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# Like Parent, Like Child: Intergenerational Patterns of Cardiovascular Risk Factors at Midlife

**Todd M. Jensen, PhD**,
School of Social Work, University of North Carolina at Chapel Hill, Chapel Hill, NC

**Naomi N. Duke, MD, PhD, MPH**,
Department of Pediatrics, Duke University, Durham, NC

**Kathleen Mullan Harris, PhD**,
Department of Sociology & Carolina Population Center, University of North Carolina at Chapel Hill, Chapel Hill, NC

**V. Joseph Hotz, PhD**,
Department of Economics, Duke University, Durham, NC

**Krista M. Perreira, PhD**
Department of Social Medicine, University of North Carolina School of Medicine, Chapel Hill, NC

## Abstract

**Purpose:** We aimed to assess the prevalence of four cardiovascular risk factors (obesity, diabetes, excessive alcohol intake, and cigarette smoking) for parents and their adult children at the same approximate midlife age. We also evaluated associations of parents' cardiovascular risk factors, childhood health exposures, and social contexts (i.e., family, school, and neighborhood) during adolescence with adult children's cardiovascular health at midlife.

**Methods:** We used data from respondents at Wave V of the National Longitudinal Study of Adolescent to Adult Health (Add Health) who had corresponding parent (mostly mothers) data from Wave I. The final sample included 10,466 adult children with a mean age of 37.8 years. Descriptive statistics and logistic regression models were estimated accounting for the Add Health sampling design.

**Results:** At similar ages (i.e., 35-45) to their parents, adult children had higher rates of excessive drinking and obesity than their parents, lower rates of diabetes, and similar rates of smoking. Adult children's health largely converged and correlated with their parents' health at similar ages. Cardiovascular risks for adult children were also significantly associated with their childhood

**Corresponding Author:** Correspondence concerning this article should be addressed to Todd M. Jensen, 325 Pittsboro St. CB# 3550, Chapel Hill, NC 27516; office phone number: 919-962-6543; toddm.jensen@gmail.com.

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health exposures and social contexts during adolescence. Some associations varied with respect to the health status of parents at Wave I.

**Conclusions:** The cardiovascular risk of parents at midlife is strongly associated with the cardiovascular risk of their adult children at midlife. The status of parents' health during adolescence can also modify the significance and magnitude of associations between childhood health exposures or adolescent social contexts and adult children's cardiovascular risk factors.

## Keywords

Cardiovascular/cardiometabolic health; obesity; diabetes; alcohol; smoking; adolescence

Our understanding of health as a complex interplay between genetic inheritance, socio-developmental context, and gene-environment interaction continues to evolve [1,2]. The intergenerational transfer of health is of increasing concern, given the enduring impact of intergenerational disadvantage as a source of persistent and widespread health disparities [3,4]. Links in the fortunes of parents and children have led to calls for using 2- and 3-generational approaches for reducing health disparities [1,5]. To inform strategies to enhance the health of future generations, the multi-generational approach emphasizes the need for research to identify factors related to parent-child concordance and discordance in health outcomes.

Current conceptions of health benefit from the integration of health, biological, and social science disciplines. Life course research has advanced our understanding of the significance of early life context in shaping health and social status in adulthood. Socially patterned environmental and economic exposures occurring during key periods of development track individuals and groups into health differentials and trajectories of health decline [6,7]. Familial genetic predispositions overlay these environmental and economic exposures [8]. Thus, incorporating the role of familial genetic predisposition in research models of adult health that also focus on early socio-environmental contexts provides a significant contribution to our knowledge of health development over the life course.

Family members share genes and ecological context. Intergenerational links in health status result from variable contributions of these and related factors including health behaviors and habits. Risks for diseases such as diabetes, cancer, and heart disease run in families, and the health trajectories of parents and their children are highly correlated [8]. For example, parental history of cardiovascular disease (CVD) is a well-established risk factor for cardiovascular events in adult offspring [9-11], and cardiovascular risk factors are observed to track from parents to their adult children [12]. Owing to the obesity epidemic, a growing research base describes the intergenerational transmission of body mass and obesity [13]. Moreover, researchers have long documented intergenerational linkages in health behaviors, such as alcoholism, drug use, smoking, and physical activity [14-17]. To understand health outcomes and health risks at individual and group levels, it is essential to study linkages in health across generations [14,17,18]. But these linkages are a complicated mixture of genetic and biological processes, patterns of behavior, and choice of social environments [19]. This complexity suggests the necessity of comprehensive data spanning biological, behavioral, and contextual domains to identify and monitor health linkages across and within

generations. Research providing evidence for the intergenerational transfer of health using longitudinal, population-representative, and multi-generational self-report data remains limited.

Most evidence of the intergenerational linkages in health are based on data from one generation derived from retrospective case-control studies or longitudinal studies that rely on offspring self-report of parental health events, such as in the case for CVD [20-23]. Such reports have been found to be unreliable due to recall bias and lead to inflated estimates of intergenerational effects [24,25]. When data on two generations are available, such as in the Framingham Study, estimates of the intergenerational correlation in CVD are reduced, but still indicate increased risk for cardiovascular events among adult children whose parents had CVD [10]. This evidence suggests a genetic predisposition for CVD that is shared by parents and children, but also implies there are shared environmental and behavioral factors.

The present study uses innovative new data from two generations to examine intergenerational associations of CVD risk. With both parent and adult child reports on CVD risk, anthropometric measurement, and family, school, and neighborhood context, the present study addresses many of the limitations of the current literature and provides insight into how the health of adult children at midlife compares to the health of their parents at midlife. [26].

