How neighborhoods shape health from adolescence to adulthood: An examination of age-varying effects

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Dedication

To my darling son, Dexter, whose spirit has brought immeasurable joy and laughter to this journey. I do it all for you. With all my love, mama.

Abstract

The neighborhood in which one resides represents an important contextual influence on health. Indeed, a large body of research has examined associations between myriad neighborhood conditions and health, often demonstrating that worse neighborhood conditions are related to worse health. However, few studies have examined when in development exposure to harmful neighborhood conditions may confer the greatest risk. In this dissertation, I aimed to address this gap in the literature by examining change in neighborhood concentrated disadvantage, depressive symptoms, and binge drinking across adolescence and emerging adulthood (Aim 1) and the age-varying associations between neighborhood concentrated disadvantage and depressive symptoms (Aim 2) and binge drinking (Aim 3) from ages 13-25. To address these questions, I leveraged a sample of 9,956 youth that participated in Waves I-III of the National Study of Adolescent to Adult Health. All participants were between 13-25-years-old at all three timepoints. The sample was predominantly female (53.18%), white (68.74%), non-Hispanic (84.42%), and resided in households where at least one caregiver had a high school diploma/GED or more (87.93%) and was employed (90.21%). Although this varied slightly across waves, participants resided in neighborhoods where, on average, 17% of residents were socioeconomically disadvantaged. To address the primary aims of this dissertation, I conducted time-varying effect models, structuring the data by age instead of timepoint, yielding estimates of the association between neighborhood concentrated disadvantage and depressive symptoms and binge drinking as a function of continuous age from 13-25. All models controlled for participant sex, race, and ethnicity,

and caregiver educational attainment, employment, and receipt of public assistance. I found that neighborhood concentrated disadvantage increased across age, likely as a function of increasing concentrated poverty across historical time. Average depressive symptoms and engagement in binge drinking varied substantially by age, with the former peaking in late adolescence and the latter peaking in mid-emerging adulthood. Neighborhood concentrated disadvantage was only associated with depressive symptoms in emerging adulthood, whereas the association between neighborhood concentrated disadvantage and binge drinking was significant only in early adolescence. These findings may prove useful for the early identification of and intervention for those most at risk for depressive symptoms and binge drinking among those residing in disadvantaged neighborhoods. Several important directions for future research include 1) the development and use of datasets appropriate for longitudinal analyses of neighborhoods and health to elucidate how associations unfold dynamically over time, 2) investigating associations between neighborhood conditions and health that extend beyond socioeconomic conditions to examine other environmental features of the neighborhood, and 3) the incorporation of structural determinants of health and health inequities that shape the neighborhood context into research designs.

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Chapter 1: Introduction

The neighborhood in which one resides is an important contextual influence on development. Extant research shows robust associations between an array of adverse neighborhood conditions (e.g., high poverty rates, high crime rates, low access to healthy food, minimal green space) and mental, physical, and cognitive health outcomes (Ding et al., 2011; Richardson et al., 2015; Taylor et al., 2020). These findings highlight the importance of 1) interventions to ameliorate the deleterious effects of exposure to adverse neighborhood conditions and 2) improving neighborhoods to prevent exposure to adverse neighborhood conditions in the first place. However, the current body of evidence linking neighborhoods to health is predominantly cross-sectional (Gresham & Karatekin, *under revision*), providing limited insight into *when* in development youth may be most at risk for poor health outcomes in the context of poor neighborhood conditions. Identifying when youth may be most at risk is critical for the design and implementation of developmentally appropriate interventions. Therefore, in this dissertation, my overarching goal is to elucidate sensitive periods in development during which the effects of poor neighborhood conditions on health may be especially pronounced. In line with this goal, my dissertation has the following specific aims.

Specific Aim 1: To characterize variation in neighborhood concentrated disadvantage, depressive symptoms, and binge drinking among adolescents and emerging adults.

Specific Aim 2: To examine the extent to which the magnitude and direction of the association between neighborhood concentrated disadvantage and depressive symptoms varies across adolescence and emerging adulthood.

Specific Aim 3: To investigate the extent to which the magnitude and direction of the association between neighborhood concentrated disadvantage and binge drinking varies across the aforementioned developmental periods.

1.1 Historical Overview of Neighborhoods and Health Research

It was over a century ago when Dr. John Snow was among the first to acknowledge the relationship between neighborhoods and health. In trying to understand the etiology of a London cholera outbreak, Snow traced the outbreak to a water pump in the Soho neighborhood (Snow, 1856), demonstrating the importance of place on health. This initial finding has led to a growing body of evidence examining relationships between neighborhood conditions and health (e.g., Arcaya et al., 2016; Oakes et al., 2015; Richardson et al., 2015). Experts in the field of neighborhood effects suggest that to date, there are three "generations" of neighborhoods and health research (Diez Roux, 2018a).

According to Diez Roux (2018a), the first generation of research on neighborhoods and health, published from the late 1980s until the early 2000s, consisted largely of studies of individual-level data with unrelated primary aims that were linked to census data via participant-reported addresses. Generally, research from this generation provided preliminary evidence that adverse neighborhood conditions are associated with myriad poor health outcomes (e.g., premature mortality, lower self-reported health,

health-risk behaviors; Jivraj et al., 2021; Kim et al., 2010; Lawrence et al., 2024). However, the cross-sectional nature of these studies, reliance on census geographies as a proxy of neighborhoods, and focus on socioeconomic conditions led to a number of methodological critiques (Oakes et al., 2015).

Nevertheless, this initial evidence provided the foundation for the second generation of neighborhoods and health research (i.e., early 2000s to 2015). This generation saw the emergence of studies explicitly designed to examine neighborhood effects on health (Diez Roux, 2018a). This direct focus on relationships between neighborhoods and health led to 1) increased attention toward the theoretical underpinnings of this work, 2) more rigorous modeling that improved causality and sought to disentangle neighborhood and family influences, 3) and extended the measurement of so-called neighborhood effects beyond socioeconomic conditions to features of the social, physical, and built environments (Diez Roux, 2018a). Across domains of neighborhood conditions, the second generation of neighborhoods and health research yielded a body of evidence demonstrating that other elements of neighborhoods in addition to socioeconomic conditions, matter for health (e.g., Renalds et al., 2010). However, this research continued to predominantly rely on cross-sectional studies, limiting causal inference and insight into the influence of neighborhood conditions across development (Diez Roux, 2018a; Oakes et al., 2015).

Thus, in the ongoing third generation of neighborhoods and health research (i.e., approximately 2016 to present), emphasis has shifted toward understanding the causal role of neighborhoods in predicting health through the use of more rigorous methods and

the incorporation of factors influencing selection into neighborhoods and the role of residential mobility. However, across these three generations of research, studies that approach associations between neighborhood conditions and health from a developmental perspective remains limited.

1.2 Neighborhoods as an Important Contextual Influence on Development

Neighborhoods are geographical places where people live that are nested within larger areas (e.g., cities, counties, states) and have social and cultural meaning to their residents (Duncan & Kawachi, 2018). As such, the neighborhood in which one resides represents an important contextual influence on health and development. Indeed, conceptual models and frameworks have long posited the importance of the neighborhood context. For instance, Bronfenbrenner's ecological systems model suggests that an individual's development is influenced by a series of interconnected environmental influences (Bronfenbrenner, 1979). In this framework, there are five levels of environmental influence, or systems, that vary in proximity to the individual: microsystem, mesosystem, exosystem, macrosystem, and chronosystem. The most proximal system, the microsystem, includes aspects of the environment that have direct contact with the individual, including factors at the family-, school-, and neighborhood-level. The mesosystem represents the interactions between microsystem influences (e.g., caregiver involvement with the school). Formal and informal social structures (e.g., the local government, extended family) are part of the exosystem, and the macrosystem represents broader contextual influences (e.g., culture, laws, governance, policy). Finally,

the chronosystem includes experiences throughout the lifetime, such as expected life transitions, historical events, and the culmination of experiences throughout the lifespan.

Beyond the organization of environmental influences into multiple system levels, one of the core tenants of Bronfenbrenner's (1979) ecological systems model is the dynamic interaction between individuals and their environment over time. Essentially, the effect of a given environmental input on an individual varies across development. The association between the environment and individual development is a function of previous and current experiences. Indeed, this is a widely held belief in developmental science. For example, Gilbert Gottlieb's (2007) model on probabilistic epigenesis suggests that development arises from reciprocal interactions between multiple system levels (e.g., genetics, behavior, environment) across development. Thus, the neighborhood is a microsystem-level context that exerts a direct influence on individuals, the effects of which may vary across development.

Another core tenant of Bronfenbrenner's ecological systems model (1979) and other conceptual frameworks (e.g., Solar & Irwin, 2010) is the influence of broader structural factors on neighborhood conditions, which in turn, shape health and development. One such conceptual framework is the World Health Organization's conceptual framework for action on social determinants of health and health inequities (Solar & Irwin, 2010). Social determinants of health are broadly conceptualized as the conditions in which people are born, work, play, and live. According to Solar and Irwin (2010), the social and political context (e.g., governance, macroeconomic policies, cultural values; termed structural determinants of health and health inequities) gives rise

to the social stratification of individuals based on characteristics such as sex/gender, race, income, education (i.e., structural stratifiers; Solar & Irwin, 2010). This social stratification results in the inequitable exposure to health-compromising factors, termed intermediary determinants of health, ultimately leading to poor health and health disparities (Solar & Irwin, 2010). Importantly, Solar & Irwin (2010) also posit bidirectional associations and causal feedback loops between structural determinants of health and health inequities, structural stratifiers, intermediary determinants of health, and health outcomes (Solar & Irwin, 2010). As such, neighborhood conditions, the neighborhoods in which individuals choose to reside, and the effect of neighborhood conditions on health are shaped by upstream, macropolitical factors.

Given the conceptual importance of the neighborhood context in shaping health, developing a robust body of empirical evidence that extends beyond *whether* neighborhoods matter for health to address *when*, *how*, and *for whom* neighborhoods matter is a critical population health goal. Historically, however, research on neighborhoods and health has emphasized *whether*, not *when* myriad neighborhood conditions are associated with various health outcomes.

1.3 Neighborhoods and Health Research from a Developmental Lens

Developmental scientists have long posited the existence of sensitive periods in development (Bornstein, 1989), during which an individual may either be at greater risk for poor outcomes or a particular experience may have a relatively strong influence on development relative to other developmental periods. Researchers have investigated associations between neighborhood conditions and health across infancy (MacKinnon et

al., 2021), childhood (Minh et al., 2017a), adolescence (Leventhal et al., 2009), and into adulthood (Huang & Sparks, 2023), but the specific developmental periods or ages at which neighborhood conditions may have increased salience for health are relatively unknown. Each study capturing associations between neighborhood conditions and health within a specific developmental period adds to a cumulative body of evidence that indicates neighborhood conditions impact health across the first two decades of life (Leventhal, 2018). However, substantial heterogeneity in the measurement of neighborhood conditions (e.g., the specific neighborhood features assessed and the operationalization of neighborhoods) make comparisons of the magnitude of effects across age difficult to ascertain. This represents a significant gap in the literature, as uncovering *when* in development a particular exposure may have a pronounced effect is critically important for early identification and intervention.

Developmentally focused neighborhood researchers have posited three competing models of the developmental timing of neighborhood effects (Anderson et al., 2014; Leventhal et al., 2015): the early childhood model, the adolescent model, and the cumulative exposure model. The early childhood model suggests that neighborhood conditions and, in particular, socioeconomic resources of the neighborhood, early in the life course are especially important for subsequent outcomes (Duncan et al., 2010). This influence may operate directly, via neighborhood institutional resources (Dupere et al., 2010), or indirectly, through the effect of neighborhood conditions on caregiver wellbeing and behaviors (Shuey & Leventhal, 2019). In contrast, the adolescent model indicates that the autonomy and exploration characteristic of adolescence increases the

salience of extrafamilial contexts, such as the neighborhood (Leventhal et al., 2009). Thus, greater exposure to harmful neighborhood conditions that is unmediated by caregivers and other adults may make the effects of these conditions on outcomes especially pervasive. Finally, the cumulative exposure model suggests that rather than exposure to poor neighborhood conditions at a specific developmental period, the accumulation of exposure to poor neighborhood conditions may be more impactful (Crowder & South, 2011). Although these models do not extend beyond the first two decades of life, the developmental challenges of adolescence (e.g., autonomy, exploration, identity) remain salient through emerging adulthood (Arnett, 2007). As such, it is possible that neighborhood exert influence through similar mechanisms in adolescence and emerging adulthood.

Few studies have explicitly focused on issues related to developmental timing within the neighborhoods and health literature (Leventhal, 2018). However, existing empirical research examining the role of developmental timing of neighborhood effects presented herein is interpreted in the context of these models. Across studies, evidence supporting the early childhood model versus the adolescent model is mixed. For example, the study by Anderson and colleagues (2019) leveraged three cohorts of children and adolescents, who completed baseline assessments at age 6-, 9-, or 12-years-old, and were followed across three timepoints. In this study, the association between neighborhood conditions (measured at baseline for each cohort) and trajectories of reading achievement, internalizing, and externalizing were examined (Anderson et al., 2019). Differences in outcome trajectories across cohorts were evaluated to assess developmental timing

(Anderson et al., 2019). The authors found that neighborhood advantage (that is, residing in a higher SES neighborhood) was associated with reading achievement across cohorts but the magnitude of this association was marginally larger in the younger cohorts, providing support for the early childhood model. This study also provided support for the adolescent model, as adolescents residing in more advantaged neighborhoods experienced faster growth in reading achievement, relative to younger children (Anderson et al., 2019). Notably, there were no between-cohort differences in the associations between neighborhood disadvantage and behavioral problems (Anderson et al., 2019). One additional study leveraged a multi-cohort design to examine the developmental timing of neighborhood effects (Gard et al., 2021). Across the two cohorts, greater neighborhood disadvantage early childhood, but not adolescence, was associated with greater amygdala reactivity to ambiguous faces at age 20; however, the magnitude of the association was attenuated when concurrent measures of disadvantage were controlled for (Gard et al., 2021). One additional study has demonstrated support for the adolescent model of neighborhood effects (Wodtke et al., 2016). Specifically, Wodtke and colleagues (2016) found that residing in a more disadvantaged neighborhood in adolescence was strongly associated with educational achievement. Although childhood residence in disadvantaged neighborhoods was also associated with educational achievement, this effect was less pronounced (Wodtke et al., 2016).

Several additional studies have drawn conclusions regarding age-specific effects of neighborhood conditions and depressive symptoms (e.g., Barr, 2018; Estrada-Martinez et al., 2019). Using the National Study of Adolescent to Adult Health (Add Health), both

Barr (2018) and Estrada-Martinez and colleagues (2019) examined associations between adolescent neighborhood conditions and trajectories of depressive symptoms by age across adolescence and into emerging adulthood. Estrada-Martinez and colleagues (2019) found that baseline neighborhood satisfaction interacted with age to predict depressive symptoms, such that the magnitude of the significant association between greater neighborhood satisfaction and lower depressive symptoms increased with age among Mexican and Puerto Rican, but not other Latino subgroups. The authors also reported that lower neighborhood SES was associated with depressive symptoms across age, increasing in strength across age (Estrada-Martinez et al., 2019). In contrast, Barr (2018) found that adolescent neighborhood disorder was associated with concurrent, but not persistent, depressive symptoms.

These studies represent an important step toward developing a deeper understanding of developmental timing in relation to neighborhood conditions and health, allowing for a direct comparison of associations across developmental periods. However, the reliance on a single observation within each developmental period (e.g., Gard et al., 2020; Wodtke et al., 2016) or a single timepoint of neighborhood conditions (e.g., Anderson et al., 2019; Barr, 2018; Estrada-Martinez et al., 2019), obscures the extent to which the magnitude of the association between neighborhood conditions and health varies by age within developmental periods. A more granular understanding of the age(s) at which youth may be most susceptible to the effects of adverse neighborhood conditions has meaningful implications for intervention (e.g., screening and identification of who is most at risk, developmentally appropriate intervention).

