Respiratory Effects of Relocating to Areas of Differing Air Pollution Levels

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We studied 110 children (59 boys and 51 girls, who were 10 yr of age at enrollment and 15 yr of age at follow-up) who had moved from communities participating in a 10-yr prospective study of respiratory health (The Children’s Health Study [CHS]) to determine whether changes in air quality caused by relocation were associated with changes in annual lung function growth rates. The subjects were given health questionnaires and underwent spirometry in their homes across six western states, according to a protocol identical to evaluations performed annually on the CHS cohort in school. Changes in annual average exposure to particulate matter with a mean diameter of 10 μm (PM$_{10}$) were associated with differences in annual lung function growth rates for FEV$_{1}$, maximal mid-expiratory flow, and peak expiratory flow rate. As a group, subjects who had moved to areas of lower PM$_{10}$ showed increased growth in lung function and subjects who moved to communities with a higher PM$_{10}$ showed decreased growth in lung function. A stronger trend was found for subjects who had migrated at least 3 yr before the follow-up visit than for those who had moved in the previous 1 to 2 yr. We conclude that changes in air pollution exposure during adolescent growth years have a measurable and potentially important effect on lung function growth and performance.

Keywords: air pollution; respiratory system; growth and development

The establishment of appropriate regulatory standards to protect the public health from ambient air pollution requires credible health information based on carefully performed studies. Long-term community studies tracking health outcomes in free-living populations (1, 2) offer a unique opportunity to make highly relevant health assessments in this regard. However, such studies may be complicated by practical limitations, such as loss to follow-up of portions of the study population through subject withdrawal or relocation (3). Subject withdrawal or relocation may inadvertently introduce bias through the loss of specific subgroups within the general study population, such as sensitive individuals who may perceive themselves as being at greater increased risk if they remain in the study area. Moreover, subjects who relocate from one region to another can experience appreciable changes in ambient pollutant exposure by moving from an area of higher pollution to one characterized by lower ambient pollutant levels or vice versa.

In 1993, the Children’s Health Study (CHS) was initiated to assess the potential chronic respiratory effects of ambient air pollution in children living in Southern California (4). Associations between air pollution and various health-related outcomes have been previously reported by several investigators (5–16). Our group recently reported that children aged 10 to 14 yr and living in areas of higher ambient air pollution (areas with higher levels of particulate matter with a mean diameter of 10 μm [PM$_{10}$], NO$_{2}$, and acids) have measurably slower annual rates of lung function growth, as measured by mean annual changes in FEV$_{1}$, forced expiratory flow at 75% of FVC (FEF$_{25}$), or maximal midexpiratory flow (MMEF) (17).

Adolescence is a time of rapid lung function growth for both males and females, with age-related increases in lung function leveling off by the late teen years for females and by the early twenties for males (18). On the basis of our previous findings relating annual lung function growth rates to air pollution exposure during adolescence, we wanted to determine whether changes in pollution exposure during this period of rapid lung growth might affect rates of growth. If ambient air pollution was demonstrably affecting longitudinal lung function growth rates, we speculated that residential relocation (along with an increase or decrease in ambient air pollution exposure) might be reflected in measurable changes in lung function performance.

METHODS

Subjects were drawn from a 10-yr longitudinal respiratory health study of children living in Southern California (4, 5), who enrolled in 1993 at the age of 10 yr or in 1994 at the age of 11 yr. Subject participation was approved by the University of Southern California Institutional Review Board for Human Studies, after receipt of written informed consent from subjects and their parents or guardians. To participate, subjects had to have been CHS participants, to have had one or more years of acceptable CHS lung function data, and to have moved away from the CHS communities at least 1 yr before follow-up. Ambient pollution data had to have been obtainable for the subject’s current residential location at the time of the follow-up study. To control costs, subjects had to have moved no farther away than California, Arizona, Nevada, Oregon, Washington, or Utah. On the basis of these criteria, 164 subjects were eligible for study. Of these, 149 subjects responded to phone or mail contact, 15 declined to participate, and 110 subjects were tested.