We focus on four adult CVD risk factors: obesity, diabetes, excessive alcohol intake, and cigarette smoking. Our choice of outcomes reflects recognition of CVD as the leading cause of morbidity and mortality in the United States (U.S.) and our outcome measures represent four of the principal risk factors for heart disease, a leading cause of death for men and women in the U.S. [27,28].

In our examination of intergenerational associations of CVD risk, we hypothesize that CVD risks of the parents will be positively associated with CVD risks in their adult children when they are at the same approximate midlife age. Additionally, we hypothesize that disadvantageous family socio-economic, school, and neighborhood contexts in early life will be positively associated with adult children's CVD risks. Because the role of disadvantaged contexts for children's future health risks might be modified by parents' health risks, we also explore parents' CVD risk as effect modifiers.

## Methods

### Data and Sample

We used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a school-based study with a nationally representative sample of adolescents in grades 7 through 12 during the 1994-1995 school year [26]. In-home data were collected from a randomly selected subset of adolescents on school rosters, yielding a total of 20,745 Add Health respondents at Wave I (W1) and a corresponding sample of 17,670 Add Health parent respondents (mostly mothers). In-home data from youth were collected across four additional waves, with Wave V (W5) being the most recent (2016-2018).

Our analysis relied on Add Health respondents from W5 ($n$ = 12,300) who had corresponding parent data available from W1 ($n$ = 10,712). After multiple imputation of missing data, our analytic sample included 10,466 (98%) adult children (49% female) whose average age was 37.8 years and racial/ethnic identities reflected national distributions (69% Non-Hispanic [NH] White; 15% NH Black; 12% Hispanic).

## Measures

**Adult Child's CVD Risk Factors.—**We created four dichotomous measures of adult children's CVD risk factors at W5: excessive drinking (i.e., drinking every or almost every day or binge drinking two or more days/month during the past 12 months) [29], smoking (i.e., any amount of cigarette use during the past 30 days), obesity (measured Body Mass Index (BMI)>=30kg/$m^2$), and diabetes (i.e., ever told by a health care provider that you have high blood sugar or diabetes).

**Parent's CVD Risk Factors.—**We used congruous measures of parents' CVD risks at W1. Parents were classified as drinking excessively when they reported drinking nearly every day or binge drinking two or more days during the past month. They were classified as smokers when they indicated that they smoked; parents classified as non-smokers indicated that they did not smoke (or that there were no smokers in the household). Parents were classified as obese when the parent respondents indicated that either the youth's biological mother or father were obese. Parents were classified as diabetic when parents indicated that either the youth's biological mother or father had diabetes.

**Adult Child's Demographic Background.—**Using data from Add Health respondents at W1 and W5, we measured the adult child's age in years, self-reported gender, and whether the child's parents were foreign-born versus U.S.-born. We categorized self-reported racial/ethnic identity as Hispanic, NH Black, NH Other (i.e., Asian, Native American or Other), and NH White.

**Childhood Health Exposures.—**Using data from Add Health parents at W1, we controlled for the adult child's birthweight measured in pounds and duration of breastfeeding during infancy (i.e., none, 1-6 months, and 6 months or longer). Using data from Add Health respondents from W1 to W4, we also created a count of seven adverse childhood experiences (ACEs) prior to age 18: (1) parental incarceration, (2) parental death, (3) witness of violent crime, (4) victim of violent crime, (5) physical abuse, (6) sexual abuse, or (7) removal from home by social services.

**Adolescent Family Context.—**Family context measures pertained to Add Health respondents' experiences at W1 (i.e., adolescence). Family socioeconomic status (SES) included family income, use of public assistance (indicated retrospectively at W3 or W4), and whether at least one parent had completed college. We also identified whether the adolescent had lived in a household with two biological/adoptive parents (vs. another family structure such as a stepfamily, single-parent family, or foster family). We averaged four items on closeness, warmth, communication satisfaction, and overall relationship satisfaction with both mothers and fathers (and only the parent respondent in single-parent

households) to create a scale of youth-reported parent-child closeness, ranging from values of 1 (“strongly disagree”/”not at all”) to 5 (“strongly agree”/”very much”) [30].

**Adolescent School Context.—**Intergenerational closure measured the percent of parents in a given school at W1 that indicated speaking with three or more of their child’s friends’ parents in the last four weeks [31]. We also measured the percent of respondents’ grademates (i.e., those in the same grade within the same school) at W1 who (a) drank excessively (i.e., any amount of drinking among those younger than 18; among those 18 and older, near daily alcohol use or being drunk two or more times in a month) during the past 12 months, (b) smoked at all during the past 12 months, or (c) exercised six or more days per week. Youth in schools without typical grading structures were clustered into four age groups (i.e., 13 or younger, 14 to 15, 16 to 17, and 18 or older). Intergenerational closure was negatively correlated with grademates’ excessive drinking ($r$=−.11), not correlated with grademates’ smoking, and positively correlated with grademates’ exercise ($r$=.50).

**Adolescent Neighborhood Context.—**All neighborhood context measures were measured at the Census tract using either reports by Add Health respondents at W1, their parents at W1, or 1990 Census data. To measure neighborhood collective efficacy, we summed reports by parents on two 4-point ordinal items, each ranging from 0 (“definitely would not”) to 4 (“definitely would): (1) whether parents would notify neighbors if neighbors’ child getting into trouble and (2) whether others would tell the parent if their child was getting into trouble. Neighborhood social cohesion measured the percent of Add Health respondents living in a tract who indicated that “people in the neighborhood look out for each other.” Following Martin and colleagues [32], the neighborhood disadvantage index was a count ranging from 0 to 5 of whether 1990 Census data indicated a tract was in the top quartile of the distribution in poverty, female-headed households, unemployment, adults without a high school diploma or equivalent, and use of public assistance/welfare. Neighborhood collective efficacy was positively correlated with both social cohesion ($r$=.16) and neighborhood disadvantage ($r$=.15).

