

# **Early Life Neighborhood Disadvantage and Blood Pressure in Early Adulthood**

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## **Introduction**

The leading cause of mortality in the United States is coronary heart disease (CHD). Hypertension, or high blood pressure, is an important risk factor for CHD. Recent estimates suggest that rates of hypertension are not just high among US adults, but nearly 1 in 5 young adults aged 24 – 32 have high blood pressure in 2008 (Nguyen 2011). Individual level socioeconomic status is highly correlated with a number of health outcomes, including hypertension (Bravemen et al 2005). Theories suggest that stressors related to low economic status may be the reason for health disparities (Pampel et al 2010, Baum et al 1999, Krueger and Chang 2008), as chronic stress exposure has been linked to biological mechanisms that can lead to worse health outcomes (Cohen et al 2006).

However, just as individual disadvantage can be detrimental to health outcomes, neighborhood level factors are important predictors of health as well (Robert 1999; Winkleby et al 2006; Anderson 1997). The stress of living in a low SES community has been connected to an increased risk of hypertension and cardiovascular risk independent of the impact of individual level SES (Mujahid et al 2008; Boardman 2004, Elliot 2000; Diez Roux et al 2001). Most research on the relationship between community level disadvantage and individual's blood pressure, however, has used cross-sectional designs; few use a life course perspective in a longitudinal design to trace the short and long-term effects of growing up in a disadvantage neighborhood on health outcomes such as blood pressure.

A growing literature indicates that early life circumstances are associated with health and susceptibility to disease in adulthood. We contribute to this literature by investigating the extent to which neighborhood disadvantage experienced prior to adulthood impacts adult health. We use data from the National Longitudinal Study of Adolescent Health (Add Health) to construct census-tract level indexes of disadvantage for two early stages in the life course: adolescence and the transition to adulthood. With repeated measures on neighborhood context, we examine how three aspects of exposure to neighborhood disadvantage—life stage timing, duration, and mobility into or out of neighborhood disadvantage—are associated with blood pressure in adulthood.

## **Methods**

We use a longitudinal design to examine how neighborhood context in adolescence and the transition to adulthood is related to adult health using data from Add Health, first collected in 1994 from a national cohort of 7 – 12 graders with follow-up interview conducted in 1996, 2002 and most recently at Wave IV in 2008-09.

Our health outcome of interest is blood pressure, measured at Wave IV, when respondents were between the ages of 24 and 32. Blood pressure often increases and remains elevated in response to chronic stress, and hypertension represents an important risk factor for stroke and cardiovascular disease. We use three measures of blood pressure. The first two, systolic blood pressure (SBP) and diastolic blood pressure (DBP), are taken directly from the measured blood pressure at Wave IV and are modeled as linear dependent variables. High SBP or DBP indicates greater biological stress to the cardiovascular system and poor health. We also construct a dichotomous hypertension variable using the clinical cut point for high blood pressure (SBP>140 or DBP>90) that also takes into account those who self-report that they have been diagnosed with hypertension or are using blood pressure medication.

Our fundamental independent variable is a constructed neighborhood disadvantage index (NDI) drawn from five census-tract level measures: proportions of female-headed households, individuals living in poverty, high-school dropouts, individuals receiving public assistance and residents unemployed. For each of these five measures, we designate an individual as living in a disadvantaged

tract if they fall into the highest quartile of all tracts for each manifestation of disadvantage, and then we sum the number of measures on which the individual's tract is disadvantaged. Thus, a respondent may live in a tract with an NDI ranging from 0 (no disadvantage) to 5 (high disadvantage). Tract-level data are available from both Waves I and III, thus we have NDIs for where respondents lived in adolescence (ages 12 – 18) and while they were transitioning to adulthood (ages 18 – 26).

The NDIs at Wave I and Wave III represent our “life stage” measures of neighborhood disadvantage. To construct the duration of neighborhood disadvantage, we sum the two indexes to form a combined NDI score representing disadvantage over the period from adolescence and through the transition to adulthood. Finally, to examine whether movement in and out of neighborhood disadvantage might matter for blood pressure in adulthood, we construct four pathways of mobility: 1) Those who start in low disadvantage (NDI scores from 0-2) in adolescence and remain in low disadvantage in early adulthood; 2) those who start in low disadvantage and move into high disadvantage (NDI score between 3-5); 3) those who move from high to low disadvantage; and 4) those who begin in high disadvantage and remain in high disadvantage.

We conduct both bivariate and multivariate analysis, controlling for age, race and sex. To observe whether neighborhood disadvantage is still important above and beyond individual disadvantage, we also test models that control for parental income and education and the adult respondent's own education and income, in addition to their marital status at Wave IV. We investigate how the duration and timing of neighborhood disadvantage (how long one is exposed to disadvantage and during which point of their early life), in addition to how various mobility pathways, are associated with blood pressure in adulthood.

The hypotheses that we examine are:

- 1) We expect longer durations of exposure to high levels of neighborhood disadvantage, indicated by those who have high NDI scores at both points in their early life course, will have an increased risk of hypertension.
- 2) We expect that the timing of the exposure to disadvantage matters as well, such that living in highly disadvantaged neighborhoods in adolescence (Wave I) will be more detrimental to health and blood pressure than a similar exposure during the transition to adulthood at Wave III. The earlier the exposure to disadvantage in the life course, the longer lasting are the negative consequences because disadvantage is often difficult to move out of (i.e., there is strong state dependence). In addition, disadvantage during the transition to adulthood may represent a transient state while the young adult is developing his path into adulthood.
- 3) Mobility patterns should matter as well, and we expect that compared to those who experience high disadvantage and no change over time, those who are able to move out of disadvantage may have better health while those who move into disadvantage may be no different.
- 4) The previously hypothesized relationships between all three aspects of exposure to neighborhood disadvantage before adulthood and blood pressure will persist, though most likely weaken, once estimates are adjusted for individual characteristics.