Of note, there is a separate but related body of literature that examines the effects of residential mobility on outcomes by age, predominantly leveraging the Moving to Opportunity study sponsored by Department of Housing and Urban Development (e.g., Gresham et al., 2024; Schmidt et al., 2017). Most participants that moved as part of the Moving to Opportunity study did indeed relocate to improved neighborhoods (H. Kim et al., 2023). However, it is difficult to disentangle the age-specific impact of better neighborhood conditions from the age-specific impact of relocation, which is accompanied by considerable disruptions to other social contexts (e.g., disruption of peer groups, new school). As such, I did not consider this body of literature in the present dissertation.

1.4 Differential Associations Between Neighborhoods and Health by Sex

Numerous studies show differences in the prevalence and incidence of depressive symptoms and substance use by sex, such that depressive symptoms were higher among females than males (Schubert et al., 2017; D. J. Smith et al., 2008) and that substance use is higher among males than females (Brady & Randall, 1999). There is also evidence to suggest that males and females may experience differential consequences of residing in an adverse neighborhood context (King et al., 2022). Specifically, one study found that females were more vulnerable to effects of neighborhood disadvantage on depressive symptoms than males (King et al., 2022). However, the moderating role of gender in associations between neighborhood characteristics and development remains unclear, with one review describing mixed findings across studies (Minh et al., 2017b). Given these findings, the inclusion of sex as a moderator of the association between

neighborhood concentrated disadvantage and depressive symptoms and substance use merits investigation.

1.5 Specific Aims

In this dissertation, my overarching goal is to provide key developmental insights into the role of the neighborhood context in shaping health from adolescence to emerging adulthood. I will do so by investigating the age-varying associations between exposure to neighborhood concentrated disadvantage and two health-related outcomes – depressive symptoms and binge drinking – via secondary analysis of the National Study of Adolescent to Adult Health (Add Health).

I elected to focus on age-varying associations in adolescence and emerging adulthood as they represent key developmental periods during which extrafamilial contexts, such as the neighborhood, may have increased salience (Leventhal, 2018). For example, adolescents may experience less direct supervision and their exposure to their neighborhood may be unmediated by caregivers and other adults, during which time they may more directly engage with negative environmental features (Leventhal, 2018). This increased exploration continues into emerging adulthood, extending the salience of the neighborhood context.

Depressive symptoms and binge drinking were selected as the outcomes of interest because adolescence and emerging adulthood also represent developmental periods of increased risk of poor mental health (Jackson & Goossens, 2020). In the United States alone, over 5 million (20%) of adolescents have had at least one major depressive episode in the past year (Substance Use and Mental Health Services

Administration, 2022). The prevalence of depressive symptoms and diagnoses among adolescents has also increased over the past 20 years (Askari et al., 2023). One meta-analysis across several cultural contexts showed that 34% of adolescents experienced depressive symptoms, with approximately 8% of adolescents having major depressive disorder (Shorey et al., 2022). Although depressive symptoms tend to peak in late adolescence (Schuler et al., 2015), a systematic review of the literature showed that in some studies, up to 32% of the general population sampled report persistent depressive symptoms that extend into emerging adulthood (Musliner et al., 2016). The increased autonomy and accompanying neurodevelopmental changes (e.g., increases in reward sensitivity and risk-taking behaviors; Romer, 2010) experienced in adolescence and emerging adulthood may also increase the risk of substance use. Indeed, a nationally representative survey among adolescents 12-17 showed that 7% of adolescents reporting drinking in the past 30 days, with over half of those adolescents (3.8%) reporting at least one incident of binge drinking (4-5 drinks on one occasion; Substance Use and Mental Health Services Administration, 2022). Research suggests early initiation of substance use in adolescence may lead to problematic substance use in emerging adulthood (Nelson et al., 2015), highlighting a need to better understand how certain risk factors, such as neighborhood concentrated disadvantage, may confer risk for problematic drinking across adolescence and emerging adulthood.

Taken together, the neighborhood context may have a pronounced effect on depressive symptoms and binge drinking during adolescence and emerging adulthood relative to other development periods. As such, a more nuanced understanding of when in

adolescence and emerging adulthood neighborhood conditions and these outcomes, and the extent to which these associations differ by biological sex, is warranted. Findings from this study will provide valuable insight into *when* and *for whom* neighborhood conditions matter, informing intervention and prevention efforts.

My specific aims were:

1.5.1 Change in the Predictor and Outcomes Across Age (Aim 1)

To examine change in neighborhood conditions, depressive symptoms, and binge drinking across adolescence and emerging adulthood (i.e., ages 13-25). I expected that neighborhood concentrated disadvantage would increase from adolescence to emerging adulthood. This expectation arose from evidence of increasing concentrated poverty from the 1990s to the 2000s (Iceland & Hernandez, 2017), when Add Health participants were 13-25, rather than inherent changes due to age. I expected that depressive symptoms would change quadratically across adolescence and emerging adulthood, initially increasing across adolescence and early emerging adulthood (i.e., ages 13-20) and declining toward the middle-to-end of emerging adulthood (i.e., ages 21-25). I further expected that depressive symptoms would peak in late adolescence, around age 17-18. With respect to binge drinking, I anticipated that the likelihood of binge drinking would increase approximately linearly from adolescence to emerging adulthood, peaking in around age 21.

1.5.2 Age-Varying Associations between Neighborhood Concentrated Disadvantage and Depressive Symptoms (Aim 2)

Aim 2a: To examine the direct age-varying association between neighborhood concentrated disadvantage and depressive symptoms from adolescence through emerging adulthood. I predicted that greater neighborhood concentrated disadvantage would be related to higher depressive symptoms across age. I further expected that this association would be strongest in late adolescence. I also expected that depressive symptoms would be higher among females than males across age.

Aim 2b: To investigate whether the age-varying association between neighborhood concentrated disadvantage and depressive symptoms varied by age. I anticipated that the magnitude of association between neighborhood concentrated disadvantage and depressive symptoms across age would be higher among females than males.

1.5.3 Age-Varying Associations between Neighborhood Concentrated Disadvantage and Binge Drinking (Aim 3)

Aim 3a: To investigate the age-varying association between neighborhood concentrated disadvantage on the likelihood of binge drinking across adolescence and emerging adulthood (i.e., ages 13-25). I anticipated that residing in a more disadvantaged neighborhood would be associated with a higher likelihood of binge drinking at all ages, but that this effect would be strongest in emerging adulthood, when risk-taking behaviors and ready access to alcohol overlap.

Aim 3b: To examine the extent to which the association the age-varying association between neighborhood concentrated disadvantage and binge drinking differs by biological sex. I expected that the effect of neighborhood concentrated disadvantage on binge drinking in emerging adulthood would be more pronounced among males than females.

Chapter 2: Method

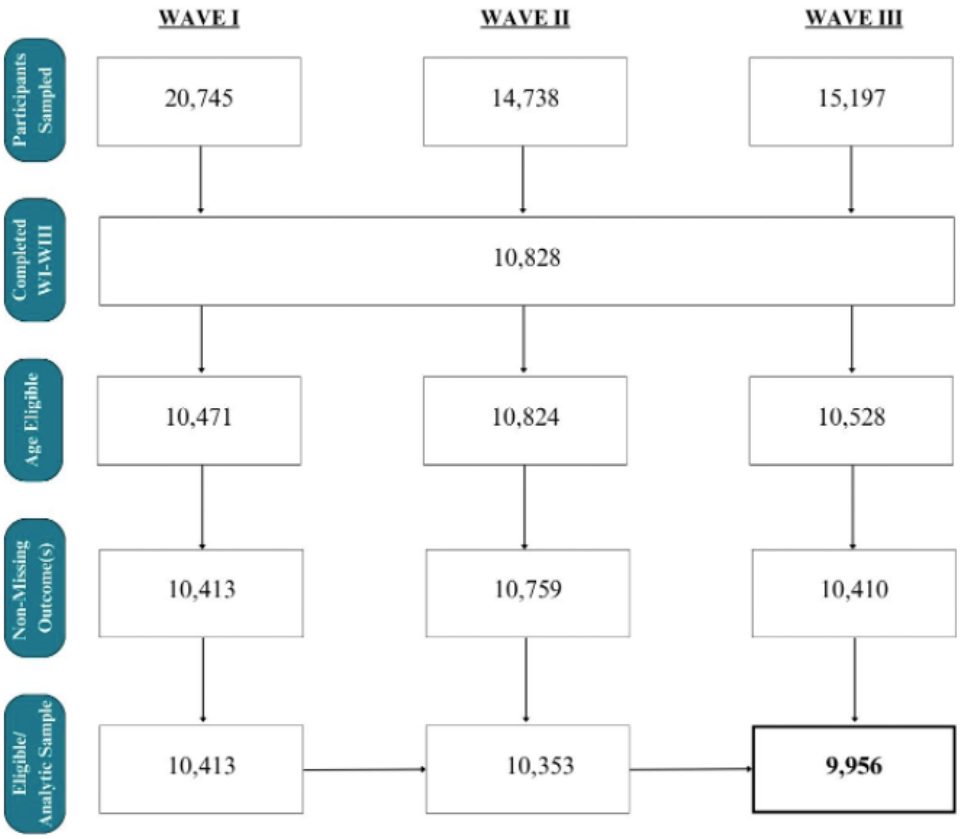
2.1 Participants

Over 90,000 adolescents in grades 7-12 participated in the National Study of Adolescent to Adult Health (Add Health; Harris, 2013a), 20,745 of whom were selected for longitudinal follow-up. At this initial timepoint (i.e., Wave I), participants were between 12-20 years of age. Of interest to the current study were the 10,828 participants that remained in the study through emerging adulthood (i.e., Wave III; aged 18-26). Due to the limited number of participants that were younger than 13 years of age, the analyses were restricted to those that were 13 years of age or older at Wave I. Additionally, as the focus of the present dissertation study is to examine the associations between neighborhood concentrated disadvantage and depressive symptoms and substance use from adolescence through emerging adulthood, I restricted the sample to only include individuals 25 and under at each timepoint. Additionally, participants were removed from the sample if they were missing either outcome (i.e., depressive symptoms or binge drinking) at any of the three timepoints (Wave I: N = 58; Wave II: N = 65; Wave III: N = 118). The eligible sample at each timepoint (i.e., those than participated in Waves I-III, were between the ages of 18-25 at each timepoint, and had non-missing outcome data)

was then merged to construct the final analytic sample (N = 9,956). See Figure 1 for a flowchart of sample selection.

Figure 1

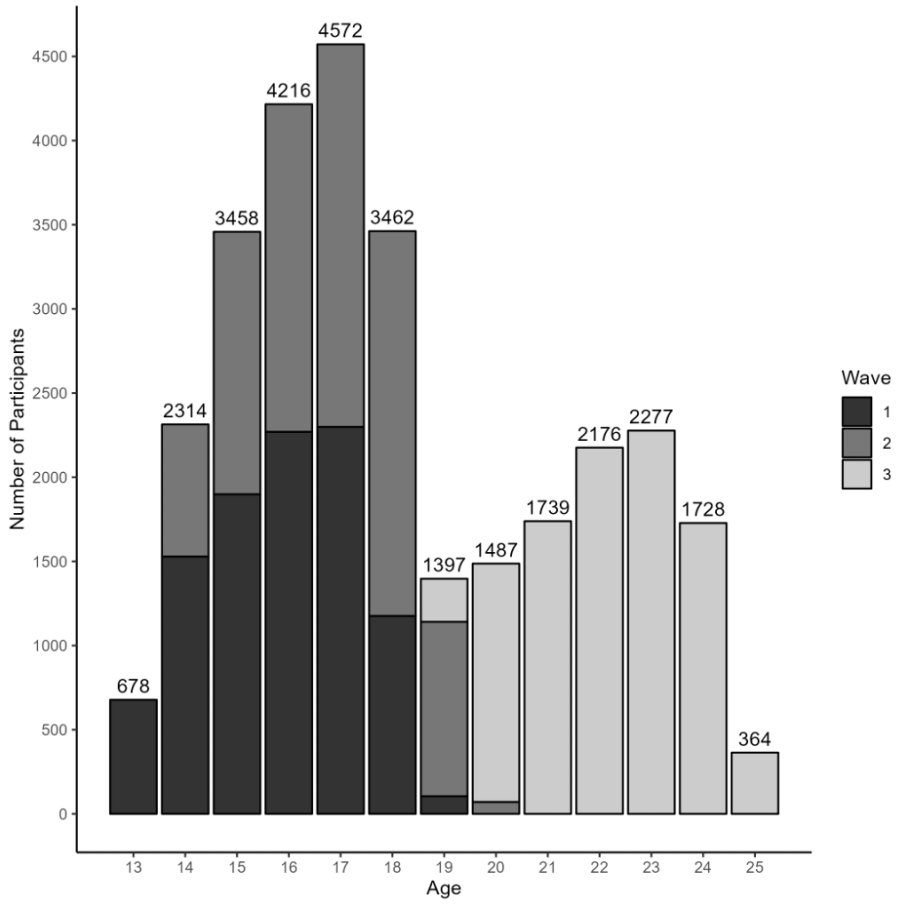
Flowchart of sample selection for each time point of the National Adolescent to Adult Health (Add Health) study, as well as the final analytic sample (N = 9,956)



The sample was 53.18% female (N = 5,295), predominantly White (68.74%, N = 6,844), and non-Hispanic or Latino (84.42%, N = 8,405), with limited variability in other racial or ethnic categories. On average, participants were 15.80 years of age at Wave I (SD = 1.44), 16.71 years of age at Wave II (SD = 1.44), and 22.15 years of age at Wave III (SD = 1.45). See Figure 2 for a breakdown of participant age, rounded to the nearest whole number, by Add Health Wave.

Figure 2

Stacked bar plot of age by Add Health timepoint among participants aged 13-25 at Waves I-III (N = 9,956)



Most participants had at least one parent/caregiver with education beyond a high school diploma or GED (63.80%, N = 6,352) and at least one working parent/caregiver (90.21%, N = 8,981). Fewer than 10% of participants belonged to a household receiving public assistance (8.93%, N = 889).

2.2 Procedure

The present study leveraged data from the Add Health study, a nationally representative cohort study originally designed to study social, behavioral, and biological

determinants of health (Harris, 2013b). Over 90,000 adolescents were recruited from select high schools in 1994-95 (Wave I; ages 12-20) to take part in the Add Health study. Participants were recruited from 80 high schools and 52 middle schools with the goal of national representation in terms of region, urbanicity, school size and type, and race and ethnicity (Harris, 2013b). A subsample of 20,745 adolescents were selected from the original ~90,000 for longitudinal follow-up. Approximately one year later (1996), 14,738 adolescents in grades 8-12 took part in Wave II of the Add Health study. In 2001-02, 15,197 participants completed Wave III (ages 18-26). Although over 15,000 participants returned for Wave IV in 2008-09 and 12,000 participants returned for Wave V in 2016-18, very few participants were below age 26 (N = 159) at Wave III and no participants were below age 26 at Wave IV. As such, Wave III and Wave IV data were outside the scope of the present study.

At each time point, participants completed surveys via computer-assisted personal interview (CAPI), and sensitive questions (e.g., depression, anxiety, substance use) were completed during the self-administered portion of the interview. At Wave I, one parent or caregiver in the home also completed surveys providing individual- and household-level demographic information. Subsequently, an array of contextual data was linked to participant data, including administrative data reflecting the neighborhood environment.

The Add Health study is led by the Carolina Population Center at the University of North Carolina – Chapel Hill (Harris et al., 2019). This secondary analysis of Add Health data was approved by the University of Minnesota's Institutional Review Board

under Dr. Theresa Osypuk's Add Health Restricted-Use Contract at the University of Minnesota.