## Analysis

We estimated the prevalence of CVD risk factors for adult children at W5, corresponding measures for parents at W1, and weighted bivariate polychoric correlations between generations. We then created a 4-category variable for each CVD risk factor to indicate whether, at similar ages, both generations had the risk factor, did not have the risk factor, or only one generation had the risk factor. These variables allowed us to evaluate intergenerational correspondence for each risk factor. Next, we regressed (logit function) each adult child’s CVD risk factor on the parent’s corresponding risk factor, controlling for child’s demographic background; childhood health exposures; and adolescent family, school, and neighborhood contexts. We then re-estimated each logistic regression after stratifying the sample by whether parents had the CVD risk or not, and examined associations between the adult children’s risk factors and early life conditions.

Using Stata 14.2 survey estimation procedures, all analyses incorporated sampling weights and accounted for within-school clustering. Only 6% of all data points were missing;

multiple imputation was used to handle missing data. In additional sensitivity analyses, we evaluated the potential influence of contextual variables representing type of community (e.g., urban, rural, suburban), school cohesion, school problems, school-level racial/ethnic identity proportions, school-level parent education, and parental community involvement. These variables were non-significant and omitted in favor of more parsimonious models. We also evaluated whether contextual influences in our model varied by the respondent's gender or race/ethnicity and found no significant interactions. Institutional Review Board processes categorized the current study as non-human subjects research.

## Results

### Prevalence of CVD Risk Factors in Parents and Adult Children

At approximately the same age, adult children at W5 had higher rates of excessive drinking than their parents at W1 (21.0% vs. 8%,), but smoking rates did not differ significantly between adult children at W5 and their parents at W1 (Table 1). Obesity was more prevalent among adult children at W5 than among their parents at W1 (41.0% vs. 23.0%); however, diabetes was slightly more prevalent among parents at W1 than among their adult children at W5 (7.6% vs. 5.8%).

### Correspondence Between CVD Risk Factors in Parents and Adult Children

Most adult children's CVD risk factors at W5 matched their parents' risk factors at W1 (Table 2)—both did not drink excessively (74%) or both did not smoke (55%). Less commonly, both drank excessively (2.4%) and both smoked (12%). Similarly, most adult children and their parents were similar with respect to either both not being obese (49.3%) or both being obese (13.0%), and both being diabetic (87.5%) or both not being diabetic (1.0%). At the same time, we did observe that adult children at W5 frequently engaged in excessive drinking (18.7%) or smoking (16.0%) when their parents at W1 had not. Moreover, 27.7% of adult children at W5 became obese even though their parents at W1 were not obese, and 4.8% became diabetic even though their parents were not diabetic.

### Associations Between Adult Child's and Parents' CVD Risk Factors

Logistic regressions highlighted the strong association between parents' health at W1 and their adult children's health at W5 (Table 3). Adult children had a higher odds of drinking excessively at W5 when a parent reported drinking excessively at W1; a higher odds of smoking at W5 when a parent reported smoking at W1; a higher odds of being obese at W5 when either their biological mother or father was obese at W1; and a higher odds of being diabetic at W5 when either their biological mother or father was diabetic at W1. These strong associations persisted with controls for the adult child's demographic background, childhood health exposures, and family, school, and neighborhood contexts during adolescence.

### Adult Children's Excessive Drinking

Among adult children at W5 whose parents were excessive drinkers at W1 (Table 4), we found lower odds of excessive drinking for females (compared to males), the children of immigrants (compared to the children of U.S. natives), and those identifying as NH Black

(compared to those identifying as NH White). Adult children with greater odds of excessive drinking at W5 lived in biological or adoptive families with two parents during adolescence, had been breast fed 6 months or longer, and lived in neighborhoods with greater social cohesion during adolescence. Among adult children at W5 whose parents were *not* excessive drinkers at W1 (Table 5), we found lower odds of excessive drinking for females and those who reported higher levels of parent-child closeness in adolescence.

### Adult Children's Smoking

Among adult children at W5 whose parents smoked at W1 (Table 4), we found lower odds of smoking for females, those identifying as Hispanic, those identifying as NH Black, and those whose parents had at least a college education at W1 compared to males, those identifying as NH White, and those whose parents had less than a college education, respectively. Adult children with greater odds of smoking at W5, regardless of parent smoking at W1, had lived through more ACEs and had grown up in more disadvantaged neighborhoods during adolescence. Among adult children at W5 whose parents did *not* smoke at W1 (Table 5), older age, being female, identifying as Hispanic or NH Black, breastfeeding for 6 months or longer in early childhood, living with a biological or adoptive two-parent family in adolescence, a higher family income in adolescence, and a parent with at least a college education at W1 were associated with lower odds of smoking. Finally, percent of grademates that smoked and neighborhood social cohesion had slight positive associations with adult children's smoking.

### Adult Children's Obesity

Among adult children at W5 with obese parents at W1 (Table 4), we found that breastfeeding 1-6 months (versus none) and higher family incomes were associated with a lower risk of obesity. ACEs and neighborhood disadvantage were associated with a higher risk of obesity. Among adult children at W5 whose parents were *not* obese at W1 (Table 5), being female, identifying as Hispanic, identifying as NH Black, birthweight, and use of public assistance during adolescence were positively associated with obesity. A parent having at least a college education at W1 was negatively associated with obesity.

