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Neighborhood Factors During Adolescence: Modest Effects On Cardiovascular Risk, Small Impact On Obesity And Depression

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Abstract

Researchers increasingly recognize the importance of neighborhoods as a potential cause of noncommunicable disease, although the long-term impacts of early neighborhood exposures are not well understood. We used data from the prospective, nationally representative National Longitudinal Study of Adolescent to Adult Health to calculate correlations in health outcomes during adulthood among individuals who attended the same school in adolescence. These upper-bound estimates suggest that the amount of variation attributable to early neighborhoods is small in the case of adult obesity and depressive symptoms and small or modest in the case of cardiovascular risk in early adulthood. Taken together, our results suggest that the benefits of neighborhood-based interventions during adolescence in alleviating the burden of these noncommunicable diseases are likely to be relatively small. Our methods also provide a useful framework for future work that seeks to produce similar estimates on other adult conditions and for future policy discussions that attempt to compare the likely impacts of early neighborhood-level interventions across various adult outcomes.

Researchers increasingly recognize the importance of neighborhoods in the development of noncommunicable diseases^{1,2} and as a source of disparities in disease burden.^{3,4} Living in a distressed neighborhood—one with high rates of poverty and crime and with lack of access to high-quality food and health services—is associated with a higher prevalence of many noncommunicable diseases, including obesity,⁵ hypertension,⁶ cardiovascular disease,^{7,8} and depression.^{9,10} These neighborhood influences have the potential to produce long-term effects through a range of mechanisms, both physical (for example, environmental exposures and the quality of the built environment) and social (for example, exposure to violence).²

Perhaps the best evidence for the existence of neighborhood influences comes from the Moving to Opportunity for Fair Housing demonstration,¹¹ a randomized trial that provided housing assistance for families living in high-poverty urban neighborhoods. Ten to fifteen

years after moving out of distressed neighborhoods, adults in the demonstration's treatment groups showed improvements in rates of obesity and diabetes,¹² physical functioning, and mental health.^{13,14}

Children of these families have shown mixed results in health outcomes, although economic outcomes may have improved.¹⁵ In long-term follow-up, there were no detectable differences between control and treatment groups in physical health outcomes.¹⁶ However, treated girls showed improvements in rates of major depression and conduct disorder, while treated boys were found to have higher rates of major depression and conduct disorder than boys in the control group.¹⁷

This work has increased the already growing interest in neighborhood interventions as a means of improving population health and reducing the burden of noncommunicable diseases.¹⁸ Examples of such interventions include housing assistance,¹⁹ changing the physical environment to encourage greater physical activity,^{20,21} and reducing the prevalence of "food deserts."^{22,23} These interventions are generally motivated by hopes of improving population health in the short run.

However, focusing on short-term improvements might underestimate potential benefits. A substantial literature has found that individuals who live in a disadvantaged neighborhood during childhood have poor physical and mental health later in life,^{24–28} which suggests that a child's neighborhood plays a role in shaping his or her future health. Understanding the magnitude of these early neighborhood influences could be valuable in setting expectations about and targeting resources for improving population health.

In this article we provide a novel estimate of the importance of neighborhoods during adolescence in the development of noncommunicable diseases later in life. Researchers have noted the difficulty of defining the total impact of neighborhoods with measurements for a limited number of neighborhood attributes,² such as the percentage of people living in poverty. Estimating neighborhood influences through direct measurement of neighborhood attributes can also lead to biased estimates because of the difficulty of separating family or individual factors from neighborhood importance per se (for example, separating the effects of poverty of one's own family from the effects of poverty of one's neighbors). Because of these issues, we took an alternative approach to estimating the total impacts of neighborhoods.

We extended into the context of adult health outcomes methods used in studies that examined the importance of a child's neighborhood in determining educational attainment.^{29,30} The purpose of the exercise was to provide an upper-bound estimate of the likely importance of adolescent neighborhoods on adult health outcomes, as opposed to understanding the particular sources of neighborhood effects (sources such as poverty, the built environment, and exposure to crime). We used the term neighborhood effects to denote associations that may or may not represent causal processes.

