available upon request. I incorporate appropriate sampling weights in all of the models.

Table 3 contains the first set of results from OLS (BMI) and logit (overweight-only, obese-only) regressions. A diagnosis of asthma “ever” in the prior survey waves is associated with a 1.248 point increase in BMI ($p < 0.01$). While lagged asthma is not significantly associated with overweight, there is a correlation between asthma and obesity of 9.0 percentage points ($p < 0.01$). Similarly, having received treatment for asthma in the previous period is associated with a slightly higher increase in BMI levels compared to asthma “ever” (1.380 percentage points, $p < 0.01$), but asthma treatment does not appear to be significantly associated with levels of overweight and obesity.

I also explore whether new reports of asthma are differentially associated with weight outcomes in the sample. Put differently, is a change in asthma status (new onset asthma or a need for asthma treatment) related to either BMI or the probability of being overweight or obese? As aforementioned, there are only two opportunities to observe weight changes or changes in asthma diagnoses (from 3rd to 5th grades and 5th to 8th grades), and I can only observe the associations between lagged new onset asthma and outcomes at eighth grade. Lagged new asthma is associated with a 1.235 point increase in BMI ($p < 0.05$) and a 6.4

percentage point increase in the probability of being overweight ($p < 0.10$). However, lagged new asthma is not significantly associated with overweight status.

The correlations between new asthma treatment and weight outcomes appear to be larger and more significant compared to new onset asthma. Lagged new asthma treatment is associated with a 1.641 point increase in BMI and a 8.2 percentage point increase in the probability of being obese ($p < 0.01$). However, counterintuitively, lagged new asthma treatment is associated with a small but significant decrease in the likelihood of being overweight (-0.4 percentage points, $p < 0.05$).

4.2. Changes in weight outcomes and granger causality

In the spirit of Granger (1969), Adams et al. (2003), and Smith (1999), I use the incidence of new weight outcomes to test for Granger noncausality of asthma on health outcomes. Here, if lagged asthma is related to future changes in weight, then I conclude that there may be evidence that asthma is Granger causal for weight outcomes. It is important to reiterate that these tests are not tests of true causality (particularly given the non-exogeneity of various health states), but simply a descriptive exercise to rule out noncausal relationships. Overall, the empirical findings in Table 4 suggest that asthma may be Granger causal for BMI and obesity. A diagnosis of asthma prior to the current wave is associated with a positive change in BMI (0.259 points, $p < 0.05$) and a 1.5 percentage point increase in the probability of becoming obese ($p < 0.10$).

There is also evidence that asthma onset is Granger causal for subsequent changes in weight status. Lagged new onset asthma corresponds to a 0.375 point increase in BMI ($p < 0.10$). However, lagged asthma onset is not associated with changes in overweight or obesity, and lagged new asthma treatment is not associated with weight outcomes.

4.3. Associations between asthma, aerobic activity and television viewing

In this section, I estimate the relationships between asthma and physical and sedentary behaviors. Given that the ECLS-K measures both asthma and obesity over a relatively short five-year time span, the influence of asthma on obesity (and visa versa) may be difficult to detect. Thus, I explore possible mechanisms through which pediatric asthma might influence future obesity. In this case, estimation samples are slightly smaller because some children's parents failed to report physical and sedentary activity at either 3rd, 5th, or 8th grades. In short, there are very few associations between lagged asthma and aerobic activity (or changes in aerobic activity), implying that asthma is not Granger causal for physical activity. Lagged diagnoses of asthma, new asthma, or newly treated asthma are uncorrelated with the number of days of aerobic activity or changes in the days of aerobic activity. Receiving treatment for asthma in the previous period has a small negative association with the number of days of aerobic activity (0.227 days, $p < 0.05$), but has no statistically significant associations with changes in days of aerobic activity (see Table 5).

I also find few associations between asthma and the amount of time that children spend watching television. There are no statistically significant associations between a lagged asthma diagnosis, lagged asthma onset, or asthma treatment and levels or changes in

television viewing behavior. However, I do find that lagged new asthma treatment is associated with watching nearly 52 min less (-0.865 h) of television per week ($p < 0.10$). Moreover, lagged new asthma treatment appears to be associated with a change of nearly 93 min less (-1.543 h) of television viewing per week ($p < 0.05$).

4.4. Relationships between weight status and asthma outcomes

In this section, I discuss the results of models that estimate the associations between weight status and asthma, where asthma serves as the outcome of interest. As before, asthma is measured in two ways: “ever diagnosed” with asthma or received treatment for asthma during the period. (See Table A.3 in Appendix A for the fully specified model of current asthma outcomes regressed on past BMI. Other results available upon request.)