2.3 Measures

2.3.1 Neighborhood Concentrated Disadvantage

To operationalize concentrated disadvantage at Waves I-III, I used five census block group level variables available at each Wave. While census block groups may not accurately represent an individual's perception of their neighborhood boundaries, they are relatively small and homogeneous areas that typically consist of between 600-3,000 people and are the smallest geographies for which the census publishes economic data. Participant addresses at each Wave were geocoded to the census block group level and linked to census data. Waves I and II were linked to 1990 decennial census data and geographic boundaries, Wave III was linked to decennial 2000 census data and geographic boundaries. At each wave, concentrated disadvantage was measured via the following indicators in accordance with previous research (Farrell, 2020; Hackman et al., 2021): proportion of families below the federal poverty line, unemployment rate among those aged 16+ that are in the workforce, proportion of single-headed households, proportion of households receiving public assistance, and proportion age 25+ without a high school diploma or GED.

To construct the measure of neighborhood concentrated disadvantage from the five indicators at each Wave, I conducted a principal component analysis to confirm one-dimensionality and to extract weights for each item (Table 1). Each item was then multiplied by its weight and the weighted items were summed to create a composite

score, with higher scores indicating greater disadvantage. Scores ranged from 0-1 and represented the proportion of residents within each neighborhood that were socioeconomically disadvantaged. Internal consistency was acceptable at each of the three Waves (α = .889, .889, and .762, respectively).

Table 1

Weights from a series of principal components analyses to create a composite of neighborhood concentrated disadvantage for Add Health Waves I-III (N = 9,956)

	Wave I	Wave II	Wave III
Proportion of families below the federal poverty line	0.24	0.24	0.29
Proportion of individuals 16+ in the workforce that are unemployed	0.23	0.23	0.23
Proportion of single-headed households	0.22	0.22	0.24
Proportion of households receiving public assistance	0.24	0.24	0.26
Proportion of individuals 25+ without a high school diploma/GED	0.21	0.21	0.24

2.3.2 Depressive Symptoms

Depressive symptoms at Waves I-III were assessed via nine items from the 20-item Center for Epidemiological Studies - Depression scale (Radloff, 1977). Although additional items were assessed at each Wave, only nine items were consistently included across the three Waves. For each item, participants (adolescents, now adults) reported how often in the last seven days they experienced symptoms such as being depressed, sad, or too tired to do things (see Appendix A for a full list of items). Two items ("you felt you were just as good as other people" and "you enjoyed life") were reverse coded. The items were rated on a 4-point Likert scale from 0 ("rarely or none of the time") to 3 ("most or all of the time"). Responses to each item were summed, with a total possible score ranging from 0-27, with higher scores indicating greater symptoms. In line with previous research (Andresen et al., 1994), a score of 10 or greater was used to indicate

clinically meaningful depressive symptoms. Internal consistency was acceptable at each timepoint (α = .791, .798, and .805 at Wave I, II, and III, respectively).

2.3.3 Binge Drinking

At each Wave, participants were asked whether they had ever had (or had since the month of their last interview) a drink of beer, wine, or liquor more than 2 or 3 times in their life (not including a sip or taste of someone else's drink). If the participant responded "yes", they were subsequently asked "over the past 12 months, on how many days did you drink 5 or more drinks in a row?" to assess binge drinking behaviors. Response options included "never" (0), "1 or 2 days in past 12 months" (1), "once or month or less (3-12 times in the past 12 months)" (2), "2 or 3 days a month" (3), "1 or 2 days a week" (4), "3 to 5 days a week" (5), and "every day or almost every day" (6). If a participant responded "no" to the initial question of having ever drank more than 2-3 sips, they were coded as "never" (0) for binge drinking. Although I intended to examine binge drinking as a count score from 0 to 6, with higher scores indicating a greater frequency of binge drinking, the variance in binge drinking was greater than the mean, indicating potential issues with overdispersion. Accordingly, responses from the single-item assessment of binge drinking were collapsed into "never" (0) or "at least once in the past 12 months" (1).

2.3.4 Biological Sex

Biological sex was reported at Wave I, where 0 = male and 1 = female.

2.3.5 Race and Ethnicity

Participant race was self-reported at Wave I as White, Black or African American, American Indian or Native American, Asian or Pacific Islander, or Other. Participants also reported whether they were of Hispanic or Latino origin. Due to limited variability across racial and ethnic categories, race and ethnicity were collapsed to create a single variable. Race/ethnicity was categorized as Non-Hispanic White (0), Non-Hispanic Black (1), Hispanic (2), and other (3). Prior to analyses, the collapsed race and ethnicity was recoded into three separate variables (i.e., dummy-coded), with non-Hispanic White as the reference group in each (i.e., Non-Hispanic Black vs. Non-Hispanic White; Hispanic vs. Non-Hispanic White, Other vs. Non-Hispanic White).

2.3.6 Caregiver Educational Attainment

At Wave I, the responding parent or caregiver reported their educational attainment as well as the educational attainment of the other parent or caregiver in the household, if applicable. Educational attainment was reported as "8th grade or less", "more than 8th grade, but did not graduate from high school", "went to a business, trade or vocational school after high school", "went to college, but did not graduate", "graduated from a college or university", "professional training beyond a 4-year college or university", or "never went to school". From this, I created a combined parental educational attainment variable reflecting the highest level of education obtained by either parent or caregiver (e.g., if parent 1 had a high school diploma and parent 2 had a 4-year degree, the combined highest level of education was coded as "graduated from a college or university"). Due to limited variability in some categories, parental educational

attainment was coded in the following categories, “less than high school” (0), “high school diploma or GED” (1), “some college” (2), “4-year college degree” (3), and “professional or graduate school” (4).

2.3.7 Caregiver Employment Status

The sampled parent or caregiver reported whether they, or the other parent/caregiver in the home, if applicable, was working outside of the home at Wave I. Responses to these two items were coded dichotomously as a single item (i.e., if one or both caregivers were employed, parental employment was coded as “yes”, else, it was coded as “no”).

2.3.8 Household Receipt of Public Assistance

At Wave I, the responding parent/caregiver reported whether they were receiving public assistance (e.g., welfare). Responses were coded dichotomously as “yes” (1) or “no” (0).

2.4 Data Analysis

All analyses were conducted in R version 4.1.3 (R Core Team, 2023). The results reported in this dissertation were reviewed for deductive disclosure risk prior to release from Add Health’s secure remote server.

2.4.1 Preliminary Data Analysis

The proportion of missing data for each variable of interest in the study was examined. As noted above, participants that had missing depressive symptom and/or binge drinking data at any of the timepoints of interest were excluded. Missing data on most variables was minimal. However, approximately 12% of participants were missing

data on one or more of the parent demographic variables (i.e., education, employment, public assistance). The appropriateness of using multiple imputation to handle missing data is a well-debated topic, with researchers raising concerns of biased estimates when multiple imputation is used with a large proportion of missing data (Dong & Peng, 2013). Yet, evidence from simulation studies suggests that when there is sufficient auxiliary information (i.e., variables not included in the analytic model), multiple imputation reduces bias relative to other missing data management approaches, even in the context of large amounts of missing data (e.g., Madley-Dowd et al., 2019). In line with this evidence, I conducted multiple imputation with 10 imputations, including auxiliary variables in the model (i.e., indicator variables of neighborhood concentrated disadvantage, demographic covariates at Waves II and III).

Following imputation, I examined descriptive statistics (i.e., mean, standard deviation, skew, kurtosis) of the key study variables. Spearman correlations were used to examine the bivariate associations between continuous and ordinal variables. To examine the bivariate associations between categorical and continuous variables (e.g., biological sex and depressive symptoms), independent samples t-tests and chi-square difference tests were conducted, depending on whether the categorical variable had 2 or 3+ levels. Variables that were significantly associated with both the predictor and outcome in the bivariate models (i.e., $p < .05$) were selected as covariates. The internal consistency of each scale was assessed, with α greater than .70 indicating acceptable reliability (Gliem & Gliem, 2003).

2.4.2 Time-Varying Effect Models

I conducted time-varying effect models (TVEM) to address my three primary research questions. TVEM is a nonparametric model that allows one to examine the how the magnitude or direction of an effect between a predictor and an outcome changes over time (Lanza et al., 2016a). In the case of an age-varying analysis, the data points were structured by age, yielding estimates of the association between a predictor and outcome at each continuous age represented in the data.

I first examined variation in neighborhood concentrated disadvantage, depressive symptoms, and binge drinking across ages 13-25 (Aim 1) via a series of intercept-only models. Intercept-only TVEM estimate the average level of a specified outcome as a function of continuous age.

To examine the age-varying associations between neighborhood concentrated disadvantage, biological sex, and their two-way interaction on depressive symptoms (Aim 2) and binge drinking (Aim 3), I specified a series of linear TVEM. I first examined the unadjusted association between neighborhood concentrated disadvantage and sex with depressive symptoms (Aim 2a; Models 1 and 2) and binge drinking (Aim 3a; Models 1 and 2). Next, I examined the adjusted effect of neighborhood concentrated disadvantage and biological sex by adding covariates to each model. TVEM accommodates time-varying and time-invariant covariates. Although certain variables are traditionally assumed to be time-invariant (e.g., biological sex), TVEM estimates how the association between a given covariate outcome changes as a function of time. As such, constructs that do not change over time may still have a time-varying effect. In line with

recommendations by Lanza and colleagues (2016b), separate models with covariates specified as time-invariant (Model 3) and time-varying (Model 4) were conducted for each outcome, and model fit was compared. When the inclusion of time-varying covariates did not improve model fit, the model with time-invariant covariates was selected in the interest of model parsimony.

Finally, the two-way interaction between neighborhood concentrated disadvantage and biological sex was added to each model to examine whether the effect of biological sex on the association between neighborhood concentrated disadvantage and depressive symptoms (Aim 2b) and binge drinking (Aim 3b) varied with age. Prior to creating the interaction term, neighborhood concentrated disadvantage was mean-centered. Significant interactions were probed to determine the age(s) at which the association between neighborhood concentrated disadvantage and the outcome differed by biological sex. A nonsignificant interaction term yields two possible interpretations. First, an interaction that is not significant may indicate that there is no sex difference in the association between neighborhood concentrated disadvantage and the outcome at any age (i.e., flat slope with estimates near zero or one, depending on the outcome). Second, sex differences in the association between neighborhood concentrated disadvantage and the outcome exist, but do not vary with age (i.e., a flat slope with estimates that do not overlap zero or one, depending on the outcome). To aid in interpretation of nonsignificant interactions, I examined the predicted estimates and accompanying 95% confidence intervals.

Of note, the TVEM for depressive symptoms were modeled as an extension of linear regression given its continuous nature. In contrast, the TVEM for binge drinking was modeled as an extension of binomial logistic regression, given that binge drinking was dichotomous.

TVEM requires the specification of knots, which determine the complexity of each coefficient function and permit flexibility in terms of linearity. More knots allow the estimated coefficient function to more flexibly fit the shape of the data, but impacts model parsimony, reducing the likelihood that the results will generalize to other samples (Lanza et al., 2016b). Thus, selecting the number of knots for each model requires a balance between model complexity and parsimony. The number of knots in a model is specified via either p-splines or b-splines. P-splines automatically select the optimum number of knots based on Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC), with lower values indicating better fit. B-splines require one to specify a series of models with different numbers of knots, selecting the optimal number of knots based on the AIC and BIC. The automatic selection via p-splines prevents overfitting but provide less detail. In contrast, b-splines are less computationally intensive/more stable and yield more detailed curves but may be prone to overfitting. Given the benefits and drawbacks to both approaches, I used b-splines for all models.

Although the minimum sample size required to detect significant effects in TVEM is not well understood, Lanza and Linden-Carmichael (2021), suggest that a power analysis using a linear regression approach is appropriate. The authors argue that TVEM is a direct extension of linear regression and thus estimating the sample size

needed to detect an association of at least .80 would provide strong evidence of adequate power. Based on a power analysis performed with guidance from the Research Methodology Consulting Center (RMCC), the Add Health sample size is sufficient to detect medium-to-large interaction effect at > 80% power.

Longitudinal sampling weights were applied to all TVEM analyses to more closely estimate population-level associations. The 95% confidence intervals of each estimate were examined, with confidence intervals that did not overlap 0 (depressive symptoms) or 1 (binge drinking) indicating statistically meaningful results.

Chapter 3: Results

3.1 Preliminary results

On average, participants resided in census block groups with relatively low levels of concentrated disadvantage at each timepoint, with mean proportion of disadvantage ranging from 0.17 (SD = 0.10) at Waves I and II to 0.19 (SD = 0.10) at Wave III. In other words, Add Health participants resided in neighborhoods where, on average, roughly 17-19% of residents were considered socioeconomically disadvantaged. At Waves I and II, participants resided in neighborhoods where 3-70% of residents were socioeconomically disadvantaged. At Wave III, the percentage of residents that were socioeconomically disadvantaged among Add Health participant neighborhoods ranged from 1-78%. At each timepoint, neighborhood concentrated disadvantage was positively skewed, and a logarithmic + 1 transformation was performed. The average level of depressive symptoms decreased at each timepoint (Wave I: M = 5.90, SD = 4.27; Wave II: M = 5.84, SD = 4.24; Wave III: M = 4.63, SD = 4.08). I performed a logarithmic

transformation on the depressive symptoms score at each timepoint due to positive skew. See Table 2 for the full descriptive statistics for all key time-invariant and time-varying characteristics.

Table 2

Descriptive statistics of key study variables for sample of National Longitudinal Study of Adolescent to Adult Health (Add Health) participants aged 13-25 at Waves I-III (N = 9,956)

	Wave I		Wave II		Wave III	
	M/N	SD/%	M/N	SD/%	M/N	SD/%
Time-Invariant						
Sex						
Male	4,4,61	46.82	-	-	-	-
Female	5,295	53.18	-	-	-	-
Race						
White	6,844	68.74	-	-	-	-
Black or African American	2,145	21.54	-	-	-	-
American Indian or Native American	194	1.95	-	-	-	-
Asian or Pacific Islander	773	7.76	-	-	-	-
Ethnicity						
Hispanic or Latino	1,551	15.58	-	-	-	-
Not Hispanic or Latino	8,405	84.42	-	-	-	-
Educational Attainment						
Less than high school	1,202	12.07	-	-	-	-
High school diploma or GED	2,402	24.13	-	-	-	-
Some college	2,960	29.73	-	-	-	-
4-year college degree	1,851	18.59	-	-	-	-
Professional or graduate school	1,541	15.48	-	-	-	-
Employed						
Yes	8,981	90.21	-	-	-	-
Not	975	9.79	-	-	-	-
Receipt of public assistance						
Yes	889	8.93	-	-	-	-
No	9,067	91.07	-	-	-	-
Time-Varying						
Age	15.80	1.44	16.71	1.44	22.15	1.45
Neighborhood concentrated disadvantage	0.17	0.10	0.17	0.10	0.19	0.10
Depressive symptoms	5.90	4.27	5.84	4.24	4.43	4.08
Ever drank > 2-3 sips						
Yes	5,410	54.34	4,775	47.96	7,670	77.04
No	4,446	44.66	5,181	52.04	2,286	22.96
Binge drinking						
Never	7,576	76.09	7,181	72.13	5,145	51.68
1 or 2 days in past 12 months	870	8.74	920	9.24	1,608	16.15
Once per month or less	562	5.64	624	6.27	1,032	10.37
2 or 3 days a month	398	4.00	476	4.78	873	8.77
1 or 2 days a week	335	3.36	458	4.60	901	9.05
3 to 5 days a week	141	1.42	187	1.88	325	3.26
Every day or almost every day	74	0.74	110	1.10	72	0.72

There were at least 350 observations at each age from 13-25, with most ages (rounded to the nearest whole number) having at least 1,000 observations (Figure 2). The number of observations at each age indicated sufficient coverage to conduct time-varying effect models.