Subjects were assigned pollution scores on the basis of annual average 24-h NO$_{2}$, daily average PM$_{10}$ mass, and average daytime (10 a.m. to 6 p.m.) O$_{3}$ levels in their current and former communities. Annual averages for former communities of residence were based on 1994 data, whereas averages for current communities of residence were represented by 1998 values. Differences between current and former community O$_{3}$, NO$_{2}$, and PM$_{10}$ levels were calculated and used in pollutant-specific analyses representing subject-specific changes in ambient exposure. Negative pollution scores reflected moves to areas of lower pollution (i.e., the current community of residence had lower ambient levels of O$_{3}$, NO$_{2}$, or PM$_{10}$ than did the former community). Similarly, positive pollution scores represented moves to communities with higher air pollution levels.

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Study participation involved completing a written questionnaire, responding to a computerized health interview, and performing maximal-effort spirometry. Spirometry was accomplished with 12-L rolling seal spirometers (Model 232; Morgan Instruments, Andover, MA) interfaced to laptop computers. Subjects performed up to seven maximal exhalations from a seated position to provide three acceptable and consistent maneuvers. From these, the largest maneuver was used to determine FEV₁ in milliliters, FVC in milliliters, MMEF in milliliters per second, and peak expiratory flow rate (PEFR) in milliliters per second. Testing was preceded and followed by flow calibrations done with a 3-L volumetric syringe (Model FVC3000; Jones Instruments, Oakbrook, IL). This testing approach has been previously described and is being used in the CHS longitudinal study (5).

Testing was performed between January and June 1998, to parallel annual CHS testing. Testing of children who moved was typically accomplished on weekend mornings in their respective homes, whereas CHS testing was performed on weekday mornings in neighborhood schools. Testing involved 110 subjects who moved, consisting of 59 boys (age: 10.2 ± 0.5 [mean ± SD] yr at baseline and 15.1 ± 0.4 yr at follow-up) and 51 girls (age: 9.9 ± 0.4 yr at baseline and 14.9 ± 0.4 yr at follow-up).

Annual average changes in lung function were individually determined by subtracting subjects’ baseline values from their follow-up values and dividing by the difference in age at testing. Linear regression was used to determine whether annual average changes in lung function correlated with average changes in pollution. Models included adjustments for sex, race, CHS entry year, annual average change in height, weight and body mass index (BMI), and the interaction of sex with annual average change in height. Hypothesis tests were performed at the p = 0.05 level, assuming a two-tailed alternative hypothesis.

RESULTS

A summary of 1994 average air pollution levels for PM₁₀, O₃, and NO₂ in the 12 CHS communities appears in Table 1. Annual PM₁₀ levels varied from 15 μg/m³ in Lompoc to more than 66 μg/m³ in Mira Loma. Annual daytime (10 a.m. to 6 p.m.) ozone levels varied from 30 ppb in Santa Maria to 71 ppb in Lake Gregory. Daily 24-h average NO₂ levels ranged from 5 ppb in Lompoc to 43 ppb in Upland. Also listed in Table 1 are the numbers of subjects from each community that participated in the follow-up study, and their mean change in pollutant score. As expected, children who originated in high-pollution communities tended to move to lower-pollution communities, giving them on average a negative pollution score. This underscores the observation that air quality in several Southern California communities has historically been poorer than in many other residential locations throughout the western United States. Conversely, there was a subset of communities (Atascadero, Santa Maria, and Lompoc) from which children who moved generally experienced a positive change in pollution score (i.e., they moved to an area of higher pollution).

As shown in Figure 1, increasing exposure to PM₁₀ was associated with decreased rates of annual growth in MMEF (p = 0.04), and PEFR (p = 0.007), and with marginally decreased rates of annual growth in FEV₁ (p = 0.06). For each increase of 10 μg/m³ in the annual average 24-h PM₁₀, annual lung function growth was estimated to decrease by 6.6 ml for FEV₁, 16.6 ml/s for MMEF, and 34.9 ml/s for PEFR. These effect estimates, as well as those for NO₂ and O₃, are shown in Table 2. Although increases in NO₂ and O₃ were also estimated to reduce lung function growth rates, none of these effects was statistically significant at the 5% level.