4.4.1. Effects of weight status on asthma levels—Table 6 describes the relationships between weight status and ever having been diagnosed with asthma. A 1-point increase in lagged BMI is associated with a 0.7 and 0.6 percentage point change in the probability of an asthma diagnosis or asthma treatment, respectively ($p < 0.01$). Though overweight is not significantly associated with the prevalence of asthma or asthma treatment, obesity is associated with large increases in the prevalence of asthma (10.7 and 8.2 percentage points respectively, $p < 0.01$).

Changes in most weight outcomes are generally positively correlated with asthma status. For example, A 1-point change in lagged BMI associated with an increase in the probability of receiving an asthma diagnosis of 1.4 percentage points ($p < 0.01$) and a 1.1 percentage point increase in the likelihood of receiving asthma treatment ($p < 0.01$). As before, being overweight is not significantly associated with asthma prevalence. However, lagged changes in obesity are associated with a 9.4 percentage point increase in asthma prevalence ($p < 0.01$) and a 7.0 percentage point increase in asthma treatment prevalence ($p < 0.05$).

4.4.2. Changes in weight outcomes and Granger causality—Even when using lagged health states, it is challenging to disentangle Granger causal relationships between weight and asthma: whereas lagged asthma prevalence is strongly associated with changes in weight outcomes, various measures of weight status have small but statistically significant associations with new onset asthma. Lagged BMI is associated with a 0.2 percentage point increase in the probability of developing new onset asthma or new onset asthma treatment. Though lagged overweight is Granger noncausal for asthma outcomes, lagged obesity is positively associated with the likelihood of receiving first-time asthma treatment (1.5 percentage points, $p < 0.10$).

I, however, fail to find evidence that the onset of weight changes in prior periods are Granger causal for current asthma outcomes. Lagged BMI is positively associated with new onset asthma and first-time asthma treatment, but the results are small and statistically insignificant. Lagged onset of overweight is negatively associated with new asthma (and positively associated with first-time asthma treatment), but the results are statistically insignificant. Lastly, the lagged onset of obesity is negatively associated with new asthma and asthma treatment, but the results are statistically insignificant.

5. Conclusion and future research directions

This study uses longitudinal data to evaluate the evidence for statistically causal relationships among asthma, activity levels, and obesity among preadolescent children. I find that a past asthma diagnosis is Granger causal for BMI and obesity. My results also suggest that lagged asthma onset is Granger causal for positive changes in BMI. However, the mechanisms underlying this relationship are unclear, as I find little evidence that asthma is correlated with physical or sedentary activity. There are no statistically significant relationships between current or lagged asthma and the number of days of aerobic activity (or changes in aerobic activity), with the exception of the finding that lagged asthma treatment has a small negative association with days of aerobic activity. While some studies report that asthmatics are less likely to exercise and are less fit, others find that asthmatics are as likely (or more likely) to engage in high levels of activity (Gannotti et al., 2007; Lang et al., 2004). One explanation might be that more active children are more likely to receive an asthma diagnosis (Gilliland et al., 2003). I also fail to find statistically significant relationships among lagged asthma, new onset asthma, or asthma treatment, and aerobic activity. However, the results indicate that new asthma treatment is Granger causal for changes in television-viewing behavior. Counterintuitively, the relationship is negative, rather than positive. One reason might be that more recently diagnosed asthmatic children are more active and less sedentary (though the results in Table 5 do not lend strong support to this idea). I also examine the correlations between asthma and obesity when asthma serves as the outcome of interest. While levels of BMI and obesity are Granger causal for new onset asthma and first-time asthma treatment, changes in weight status are not Granger causal for asthma outcomes.

Taken together, the results suggest a more modest and nuanced relationship between pediatric asthma and obesity in childhood than previous research would imply (Gilliland et al., 2003; Zhang et al., 2010). While lagged weight is associated with the new onset of asthma, the *onset* of overweight or obesity does *not* necessarily imply the future onset of asthma – at least in the short term. This is a subtle distinction, but one worth emphasizing in this context. Moreover, lagged asthma levels are Granger causal for obesity, but only lagged new onset asthma is correlated with BMI. This finding implies that the lagged onset of asthma is positively associated with upward creep in BMI, though the mechanisms are unclear.