To provide evidence on the importance of the adolescent neighborhood's influences on later health outcomes, we estimated correlations in the health outcomes of young adults among people who lived in the same neighborhoods during adolescence. To the extent that

neighborhoods affect later health, people who shared the same neighborhood in adolescence should be more similar in their health outcomes during young adulthood than people who did not share the same neighborhoods in adolescence. The correlations we estimated assessed the magnitude of this similarity, with a correlation of 0 suggesting no impact of the shared neighborhood environment and a correlation of 1 suggesting the maximum impact. This is consistent with two explanations: Adolescents within a neighborhood have similar family and individual influences that cause later disease; and the shared neighborhood exposures are the source of the similarity. While large correlations can be suggestive of neighborhood influences, small correlations would be inconsistent with the existence of important neighborhood effects.

The estimated correlations produced an upper-bound estimate of the influence of neighborhoods because the analysis was unable to separate the influence of shared neighborhood environments from the effects of shared family backgrounds in circumstances where families were not randomly assigned to neighborhoods.²⁷

For example, if rich families are likely to live in health-enhancing neighborhoods, the estimated correlations will combine the effects of family-level income on health with the effects of neighborhood-level attributes on health.

To provide additional context for our results, we also compared schoolmate correlations in adult health outcomes with sibling correlations. The latter correlations combine the impacts of sharing a family environment and sharing a neighborhood. Thus, they are expected to be larger in magnitude than the schoolmate correlations and to provide upper bound effects of sharing both a neighborhood and family environment (including genes).

The upper-bound effects we estimated are useful for policy makers in several circumstances. First, finding upper-bound estimates that are small in magnitude for a particular adult health outcome suggests a likely lack of early neighborhood influence on the health outcome. Second, it is possible to compare correlations across health outcomes to gain a sense of the potential differences in the impacts of neighborhoods on later health outcomes (for example, obesity versus depression). This, in turn, might suggest the types of neighborhood interventions that are likely to be most effective (for example, improving built environments versus tighter controls on noise pollution to reduce stress).

Study Data And Methods

DATA

Our data came from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is a nationally representative sample of over 15,000 individuals who were enrolled in middle or high school in 1994. The participants have been surveyed in four waves, from 1994 to 2009. A complete description of the study design is available elsewhere.³¹

The study's primary sampling unit is schools, instead of individuals or households within a geographic area. Our approach took advantage of the geographically based local catchment

areas of most high schools in the United States to capture information for individuals within geographic neighborhoods (that is, census tracts) as well as the influence of sharing a common school environment for approximately eight hours a day for 180 days a year during adolescence. Our calculations (available upon request) suggest that nearly 66 percent of the sample attended schools in which all students lived in at most two distinct census tracts and that nearly 95 percent of the sample attended schools in which all students lived in fewer than five distinct census tracts.

The study's design also identifies sibling pairs who attended the same school during the data collection window. This allowed us to conduct a comparative analysis of sibling correlations in later health outcomes that combined the influence of neighborhoods or schools and the influence of families.

Because the Add Health data are nationally representative, the sociodemographic characteristics of the sample mirror those of other large representative data sets. Exhibit 1 presents summary statistics for the schoolmate-pair data set.

METHODS

Following previous research,^{29,30} we calculated correlations in health outcomes during young adulthood between pairs of individuals who attended the same middle or high school. A second comparative analysis estimated correlations in health outcomes during young adulthood for siblings in our sample. We described above why this approach was expected to estimate the upper-bound influence of neighborhoods. A more formal explanation is provided in the online Appendix.³²

As additional clarification, we outline some examples of potential neighborhood effects of interest and how they were captured in our analysis. Neighborhood air quality might affect an adolescent's likelihood of developing pulmonary disorders later in life. This effect would apply to everyone living within a neighborhood—for example, siblings and schoolmates. Thus, we would expect former schoolmates to be more similar in their pulmonary outcomes, compared to others. Furthermore, adolescents living in a household with a parent who smokes experience increased risk of developing pulmonary disorders. One would expect siblings within a house-hold in which a parent smokes to have similar risk. However, the extent to which schoolmates have similar risk depends on the clustering of smokers within a neighborhood. Thus, these neighborhood risks as well as family-level risks that happen to be clustered in particular neighborhoods (but are not necessarily neighborhood effects) would be captured in our schoolmate correlations, providing an upper bound that conflated the neighborhood-level (air quality) and family-level (parental smoking status) contributors to these children's later health outcomes.

If family or individual factors (such as parental smoking status) were observed and statistically controlled for in the models, the neighborhood correlation would decrease. However, the inability to measure and separately estimate all family-and neighborhood-level contributors to health is a motivation for our focus on acknowledging our estimates as providing an upper bound.