We then assessed the importance of community of origin in predicting changes in annual lung function growth. Based on PM₁₀ data collected during the early years of the study (and summarized in Table 1), we divided the 12 originating communities into tertiles of “high,” “medium,” and “low.” Changes in annual average lung function growth across these strata were compared with changes in PM₁₀ levels between former and current communities of residence. For subjects who moved from communities of high or low PM₁₀, changes in PM₁₀ levels between the original and current communities of residence (in either a positive or negative direction) were reflected in statistically significant changes in rates of annual growth in MMEF (Figure 2). Most subjects originating in communities of medium PM₁₀ experienced modest changes in PM₁₀, and for these subjects no significant association was detected between annual lung function growth and change in PM₁₀ levels. Similar associations were observed for PEFR and FEV₁. We also explored the potential association between annual lung function growth rate and NO₂ or O₃ levels, but none of these was significant (p = 0.06). As shown in Table 2, PM₁₀, O₃, and NO₂ were associated with decreased growth rates of both MMEF and PEFR, with NO₂ being the most consistently associated with decreased lung function growth.

### Table 1. Summary of Ambient PM₁₀, O₃, NO₂ Concentrations for Subject Baseline Communities and Relative Change in Pollutants as a Result of Subject Relocation

<table>
<thead>
<tr>
<th>Subject’s Originating Children’s Health Study Community</th>
<th>ML</th>
<th>UPL</th>
<th>RIV</th>
<th>LB</th>
<th>SD</th>
<th>LEL</th>
<th>LAN</th>
<th>ALP</th>
<th>LG</th>
<th>ATA</th>
<th>SM</th>
<th>LOM</th>
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<tbody>
<tr>
<td>No. of subjects</td>
<td>12</td>
<td>8</td>
<td>14</td>
<td>10</td>
<td>10</td>
<td>6</td>
<td>10</td>
<td>11</td>
<td>6</td>
<td>7</td>
<td>9</td>
<td></td>
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<tr>
<td>Baseline Levels*</td>
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<tr>
<td>PM₁₀ (μg/m³)</td>
<td>66.2</td>
<td>46.0</td>
<td>43.4</td>
<td>38.0</td>
<td>36.6</td>
<td>34.6</td>
<td>28.7</td>
<td>23.9</td>
<td>21.9</td>
<td>21.2</td>
<td>19.8</td>
<td>15.0</td>
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<tr>
<td>NO₂ (ppb)</td>
<td>25.7</td>
<td>43.2</td>
<td>28.7</td>
<td>35.6</td>
<td>39.8</td>
<td>19.8</td>
<td>19.2</td>
<td>12.9</td>
<td>7.2</td>
<td>13.4</td>
<td>11.2</td>
<td>4.6</td>
</tr>
<tr>
<td>O₃ (ppb)</td>
<td>56.7</td>
<td>54.0</td>
<td>63.0</td>
<td>30.7</td>
<td>60.8</td>
<td>58.9</td>
<td>47.3</td>
<td>58.1</td>
<td>70.8</td>
<td>42.2</td>
<td>30.4</td>
<td>41.0</td>
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<td>Change in Pollutant Score†</td>
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<tr>
<td>PM₁₀</td>
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<td></td>
</tr>
<tr>
<td>Mean</td>
<td>−32.9</td>
<td>−9.7</td>
<td>−8.7</td>
<td>−18.7</td>
<td>−0.5</td>
<td>−24.</td>
<td>2.1</td>
<td>0.5</td>
<td>11.0</td>
<td>8.6</td>
<td>11.4</td>
<td>9.5</td>
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<tr>
<td>Range, min</td>
<td>−48.9</td>
<td>−17.0</td>
<td>−21.5</td>