All time-varying effect models controlled for caregiver education, employment, and receipt of public assistance, and participant sex, race, and ethnicity. The bivariate associations between the selected covariates and the constructs of interest are presented in Tables 3 and 4.

Table 3

Spearman correlations of bivariate associations between key continuous and ordinal study variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
Wave I														
1. Parent education	-													
2. Parent employment	**.26**	-												
3. Public assistance	**-.25**	**-.38**	-											
4. Age	**-.07**	**-.02**	**-.02**	-										
5. NCD	**-.32**	**-.21**	**.25**	**.02**	-									
6. Depressive symptoms	**-.14**	**-.05**	**.07**	**.13**	**.09**	-								
7. Binge drinking	**-.06**	-.01	.01	**.22**	**-.05**	**.16**	-							
Wave II														
8. Age	**-.07**	**-.02**	-.02	**.99**	**.02**	**.13**	**.22**	-						
9. NCD	**-.32**	**-.22**	**.25**	**.02**	**.98**	**.09**	**-.05**	**.03**	-					
10. Depressive symptoms	**-.13**	**-.06**	**.07**	**.10**	**.08**	**.57**	**.12**	**.10**	**.09**	-				
11. Binge drinking	**-.02**	**.03**	**-.03**	**.18**	**-.11**	**.10**	**.48**	**.18**	**-.10**	**.11**	-			
Wave III														
12. Age	**-.07**	**-.02**	-.01	**.99**	**.02**	**.13**	**.23**	**.99**	**.02**	**.10**	**.18**	-		
13. NCD	**-.21**	**-.14**	**.17**	.01	**.57**	**.09**	**-.05**	**.02**	**.56**	**.07**	**-.09**	**.02**	-	
14. Depressive symptom	**-.09**	**-.04**	**.07**	.01	**.04**	**.33**	**.02**	.01	**.05**	**.37**	**.02**	.00	**.06**	-
15. Binge drinking	**.11**	**.09**	**-.09**	-.00	**-.20**	**-.06**	**.18**	-.00	**-.20**	**-.04**	**.23**	-.00	**-.18**	-.01

Note. Bold indicates $p < .05$. NCD = neighborhood concentrated disadvantage.

Table 4

Results from a series of independent samples t-tests of the bivariate associations between biological sex, race, and ethnicity with neighborhood concentrated disadvantage and depressive symptoms, and chi-square difference tests of the bivariate associations between biological sex, race, and ethnicity and binge drinking

	Sex			**Race**			**Ethnicity**		
	t / χ^2	**df**	p	t / χ^2	**df**	p	t / χ^2	**df**	p
Wave I									
NCD	4.00	9,917	.000	33.25	4,594.10	.000	14.19	2,256.20	.000
Depressive Symptoms	16.82	9,892.90	.000	9.05	5,898.10	.000	6.48	2,131.90	.000
Binge Drinking	35.36	1	.000	36.76	1	.000	274.45	1	.000
Wave II									
NCD	4.01	9,915	.000	32.61	4,613.60	.000	14.44	2,246.70	.000
Depressive Symptoms	16.75	9,901.50	.000	8.57	5,916.10	.000	7.77	2,128.40	.000
Binge Drinking	186.54	1	.000	302.85	1	.000	571.00	1	.000
Wave III									
NCD	3.70	9,879.70	.000	28.56	4,888.50	.000	13.73	2,192.50	.000
Depressive Symptoms	10.55	9,943.40	.000	8.05	5,831.40	.000	3.95	2,073.20	.000
Binge Drinking	14.98	1	.000	9.57	1	.002	9.25	1	.002

Note. NCD = neighborhood concentrated disadvantage.

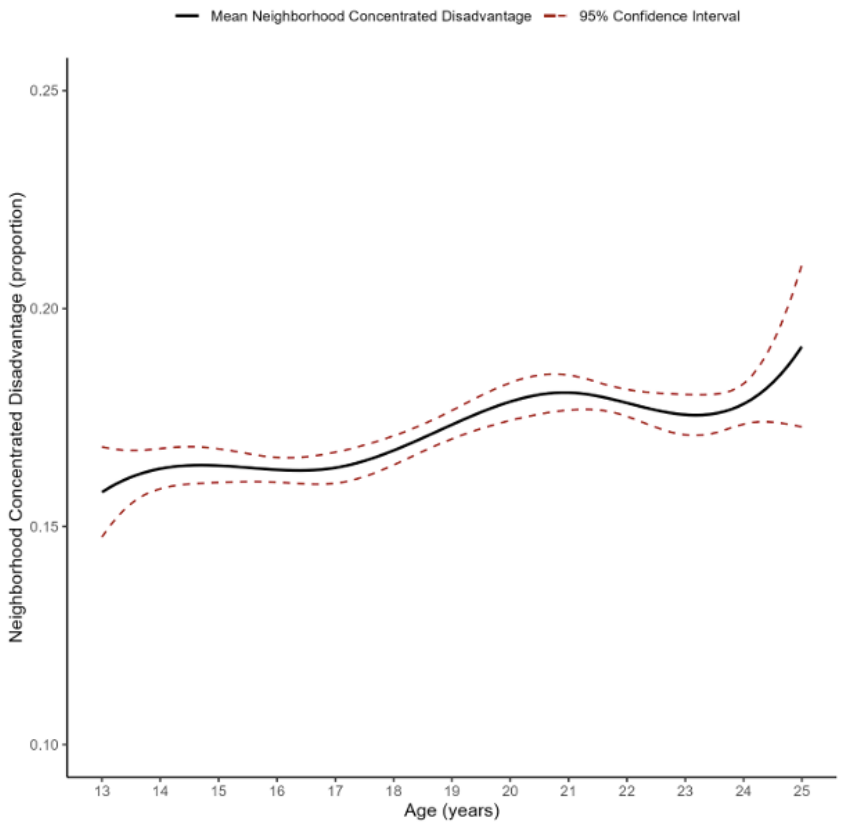
3.2 Variation in Neighborhood Concentrated Disadvantage, Depressive Symptoms, and Binge Drinking Across Age (Aim 1)

3.2.1 Neighborhood Concentrated Disadvantage Across Age (Aim 1a)

As expected, neighborhood concentrated disadvantage increased across ages 13-25 (Figure 3).

Figure 3

Plot of intercept-only time-varying effect model predicting neighborhood concentrated disadvantage across ages 13-25 (N = 9,956)



Specifically, mean neighborhood concentrated disadvantage grew from 0.16 (95% CI = 0.15, 0.17) at age 13 to 0.19 (95% CI = 0.17, 0.21) at age 25. In other words, on average, adolescents lived in neighborhoods where approximately 16% of residents were

socioeconomically disadvantaged but this increased to 19% by the end of emerging adulthood. This increase occurred approximately linearly across age; however, average neighborhood concentrated disadvantage was relatively stable from 13-17-years-old. Of note, the 95% confidence intervals approaching age 25 widened, indicating a greater degree of uncertainty in estimated mean neighborhood concentrated disadvantage relative to other ages included in the model. Model fit criteria for the optimal number of knots are presented in Table 5.

Table 5

Model fit information from linear time-varying effect models predicting neighborhood concentrated disadvantage across ages 13-25, with 1-6 knots specified for the b-spline

Knots	AIC	BIC
1	-47,000.44	-46,979.82
2	**-4,7030.78**	**-46,980.66**
3	-4,7010.47	-46,967.26
4	-4,7027.28	-46,972.75
5	-4,7028.85	-46,971.14
6	-4,7027.07	-46,966.94

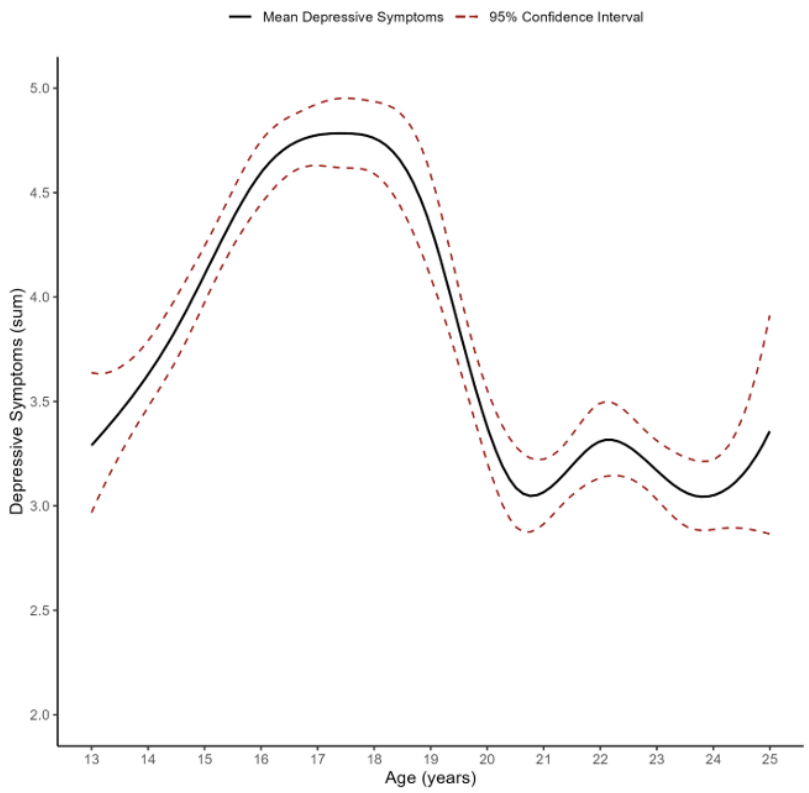
Note. Bold indicates selected model. AIC = Akaike Information Criterion. BIC = Bayesian Information Criterion.

3.2.2 Depressive Symptoms Across Age (Aim 1b)

Average depressive symptoms increased across adolescence, declining in early emerging adulthood (Figure 4). The functional form was roughly quadratic. These findings aligned, in part, with my expectations; depressive symptoms peaked in late adolescence and subsequently declined. However, the decline in depressive symptoms during early emerging adulthood was much steeper than expected, with only minor fluctuations thereafter.

Figure 4

Plot of intercept-only time-varying effect model predicting depressive symptoms across ages 13-25 (N = 9,956)



At age 13, participants reported relatively low depressive symptoms, on average (β = 3.29, 95% CI = 2.97, 3.64). Depressive symptoms then increased sharply until age 17 (β = 4.78, 95% CI = 4.63, 4.92), peaking at 17.36 (β = 4.78, 95% = 4.62, 4.95). Despite this increase, average depressive symptoms remained at subclinical levels. Depressive symptoms then declined sharply until age 20.88, returning to early adolescent levels (β = 3.05, 95% CI = 2.89, 3.22). Thereafter, there were only slight fluctuations in average depressive symptoms from age 21 (β = 3.07, 95% CI = 2.91, 3.22) through age 25 (β = 3.36, 95% CI = 2.87, 3.91). The model with seven knots yielded the lowest AIC and BIC (Table 6, Model 0).

Table 6

Model fit information from a series of time-varying effect models predicting depressive symptoms across ages 13-25

Model	Knots	AIC	BIC
Depressive Symptoms Intercept-Only (Model 0)	1	84915.12	84958.14
	2	84853.76	84904.00
	3	84873.28	84929.50
	4	84839.24	84897.79
	5	94926.88	84897.63
	6	84841.02	84905.48
	7	**84815.49**	**84890.32**
	8	84821.52	84899.38
	9	84814.81	84897.23
	10	84820.32	84902.85
NCD → Depressive Symptoms (Model 1)	3	84659.44	84746.62
	4	84621.84	84716.50
	5	84622.51	84725.18
	6	84620.10	84722.13
	7	**84593.72**	**84707.55**
	8	84600.06	84716.97
Sex → Depressive Symptoms (Model 2)	3	84312.41	84394.73
	4	84271.88	84369.83
	5	84257.12	84367.79
	6	8327153	84380.43
	7	**84240.57**	**84363.72**
	8	84248.39	84372.75
NCD and Sex → Depressive Symptoms (Model 3)	3	83630.87	83764.77
	4	83593.82	83739.73
	5	83583.37	83741.46
	6	83593.42	83777.58
	7	**83566.10**	**83766.63**
	8	83572.33	83775.60
NCD and Sex → Depressive Symptoms (Model 4)	3	83627.59	83793.02
	4	83589.74	83795.79
	5	83578.94	83774.36
	6	83590.22	83819.57
	7	**83562.40**	**83810.55**
	8	83568.63	83821.30
NCD x Sex → Depressive Symptoms (Model 5)	3	83626.44	83783.77
	4	83589.37	83757.09
	5	83579.52	83757.28
	6	83591.43	83770.00
	7	**83565.03**	**83756.93**
	8	83571.88	83769.11
	9	83566.19	83771.75
	10	83570.80	83783.90
	31	83562.12	83890.09

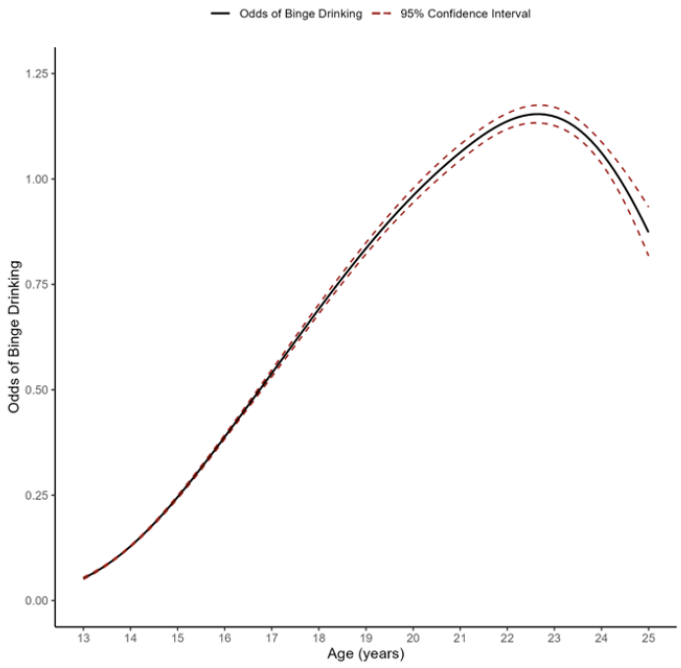
Note. The number of knots selected for each model is bolded. Model 1 examines the unadjusted association between neighborhood concentrated disadvantage and depressive symptoms; Model 2 examines the unadjusted association between biological sex and depressive symptoms; Model 3 examines the associations between neighborhood concentrated disadvantage and sex with depressive symptoms, with covariates specified as time-invariant; Model 4 examines the associations between neighborhood concentrated disadvantage and sex with depressive symptoms, with covariates specified as time-varying; Model 5 examined the two-way interaction between neighborhood concentrated disadvantage and biological sex on depressive symptoms.

3.2.3 Binge Drinking Across Age (Aim 1c)

The likelihood of binge drinking increased across adolescence and early emerging adulthood, declining toward the end of emerging adulthood, partially aligning with my expectations (Figure 5).

Figure 5

Plot of intercept-only time-varying effect model predicting likelihood of binge drinking across ages 13-25 (N = 9,956)



Across ages approximately 13-25, the likelihood of binge drinking was quadratic. The predicted probability of binge drinking in the past 12 months increased from 4.8% at age 13 (OR = 0.05, 95% CI = 0.5, 0.06) to 53.5% at age 22.94 (OR = 1.15, 95% CI = 1.13, 1.17). From age 22.57 (OR = 1.14, 95% = 1.12, 1.16) through age 25 (OR = 0.92, 95% CI = 0.87, 0.97) the probability of binge drinking decreased from 53.27% to 46.52%. Of note, the likelihood of binge drinking in the past 12 months was relatively low across age, but particularly during adolescence. The intercept-only model of likelihood of binge

drinking across age indicated that two knots provided optimal fit to the data (Table 7, Model 0).