By comparing sibling and schoolmate correlations, we accomplish two goals. First, we provide an upper-bound estimate on the importance of adolescent neighborhood and family influences on cardiovascular risk, obesity, and depressive symptoms later in life. Second, we can assess the relative importance of total (both observed and unobserved) family and neighborhood influences on these health outcomes. This makes our study an advance on prior work, which has typically examined only a small number of neighborhood or family measures.

Several types of results would be informative. If the upper-bound estimates based on schoolmate correlations and sibling correlations were small for a particular adult health outcome, we might infer that early environmental interventions would have limited effects on the adult health outcome. However, if schoolmate correlations were small and sibling correlations were large, we might infer that early family-based interventions could be effective and early neighborhood-based interventions are likely to be ineffective for the specific health outcome.

If we found large correlations in both school-mates and siblings for a particular health outcome, we might infer that there was strong evidence of early influences on the health outcome but conclude that further studies were necessary to uncover the specific source or sources of the effects. For example, a correlation of 0.01 in the schoolmate pairs would suggest that at most 1 percent of the variation in a given outcome could be attributed to neighborhood-level effects.

OUTCOMES

We first examined three outcomes relevant to the development of noncommunicable disease: cardiovascular risk, obesity, and scores on standardized depression scales.

CARDIOVASCULAR DISEASE: We used the thirty-year risk for hard cardiovascular disease—that is, myocardial infarction, stroke, or death from cardiovascular problems—based on the Framingham Heart Study, as our measure for cardiovascular risk.³³ These scores are estimates of the probability that an individual will experience hard cardiovascular disease within the next thirty years. The scores were developed based on a prospective cohort of individuals ages 20–59 at baseline who were followed for thirty years. While the members of our population were younger on average than the original Framingham cohort, they were within the range of ages used to develop these scores.

Ours was not the first study to estimate thirty-year risk among the Add Health sample. Cari Jo Clark and coauthors also estimated the thirty-year risk for cardiovascular disease among the sample and calculated risk scores similar to those we used in our analysis.³⁴

Framingham Heart Study scores are calculated from an algorithm whose inputs are sex, age in years, systolic blood pressure, use of antihypertensive treatment, current smoking status, diagnosis of diabetes, and body mass index (BMI). Trained field interviewers measured height, weight, and systolic blood pressure of the Add Health population during wave 4 of the study.

The data do not contain direct measures of treatment. Therefore, use of hypertensive treatment was assumed if a respondent reported ever having been told by a doctor or nurse that he or she had hypertension or high blood pressure. An analogous assumption was made for treatment for diabetes. Respondents who reported regularly smoking in the previous thirty days were defined as smokers. Respondents with a previous diagnosis of cancer or heart disease were excluded from the analysis, as were respondents with one or more of the inputs missing.

OBESITY: Obesity was defined as having a BMI of over 30. A respondent's BMI was calculated based on his or her measured height and weight during wave 4 of the study.

DEPRESSIVE SYMPTOMS: Depressive symptoms were measured using a modified version of the Center for Epidemiologic Studies—Depression (CES-D) scale,³⁵ a standardized measure based on self-reports of feelings over the previous seven days. Add Health participants rated their agreement with statements such as, “During the past seven days I felt depressed.” Scores across these statements were summed, with higher scores indicating more intense depressive feelings. The CES-D scale has been validated as a reliable measure for depressive symptoms among young adults, despite using information from only the previous seven days.³⁶

OUTCOME BENCHMARKS: Our approach was based on previous research that examined differences in educational attainment and labor-market outcomes among neighboring and sibling pairs.^{29,37} Thus, we benchmarked our results against these studies by also calculating correlations in educational attainment among school-mate pairs and among sibling pairs in the Add Health sample. This allowed us to compare our estimates with the results of previous work and to compare the relative importance of family and neighborhood influences on health and on educational outcomes.

LIMITATIONS

This study had several limitations. First, we could not directly estimate correlations in cardiovascular disease because respondents were generally too young to have developed a clinically relevant disease. As a result, we used Framingham thirty-year risk measures, which were developed using a cohort that might be different from the sample we based our estimates on. However, the American College of Cardiology Foundation and the American Heart Association have endorsed the use of Framingham thirty-year risk measures as a general measure for cardiac risk.³⁸

Second, our outcome measure used self-reports of medical diagnoses that might contain measurement error. However, in unreported results using BMI or systolic blood pressure as our outcomes, we still found negligible potential for neighborhood effects. This provides evidence that our conclusions are robust to the outcome used.