### Adult Children's Diabetes

Because of the small number of parents and adult children with diabetes, we had limited power to detect risk factors associated with diabetes. Among adult children at W5 whose parents had diabetes at W1 (Table 4), collective efficacy was negatively associated with adult children's diabetes. Among adult children at W5 whose parent did *not* have diabetes at W1 (Table 5), being female, higher birthweight, and breastfeeding 1-6 months (versus none) were associated with lower odds of diabetes. Identifying as NH Black and living in a two-parent biological or adoptive family during adolescence were associated with higher odds of diabetes.

## Discussion

This study examined intergenerational linkages between parent's and children's health at the same approximate life course stage—midlife. Conditional on these intergenerational

linkages, we then explored how background characteristics, childhood health exposures, and social contexts (i.e., family, school, and neighborhood) during adolescence were associated with health at midlife. As hypothesized, we found that parents' cardiovascular risks during midlife were positively associated with their adult children's cardiovascular risks during midlife. Moreover, adult children's cardiovascular risks were significantly associated with their childhood health exposures and social contexts during adolescence and these associations varied depending on the health status of their parents at W1.

Overall, our study showed that the midlife cardiovascular risk of adult children can be foreshadowed by the cardiovascular risk of their parents at midlife. Previous research has found predispositions for CVD and some cardiovascular risk factors are shared by parents and their children [9-18]. However, many of these studies relied on either adolescent's reports or adult children's retrospective cross-sectional reports of their parents disease histories and risk factors [20-25]. Our study utilizes data collected separately from parents and from their children in a nationally-representative longitudinal study.

We also document that associations between parent and child cardiovascular risk at midlife result, in part, from the influence of parent's health and health behaviors on their children's health exposures [4,5,19]. We found that children's birthweight, breastfeeding duration, and exposures to ACEs each contributed to their adult cardiovascular risk. Previous studies also have found associations between low birthweight and obesity, between high birthweight and diabetes, between short breastfeeding duration and obesity, and between exposure to ACEs and a variety of cardiovascular risk factors [33-37].

Moreover, parents' health and lifestyle can influence the family, school, and neighborhood contexts in which children live [5,32]. Consistent with this influence, we found that family, school, and neighborhood contexts in adolescence had long-lasting effects associated with adult cardiovascular risk factors up to 20 years later. The influence of family structure and SES on health is perhaps one of the most enduring findings in the literature [3,5,8,30]. Though previous research is inconclusive about the influence of school and neighborhood context on health, previous studies have found associations between peer smoking and smoking initiation in adolescence as well as between neighborhood disadvantage and both smoking and obesity [7,32,38].

Although our analysis begins to shed light on the intergenerational linkages in cardiovascular health, our data lack explicit controls for genetic factors. Future research on the intergenerational transmission of CVD would benefit from the use of molecular data from parents and/or their children that can be used to create polygenic scores for specific risks of CVD and risk factors (e.g., obesity, smoking, alcohol) [2]. Another study limitation is the use of self-reported items, which could yield measurement error. Additionally, our analysis is limited to data on only two generations. Future research would benefit from multigenerational data extending to grandparents and even great grandparents [1,20-23]. Finally, we found no significant variations in associations by race/ethnicity or gender. However, even in a large sample such as Add Health, the sample sizes of minority populations can be relatively small and prohibit more nuanced analysis of ethnic

subpopulations and intersectionalities. Thus, intergenerational data focused on racial/ethnic minority groups in the U.S. are sorely needed.

Although family genetic predispositions are not easily changed, programs and policies can be designed to influence childhood health exposures such as birthweight, breastfeeding duration, and ACEs [1,4,5]. Furthermore, our results on the long-lasting influence of family SES support research indicating that policies and programs in which investment in families with young children have the potential to yield high economic and social returns [4,6]. Similarly, our results on the long-lasting association between peer smoking behaviors and adult children's smoking behavior suggests that schools might have a major influence on health by instituting policies and practices in schools to discourage smoking [7,28]. Finally, our results on the long-lasting influence of neighborhood context suggest that families can potentially benefit from place-based initiatives to improve infrastructure in disadvantaged communities and moving to opportunity programs which enable families to move to less disadvantaged neighborhoods [28,32,39,40].

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### Implications and Contribution

By capitalizing on representative intergenerational data, findings highlight important similarities and differences between parents and adult children at similar stages of the life course. Findings identify childhood socioeconomic conditions and contexts that shape the contours of adult children's health, with implications for strategies to enhance the health of future generations.

**Table 1.**

Intergenerational comparisons of Add Health parents' cardiovascular risk at W1 and their adult children's cardiovascular risk at W5 (N=10,466)

| | Parent Age: *M* = 41.43 (SD = 6.47) | | | Adult Child Ages: *M* = 37.75 (SD = 1.74) | | | | |
|---|---|---|---|---|---|---|---|---|
| | % | 95% CI | | % | 95% CI | | *r* | *p*-value |
| **Excessive Drinking** | | | | | | | | |
| No | 92.4 | 91.4 | 93.2 | 78.8 | 77.4 | 80.2 | 0.17 | <.001 |
| Yes | 7.6 | 6.8 | 8.6 | 21.2 | 19.8 | 22.6 | | |
| **Smoking** | | | | | | | | |
| No | 70.9 | 68.3 | 73.3 | 72.4 | 70.5 | 74.2 | 0.29 | <.001 |
| Yes | 29.1 | 26.7 | 31.7 | 27.6 | 25.8 | 29.5 | | |
| **Obesity** | | | | | | | | |
| No | 77.0 | 75.8 | 78.2 | 59.3 | 57.4 | 61.1 | 0.30 | <.001 |
| Yes | 23.0 | 21.8 | 24.2 | 40.7 | 38.9 | 42.6 | | |
| **Diabetes** | | | | | | | | |
| No | 92.4 | 91.4 | 93.2 | 94.2 | 93.4 | 94.9 | 0.26 | <.001 |
| Yes | 7.6 | 6.8 | 8.6 | 5.8 | 5.1 | 6.6 | | |