While this study has a number of strengths over previous studies (longitudinal data, precise measures of height and weight), there are several important limitations to consider. First, I lack important early data on asthma incidence and treatment. In a recent study using cross-sectional data, Suglia et al. (2011) detected relationships between asthma and obesity in a sample of children who were only three years of age. The ECLS-K survey only collects asthma data from 3rd grade on, and this limitation may have caused me to miss important relationships between obesity and asthma in early childhood. A goal of future research will be to examine asthma and obesity in younger children (e.g. from birth to kindergarten) so that I can observe the effects of early-life asthma diagnoses.

Second, measurement error is an important issue. Undiagnosed asthma is fairly common, and the ECLS-K survey does not contain data on wheezing or other asthma-related symptoms or whether a child has allergic or non-allergic asthma. Fletcher et al. (2010) report that correcting for measurement error using instrumental variable (IV) methods results in substantially larger estimates of the impact of asthma on adult health outcomes. Because poor and minority children are more likely to be underdiagnosed with asthma (Leong, 2006), I include a measure for Medicaid status. Despite the inclusion of Medicaid status, measurement error may have resulted in downwardly biased coefficient estimates on asthma in this study. In addition, previous researchers have found that while BMI is a convenient measure of obesity, it is not necessarily the best indicator of fatness, particularly among African-Americans (Burkhauser and Cawley, 2008; Wada and Tekin, 2010). However, in a study of obesity and asthma in school-aged children, Figueroa-Munoz et al. (2001) suggest that BMI may provide a better measure of fatness compared to skinfold thickness for studies of asthma and obesity because the increased amount of muscle and bone in obese children may influence the accuracy and repeatability of caliper measurements. Lastly, parental measures of children's activity levels are subject to measurement error (Dollman et al., 2009; Gortmaker et al., 1990) and may fail to accurately capture children's actual behaviors.

One of the more prominent issues is omitted variable bias. Granger causality is limited in its ability to detect anything beyond statistical causality. I include a number of SES-related variables in the models: child race/ethnicity, income, food insecurity, maternal education, and maternal marital status. While many of these variables are strongly related to both asthma and obesity, omitted characteristics of the home and neighborhood environment are likely also correlated with both asthma and obesity. There are unobserved factors related to socioeconomic status that are common to both asthma and obesity that would explain existing relationships. For example, poor housing conditions are positively associated with both asthma and obesity. Moreover, some studies suggest that the relationships between obesity and asthma vary substantially by race (Black et al., 2012).

Despite these caveats, this work provides an important step in disentangling the temporal relationships between obesity and asthma. The finding that asthma onset is related to subsequent changes in BMI supports the conclusions of Fletcher et al. (2010), Beckett et al. (2001), and Hasler et al. (2006). Further research should continue to explore the mechanisms through which childhood asthma may affect obesity and other health and economic outcomes, in addition to addressing causal relationships.

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Appendix A

See Tables A.1–A.3

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Table 1 Summary statistics-full sample ($N = 6452$).

Variable ^{a,b,c}	3rd grade	5th grade	8th grade
Child health outcomes/inputs			
Asthma, ever	0.12	0.16	0.20
New asthma	N/A	0.04	0.04
Treated asthma	0.09	0.11	0.11
Newly treated asthma	N/A	0.04	0.04
Overweight	0.20	0.24	0.21
Obese	0.13	0.15	0.15
Normal weight	0.67	0.61	0.64
Became overweight	N/A	0.10	0.09
Became obese	N/A	0.04	0.04
Became normal weight	N/A	0.03	0.09
BMI	18.50	20.45	22.81
BMI	N/A	1.96	2.36
Hay fever at 3rd grade	0.13	0.13	0.13
Child characteristics			
Child low birth weight	0.09	0.09	0.09
Child male	0.52	0.52	0.52
Child White, non-Hispanic	0.63	0.63	0.63
Child Black, non-Hispanic	0.13	0.13	0.13
Child Latino	0.17	0.17	0.17
Child Asian, non-Hispanic	0.04	0.03	0.03
Child other, non-Hispanic	0.04	0.04	0.04
Maternal characteristics			
Mom less than high school	0.11	0.09	0.09
Mom high school graduate	0.24	0.23	0.22
Mom some college	0.36	0.36	0.37
Mom college graduate	0.30	0.31	0.32
Education missing	0.03	0.03	0.03
Mom married	0.79	0.77	0.77
Marital status missing	0.03	0.02	0.02
Food insecure	0.07	0.10	0.09
Log income at kindergarten	10.48	10.48	10.48
Income missing	0.26	0.26	0.26
Medicaid	0.14	0.12	0.14
Medicaid missing	0.05	0.06	0.05
Geographic characteristics			
City	0.36	0.34	0.32
Suburban	0.42	0.43	0.43
Rural	0.23	0.24	0.25