Table 7

Model fit information from a series of time-varying effect models predicting binge drinking across ages 13-25

Model	Knots	AIC	BIC
Binge Drinking Intercept-Only (Model 0)	1	35203.02	35238.96
	2	**35204.43**	**35246.70**
	3	35205.72	35247.04
	4	35204.15	35248.44
	5	35206.28	35253.43
	6	35200.16	35254.40
	7	35203.29	35257.29
NCD → Binge Drinking (Model 1)	2	34748.10	34818.72
	3	34747.47	34818.74
	4	**34742.59**	**34818.48**
	5	34745.49	34825.93
	6	34739.69	34827.35
	7	34742.60	34832.47
Sex → Binge Drinking (Model 2)	1	34813.36	34880.88
	2	**34812.91**	**34887.75**
	3	34815.59	34892.41
	4	34812.91	34894.02
	5	34813.16	34900.25
	6	34806.06	34904.42
	7	34809.38	34909.44
NCD and Sex → Binge Drinking (Model 3)	3	33674.77	33824.43
	4	33672.04	33827.66
	5	33671.41	33836.03
	6	**33665.67**	**33841.27**
	7	33668.37	33848.49
	8	33666.33	33853.81
NCD and Sex → Binge Drinking (Model 4)	3	33510.73	33798.87
	4	33508.21	33804.29
	5	33504.95	33821.64
	6	**33489.14**	**33831.47**
	7	33492.98	33850.27
	8	33489.77	33860.74
NCD x Sex → Binge Drinking (Model 5)	3	33509.11	33807.24
	4	33506.71	33813.14
	5	33502.43	33830.48
	6	**33486.18**	**33839.79**
	7	33489.90	33857.99
	8	33486.93	33868.65
	9	33489.15	33875.53
	10	33489.05	33884.85

Note. The number of knots selected for each model is bolded. Model 1 examines the unadjusted association between neighborhood concentrated disadvantage and likelihood of binge drinking; Model 2 examines the unadjusted association between biological sex and likelihood of binge drinking; Model 3 examines the associations between neighborhood concentrated disadvantage and sex with likelihood of binge drinking, with covariates specified as time-invariant; Model 4 examines the associations between neighborhood concentrated disadvantage and sex with likelihood of binge drinking, with covariates specified as time-varying; Model 5 examined the two-way interaction between neighborhood concentrated disadvantage and biological sex on likelihood of binge drinking.

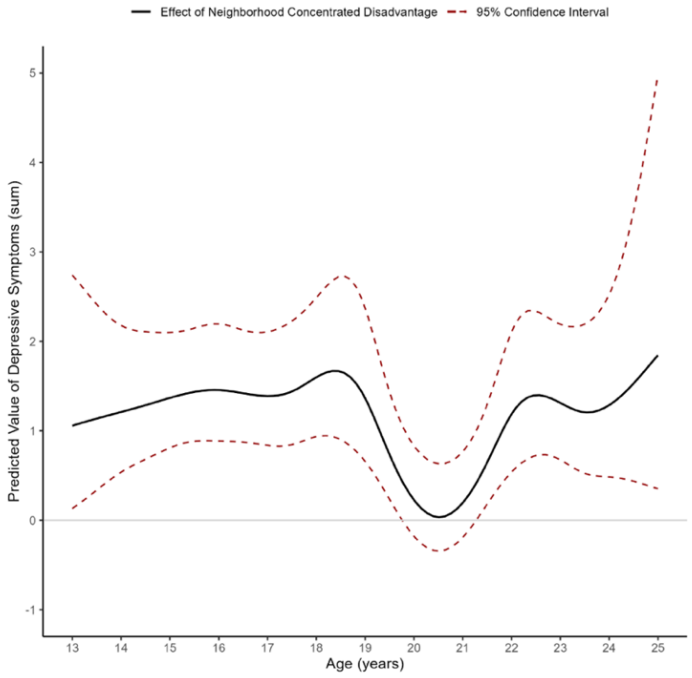
3.3 Age-Varying Associations with Depressive Symptoms (Aim 2)

3.3.1 Association Between Neighborhood Concentrated Disadvantage and Depressive Symptoms (Aim 2a)

In the model unadjusted for covariates, residing in a neighborhood with higher levels of concentrated disadvantage was associated with greater depressive symptoms across most of adolescence and emerging adulthood (Figure 6).

Figure 6

Plot of time-varying effect model predicting depressive symptoms from neighborhood concentrated disadvantage across ages 13-25, unadjusted for covariates (N = 9,956)



As shown in the figure, residing in a neighborhood with greater concentrated disadvantage was significantly associated with higher depressive symptoms from age 13 ($\beta = 1.06$, 95% CI = 0.13, 2.74) until age 19.67 ($\beta = 0.54$, 95% CI = 0.08, 1.18). From 19.79 ($\beta = 0.41$, 95% CI = -0.02, 1.03) through age 21.24 ($\beta = 0.38$, 95% CI = -0.03, 0.96), the association between neighborhood concentrated disadvantage and depressive

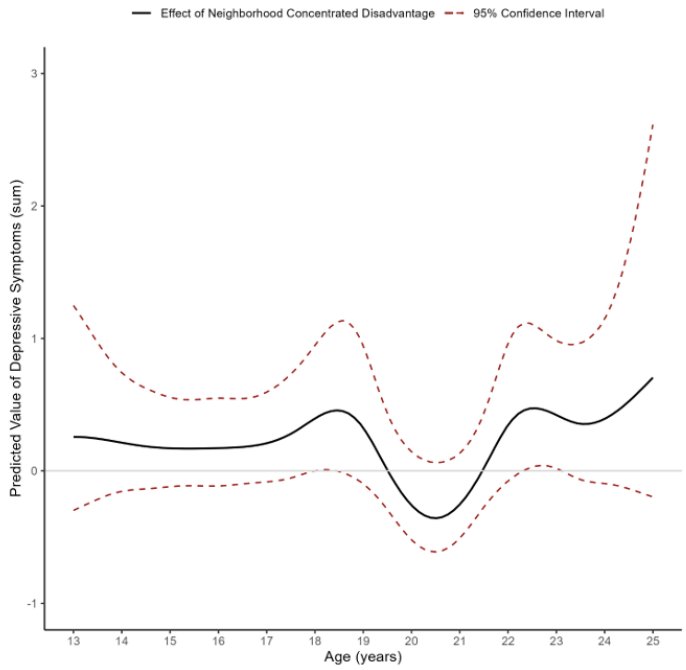
symptoms was not significant. Residing in a neighborhood with higher concentrated disadvantage was then again associated with higher depressive symptoms from age 21.36 (β = 0.50, 95% CI = 0.07, 1.10) through 25 (β = 1.84, 95% CI = 0.35, 4.99). Age 25 is also when the association between neighborhood concentrated disadvantage and depressive symptoms was strongest, although the 95% confidence intervals were relatively wide, suggesting uncertainty in the estimates at ages 24-25.

The unadjusted time-varying effect model predicting depressive symptoms from neighborhood concentrated disadvantage with seven knots had optimal fit to the data (Table 6, Model 1).

However, when covariates were introduced into the model, the association between neighborhood concentrated disadvantage and depressive symptoms was attenuated, only remaining significant at very narrow age intervals (Figure 7). The magnitude of the effect across these ages was small, whereby a 10% increase in neighborhood concentrated disadvantage was associated with an increase in depressive symptom scores ranging from 0.041 to 0.184.

Figure 7

Plot of time-varying effect model predicting depressive symptoms from neighborhood concentrated disadvantage across ages 13-25, adjusted for biological sex and caregiver sociodemographic characteristics (N = 9,956)



Specifically, greater neighborhood concentrated disadvantage was associated with greater depressive symptoms from age 18.09 (β = 0.41, 95% CI = 0.01, 0.99) until age 18.33 (β = 0.45, 95% CI = 0.01, 1.09) and from age 22.33 (β = 0.46, 95% CI = 0.004, 1.12) until age 23.06 (β = 0.41, 95% CI = 0.01, 0.97). The magnitude of the association was strongest at age 22.57 (β = 0.47, 95% CI = 0.04, 1.09). Neighborhood concentrated disadvantage was not a significant predictor of depressive symptom at any other ages in the adjusted model, as the 95% confidence intervals overlapped with zero. The observed pattern of findings across age contradicted my expectations (i.e., a significant association across age that was strongest in late adolescence).

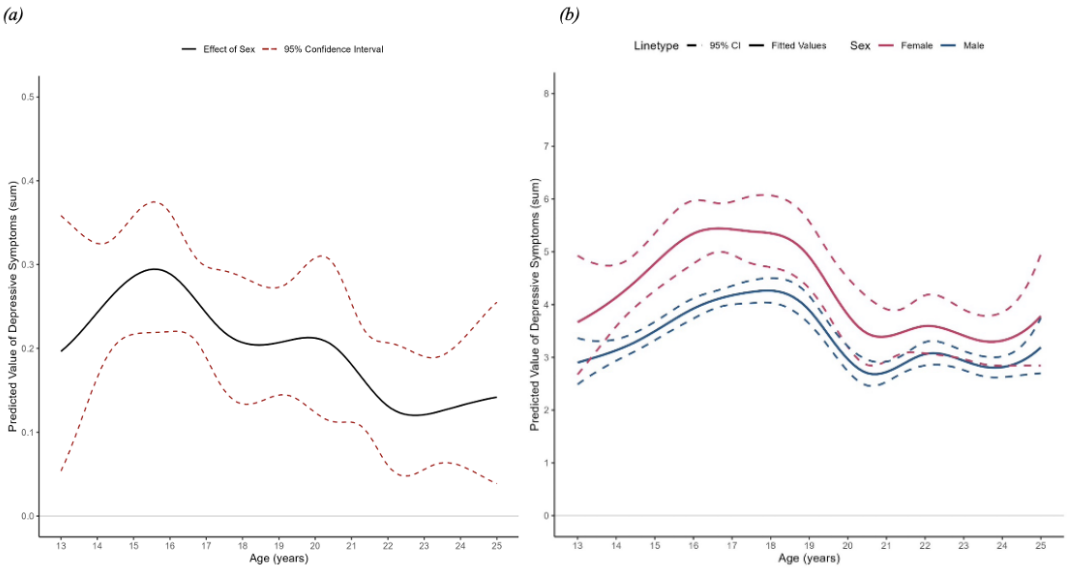
With respect to model selection for the adjusted model, I conducted two series of models with these covariates specified as time-invariant (Table 6, Model 3) and time-varying (Table 6, Model 4). For both sets of models, the model with six knots had the best fit to the data. However, when comparing model fit across the six-knot models with time-invariant and time-varying covariates, the AIC was lower for the model with time-varying covariates, but the BIC was lower for the model with covariates specified as time-invariant. Since the model fit indicators were inconclusive for selecting the best model, I visually inspected the estimates and coefficient plots of the covariates from the two competing models. The model with the covariates specified as time-varying showed that the effect of caregiver employment and receipt of public assistance did not vary across age. Additionally, the pattern of results across the two models did not change. As such, I selected the model with time-invariant covariates, as it was the more parsimonious model (i.e., Model 3).

3.3.2 Association Between Biological Sex and Depressive Symptoms (Aim 2a)

Biological sex was significantly associated with depressive symptoms across adolescence and emerging adulthood (Figure 8a). In line with my expectations, females reported higher depressive symptoms than males from ages 13-25 (Figure 8b).

Figure 8

Plot of time-varying effect model predicting depressive symptoms from biological sex across ages 13-25, unadjusted for covariates (N = 9,956). Panel A represents the overall effect of biological sex. Panel B shows the slopes of male (blue) and female (pink) depressive symptoms across age.



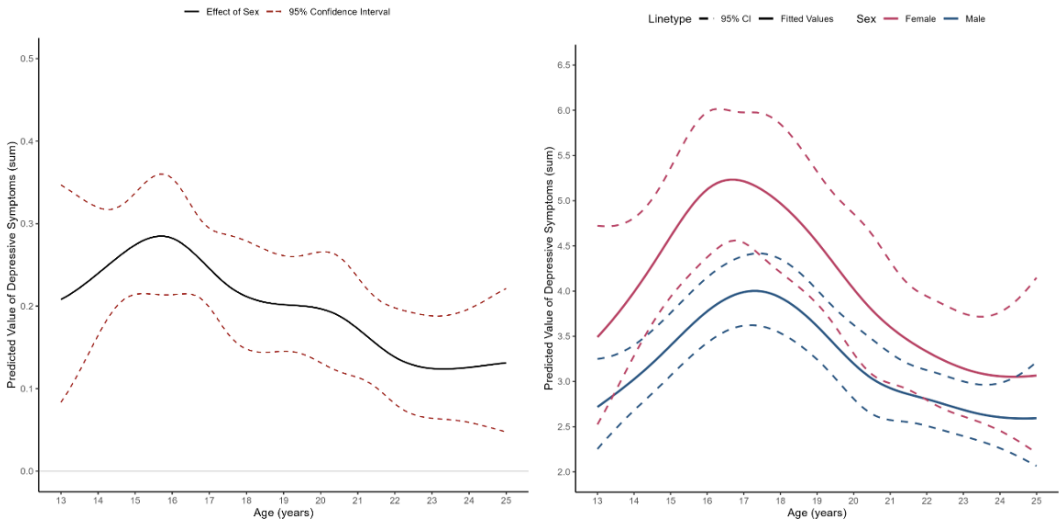
The magnitude of the association between biological sex and depressive symptoms varied with age, with the strongest difference observed at age 15.54 (overall predicted difference $\beta = 0.29$, 95% CI = 0.22, 0.37; $\beta_{Male} = 3.74$, 95% CI = 3.55, 3.93; $\beta_{Female} = 5.13$, 95% CI = 4.55, 5.78). In contrast, the association between biological sex and depressive symptoms was weakest at age 22.59 ($\beta = 0.12$, 95% CI = 0.05, 0.20). It should be noted that there were several ages at which the 95% confidence intervals for males and females overlapped, indicating uncertainty regarding the specific estimated values at those ages. Specifically, the 95% confidence intervals overlapped from age 13 ($\beta_{Male} = 2.90$, 95% CI = 2.49, 3.36; $\beta_{Female} = 3.67$, 95% CI = 2.67, 4.93) through age 13.73 ($\beta_{Male} = 3.06$, 95% CI = 2.83, 3.31; $\beta_{Female} = 3.99$, 95% CI = 3.34, 4.74). As indicated by a lower AIC and BIC, the unadjusted model predicting depressive symptoms from biological sex with seven knots had the best fit to the data (Table 6, Model 2).

Biological sex remained a significant predictor of depressive symptoms across ages 13-25 in the adjusted model (Figure 9a; Age 13: $\beta = 0.23$, 95% CI = 0.14, 0.32; Age 25: $\beta = 0.13$, 95% CI = 0.06, 0.20), with females consistently reporting higher depressive symptoms than males (Figure 9b).

Figure 9

Plot of time-varying effect model predicting depressive symptoms from biological sex across ages 13-25, adjusted for neighborhood concentrated disadvantage and caregiver sociodemographic characteristics (N = 9,956). Panel A represents the overall adjusted effect of sex on depressive symptoms. Panel B shows the slopes of male (blue) and female (pink) depressive symptoms across age.