Notes: Ns are unweighted and percentages are weighted. Adult children's health measures are self-reported. Parents' health measures are self-reported by the primary caregiver (typically the mother). Excessive drinking and smoking behavior is measured for only the primary caregiver. Obesity and diabetes are measured for both biological parents. Significant differences in parent and adult child proportions are indicated when 95% confidence intervals do not overlap. Polychoric correlations (*r*) were estimated to accommodate binary variables. P-values correspond to correlation analyses.

**Table 2.**

Correspondence between parents' cardiovascular risk at W1 and adult child cardiovascular risk at W5 (N=10,466)

| | % | 95% CI | |
|---|---|---|---|
| **Excessive Drinking** | | | |
| Both do not drink excessively | 73.5 | 72.0 | 74.9 |
| Both drink excessively | 2.4 | 2.0 | 2.9 |
| Parent drinks excessively, child does not | 5.4 | 4.7 | 6.2 |
| Parent does not drink excessively, child does | 18.7 | 17.4 | 20.1 |
| **Smoking** | | | |
| Both do not smoke | 54.7 | 52.1 | 57.3 |
| Both smoke | 11.6 | 10.2 | 13.4 |
| Parent smokes, child does not | 17.6 | 16.2 | 19.0 |
| Parent does not smoke, child does | 16.1 | 15.0 | 17.2 |
| **Obesity** | | | |
| Both not obese | 49.3 | 47.6 | 51.0 |
| Both obese | 13.0 | 11.9 | 14.1 |
| Parent obese, child not obese | 10.0 | 9.2 | 11.0 |
| Parent not obese, child obese | 27.7 | 26.2 | 29.2 |
| **Diabetes** | | | |
| Both not diabetic | 87.6 | 86.3 | 88.7 |
| Both diabetic | 1.0 | 0.8 | 1.4 |
| Parent diabetic, child not diabetic | 6.7 | 5.9 | 7.5 |
| Parent not diabetic, child diabetic | 4.8 | 4.2 | 5.4 |

Notes: Ns are unweighted and percentages are weighted. Adult children's health measures are self-reported. Parents' health measures are self-reported by the primary caregiver (typically the mother). Excessive drinking and smoking behavior is measured for only the primary caregiver. Obesity and diabetes are measured for both biological parents.

**Table 3.**

Adult child's cardiovascular risk at W5 regressed on parent cardiovascular risk at W1, child characteristics, and family, school, and neighborhood contexts in childhood (i.e. <18 years), full sample (N=10,466)

| | | | Drinks Excessively | | | Smokes | | | Obese | | | Diabetic | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **M/%** | **(SE)** | **OR** | **95% CI** | | **OR** | **95% CI** | | **OR** | **95% CI** | | **OR** | **95% CI** | |
| **Parent's Cardiovascular Risk (W1)** | | | | | | | | | | | | | | |
| Parent Drinks Excessively | | | **1.75** | 1.38 | 2.22 | | | | | | | | | |
| Parent Smokes | | | | | | **1.66** | 1.46 | 1.90 | | | | | | |
| Parent Obese | | | | | | | | | **2.28** | 1.98 | 2.63 | | | |
| Parent Diabetic | | | | | | | | | | | | **2.25** | 1.62 | 3.14 |
| **Adult Child's Demographic Background** | | | | | | | | | | | | | | |
| Adult Child Age | 37.75 | 0.12 | 0.97 | 0.92 | 1.01 | **0.92** | 0.89 | 0.96 | 1.03 | 0.99 | 1.06 | 1.00 | 0.94 | 1.06 |
| Adult Child Female (vs Male) | 0.49 | 0.01 | **0.46** | 0.40 | 0.52 | **0.71** | 0.62 | 0.82 | 1.10 | 0.97 | 1.25 | 0.80 | 0.62 | 1.01 |
| Foreign-Born Parent (vs U.S.-Born Parent) | 0.15 | 0.02 | 0.77 | 0.57 | 1.03 | 0.83 | 0.64 | 1.10 | 0.86 | 0.68 | 1.09 | 0.82 | 0.54 | 1.25 |
| Adult Child Race | | | | | | | | | | | | | | |
| Hispanic | 0.12 | 0.02 | 1.17 | 0.83 | 1.64 | **0.60** | 0.45 | 0.79 | **1.30** | 1.04 | 1.63 | 1.22 | 0.81 | 1.83 |
| Non-Hispanic Black | 0.15 | 0.02 | 0.92 | 0.73 | 1.16 | **0.63** | 0.51 | 0.78 | **1.29** | 1.08 | 1.54 | **1.61** | 1.17 | 2.23 |
| Non-Hispanic Other | 0.04 | 0.01 | 0.77 | 0.50 | 1.19 | 0.84 | 0.56 | 1.25 | 0.96 | 0.68 | 1.37 | 1.73 | 0.87 | 3.45 |
| Non-Hispanic White | 0.69 | 0.03 | *ref.* | | | *ref.* | | | *ref.* | | | *ref.* | | |
| **Childhood Health Exposures** | | | | | | | | | | | | | | |
| Adult Child's Birthweight (lbs.) | 7.47 | 0.03 | 0.96 | 0.90 | 1.02 | 0.99 | 0.94 | 1.04 | **1.09** | 1.03 | 1.14 | **0.89** | 0.80 | 0.99 |
| Adult Child Breastfed | | | | | | | | | | | | | | |
| None | 0.54 | 0.01 | *ref.* | | | *ref.* | | | *ref.* | | | *ref.* | | |
| 1-6 Months | 0.25 | 0.01 | 1.18 | 1.00 | 1.40 | 1.03 | 0.88 | 1.21 | **0.82** | 0.72 | 0.95 | **0.61** | 0.44 | 0.85 |
| 6 Months or Longer | 0.21 | 0.01 | 1.03 | 0.83 | 1.26 | **0.78** | 0.64 | 0.96 | **0.71** | 0.61 | 0.82 | 0.72 | 0.50 | 1.02 |
| Adverse Childhood Experiences | 0.68 | 0.02 | 1.07 | 0.98 | 1.17 | **1.26** | 1.18 | 1.34 | 1.06 | 1.00 | 1.13 | 1.04 | 0.91 | 1.19 |
| **Family Context** | | | | | | | | | | | | | | |
| Two Biological/Adoptive Parents (vs Other) | 0.58 | 0.01 | 1.15 | 0.98 | 1.34 | **0.80** | 0.70 | 0.91 | 0.95 | 0.84 | 1.09 | 1.24 | 0.97 | 1.59 |
| Family Income (in ten-thousands) | 4.65 | 0.19 | 1.01 | 0.99 | 1.02 | **0.98** | 0.97 | 1.00 | **0.97** | 0.95 | 1.00 | 0.98 | 0.93 | 1.02 |
| Family Public Assistance Use | 0.17 | 0.01 | 0.92 | 0.75 | 1.13 | 1.09 | 0.89 | 1.33 | 1.14 | 0.96 | 1.36 | 1.09 | 0.82 | 1.46 |
| Parent College or More (vs Less) | 0.32 | 0.02 | 1.08 | 0.93 | 1.25 | **0.72** | 0.60 | 0.86 | **0.78** | 0.66 | 0.91 | 0.77 | 0.54 | 1.09 |