Variable ^{a,b,c}	3rd grade	5th grade	8th grade
Location missing	0.04	0.09	0.13
Physical and sedentary behavior			
No. days of 20 min of aerobic activity	3.94	3.74	5.62
No. days aerobics missing	0.01	0.01	0.02
in no. days aerobic activity	N/A	−0.19	1.87
Hours television per week	6.57	7.12	7.27
No. hours television missing	0.01	0.01	0.03
in weekly hours television	N/A	0.52	0.20

^a All health variables are expressed in percentages except BMI and BMI, which are continuous.

^b All child, family, and location variables are expressed as means.

^c Summary statistics weighted using sampling weights provided in ECLS-K data.

Table 2

Asthma, weight status, and physical activity (N = 12,904)^a.

	Asthma		New asthma		Asthma treated		New treatment	
	Yes	No	Yes	No	Yes	No	Yes	No
Weight outcomes								
BMI	23.08	21.47	23.06	21.47	23.34	21.56	23.41	21.70
Overweight	0.22	0.23	0.24	0.21	0.22	0.22	0.22	0.22
Obese	0.24	0.14	0.23	0.14	0.27	0.14	0.27	0.15
Activity measures								
Days aerobics	4.70	4.63	4.69	4.64	4.61	4.65	4.74	4.64
Hours television	7.81	7.26	7.01	7.37	8.01	7.28	7.67	7.35

^a All health variables are expressed in percentages except BMI and ABMI, which are continuous.

Table 3

Effect of asthma on weight status at time *t*-levels (*N* = 12,904).

	BMI			Overweight			Obese		
	APE	Std. Error		APE	Std. Error		APE	Std. Error	
Asthma ever									
Asthma _{<i>t-1</i>}	1.248***	(0.302)		-0.010	(0.019)		0.090***	(0.022)	
1st Lag sig.	Yes			No			Yes		
New asthma									
New asthma _{<i>t-1</i>}	1.235**	(0.594)		-0.009	(0.042)		0.064*	(0.035)	
1st Lag sig.	Yes			No			Yes		
Asthma treatment									
Treatment _{<i>t-1</i>}	1.380***	(0.333)		0.006	(0.022)		0.006	(0.030)	
1st Lag sig.	Yes			No			No		
New treatment									
Treatment _{<i>t-1</i>}	1.641***	(0.627)		-0.004**	(0.050)		0.082***	(0.035)	
1st Lag sig.	Yes			Yes			Yes		

Note: Standard errors in parentheses. *N* = 6452 where new asthma treatment and new asthma are regressors.

***
p < 0.01.
**
p < 0.05.
*
p < 0.10.

Table 4

Effect of asthma on changes in weight status ($N = 6452$).

	BMI		Overweight		Obese	
	APE	Std. Error	APE	Std. Error	APE	Std. Error
Asthma ever $N = 12,904$						
Asthma _{<i>t-1</i>}	0.259**	(0.104)	-0.003	(0.011)	0.015*	(0.008)
1st Lag sig.	Yes		No		Yes	
New asthma						
New asthma _{<i>t-1</i>}	0.375*	(0.222)	0.004	(0.028)	-0.025	(0.016)
1st Lag sig.	Yes		No		No	
Asthma treatment						
Asthma _{<i>t-1</i>}	0.184	(0.132)	-0.005	(0.015)	0.006	(0.009)
1st Lag sig.	No		No		No	
New treatment						
New treatment _{<i>t-1</i>}	0.039	(0.280)	0.001	(0.027)	-0.028	(0.018)
1st Lag sig.	No		No		No	

Note: Standard errors in parentheses.

***
 $p < 0.01$.
**
 $p < 0.05$.
*
 $p < 0.10$.

Table 5

Effect of asthma on physical and sedentary activity.