(a) (b)



The age at which the association was strongest was similar to the unadjusted model (age 15.79; $\beta = 0.27$, 95% CI = 0.22, 0.32; $\beta_{Male} = 2.72$, 95% CI = 2.40, 3.07; $\beta_{Female} = 3.74$, 95% CI = 3.16, 4.39). This indicates that depressive symptoms were 37.50% higher among 16-year-old females than males. The weakest difference in depressive symptoms by sex was at age 24.03 ($\beta = 0.13$, 95% = 0.06, 0.19), with males reporting an average depressive symptom score of 2.19 (95% CI = 1.82, 2.62) and females reporting an average score of 2.60 (95% = 2.00, 3.32); depressive symptoms were 18.72% higher

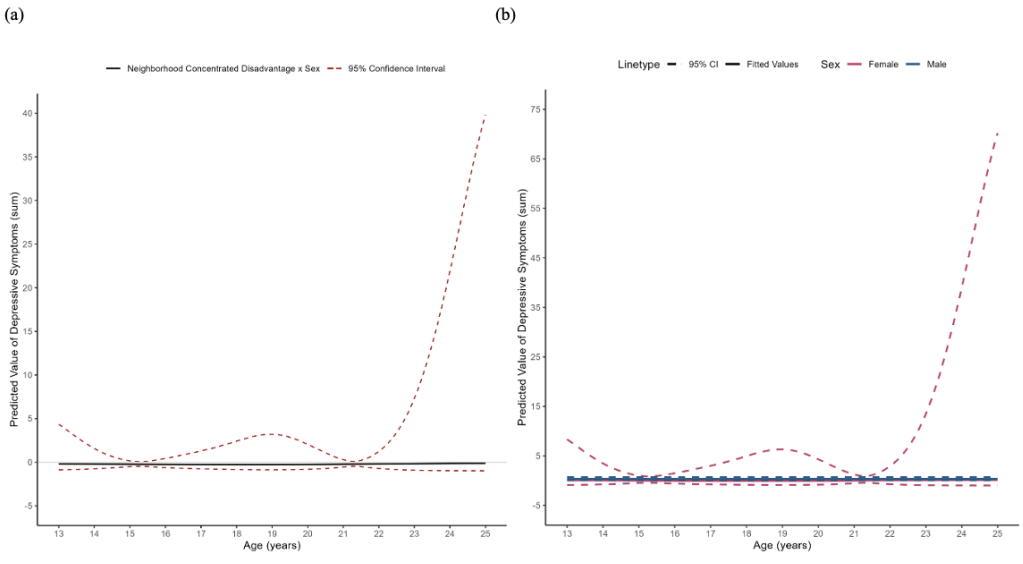
among females than males, even when the predicted difference was weakest. Model selection details are reported in the section 3.3.1 above (Table 6, Model 3).

3.3.3 Associations Between Neighborhood Concentrated Disadvantage and Depressive Symptoms by Biological Sex (Aim 2b)

The association between neighborhood concentrated disadvantage and depressive symptoms did not significantly vary by biological sex across age (Figures 10a and 10b), contradicting my expectation. Because the estimated interaction coefficient was both flat and near zero across ages 18-25, I determined there was likely no sex difference in the association between neighborhood concentrated disadvantage and depressive symptoms at any age.

Figure 10

Plot of interaction between neighborhood concentrated disadvantage and biological sex on depressive symptoms across ages 13-25 (N = 9,956). Panel A shows the predicted interaction effect. Panel B shows the slopes of the effect of neighborhood concentrated disadvantage on depressive symptoms among males (blue) and females (pink).



Initially, I varied the number of knots from 1-10 for the model predicting depressive symptoms from the two-way interaction between neighborhood concentrated

disadvantage and biological sex. The model with seven knots showed optimal fit to the data (Table 6, Model 5). However, a cursory examination of the plotted interaction showed no variability (Figure 10a). As such, I specified additional models with up to 35 knots. The AIC was lower in the model with 31 knots than the model with seven knots, however, there was still no variability in the estimated interaction coefficient. Accordingly, I interpreted the findings from the model specifying seven knots, as it represented a better balance between model fit and parsimony.

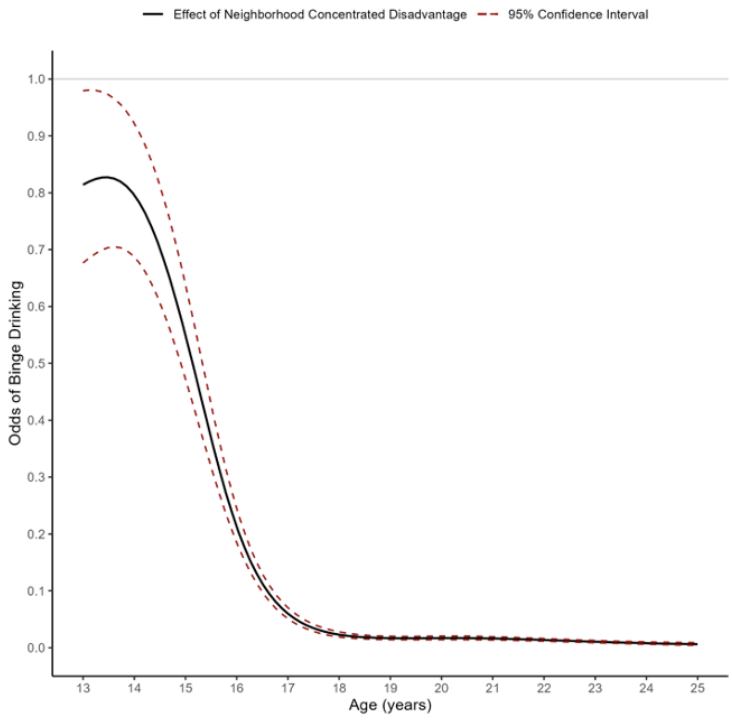
3.4 Age-Varying Associations with Binge Drinking (Aim 3)

3.4.1 Association Between Neighborhood Concentrated Disadvantage and Binge Drinking (Aim 3a)

Surprisingly, greater neighborhood concentrated disadvantage was associated with a *lower* likelihood of binge drinking across ages 13-25 in the model unadjusted for covariates (Figure 11).

Figure 11

Plot of time-varying effect model predicting the likelihood of binge drinking from neighborhood concentrated disadvantage across ages 13-25, unadjusted for covariates (N = 9,956)



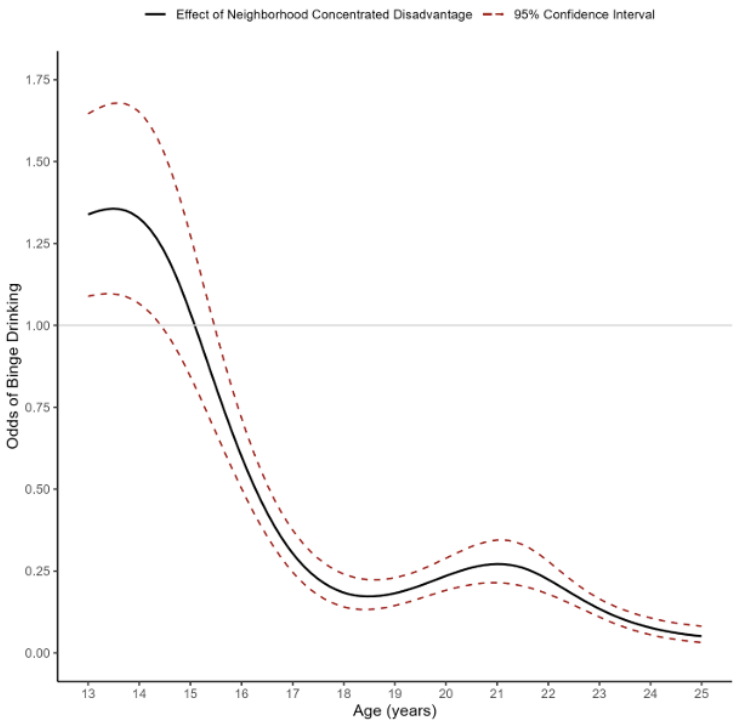
Although residing in a neighborhood with higher concentrated disadvantage was associated with a lower likelihood of binge drinking across all observed ages, the likelihood of binge drinking was highest at age 13.48 (OR = 0.83, 95% CI = 0.70, 0.97) relative to other observed ages. The unadjusted model predicting likelihood of binge drinking from neighborhood concentrated disadvantage with four knots had the best fit to the data (Table 7, Model 1).

More in line with my expectations, the adjusted model showed that residing in a more disadvantaged neighborhood was associated with a higher likelihood of binge drinking. However, this was only observed in early adolescence (Figure 12),

contradicting my prediction that the association would be significant across ages, and that the association would be strongest in emerging adulthood.

Figure 12

Plot of time-varying effect model predicting likelihood of binge drinking from neighborhood concentrated disadvantage across ages 13-25, adjusted for biological sex and caregiver sociodemographic characteristics (N = 9,956)



From age 13 (OR = 1.47, 95% CI = 1.18, 1.83) through age 14.33 (OR = 1.25, 95% CI = 1.01, 1.56), residing in a neighborhood with greater concentrated disadvantage was associated with a higher likelihood of binge drinking in the past 12 months, with the greatest likelihood observed around age 13. During this age span, a one-unit increase in neighborhood disadvantage increased the odds of binge drinking between 25-47%. At age 15.54 (OR = 0.77, 95% CI = 0.64, 0.93) through age 25 (OR = 0.05, 95% CI = 0.03,

0.08), greater neighborhood concentrated disadvantage was associated with a lower likelihood of binge drinking.

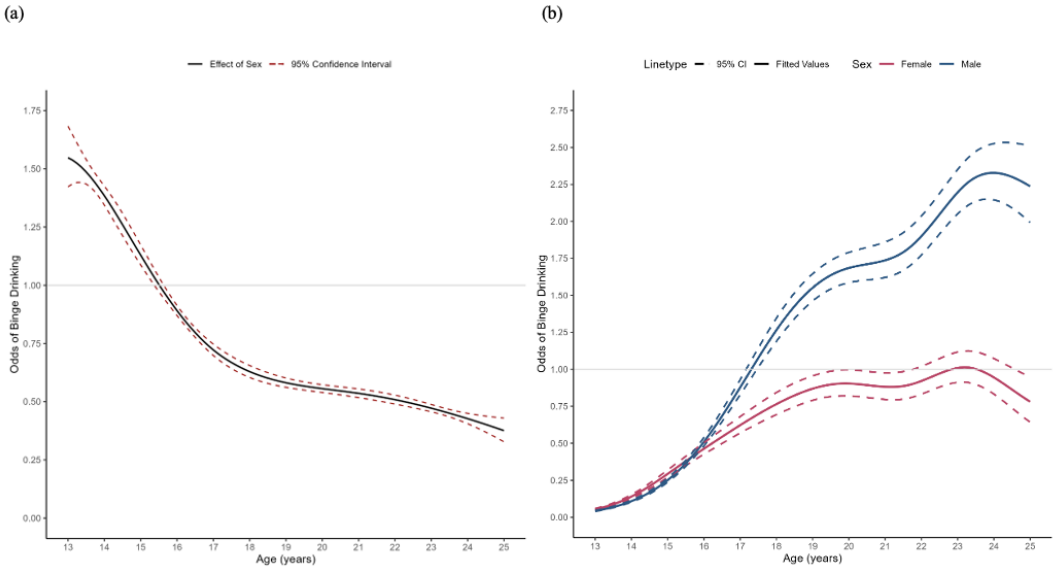
I again examined model fit with covariates specified as time-invariant (Table 7, Model 3) and time-varying (Table 7, Model 4). For both the series of models with covariates specified as time-invariant and time-variant, the model with six knots had optimal fit to the data. However, the AIC and BIC were lower in the model with time-varying covariates than in the model with time-invariant covariates. Moreover, a visual inspection of the estimates and coefficient plots of the time-invariant and time-varying models with six knots showed that one or more of the covariates had a time-varying effect. Accordingly, I selected the model with time-varying covariates.

3.4.2 Association between Biological Sex and Binge Drinking (Aim 3a)

Biological sex was a significant predictor of binge drinking in the past 12 months across most of adolescence and emerging adulthood (Figure 13a). In early adolescence, females were more likely to report binge drinking than males, differing from my expectations (Figure 13b). Males were more likely than females to report binge drinking in mid-to-late adolescence and emerging adulthood.

Figure 13

Plot of time-varying effect model predicting likelihood of binge drinking from biological sex across ages 13-25, unadjusted for covariates (N = 9,956). Panel A represents the overall effect of biological sex. Panel B shows the slopes of the likelihood of binge drinking among males (blue) and females (pink) across age.

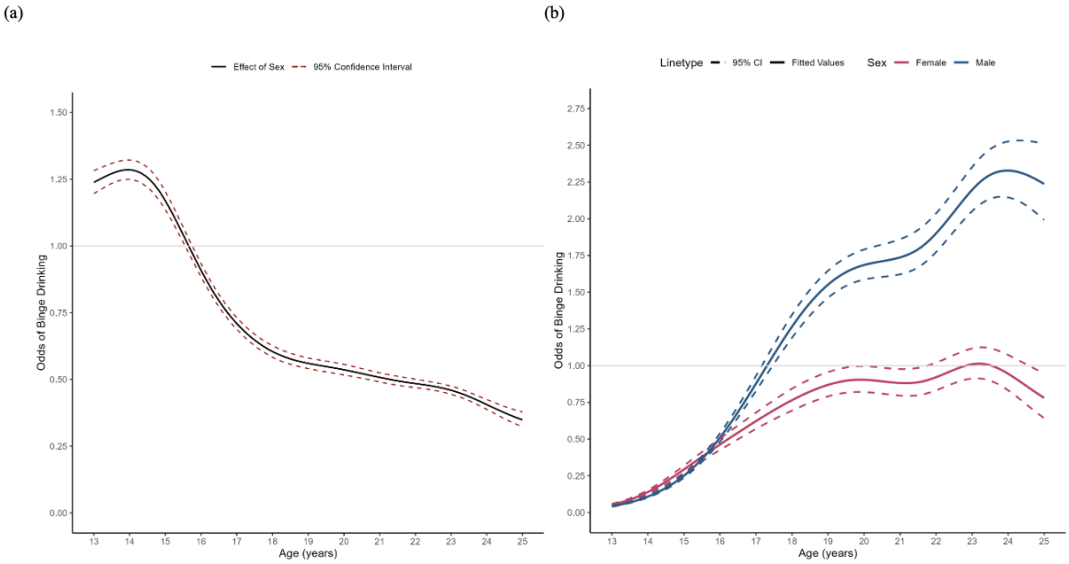


Specifically, from age 13 (OR_{Male} = 0.03, 95% CI = 0.03, 0.03; OR_{Female} = 0.04, 95% CI = 0.03, 0.05) until age 15.42 (OR_{Male} = 0.29, 95% CI = 0.28, 0.30; OR_{Female} = 0.30, 95% CI = 0.28, 0.32) the likelihood of binge drinking was higher among females than males. Beginning at age 15.79 (OR_{Male} = 0.39, 95% CI = 0.37, 0.40; OR_{Female} = 0.37, 95% CI = 0.35, 0.39) through age 25 (OR_{Male} = 3,66, 95% CI = 3.23, 4.14; OR_{Female} = 1.29, 95% CI = 1.05, 1.60), males were more likely to engage in binge drinking than females. The magnitude of the association between biological sex and likelihood of binge drinking was largest at age 25 (OR = 0.35, 95% CI = 0.32, 0.39; OR_{Male} = 3.66, 95% CI = 3.23, 4.14; OR_{Female} = 1.29, 95% CI = 1.05, 1.60), such that females were 65% less likely than males to report binge drinking. The model of the unadjusted associated between biological sex and binge drinking with two knots had the best fit to the data (Table 7, Model 2).

Biological sex remained a significant predictor of binge drinking across most ages of interest in the adjusted model (Figure 14a). Aligning with the unadjusted model, females were more likely to report binge drinking than males in early adolescence, but males were more likely than females to report binge drinking in mid-to-late adolescence and emerging adulthood (Figure 14b).