### **Preliminary Results:**

Table 1 presents the weighted means and standard deviations for each of the variables used in our analysis. Mean SBP (124.95) and DBP (79.32) are close to clinically defined healthy levels of blood pressure (120/80). More than one in four respondents (26.30%) are considered hypertensive, consistent with other estimations using Add Health (Nguyen et al 2011). Most of our sample experiences little or no disadvantage in both Waves I and III, with a mean cumulative NDI score of 2.21 (or a little more

than 2 indicators of disadvantage out of 10 over time). In addition, the modal mobility pathway group is those who remain in low disadvantage (78.10%).

Table 2 shows the bivariate relationships between Wave I, Wave III and Cumulative (Waves I + III) disadvantage and hypertension. Wave I disadvantage matters most, with higher NDI scores increasing odds of hypertension. Cumulative disadvantage has a significant association with hypertension, with each additional increase in NDI increasing odds of being hypertensive by 3.8%. Finally, mobility pathways matter as well for adult hypertension, as those who go from high to low disadvantage or remain high in both waves have much higher risks of hypertension compared to those who remain in low disadvantaged neighborhoods.

Table 3 expands on the previous table, contrasting bivariate correlations for all three outcomes with the correlations found in the fully controlled multivariate models. For SBP and DBP, we report the OLS coefficients for the various independent variables. Logistic odds ratios are reported for the binary hypertension outcome. Again, disadvantage in adolescence (Wave I) matters more than during the transition to adulthood (Wave III), increasing DBP and hypertension. The coefficients of Wave I disadvantage on DBP and hypertension weaken slightly but still remain significant when controlling for individual factors. Bivariate analysis suggests that the high to high (non)mobility pathway is the most significantly different from remaining at low disadvantage over time. However, once controlling for individual factors, those who begin low and then experience high disadvantage experience increased SBP and DBP in adulthood.

## Conclusion

Considering the bivariate analyses, we see evidence for the first three hypotheses. More cumulative exposure to disadvantage increases blood pressure, though exposure earlier in the life course, in adolescence, seems to matter more. The bivariate mobility pathway analysis confirms this, as both pathways that increased blood pressure significantly began in high disadvantage. As hypothesized, we find that controlling for individual risk factors weakens the relationship between neighborhood disadvantage and individual blood pressure. However, the relationship persists, such that we can conclude that neighborhood level disadvantage does constitute an important risk factor and likely elevated stress, above and beyond individual disadvantage, that contributes to poor health by increasing blood pressure in early adulthood. Future research will aim to investigate the specific neighborhood level mechanisms related to stress that impact health.

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Table 1: Means and Standard Errors of Variables Used in Analysis

	Mean	Standard Error
<b>Dependent Variables</b>		
Systolic Blood Pressure (SBP)	124.951	0.218
Diastolic Blood Pressure (DBP)	79.321	0.172
Hypertensive (BPHT)	0.263	0.006
<b>Independent Variables</b>		
Neigh Disadv Wave I	1.126	0.129
Neigh Disadv Wave III	1.091	0.083
Duration of Neigh Disadv	2.217	0.204
Mobility: Low to High	0.050	0.028
Mobility: High to Low	0.102	0.019
Mobility: High to High	0.067	0.012
<b>Control Variables</b>		
Black	0.161	0.022
Asian	0.037	0.008
Native American/Other	0.004	0.001
Hispanic	0.117	0.017
Female	0.492	0.007
First Generation	0.041	0.007
Second Generation	0.109	0.011
<i>Wave I</i>		
Lived with Biological Two-Parent	0.567	0.014
Parent Education, College Degree+	0.333	0.019
Parent Income Below 1994 Poverty Line	0.135	0.011
<i>Wave IV</i>		
Age	28.316	0.119
Ever Married	0.495	0.014
Education, College Degree+	0.317	0.018
Adult Income Below 2008 Poverty Line	0.114	0.007

Table 2: Bivariate Relationship Between Neighborhood Disadvantage Measures and the Odds of Hypertension. Odd Ratios for Hypertension

Neigh Disadv Wave I	1.086***	
Neigh Disadv Wave III	0.985	
Duration of Neigh Disadv		1.038***
Mobility Pathways (reference= low to low)		
Low to High		0.945
High to Low		1.268**
High to High		1.345***

Table 3: The Relationship Between Neighborhood Disadvantage and Blood Pressure, Bivariate and Fully Controlled Models

	<b>Bivariate</b>			<b>Multivariate</b>		
	SBP	DBP	BPHT	SBP	DBP	BPHT
Neigh Disadv Wave I	0.244	.265**	1.086***	0.086	.238**	1.068***
Neigh Disadv Wave III	0.174	0.013	0.985	0.23	0.071	1.002
Duration of Neigh Disadv	.212***	.149***	1.038***	0.118	.124*	1.036**
Mobility Pathways (reference= low to low)						
Low to High	1.585	0.723	0.945	2.687**	1.480*	1.082
High to Low	0.646	0.963	1.268**	-0.0296	0.486	1.144
High to High	1.557***	.916**	1.345***	0.969	0.618	1.275**