	N = 12,551 Days aerobics		N = 6243 Days aerobics		N = 12,582 Hours TV		N = 6242 Hours TV	
	APE	Std. Error	APE	Std. Error	APE	Std. Error	APE	Std. Error
Asthma ever								
Asthma _{t-1}	-0.129	(0.083)	0.055	(0.077)	0.338	(0.247)	0.045	(0.245)
1st Lag sig.	No		No		No		No	
New asthma								
New asthma _{t-1}	-0.144	(0.162)	0.094	(0.259)	-0.223	(0.521)	-0.294	(0.565)
1st Lag sig.	No		No		No		No	
Asthma treatment								
Asthma _{t-1}	-0.227**	(0.095)	0.036	(0.102)	0.231	(0.306)	-0.042	(0.341)
1st Lag sig.	Yes		No		No		No	
New treatment								
New Treatment _{t-1}	-0.026	(0.157)	0.034	(0.256)	-0.865*	(0.518)	-1.543**	(0.643)
1st Lag sig.	No		No		Yes		Yes	

Note: Standard errors in parentheses. N = 6243 where new asthma treatment and new asthma are regressors on changes in aerobic activity. N = 6242 where new asthma treatment and new asthma are regressors on changes in television viewing.

***
p < 0.01.

**
p < 0.05.

*
p < 0.10.

Table 6

Effects of weight status on asthma ($N = 12,904$).

	Asthma			New asthma			Asthma treatment			New treatment		
	APE	Std. Error		APE	Std. Error		APE	Std. Error		APE	Std. Error	
BMI												
BMI _{<i>t-1</i>}	0.007***	(0.001)		0.002***	(0.001)		0.006***	(0.001)		0.002***	(0.001)	
1st Lag sig.	YES			YES			YES			YES		
BMI												
BMI _{<i>t-1</i>}	0.014***	(0.004)		0.002	(0.001)		0.011***	(0.003)		0.002	(0.002)	
1st Lag sig.	Yes			No			Yes			No		
Overweight												
Overweight _{<i>t-1</i>}	-0.002	(0.014)		-0.004	(0.006)		0.015	(0.012)		0.001	(0.007)	
1st Lag sig.	No			No			No			No		
Became overweight												
Became overweight _{<i>t-1</i>}	-0.039	(0.033)		0.007	(0.014)		-0.013	(0.028)		0.007	(0.014)	
1st Lag sig.	No			No			No			No		
Obese												
Obese _{<i>t-1</i>}	0.107***	(0.021)		0.004	(0.014)		0.082***	(0.018)		0.015*	(0.009)	
1st Lag sig.	Yes			No			Yes			Yes		
Became obese												
Became obese _{<i>t-1</i>}	0.094***	(0.036)		-0.019	(0.020)		0.070**	(0.028)		-0.024	(0.018)	
1st Lag sig.	Yes			No			Yes			No		

Note: Standard errors in parentheses, $N = 6452$ where changes in weight are regressors, $N = 6452$ where new asthma treatment and new asthma are outcomes.

***
 $p > 0.01$.

**
 $p > 0.05$.

*
 $p > 0.10$.

Table A.1

Derivation of final sample.

	Criteria for inclusion	Remaining sample size
1	Interviewed at baseline	21,396
2	Low birth weight status	20,525
3	Child race and gender	20,506
4	Parent assessment, 8th grade	8681
5	Child assessment, 8th grade	8325
6	Asthma, 3rd, 5th and 8th grades	7781
7	Weight and height, 3rd, 5th, 8th grades	6452
8	Final sample	6452 (27.9%)

Table A.2

Effect of asthma on weight outcomes ($N = 12,904$).

	BMI		Overweight		Obese	
	APE	Std. Error	APE	Std. Error	APE	Std. Error
Asthma ₋₁	1.248***	(0.302)	-0.010	(0.019)	0.090***	(0.022)
Low birth weight	-1.183***	(0.282)	-0.048**	(0.020)	-0.054***	(0.016)
Male	-0.016	(0.179)	0.002	(0.013)	0.035***	(0.012)
Child Black	1.469***	(0.388)	-0.008	(0.024)	0.086***	(0.029)
Child Latino	0.899***	(0.329)	0.017	(0.025)	0.073**	(0.028)
Child other race	0.729***	(0.241)	0.017	(0.019)	0.055***	(0.019)
Mom high school graduate	-0.782**	(0.372)	-0.048**	(0.023)	-0.022	(0.020)
Mom some college	-1.136***	(0.357)	-0.064**	(0.024)	-0.038**	(0.019)
Mom college degree	-1.705***	(0.371)	-0.094***	(0.024)	-0.060***	(0.021)
Education missing	-1.232**	(0.579)	-0.107***	(0.027)	-0.027	(0.030)
Medicaid	-0.037	(0.293)	-0.011	(0.020)	0.012	(0.017)
Medicaid missing	-0.001	(0.379)	0.019	(0.029)	-0.024	(0.024)
Married	-0.063	(0.247)	-0.026	(0.020)	0.007	(0.016)
Marital status missing	-0.338	(0.582)	-0.005	(0.043)	-0.027	(0.032)
Food insecure	0.648**	(0.301)	0.021	(0.022)	0.023	(0.018)
Income at kindergarten	-0.171**	(0.081)	-0.012**	(0.005)	-0.004	(0.004)
Income missing	0.442*	(0.228)	-0.005	(0.016)	0.025	(0.016)
Lives in city	-0.520*	(0.270)	-0.023	(0.017)	-0.007	(0.017)
Lives in suburb	-0.796***	(0.236)	-0.020	(0.016)	-0.032**	(0.015)
Location missing	-0.737**	(0.314)	-0.006	(0.025)	-0.036**	(0.018)
Hay fever at 3rd grade	0.205	(0.303)	-0.005	(0.020)	0.023	(0.021)
8th grade	2.342***	(0.053)	-0.029***	(0.009)	-0.001	(0.006)