| | | | Drinks Excessively | | | Smokes | | | Obese | | | Diabetic | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **M/%** | **(SE)** | **OR** | **95% CI** | | **OR** | **95% CI** | | **OR** | **95% CI** | | **OR** | **95% CI** | |
| Parent-Child Closeness | 4.24 | 0.02 | **0.89** | 0.79 | 0.99 | 0.94 | 0.84 | 1.04 | 1.01 | 0.92 | 1.10 | **0.86** | 0.75 | 0.98 |
| **School Context** | | | | | | | | | | | | | | |
| Intergenerational Closure | 0.35 | 0.01 | 1.00 | 1.00 | 1.01 | 1.00 | 0.99 | 1.01 | 1.00 | 0.99 | 1.00 | 0.99 | 0.98 | 1.00 |
| Grademates Excessive Drinking (%) | 51.00 | 1.00 | 1.00 | 1.00 | 1.01 | - | - | - | - | - | - | - | - | - |
| Grademates Smoked (%) | 36.00 | 1.00 | - | - | - | **1.01** | 1.01 | 1.02 | - | - | - | - | - | - |
| Grademates Exercise 6+ Days/Wk (%) | 42.00 | 1.00 | - | - | - | - | - | - | 1.01 | 1.00 | 1.01 | 1.00 | 0.98 | 1.01 |
| **Neighborhood (tract-level) Context** | | | | | | | | | | | | | | |
| Neighborhood Collective Efficacy | 6.23 | 0.03 | 1.05 | 0.93 | 1.18 | 0.92 | 0.83 | 1.01 | **1.12** | 1.00 | 1.25 | 1.00 | 0.82 | 1.23 |
| Neighborhood Social Cohesion (%) | 73.00 | 1.00 | **1.01** | 1.00 | 1.01 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 0.99 | 1.01 |
| Neighborhood Disadvantage Index | 1.16 | 0.13 | 0.98 | 0.92 | 1.04 | **1.08** | 1.03 | 1.14 | **1.07** | 1.03 | 1.11 | 1.06 | 0.97 | 1.14 |

Abbreviations: M, Mean; SE, Standard Error; OR, odds ratio; CI, Confidence Interval. Notes: Estimates are weighted and account for sample design. Bold font indicates statistical significance at $p < .05$.

**Table 4.**

Adult child cardiovascular risk at W5 regressed on adult child characteristics, and family, school, and neighborhood contexts in childhood, stratified analysis of parents who did have the cardiovascular risk at W1