<td>−22.9</td>
<td>−15.8</td>
<td>−29.</td>
<td>−8.2</td>
<td>−4.0</td>
<td>−4.9</td>
<td>−1.5</td>
<td>2.2</td>
<td>0.6</td>
</tr>
<tr>
<td>Range, max</td>
<td>−21.1</td>
<td>5.5</td>
<td>20.5</td>
<td>5.0</td>
<td>21.8</td>
<td>14.7</td>
<td>10.3</td>
<td>28.1</td>
<td>32.4</td>
<td>27.0</td>
<td>28.4</td>
<td>37.5</td>
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<tr>
<td>NO₂</td>
<td></td>
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</tr>
<tr>
<td>Mean</td>
<td>−1.3</td>
<td>−13.4</td>
<td>−1.6</td>
<td>−0.3</td>
<td>−14.1</td>
<td>7.7</td>
<td>1.8</td>
<td>9.2</td>
<td>16.5</td>
<td>4.4</td>
<td>7.6</td>
<td>13.9</td>
</tr>
<tr>
<td>Range, min</td>
<td>−10.6</td>
<td>−28.5</td>
<td>−13.6</td>
<td>−12.7</td>
<td>−28.2</td>
<td>0.6</td>
<td>−7.6</td>
<td>−1.3</td>
<td>8.0</td>
<td>−5.5</td>
<td>−4.1</td>
<td>5.4</td>
</tr>
<tr>
<td>Range, max</td>
<td>19.5</td>
<td>0.2</td>
<td>19.3</td>
<td>4.6</td>
<td>3.7</td>
<td>23.9</td>
<td>11.5</td>
<td>20.5</td>
<td>32.8</td>
<td>13.8</td>
<td>32.2</td>
<td>25.1</td>
</tr>
<tr>
<td>O₃</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Mean</td>
<td>−11.0</td>
<td>−7.6</td>
<td>−20.5</td>
<td>−7.4</td>
<td>−14.5</td>
<td>−13.5</td>
<td>−3.7</td>
<td>−17.0</td>
<td>−27.0</td>
<td>1.8</td>
<td>11.7</td>
<td>−0.6</td>
</tr>
<tr>
<td>Range, min</td>
<td>−29.2</td>
<td>−14.4</td>
<td>−42.1</td>
<td>−0.4</td>
<td>−23.2</td>
<td>−22.1</td>
<td>−11.3</td>
<td>−30.3</td>
<td>−44.1</td>
<td>−10.8</td>
<td>2.5</td>
<td>−10.7</td>
</tr>
<tr>
<td>Range, max</td>
<td>2.3</td>
<td>−0.1</td>
<td>2.1</td>
<td>27.8</td>
<td>−5.3</td>
<td>−5.1</td>
<td>5.8</td>
<td>−1.5</td>
<td>10.0</td>
<td>8.9</td>
<td>22.7</td>
<td>6.8</td>
</tr>
</tbody>
</table>

Definition of abbreviations: ALP = Alpine; ATA = Atascadero; LAN = Lancaster; LB = Long Beach; LE = Lake Elsmore; LG = Lake Gregory; LOM = Lompoc; M = Mira Loma; RIV = Riverside; SD = San Dimas; SM = Santa Maria; UPL = Upland.

* Mean 1994 ambient pollution levels for CHS community monitoring station.† Mean 1998 ambient pollution level in community of relocation (follow-up testing) and 1994 (baseline) pollutant level.
and time elapsed since moving. To perform this analysis, we
partitioned the study population into two groups, consisting
of: (1) those who had moved away from their former com-
communities within the past 1 or 2 yr, and (2) those who had moved
away from their former communities at least 3 to 5 yr previ-
ously. A trend toward an increasing PM10 effect with increas-
ing time away from the former community of residence was
suggested for all lung function measures, but observed differ-
ences between the two groups were not statistically significant.

We compared several baseline medical and residential his-
tory characteristics of the 110 subjects who constituted the
movers group in the study with those of their CHS peers who
remained in the CHS communities and continued their study
participation ("stayers"). As summarized in Table 3, there
were no marked differences between the two groups in an-
thropomorphic, health history, or home exposure variables.
There was a significant difference in the distribution of race
remained in the CHS communities and continued their study
between the two groups were not statistically significant.