Note: Standard errors in parentheses.

***
p > 0.01.

**
 $p > 0.05$.

*
 $p > 0.10$.

Green

Table A.3

Effect of BMI on asthma outcomes ($N = 12,904$).

	Asthma, ever			New asthma			Asthma treatment			New treatment		
	APE	Std. Error		APE	Std. Error		APE	Std. Error		APE	Std. Error	
BMI _{t-1}	0.007***	(0.001)		0.002***	(0.001)		0.006***	(0.001)		0.002***	(0.001)	
Low birth weight	0.036	(0.024)		0.018*	(0.010)		0.029	(0.019)		0.011	(0.010)	
Male	0.064***	(0.014)		0.001	(0.005)		0.052***	(0.011)		0.001	(0.006)	
Child Black	0.035	(0.026)		-0.013*	(0.007)		0.019	(0.020)		0.000	(0.009)	
Child Latino	-0.001	(0.027)		-0.000	(0.010)		-0.002	(0.023)		-0.001	(0.011)	
Child other Race	-0.023	(0.018)		-0.011*	(0.007)		-0.024*	(0.013)		-0.012*	(0.007)	
Mom high school graduate	0.025	(0.029)		-0.001	(0.010)		0.014	(0.023)		0.003	(0.012)	
Mom some college	0.050*	(0.029)		-0.002	(0.010)		0.047*	(0.024)		0.005	(0.012)	
Mom college degree	0.025	(0.030)		0.003	(0.011)		0.031	(0.027)		0.007	(0.013)	
Education missing	-0.021	(0.038)		0.030	(0.027)		-0.021	(0.033)		0.014	(0.026)	
Medicaid	0.027	(0.019)		0.011	(0.008)		0.035**	(0.015)		0.016*	(0.009)	
Medicaid missing	-0.027	(0.030)		-0.018	(0.016)		-0.056**	(0.025)		-0.012	(0.016)	
Mom married	-0.008	(0.017)		-0.013	(0.008)		-0.020	(0.014)		-0.014*	(0.008)	
Marital status missing	0.060	(0.055)		-0.020***	(0.008)		-0.015	(0.031)		-0.006	(0.017)	
Food Insecure	0.038*	(0.022)		0.010	(0.010)		0.033*	(0.017)		0.014	(0.012)	
Income at Kindergarten	-0.006	(0.005)		0.001	(0.002)		-0.004	(0.004)		-0.001	(0.002)	
Income missing	-0.020	(0.017)		0.016**	(0.007)		-0.009	(0.013)		0.011	(0.007)	
Lives in City	0.001	(0.019)		0.000	(0.007)		0.031*	(0.017)		0.005	(0.008)	
Lives in suburb	-0.008	(0.017)		-0.011*	(0.006)		0.002	(0.013)		-0.005	(0.007)	
Location missing	0.014	(0.024)		0.010	(0.012)		0.035	(0.023)		0.014	(0.014)	
Hay fever at 3rd grade	0.220***	(0.027)		0.044***	(0.010)		0.140***	(0.020)		0.044***	(0.012)	
8th grade	0.021***	(0.005)		-0.002	(0.006)		-0.013**	(0.006)		-0.006	(0.006)	

Note: Standard errors in parentheses. $N = 6452$ where new asthma treatment and new asthma are regressors.

***
**
*
p > 0.01.

**
 $p > 0.05$.

*
 $p > 0.10$.

Green