Figure 14

Plot of time-varying effect model predicting likelihood of binge drinking from biological sex across ages 13-25, adjusted for neighborhood concentrated disadvantage and caregiver sociodemographic characteristics (N = 9,956). Panel A represents the overall adjusted effect of sex on likelihood of binge drinking. Panel B shows the slopes of the likelihood of binge drinking among males (blue) and females (pink) across age.



Model selection for the adjusted model is presented above in Section 3.4.2.

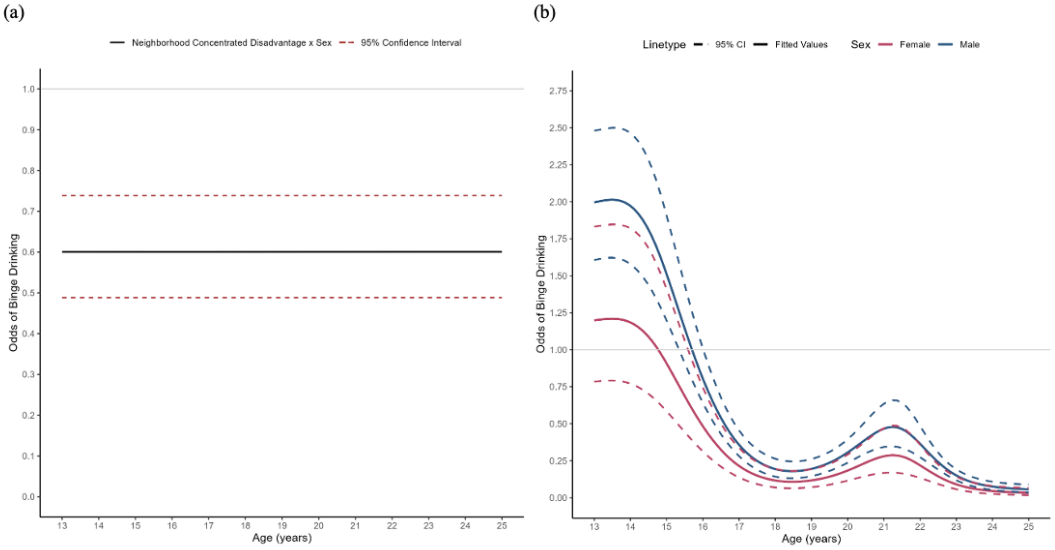
3.4.3 Associations Between Neighborhood Concentrated Disadvantage and Binge Drinking by Biological Sex (Aim 3b)

The two-way interaction between neighborhood concentrated disadvantage and biological sex was not significant. Although the 95% confidence interval did not overlap one, the slope of the interaction between neighborhood concentrated disadvantage and biological sex was flat (Figure 15a), indicating that the sex difference in the association

between neighborhood concentrated disadvantage and binge drinking did not vary by age. This was verified via a plot of the slope of the association between neighborhood concentrated disadvantage and binge drinking separately for males and females, which showed overlapping 95% confidence intervals across ages 13-25 (Figure 15b). However, the predicted estimates and 95% confidence intervals did not overlap one, suggesting that there is indeed a sex difference in the association between neighborhood concentrated disadvantage and binge drinking that is constant across age.

Figure 15

Plot of interaction between neighborhood concentrated disadvantage and biological sex on likelihood of binge drinking across ages 13-25 (N = 9,956). Panel A shows the predicted interaction effect. Panel B shows the slopes of the effect of neighborhood concentrated disadvantage on likelihood of binge drinking among males (blue) and females (pink)



For the model predicting binge drinking from the two-way interaction between neighborhood concentrated disadvantage and biological sex, six knots provided optimal fit to the data (Table 7, Model 5).

Chapter 4: Discussion

4.1 Summary of Findings

There were several notable findings in this dissertation. I summarize these core findings and their alignment with my expectations, based on previous research, below.

4.1.1 Variation in Neighborhood Concentrated Disadvantage, Depressive Symptoms, and Binge Drinking (Aim 1)

My investigation into change in neighborhood concentrated disadvantage, depressive symptoms, and binge drinking (Aims 1a-1c) yielded several key findings; discussed in detail, by construct, below.

4.1.1.1 Neighborhood Concentrated Disadvantage (Aim 1a)

Neighborhood concentrated disadvantage increased slightly across age. This increase across age, which aligned with my expectations, is likely reflective of rising concentrated poverty over historical time (Iceland & Hernandez, 2017). After marked reductions in concentrated poverty throughout the 1980s, the 1990s and early 2000s were characterized by significant increases in concentrated poverty (Iceland & Hernandez, 2017). The approximately linear growth in neighborhood concentrated disadvantage across age observed in this dissertation is well-aligned with this evidence.

The relative stability in neighborhood concentrated disadvantage across 13-18-years-old observed in the present study may be attributable to the specific contextual data sources linked to Add Health participant data. Both Wave I and II of the Add Health study were linked to the 1990 census, when participants were aged 13-19 and 14-20, respectively, whereas Wave III was linked to the 2000 census, when participants were

aged 19-25. Because both timepoints were linked to the same data source (i.e., 1990 decennial census), individual values of neighborhood concentrated disadvantage only varied for those that moved to a different census block group from Wave I to Wave II, which only applied to approximately 5% of the sample in this dissertation. When the data source changed from the 1990 decennial census to the 2000 decennial census, which spanned ages 19-25, neighborhood concentrated disadvantage increased relatively steadily.

4.1.1.2 Depressive Symptoms (Aim 1b)

Depressive symptoms increased across adolescence, with participants reporting the highest level of symptoms in late adolescence, as expected. Depressive symptoms decreased sharply in early emerging adulthood, diverging from my prediction that depressive symptoms would not begin to decline until approximately 21-years-old. In general, this pattern of change across age aligns with national estimates (Substance Use and Mental Health Services Administration, 2022). Indeed, the United States Substance Abuse and Mental Health Services Administration's (SAMHSA) annual National Survey on Drug Use and Health (NSDUH) shows a strikingly similar pattern of incidence of a major depressive episode in the past 12 months across ages 13-25 to that of the current study (Substance Use and Mental Health Services Administration, 2022). According to the most recent NSDUH (Substance Use and Mental Health Services Administration, 2022), the percentage of youth reporting a major depressive episode increased across adolescence, with the highest incidence reported among 16-17-year-olds.

4.1.1.3 Binge Drinking (Aim 1c)

The likelihood of engaging in binge drinking changed quadratically across adolescence and emerging adulthood. The increase in binge drinking engagement across adolescence and emerging adulthood aligned with my expectation. However, peak engagement in binge drinking was slightly later than expected (i.e., at ~23 rather than 21). The decline in binge drinking from ages 23-25 was also not expected. However, this pattern of binge drinking across age generally aligned with national estimates (Substance Use and Mental Health Services Administration, 2022). According to the NSDUH, binge alcohol use in the past 12 months was quite low among 12-13-year-olds, rising steadily through age 25. The percentage of emerging adults that reported binge drinking in the NSDUH reflects the peak in binge drinking identified by this study but does not capture the decline toward the end of emerging adulthood (Substance Use and Mental Health Services Administration, 2022). More granular estimates of binge drinking across 21-25 were not available via the NSDUH. However, the adult survey shows that binge drinking is lower among adults aged 26 and beyond, aligning with the decline from ages 23-25 observed in the present study.

4.1.2 Age-Varying Associations with Depressive Symptoms (Aim 2)

4.1.2.1 Association between Neighborhood Concentrated Disadvantage and Depressive Symptoms Across Ages 13-25 (Aim 2a)

Greater neighborhood concentrated disadvantage was associated with higher depressive symptoms across most of adolescence and emerging adulthood in unadjusted analyses. However, in analyses controlled for sociodemographic characteristics, this

significant association was only observed at very specific ages (i.e., late adolescence and mid-emerging adulthood). This change in findings between the unadjusted and adjusted models suggests that the significant association between neighborhood concentrated disadvantage and depressive symptoms may be driven by household-level SES in adolescence, but not emerging adulthood. Although there is limited comparable evidence spanning adolescence and emerging adulthood, this aligns, in part, with the study by Estrada-Martinez and colleagues (2019), which showed that the magnitude of the association between neighborhood conditions and depressive symptoms increases from adolescence through emerging adulthood.

However, some findings of the present study diverged from previous research (Barr, 2018; Estrada-Martinez et al., 2019). In contrast to the significant association between baseline neighborhood satisfaction and depressive symptoms across the entirety of emerging adulthood observed by Estrada-Martinez and colleagues (2019), neighborhood conditions were unrelated to depressive symptoms across adolescence and from approximately ages 18-22-years-old in the present study. One possible explanation for this difference arises from methodological differences in the assessment of neighborhood conditions. Previous studies have assessed associations between adolescent neighborhood conditions and trajectories of the outcome across age (Barr, 2018; Estrada-Martinez et al., 2019). Both studies (Barr, 2018; Estrada-Martinez et al., 2019) and the present dissertation investigated developmental timing of neighborhood effects. However, by including only baseline neighborhood conditions, the previous studies have, in effect, examined whether adolescent neighborhood conditions have a persistent impact

on depressive symptoms into adulthood. In contrast, I leveraged repeated measures of both neighborhood socioeconomic conditions and depressive symptoms to examine age-varying associations between these constructs. Multilevel modeling, as was used in previous research (Barr, 2018; Estrada-Martinez et al., 2019), differs from time-varying effect models in that the association across time is assumed to have a specific functional form in multilevel models, rather than varying dynamically as in TVEM (Vasilenko et al., 2014). As such, I was able to more directly estimate how neighborhood conditions at specific ages in adolescence and emerging adulthood were associated with depressive symptoms at each corresponding age.

Across both the unadjusted and adjusted models, neighborhood concentrated disadvantage was not significantly associated with depressive symptoms between ages 18-22. This specific interval aligns with the ages at which many emerging adults attend college (National Center for Education Statistics, 2024), during which time one's neighborhood context may change substantially. Indeed, in 2000, when Add Health participants were at least 19 years old, approximately 75% of students no longer lived at home (Horn et al., 2002). Given that most Add Health participants transitioned from high school into college, this contextual change may explain the nonsignificant association observed in the present study among 18-22-year-olds.

In sum, the transition to adulthood may represent a period during which neighborhood socioeconomic conditions have a more pronounced impact on depressive symptoms, relative to adolescence.

4.1.2.2 Association between Biological Sex and Depressive Symptoms Across Ages 13-25 (Aim 2a)

In line with my expectations, there was a significant association between biological sex and depressive symptoms across ages 13-25, such that females had significantly higher depressive symptoms than males. The magnitude of this difference was strongest around age 16. These findings were consistent across the unadjusted model and the model that adjusted for sociodemographic characteristics and were in concordance with national estimates (Substance Use and Mental Health Services Administration, 2022). Indeed, data from the National Survey on Drug Use and Health shows that a larger percentage of females report a past year major depressive episode than males at each of the reported age intervals (i.e., ages 12-17, 18-20, 21-25; (Substance Use and Mental Health Services Administration, 2022).

4.1.2.3 Age-Varying Interaction Between Neighborhood Concentrated Disadvantage and Biological Sex on Depressive Symptoms (Aim 2b)

Contrary to expectations, the age-varying effect of biological sex on the association between neighborhood concentrated disadvantage and depressive symptoms was not significant. This aligns with other Add Health research, which shows that the trajectory of the associations between three measures of neighborhood conditions (i.e., advantage, disadvantage, and disorder) and depressive symptoms does not differ by sex (Barr, 2018). In general, one review showed that evidence of moderation of the association between neighborhood conditions and depressive symptoms by sex/gender findings is mixed in terms of significance and direction of effects (Mair et al., 2008). This

discrepancy across studies may be partially due to differences in the specific environmental features of the neighborhood investigated, which varied across studies included in the review (e.g., violence, walkability, socioeconomic conditions; Mair et al., 2008). For example, two studies found that the association between neighborhood social factors (i.e., perceptions of safety/fear) and depression may be stronger among males than females (e.g., Assari et al., 2015; Assari & Caldwell, 2017). Together, these findings suggest that the impact of some environmental exposures on depressive symptoms may differ by sex, but not others. More research is needed to clarify what aspects of the neighborhood environment may have a more pronounced effect on depressive symptoms by sex.

4.1.3 Age-Varying Associations with Binge Drinking (Aim 3)

4.1.3.1 Association between Neighborhood Concentrated Disadvantage and Binge Drinking Across Ages 13-25 (Aim 3a)

Interestingly, greater neighborhood concentrated disadvantage was associated with a *lower* likelihood of engaging in binge drinking in the past year across adolescence and emerging adulthood in unadjusted analyses. However, in the model adjusted for sociodemographic characteristics, residing in a neighborhood with a higher level of disadvantage was associated with an *increased* likelihood of binge drinking in early adolescence (i.e., from approximately 13 to 14.5 years of age). Beyond early adolescence, residing in a more disadvantaged neighborhood was associated with a *lower* likelihood of binge drinking, as in the unadjusted model. The age(s) at which neighborhood

disadvantage was associated with a greater likelihood of binge drinking and the subsequent change in the direction of effects did not align with my expectations.

I identified only one study that explicitly examined the role of developmental timing in the association between neighborhood conditions and substance use behaviors (Barr, 2018b). Barr (2018b) found that greater adolescent neighborhood disadvantage was associated with higher initial monthly alcohol consumption, but not drinking status nor binge drinking. Adolescent neighborhood disadvantage was also associated with drinking behaviors in another study, although this association was observed across adolescence and emerging adulthood (Reboussin et al., 2019). Together with the results of the current study, research suggests that neighborhood disadvantage is a meaningful predictor of drinking behaviors (e.g., binge drinking, monthly consumption), but only in early adolescence.

Less clear is the association between neighborhood disadvantage and binge drinking in emerging adulthood. In this dissertation, greater neighborhood concentrated disadvantage was associated with a *lower* likelihood of engaging in binge drinking from ages 15 to 25. Yet, previous research suggests that greater disadvantage is associated with drinking behaviors across emerging adulthood, as Barr (2018b) found that greater disadvantage was associated with a slower decline in monthly consumption and heavy drinking into adulthood. Moreover, Reboussin and colleagues (2019) found that neighborhood disadvantage was associated with a higher likelihood of drinking through emerging adulthood. In both studies, however, adolescent disadvantage was associated with trajectories of alcohol use, demonstrating the persistent impact of adolescent

exposure. Instead, this study demonstrated the concurrent association between harmful neighborhood conditions and drinking behaviors at specific ages throughout adolescence and emerging adulthood. This methodological distinction may contribute to the discrepant findings across studies.

It is important to note that although greater disadvantage was associated with a *lower* likelihood of engaging in binge drinking, the findings of this study do not suggest residing in a disadvantaged neighborhood is beneficial. Despite a lower likelihood of engaging in binge drinking, residence in disadvantaged neighborhoods is linked to more alcohol related problems (e.g., fights while under the influence of alcohol, legal consequences, poor health; (Jones-Webb & Karriker-Jaffe, 2013).

4.1.3.2 Association between Biological Sex and Binge Drinking Across Ages 13-25 (Aim 3a)

Across nearly the entirety of adolescence and emerging adulthood, biological sex was significantly associated with the likelihood of binge drinking in both the unadjusted and adjusted models. In early adolescence (i.e., approximately 13-15.5), females were more likely than males to engage in binge drinking behaviors. However, from late adolescence through emerging adulthood, males were more likely to report binge drinking than females and the magnitude of this difference increased across age. Generally, these findings align with national estimates of binge drinking by gender and across age (Substance Use and Mental Health Services Administration, 2022). For example, the percentage of females that reported binge drinking from 12-17 was slightly higher than the percentage of males. Although the percentage of males and females

reporting binge drinking was nearly equal in the NSDUH from ages 18-25, diverging from the findings of this dissertation, males aged 21+ were more likely to report binge drinking than females (Substance Use and Mental Health Services Administration, 2022). It is important to note that binge drinking in Add Health was conceptualized as five or more drinks in a single occasion but in the NSDUH, binge drinking is considered five or more drinks for males and only four or more drinks for females. The discrepancy in the operationalization of binge drinking between Add Health and the NSDUH may partially account for the divergent findings. More granular estimates, such as those presented above for binge drinking by age across sex, were not available, so the exact ages at which females were more likely than males to engage in binge drinking, and vice versa, using a nationally representative dataset could not be directly compared to the findings from this dissertation. However, Barr (2018b) showed that monthly alcohol consumption was initially higher among females than males in early adolescence but that this pattern switched across adolescence and into emerging adulthood, aligning with my findings.