TABLE 2. EFFECT OF CHANGES IN PM10, NO2, or O3 ON AVERAGE ANNUAL LUNG FUNCTION GROWTH RATES

<table>
<thead>
<tr>
<th></th>
<th>PM10 24-h Average</th>
<th>NO2 24-h Average</th>
<th>O3 10 a.m. to 6 p.m., Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVC, ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean change1</td>
<td>-1.8</td>
<td>-2.7</td>
<td>-1.4</td>
</tr>
<tr>
<td>95% CI</td>
<td>-9.1, 5.5</td>
<td>-12.9, 7.5</td>
<td>-10.8, 8.0</td>
</tr>
<tr>
<td>FEV1, ml</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean change1</td>
<td>-6.6</td>
<td>-8.2</td>
<td>0.1</td>
</tr>
<tr>
<td>95% CI</td>
<td>-13.5, 0.3</td>
<td>-17.8, 1.4</td>
<td>-8.7, 8.9</td>
</tr>
<tr>
<td>MMEF, ml/s</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean change1</td>
<td>-16.6*</td>
<td>-10.7</td>
<td>-3.4</td>
</tr>
<tr>
<td>95% CI</td>
<td>-32.1, -1.1</td>
<td>-3.8, 11.4</td>
<td>23.6, 16.8</td>
</tr>
<tr>
<td>PEFR, ml/s</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean change1</td>
<td>-34.9*</td>
<td>-23.6</td>
<td>-8.9</td>
</tr>
<tr>
<td>95% CI</td>
<td>-59.8, -10.0</td>
<td>-59.5, 12.3</td>
<td>-41.6, 23.8</td>
</tr>
</tbody>
</table>

Definition of abbreviations: MMEF = maximal midexpiratory flow; PEFR = peak expi-
atory flow rate; PM10 = particulate matter with a mean diameter of 10 μm.
* Changes shown are per 10 units of pollutant, respectively.
† p < 0.05.
‡ p < 0.01.

Figure 1. Effect of changes in PM10 on adjusted annual lung function growth for FEV1, FVC, MMEF, and PEFR in all moved subjects studied.

DISCUSSION

The association reported here between PM10 and growth in
lung function is consistent with results recently reported by our
group for the larger cohort of subjects active in the CHS who
remain in their original communities (17). That investigation
found that children living in areas of higher ambient PM10-
NO₂, and acids had lower rates of annual lung function growth. Results from the current investigation indicate that during the teen years of development, the rate of lung function growth can be altered by a large change in exposure to air pollution.

Our observations about longer-term effects of pollutants on respiratory health in subjects moving to different areas of ambient air pollution are consistent with a report by Kinney and Lippmann (19). Their study assessed the respiratory health of U.S. Military Academy cadets who trained in several different regions of the United States during summer and were therefore exposed to different levels and kinds of regional air pollution. Kinney and Lippmann observed seasonal declines in respiratory function, and related the observed changes to outdoor exposure to ozone and particles.

Our current study found changes in lung function of a similar magnitude to that in our analyses of 4 yr of follow-up of subjects still actively participating in the longitudinal health study (17). In the current study, only PM₁₀ was identified as playing a statistically significant role. The importance of acids could not be assessed because of the scarcity of available data on ambient acid levels in the communities to which our subjects had moved. The results of this study, together with those of the numerous previously reported investigations of PM₁₀ and its association with increased morbidity and mortality (20–23), underscore the national concern about particulate exposure and its relation to public health.

In the present study, we could not show that the duration of the period since the subject moved to a new community was statistically associated with changes in observed rates of annual lung function growth. The data did, however, show a trend consistent with this hypothesis. A more convincing test of this hypothesis would require larger sample sizes or longer follow-up periods. Plans are currently being made to accomplish this.