Third, our method could not identify the specific factors that influence cardiovascular disease at each level. Sibling correlations combine family, neighborhood, and school influences and do not reveal information about the process. Family and community have a stronger influence on an adolescent's future schooling than on young adult health outcomes.

(that is, genetic or otherwise) through which familial or nonfamilial influences cause siblings to be more alike than two individuals picked at random from the population.³⁰

Finally, mobility across neighborhoods during childhood might increase measurement error in the exposure variable and thus attenuate the correlations. However, this effect has been shown to be small in related research.^{30,39}

Study Results

At wave 4 of Add Health, the mean Framingham Risk Score in our sample was 0.05 (Exhibit 1). Forty percent of people in the sample were obese, and the mean depressive scale score was 2.63. Participants had completed an average of 14.19 years of schooling.

The correlation for cardiovascular risk for the schoolmate pairs was 0.05 and for siblings was 0.29 (Exhibit 2). The schoolmate pair correlation suggests that the upper-bound proportion of variance in adult cardiovascular risk that could be attributed to neighborhood influences from adolescence is 5 percent. One way to interpret this result is that interventions that completely equalized adolescent neighborhood environmental factors related to later cardiovascular risk would likely leave at least 95 percent of the variation in cardiovascular risk unaffected. Likewise, the sibling pair correlation suggests that up to 30 percent of the variance in cardiovascular risk can be attributed to the combination of neighborhood and family influences.

For adult obesity the correlations were 0.02 for the schoolmate pair and 0.29 for the sibling pair. For the depressive scale scores they were 0.01 and 0.12, respectively (Exhibit 2). The very small schoolmate correlations suggest a very limited role for adolescent neighborhood conditions in explaining population variation in adult obesity and adult depressive symptoms.

The correlation in educational attainment at wave 4 was 0.51 for siblings and 0.08 for school-mates (Exhibit 3). Our sibling correlations were remarkably similar to estimates from other data sets. Bhashkar Mazumder found a correlation of 0.6 in years of schooling among sibling pairs,³⁷ and Gary Solon and coauthors found correlations of approximately 0.5 among sibling pairs and 0.1 among former neighbors.²⁹

These estimates suggest a larger, but still relatively modest, role for policies that equalize neighborhood conditions across the population to reduce variation in educational attainments, compared to the role of such policies in reducing variation in health outcomes. Our estimates mirror those in the literature, suggesting that complete equalization would leave over 90 percent of the variation in educational attainment remaining.

Discussion

Our results demonstrate positive correlations across all pair types. This is unsurprising, given the many ways that shared family and neighborhood environments might affect individuals' future health status. Also unsurprising is the finding that correlations of the sibling pairs were consistently larger than those of the schoolmate pairs. That difference partially reflects

the fact that individuals from the same family will be more similar than individuals from the same neighborhood.

Our goal was to compare the relative importance of neighborhood influences in the development of a variety of noncommunicable diseases. To do this, we compared the relative size of the sibling and schoolmate pair correlations across outcomes. A large sibling correlation combined with a small schoolmate correlation would suggest that family effects were likely a principal governing force in determining outcomes and that adolescent neighborhood and school effects were likely small.^{29,30}

Our results suggest that this is the case for adult obesity and depressive symptoms. Our upper-bound estimates suggest that less than 1 percent of the variation in depressive symptoms and only 2 percent of the variation in obesity among our sample could be explained by shared neighborhoods in adolescence. We found a larger, but still modest, possible importance of adolescent neighborhoods in determining adult cardiovascular risk.

We found much larger results among our educational outcomes than among health outcomes. This difference suggests that family and community have a stronger influence on an adolescent's future schooling, where our estimates replicate those in the literature,^{29,37} than on young adult health outcomes. Specifically, up to 10 percent of the variation in education might be explained by neighborhoods of origin and up to 50 percent by the combination of family and neighborhood factors.