| | Drinks Excessively (N=715) | | | Smokes (N=2,703) | | | Obese (N=2,333) | | | Diabetic (N=733) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | |
| **Adult Child's Demographic Background** | | | | | | | | | | | | |
| Adult Child Age | 0.96 | 0.82 | 1.12 | 0.93 | 0.87 | 1.00 | 1.00 | 0.93 | 1.08 | 1.09 | 0.91 | 1.29 |
| Adult Child Female (vs Male) | **0.36** | 0.21 | 0.61 | **0.76** | 0.61 | 0.94 | 0.94 | 0.76 | 1.17 | 1.12 | 0.66 | 1.89 |
| First or Second Generation Immigrant (vs Third) | **0.22** | 0.07 | 0.71 | 0.65 | 0.39 | 1.09 | 1.03 | 0.61 | 1.74 | 0.78 | 0.30 | 2.07 |
| Adult Child Race | | | | | | | | | | | | |
| Hispanic | 0.77 | 0.26 | 2.28 | **0.54** | 0.33 | 0.89 | 0.59 | 0.34 | 1.04 | 1.90 | 0.52 | 6.99 |
| Non-Hispanic Black | **0.29** | 0.12 | 0.70 | **0.48** | 0.31 | 0.75 | 1.12 | 0.83 | 1.51 | 2.00 | 0.80 | 4.98 |
| Non-Hispanic Other | 0.37 | 0.12 | 1.15 | 1.17 | 0.46 | 2.92 | 1.34 | 0.62 | 2.88 | 2.69 | 0.74 | 9.81 |
| Non-Hispanic White (ref.) | *1.00* | - | - | *1.00* | - | - | *1.00* | - | - | *1.00* | - | - |
| **Childhood Health Exposures** | | | | | | | | | | | | |
| Adult Child's Birthweight (lbs.) | 0.82 | 0.67 | 1.01 | 0.97 | 0.89 | 1.05 | 1.02 | 0.92 | 1.13 | 0.90 | 0.71 | 1.14 |
| Adult Child Breastfed | | | | | | | | | | | | |
| None (ref.) | *1.00* | - | - | *1.00* | - | - | *1.00* | - | - | *1.00* | - | - |
| 1-6 Months | 1.63 | 0.84 | 3.15 | 0.99 | 0.73 | 1.34 | **0.70** | 0.55 | 0.90 | 0.76 | 0.34 | 1.68 |
| 6 Months or Longer | **2.51** | 1.29 | 4.88 | 0.76 | 0.50 | 1.16 | 0.84 | 0.62 | 1.15 | 0.56 | 0.24 | 1.29 |
| Adverse Childhood Experiences | 1.08 | 0.87 | 1.33 | **1.21** | 1.09 | 1.33 | **1.17** | 1.01 | 1.37 | 0.92 | 0.71 | 1.21 |
| **Family Context** | | | | | | | | | | | | |
| Two Biological/Adoptive Parents (vs Other) | **1.91** | 1.09 | 3.36 | 0.95 | 0.76 | 1.19 | 1.01 | 0.79 | 1.29 | 1.07 | 0.56 | 2.06 |
| Family Income (in ten-thousands) | 0.99 | 0.96 | 1.02 | 0.99 | 0.96 | 1.02 | **0.94** | 0.91 | 0.98 | 0.97 | 0.88 | 1.07 |
| Any Family Public Assistance Use (vs. None) | 1.42 | 0.81 | 2.47 | 1.12 | 0.85 | 1.49 | 0.92 | 0.69 | 1.23 | 0.79 | 0.36 | 1.72 |
| Parent College or More (vs Less) | 0.79 | 0.42 | 1.50 | **0.67** | 0.46 | 0.98 | 0.78 | 0.61 | 1.00 | 0.52 | 0.25 | 1.08 |
| Parent-Child Closeness | 1.07 | 0.77 | 1.48 | 0.96 | 0.81 | 1.12 | 1.10 | 0.92 | 1.33 | 0.73 | 0.49 | 1.09 |
| **School Context** | | | | | | | | | | | | |
| Intergenerational Closure | 0.99 | 0.96 | 1.01 | 1.00 | 0.99 | 1.01 | 1.00 | 0.98 | 1.01 | 0.99 | 0.96 | 1.01 |
| Grademates Excessive Drinking (%) | 1.00 | 0.98 | 1.03 | - | - | - | - | - | - | - | - | - |

| | Drinks Excessively (N=715) | | | Smokes (N=2,703) | | | Obese (N=2,333) | | | Diabetic (N=733) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | |
| Grademates Smoked (%) | - | - | - | 1.01 | 1.00 | 1.02 | - | - | - | - | - | - |
| Grademates Exercise 6+ Days/Week (%) | - | - | - | - | - | - | 1.01 | 0.99 | 1.02 | 1.02 | 0.98 | 1.06 |
| **Neighborhood (tract-level) Context** | | | | | | | | | | | | |
| Neighborhood Collective Efficacy | 0.82 | 0.56 | 1.19 | 0.93 | 0.77 | 1.14 | 1.06 | 0.89 | 1.27 | **0.65** | 0.44 | 0.96 |
| Neighborhood Social Cohesion (%) | **1.02** | 1.00 | 1.03 | 1.00 | 0.99 | 1.00 | 1.00 | 0.99 | 1.01 | 1.00 | 0.99 | 1.01 |
| Neighborhood Disadvantage Index | 1.00 | 0.86 | 1.16 | **1.11** | 1.02 | 1.21 | **1.15** | 1.05 | 1.25 | 1.03 | 0.87 | 1.23 |
| **Subsample** | Parents who drank excessively | | | Parents who smoked | | | Parents who were obese | | | Parents who were diabetic | | |

Abbreviations: OR, odds ratio; CI, Confidence Interval. Notes: Estimates are weighted and account for sample design. Bold font indicates statistical significance at $p < .05$.