The analyses reported here suggest that previously observed changes in annual lung function growth rates (17) may be reversible during the period of rapid lung growth accompanying physical development during the teen years. Differences in annual respiratory growth rates during adolescence may be important predictors of respiratory health in later adult years. Hibbert and coworkers have argued that the development of several lung function indices in healthy children follows a consistent “track,” increasing at a constant rate relative to those

![Figure 2. Effect of changes in PM₁₀ on annual lung function growth for MMEF, stratified by tertiles of PM₁₀ concentrations in subjects’ baseline community of origin.](image-url)
of other healthy children (24). Data from the Six Cities Studies (25) also support the concept of tracking, but suggest that adolescents might deviate from predicted growth curves because of variations in the onset of the adolescent growth spurt. Nevertheless, the investigators who reported these data suggested that tracking was informative and of potential clinical use. Lebowitz and colleagues (26) reported that childhood respiratory illness and smoking (either active or passive) were important factors in childhood respiratory growth, but that with the exception of active smoking, these factors had negligible effects on tracking of subjects’ values over time. Conversely, Borsboom and colleagues reported that the large intradividual variation in the timing of growth spurts and the rates of growth precluded the application during adolescence of any averaged values to the study of ventilatory function (27). In their study of Dutch schoolboys, the authors found that the large variability in age of peak growth, and the time lag between growth in height and ventilatory function, were not adequately explained in commonly used reference values.

Among respiratory pollutants, tobacco smoke has been shown to adversely affect the growth of respiratory function in children. In a study of New Zealand children 9 to 15 yr of age who were exposed to passive smoking, Sherrill and associates reported a reduction in growth of the ratio of FEV1 to VC, but no significant changes in absolute FEV1 or VC related to either active or passive smoking (28). In a longitudinal health investigation of children in East Boston, Tager and coworkers reported a clear decline among children 9 to 14 yr old at the time of annual examination, in the predicted percent growth for those who were smokers versus those who were nonsmokers (29). In that study, predicted FEV1 decreased by 2.8% per year in children who smoked as compared with those who did not smoke. Slightly larger decreases were predicted for FEF25–75.

To compare the relative effects observed in the study by Tager and colleagues with the findings in our current work, we considered a white male CHS subject of average height, weight, and BMI who was a mover. For this hypothetical individual, an annual growth rate of 287 ml/yr in FEV1 and 291 ml/s in MMEF would be expected. On the basis of the results presented in Table 2, we would predict a reduction of 2.3% per year in FEV1 growth and a reduction of 5.7% per year in MMEF growth for every 10 μg/m3 increase in PM10 for this child. This would suggest that ambient air pollution exposure has a similar magnitude of effect on lung function development to that previously observed for children who are active smokers.

Previous studies of the etiology of respiratory disease have suggested that individuals with limited respiratory capacity are at increased risk for earlier onset of a range of respiratory maladies. The reduction in annual respiratory growth rates with increased pollution exposure observed in the present study may reduce the level of lung function attained and lead ultimately to an increased risk of respiratory events in adulthood. However, it is also possible that early deficits will be reversed with subsequent accelerations in growth rate or a longer growth period. Earlier work by Borsboom and coworkers revealed that adolescents with a history of respiratory symptoms exhibited annual lung growth on a growth curve parallel with but lower than that of asymptomatic peers (30). Burrows and colleagues have suggested that “as the twig is bent, the tree inclines” (31). Our data suggest that annual lung function growth rates can change with exposure to pollutants, but whether these changes in rates somehow compensate for slowed growth is unknown. Drawing upon data for the respiratory effects of smoking (32), one might conclude that in the absence of the exposure insult, a return to nominal (but not accelerated) rates of growth (or decline) would ensue. If this is true, then periods of slowed lung growth, even during periods of peak respiratory growth (such as those experienced by males and females in their teen years), may have lifelong implications for health.

The results reported here support the view that changes in ambient pollution levels (in this case, PM10) may have measurable effects on longer-term lung function (and health) outcomes. The relative importance of the changes observed in the present study, and the potential health implications of these observed changes for the later lives of children, support the need for studies to confirm and extend these observations.

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