Conclusion

This study estimated the importance of neighborhood influences during adolescence in the development of a range of noncommunicable diseases during adulthood. Our results might be interpreted as upper-bound estimates of the effect of a neighborhood in adolescence on our set of noncommunicable diseases in early adulthood. We conclude that neighborhood factors during adolescence have modest effects on adult cardiovascular risk but likely have small effects on the risk of obesity and depressive symptoms in adulthood. Furthermore, elucidating the exact mechanisms through which family background and experience, such as family income, influence cardiovascular health, as well as how neighborhood environmental factors, such as neighborhood-level income, influence longer-term health outcomes, might help explain the association between neighborhood and cardiovascular health. ■

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design of the original survey in 1994. Information on how to obtain Add Health data files is available on the Add Health website (<http://www.cpc.unc.edu/addhealth>).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Family and community have a stronger influence on an adolescent's future schooling than on young adult health outcomes.

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Outcomes And Demographic Characteristics Of A Schoolmate-Pair Sample From The National Longitudinal Study Of Adolescent To Adult Health

EXHIBIT 1

	Mean or percentage	Standard deviation	Minimum	Maximum
OUTCOMES				
Framingham Risk Score for 30-year hard CVD ^a	0.05	0.04	0.01	0.65
Depressive symptoms ^b	2.63	2.57	0	15
Obesity at wave 4 ^c	40%	— ^d	0	1
Years of schooling	14.19	1.96	8.00	21.00
DEMOGRAPHIC CHARACTERISTICS				
Age at wave 4 (years)	29.60	1.51	24.25	34.67
Mother's years of schooling	12.99	2.45	0.00	17.00
Female	52%	— ^d	0	1
White	38%	— ^d	0	1
Black	21%	— ^d	0	1
Hispanic	26%	— ^d	0	1
Other race/ethnicity	15%	— ^d	0	1
Family income (\$10,000s)	4.47	3.43	0.00	99.00
Parents married	72%	— ^d	0	1

SOURCE Authors' analysis of data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; see Note 31 in text).

NOTES Framingham Risk Scores are on a scale of 0.00 to 1.00 and can be interpreted as predicted probabilities of a serious cardiovascular event in the next thirty years. Depressive symptoms are based on a subset of the nine-item Center for Epidemiologic Studies—Depression scale (see Note 35 in text), whose scores are on a scale of 0 to 27. Higher scores indicate a more depressed mood in the previous thirty days and are not an indicator of clinical depression. For each outcome or demographic characteristic, there were 1,852,138 observations, except as noted. Hard CVD is hard cardiovascular disease (myocardial infarction, stroke, or death from cardiovascular problems). Wave 4 is the most recent wave of Add Health.

^a1,655,503 observations.

^b1,880,500 observations.

^c1,779,317 observations. Obesity is body mass index >30.

^dNot applicable.

EXHIBIT 2

Correlations In Noncommunicable Diseases, By Pair Type

Type of pair	Observations	Correlation	95% CI
FRAMINGHAM RISK SCORE FOR 30-YEAR HARD CARDIOVASCULAR DISEASE			
School	1,622,900	0.0524	(0.0509, 0.0538)
Sibling	2,531	0.2882	(0.2384, 0.3422)
Obesity at wave 4			
School	1,807,300	0.0199	(0.0184, 0.0214)
Sibling	2,781	0.2901	(0.2337, 0.3401)
CENTER FOR EPIDEMIOLOGIC STUDIES—DEPRESSION SCALE			
School	1,880,500	0.0076	(0.0062, 0.0091)
Sibling	2,860	0.1150	(0.0600, 0.1716)

SOURCE Authors’ analysis of data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; see Note 31 in text).

NOTES The correlations are those for each pair-type set of individuals as calculated by Gary Solon and coauthors in their weighting scheme 1 (see Note 29 in text). This weighting scheme estimates covariances by giving equal weight to each group (for example, each sample family within a neighborhood), regardless of the number of individuals in the group. Framingham Risk Scores and the Center for Epidemiologic Studies—Depression Scale are explained in the notes to Exhibit 1. Obesity is body mass index >30. Wave 4 is the most recent wave of Add Health. CI is confidence interval.

EXHIBIT 3

Correlations In Years Of Schooling, By Pair Type

Type of pair	Observations	Correlation	95% CI
School	1,881,400	0.0841	(0.0828, 0.0856)
Sibling	2,862	0.5070	(0.4493, 0.5616)

SOURCE Authors' analysis of data from the National Longitudinal Study of Adolescent to Adult Health (see Note 31 in text).

NOTES The correlations are explained in the notes to Exhibit 2. CI is confidence interval.