**Table 5.**

Adult child cardiovascular risk at W5 regressed on adult child characteristics, and family, school, and neighborhood contexts in childhood, stratified analysis of parents who did not have the cardiovascular risk at W1

| | Drinks Excessively (N=9,582) | | | Smokes (N=7,589) | | | Obese (N=7,887) | | | Diabetic (N=9,376) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | |
| **Adult Child's Demographic Background** | | | | | | | | | | | | |
| Adult Child Age | 0.96 | 0.92 | 1.01 | **0.92** | 0.87 | 0.97 | 1.04 | 1.00 | 1.08 | 0.98 | 0.92 | 1.05 |
| Adult Child Female (vs Male) | **0.46** | 0.40 | 0.53 | **0.69** | 0.58 | 0.83 | **1.17** | 1.01 | 1.35 | **0.73** | 0.56 | 0.97 |
| First or Second Generation Immigrant (vs Third) | 0.84 | 0.62 | 1.14 | 0.91 | 0.67 | 1.23 | 0.84 | 0.65 | 1.09 | 0.81 | 0.52 | 1.25 |
| Adult Child Race | | | | | | | | | | | | |
| Hispanic | 1.18 | 0.83 | 1.67 | **0.63** | 0.46 | 0.87 | **1.58** | 1.21 | 2.05 | 1.17 | 0.76 | 1.78 |
| Non-Hispanic Black | 1.02 | 0.80 | 1.30 | **0.75** | 0.59 | 0.96 | **1.37** | 1.11 | 1.69 | **1.56** | 1.00 | 2.43 |
| Non-Hispanic Other | 0.79 | 0.50 | 1.26 | 0.80 | 0.51 | 1.27 | 0.85 | 0.57 | 1.27 | 1.70 | 0.87 | 3.32 |
| Non-Hispanic White (ref.) | *1.00* | - | - | *1.00* | - | - | *1.00* | - | - | *1.00* | - | - |
| **Childhood Health Exposures** | | | | | | | | | | | | |
| Adult Child's Birthweight (lbs.) | 0.98 | 0.92 | 1.05 | 1.00 | 0.93 | 1.08 | **1.10** | 1.04 | 1.17 | **0.89** | 0.80 | 0.99 |
| Adult Child Breastfed | | | | | | | | | | | | |
| None (ref.) | *1.00* | - | - | *1.00* | - | - | *1.00* | - | - | *1.00* | - | - |
| 1-6 Months | 1.17 | 0.99 | 1.39 | 1.05 | 0.86 | 1.29 | 0.88 | 0.75 | 1.04 | **0.57** | 0.41 | 0.81 |
| 6 Months or Longer | 0.96 | 0.78 | 1.20 | **0.79** | 0.64 | 0.99 | **0.68** | 0.57 | 0.81 | 0.72 | 0.49 | 1.04 |
| Adverse Childhood Experiences | 1.06 | 0.96 | 1.16 | **1.32** | 1.20 | 1.45 | 1.02 | 0.95 | 1.10 | 1.06 | 0.93 | 1.22 |
| **Family Context** | | | | | | | | | | | | |
| Two Biological/Adoptive Parents (vs Other) | 1.08 | 0.91 | 1.27 | **0.73** | 0.61 | 0.88 | 0.96 | 0.82 | 1.11 | **1.32** | 1.01 | 1.74 |
| Family Income (in ten-thousands) | 1.01 | 0.99 | 1.02 | **0.98** | 0.95 | 1.00 | 0.98 | 0.96 | 1.00 | 0.98 | 0.93 | 1.03 |
| Any Family Public Assistance Use (vs. None) | 0.90 | 0.72 | 1.13 | 1.05 | 0.81 | 1.36 | **1.27** | 1.04 | 1.55 | 1.18 | 0.84 | 1.65 |
| Parent College or More (vs Less) | 1.08 | 0.92 | 1.27 | **0.74** | 0.61 | 0.91 | **0.77** | 0.64 | 0.93 | 0.81 | 0.55 | 1.21 |
| Parent-Child Closeness | **0.87** | 0.76 | 0.98 | 0.92 | 0.81 | 1.06 | 0.98 | 0.88 | 1.08 | 0.88 | 0.76 | 1.02 |
| **School Context** | | | | | | | | | | | | |
| Intergenerational Closure | 1.01 | 1.00 | 1.01 | 1.00 | 0.99 | 1.01 | 1.00 | 0.99 | 1.01 | 0.99 | 0.98 | 1.01 |
| Grademates Excessive Drinking (%) | 1.00 | 1.00 | 1.00 | - | - | - | - | - | - | - | - | - |

| | Drinks Excessively (N=9,582) | | | Smokes (N=7,589) | | | Obese (N=7,887) | | | Diabetic (N=9,376) | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | | **OR** | ***95% CI*** | |
| Grademates Smoked (%) | - | - | - | **1.02** | 1.01 | 1.02 | - | - | - | - | - | - |
| Grademates Exercise 6+ Days/Week (%) | - | - | - | - | - | - | 1.01 | 1.00 | 1.01 | 0.99 | 0.98 | 1.01 |
| **Neighborhood (tract-level) Context** | | | | | | | | | | | | |
| Neighborhood Collective Efficacy | 1.07 | 0.95 | 1.21 | **0.88** | 0.77 | 0.99 | 1.14 | 0.99 | 1.31 | 1.12 | 0.88 | 1.43 |
| Neighborhood Social Cohesion (%) | 1.00 | 1.00 | 1.00 | **1.01** | 1.00 | 1.01 | 1.00 | 0.99 | 1.00 | 1.00 | 0.99 | 1.01 |
| Neighborhood Disadvantage Index | 0.97 | 0.91 | 1.04 | **1.06** | 1.01 | 1.12 | 1.05 | 1.00 | 1.10 | 1.05 | 0.96 | 1.16 |
| **Subsample** | Parents who did not drink excessively | | | Parents who did not smoke | | | Parents who were not obese | | | Parents who were not diabetic | | |

Abbreviations: OR, odds ratio; CI, Confidence Interval. Notes: Estimates are weighted and account for sample design. Bold font indicates statistical significance at $p < .05$.