4.1.3.3 Age-Varying Interaction Between Neighborhood Concentrated Disadvantage and Biological Sex on Binge Drinking (Aim 3b)

The moderating role of biological sex on the association between neighborhood concentrated disadvantage and binge drinking did not differ by age. This was unexpected, as the gap in binge drinking behaviors by sex increases with age (Substance Use and Mental Health Services Administration, 2022). However, although the slope of the interaction coefficient on binge drinking across age was flat, the 95% confidence intervals did not overlap one. This suggests that the association between neighborhood

concentrated disadvantage and binge drinking *does* differ by sex, but this sex difference is consistent across age. Although the non-age-varying interaction between neighborhood concentrated disadvantage and binge drinking is outside the scope of this dissertation, the interaction coefficient suggests that the association between neighborhood disadvantage and binge drinking is stronger among males than females. The finding aligns with previous research, which suggests neighborhood conditions are only related to binge drinking among men (Matheson et al., 2012).

4.2 Practical Implications

Beyond highlighting the deleterious effect of neighborhood concentrated disadvantage on health more broadly, the present study provides key insights into *when* those residing in disadvantaged neighborhoods may be most at risk for depressive symptoms and substance use. More specifically, these findings reveal that neighborhood concentrated disadvantage is only associated with greater depressive symptoms during the transition to adulthood. In contrast, neighborhood concentrated disadvantage was associated with early binge drinking initiation. Knowing when adolescents and emerging adults may be most at risk for the negative consequences of neighborhood concentrated disadvantage is key for early identification and subsequent intervention. In a world with limited resources, the ability to more directly target those imminently at risk for poor health outcomes is essential.

Additionally, a clearer picture of when in development neighborhood concentrated poverty may confer a risk to health is useful for the tailoring of and implementation of interventions. For instance, research suggests that the positive

association between neighborhood disadvantage and alcohol-related problems across development may operate through mechanisms of lower school achievement and educational attainment (Karriker-Jaffe et al., 2017). The relative strength of the association between neighborhood concentrated disadvantage and binge drinking observed in the present study suggests that targeting youth achievement in early adolescence relative to mid-to-late adolescence and emerging adulthood may attenuate this effect.

Although neighborhood concentrated disadvantage had a relatively small impact on depressive symptoms and binge drinking at very specific ages, these findings reiterate the importance of policies and programs aimed at reducing concentrated poverty. Such place-based interventions that focus on neighborhood revitalization may reduce concentrated poverty and related factors (e.g., crime, disorder) and, in turn, improve health outcomes.

4.3 Directions for Future Research

As highlighted in the introduction, longitudinal, developmentally informed research examining associations between neighborhood conditions and health is limited (Arcaya et al., 2016). This dissertation represents a significant step toward improving our understanding of the role of developmental timing of exposure to neighborhood conditions in shaping health but additional research to this end is warranted. However, there are several significant methodological challenges to longitudinal studies of neighborhood effects (Oakes, 2004; Oakes et al., 2015) that warrant careful consideration and may explain, in part, the dearth of evidence in this area.

4.3.1.1 Challenges to Longitudinal Neighborhood Effects Research

One significant challenge faced by those conducting neighborhoods and health research is the availability of data suitable for the longitudinal analysis of neighborhood effects, either via primary data collection efforts or secondary analyses. Beyond the need for multiple timepoints of individual-level data, robust analyses of longitudinal neighborhood effects require one to account for the ways in which neighborhood exposures may change over time (Diez Roux, 2018b). In addition to change due to relocation, researchers relying on administrative data to measure neighborhoods must grapple with changes in the operationalization of neighborhood boundaries over time. Specifically, census geographic boundaries, and their associated data, often differ from one census year to another (Gregory & Ell, 2005) and are thus not comparable across time. To address these complexities, novel approaches to normalize geographic boundaries across time have emerged. Despite the advent of databases of normalized administrative data (e.g., Neighborhood Change Database; Geolytics, 2021), linkage between these data and person-level data, particularly among large, nationally representative samples, remains limited.

Further still, neighborhood conditions assessed via census data, which represents nearly 70% of recent studies (Gresham & Karatekin, *under revision*), may not reflect neighborhood conditions at the timepoint of interest. For example, if an individual participated in a study and provided their current address in 1994, as was the case for most Wave I Add Health participants, data from the 1990 decennial census may be used to approximate conditions for said timepoint. In longitudinal studies focused on

developmental processes, this is especially problematic. With respect to Add Health, a study rich with data across five timepoints, the 1990 decennial census was linked to both Waves I and II (Harris et al., 2019). Accordingly, neighborhood conditions in 1989-90 were intended to be reflective of neighborhood conditions roughly half a decade or more later in 1994-95 and 1996. In addition to the possible misestimation of neighborhood conditions at Wave I and Wave II, the only youth that had variation in neighborhood conditions across the two timepoints were those that relocated to a different census block group or tract. This effectively inhibits insight into how *change* in neighborhood conditions is associated with *change* in an outcome.

To summarize, beyond the need for longitudinal research on neighborhoods and health more broadly, analyses of spatial data that address 1) changing geographic boundaries and 2) alignment between the study year and administratively linked data.

4.3.1.2 Addressing Challenges to Longitudinal Neighborhood Effects Research: Add Health Ancillary Study

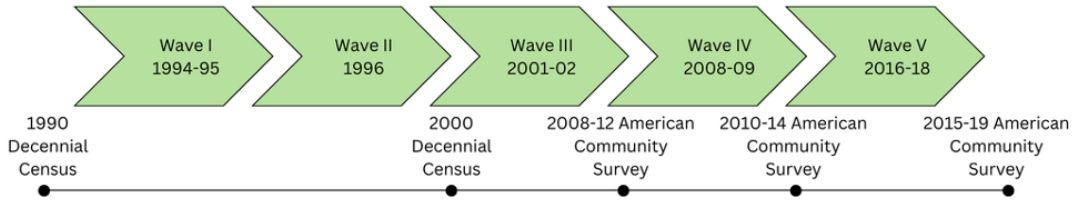
As part of this dissertation, I proposed to examine how the association between neighborhood concentrated disadvantage and health unfolds across adolescence and emerging adulthood in addition to the aims presented herein. However, the changing geographic boundaries across data sources and the misalignment between the data collection year and the census data year hindered my ability to perform a robust analysis. To address these challenges, I constructed a dataset of neighborhood concentrated disadvantage for linkage to Add Health that is appropriate for longitudinal analyses in

collaboration with Dr. Theresa Osypuk and the University of North Carolina's Carolina Population Center.

The raw data were obtained from the Integrated Public Use Microdata Series National Historical Geographic Information System (IPUMS NHGIS; Manson et al., 2024). I then standardized the data to 2010 census block group boundaries by multiplying each variable by the appropriate population weights. Next, conducted linear interpolation to calculate values for intercensal years that align with the timing of Add Health data collection, using two data source points that are most proximal to the data collection year (Figure 16).

Figure 16

Data sources used for geographic standardization and linear interpolation of neighborhood concentrated disadvantage variables at each of the five Add Health timepoints.



Note. Data from the 2008-12, 2010-14, and 2015-19 American Community Surveys were not geographically standardized, as these data already relied on 2010 census boundaries.

Finally, I conducted a principal component analysis of the five indicator variables for neighborhood concentrated disadvantage at each wave. The weights extracted from the principal component analyses were used to create a weighted composite reflecting neighborhood concentrated disadvantage, with higher scores indicating greater disadvantage. These data were linked to all participants with a residential address at any

of the five Add Health timepoints. These data will be disseminated for the benefit of all Add Health users.

4.4 Strengths and Limitations

This dissertation had several strengths. First and foremost, the study conducted as part of this dissertation leveraged innovative methods to provide key insights into *when* in development adolescents and emerging adults may be most strongly impacted by residing in a disadvantaged neighborhood.

The use of the National Study of Adolescent to Adult Health (Add Health) to examine the aims of this dissertation represents another strength, as nearly 10,000 participants with data at three timepoints were eligible for inclusion. Relatedly, Add Health longitudinal sampling weights were applied to the primary analyses, approximating population-level (i.e., U.S. adolescents and emerging adults) effects. The initial sampling strategy of Add Health also permitted an examination of associations between neighborhood concentrated disadvantage and depressive symptoms and binge drinking across a 12-year continuous span of ages, as a wide interval of ages were recruited at baseline (i.e., students in grades 8-12). This provided excellent coverage of age across adolescence and emerging adulthood (i.e., ages 13-25), two important developmental periods of increased risk of the onset of mental health conditions and problematic substance use behaviors (Jackson & Goossens, 2020; Romer, 2010). This dissertation study was further strengthened by the application of time-varying effect models to examine the question(s) of interest.

The application of time-varying effect models to longitudinal, behavioral data to examine age-varying effects is relatively recent (Lanza & Linden-Carmichael, 2021) and has not yet been applied to neighborhoods and health research. TVEM provides an advantage over other methodological approaches to investigate the developmental timing of neighborhood effects on health, as it permits a more granular investigation of the variation in the strength and direction of associations across development. Specifically, by yielding estimates of the average association between the predictor and outcome at each age observed in the data, TVEM extends insight into developmental timing to within developmental periods of interest, whereas other approaches have provided comparisons between different developmental stages (e.g., Anderson et al., 2019). Moreover, TVEM is a nonparametric approach that easily accommodates non-linearity and does not assume non-independence of observations.

Despite these strengths, the findings from this dissertation should be interpreted in the context of several limitations. One key limitation of this dissertation is that it represents a secondary analysis of previously collected data and as such, the measures used in this study were, in part, limited to those available from Add Health. With respect to covariates, there are likely factors beyond family socioeconomic conditions and demographic characteristics that impact the association between neighborhood concentrated disadvantage and depressive symptoms and binge drinking. For example, controlling for other adverse childhood experiences may have influenced the observed pattern of results. However, a broader adversity measure was not available in Add Health (Harris et al., 2019). Of note, several adversities were assessed at Wave III, when

participants were emerging adults (e.g., “were you ever in an adoptive or foster placement in which you experienced neglect, or physical or sexual abuse?”) but these or similar adversities were not assessed at baseline, impeding their inclusion in the analyses.

Expanded definitions of adverse childhood experiences questionnaires often include neighborhood experiences (e.g., Karatekin & Hill, 2019) in addition maltreatment and household dysfunction (Felitti et al., 1998). Of debate within the adversity literature is the best way to conceptualize and measure various measures of adversity, including the value of so-called “lumping” (i.e., combining adversities across domains and contexts to create a single summary score) and “splitting” (i.e., examining domain-specific adversities) approaches. Advocates for adversity-lumping highlight the co-occurrence of many adversities, highlighting the difficult of truly estimating the effect of a single adverse experience or domain of adversity. In contrast, those in support of splitting adverse childhood experiences draw attention toward the potential unique mechanisms that may explain associations between specific adversities and poor outcomes (Smith & Pollak, 2021). While the benefits and drawbacks of lumping versus splitting adversities is beyond the scope of this dissertation, controlling for other adversities in the analyses would have allowed for a closer approximation of the unique effect of neighborhood concentrated disadvantage on depressive symptoms and binge drinking.

Additionally, the measurement of key constructs of interest varied across time in Add Health, likely in the interest of reducing participant burden due to the wide array of measures assessed at each wave. For instance, the Center for Epidemiological Studies – Depression (CES-D) scale was used to assess depressive symptoms in the present

dissertation. Originally a 20-item measure (Radloff, 1977), with a well-validated 10-item short form version (Andresen et al., 1994), only nine items were consistently assessed across Waves I-III of Add Health. Although the adapted nine-item version used in this study had good internal consistency at each timepoint (i.e., α = .79, .80, .81) and has been used in other Add Health research (Lee & Estrada-Martinez, 2020), the reduced number of items makes it difficult to interpret and compare summary scores. For instance, researchers have published recommended cutoff scores to indicate clinically meaningful symptoms of depression for both the CES-D (Radloff, 1977) and CES-D-10 (Björgvinsson et al., 2013) hat may not accurately map onto the nine-item version.

This study was also limited by its exclusive focus on neighborhood socioeconomic conditions, a well-studied domain of neighborhood conditions. Indeed, a recent scoping review found that over 60% of all studies on neighborhoods and health conducted among children, adolescents, and emerging adults focused, exclusively or in part, on neighborhood socioeconomic conditions. Neighborhood features beyond socioeconomic conditions (e.g., social disorder, built environment) may have unique timing-related impacts on health and should be investigated.

This study was also limited by the use of census measures of the neighborhood context. Previous research suggests that perceptions of the neighborhood environment may be more strongly related to mental health outcomes than objective, administrative data (Zhang et al., 2019). Although perceptions of neighborhood safety and social cohesion were assessed in Add Health, these data were only available at Waves I and II and as such, analyses of these constructs would have been restricted to adolescence.

Additionally, the use of census data and accompanying geographies subject the findings of this dissertation to the modifiable areal unit problem (Openshaw, 1984). The modifiable areal unit problem refers to a form of statistical bias in the analysis of spatial data where the pattern of results differs significantly by the selected geographic resolution (Openshaw, 1984). This is a well-recognized problem in neighborhood effects studies, where the assessment of "neighborhood" effects can represent police jurisdictions, census block groups, census tracts, and even zip codes (Gresham & Karatekin, *under revision*). In addition, the reliance on census administrative data (i.e., the 1990 decennial census at Waves I and II and the 2000 decennial census at Wave III) meant that values of neighborhood concentrated disadvantage were not reflective of conditions at the year of Add Health assessment (i.e., 1994-95, 1996, 2001-02; Harris et al., 2019). Although neighborhood socioeconomic conditions are relatively stable, the 1990's and early 2000's represent a period during which concentrated poverty increased significantly (Iceland & Hernandez, 2017). It remains unclear the extent to which this may have impacted the findings of this study.

The ability to draw meaningful conclusions from this study was also limited by the relatively small number of adolescents and emerging adults that reported binge drinking in the past 12 months. I originally intended to examine the age-varying association between neighborhood concentrated disadvantage and past 12-month binge drinking *frequency*. However, high engagement in binge drinking was relatively rare in the present sample, leading to a larger variance than mean in the outcome across age (i.e., overdispersion). Accordingly, I dichotomized the binge drinking to estimate the

likelihood of binge drinking overall, and its association with neighborhood concentrated disadvantage, in the past 12 months. Yet, past 12-month binge drinking remained relatively rare at younger ages, as indicated by wider confidence intervals, yielding a higher degree of uncertainty in estimated effects.

Chapter 5: Conclusions

In this dissertation, I demonstrated that the age at which residing in a disadvantaged neighborhood is associated with health varies by the outcome examined. In doing so, I employed an innovative approach to examine the role of developmental timing in shaping neighborhood effects on health, structuring three waves of longitudinal data by age to elucidate when in development neighborhood concentrated disadvantage confers the greatest risk. This, coupled with the use of repeated measures for both neighborhood conditions and each outcome, providing novel insights not afforded by prior work. Findings revealed that the effect of neighborhood concentrated disadvantage on depressive symptoms is most pronounced during the transition to adulthood, whereas the effect on binge drinking is strongest in early adolescence. Moreover, although the association between neighborhood concentrated disadvantage and binge drinking is stronger among males than females, the moderating role of sex is equivalent across age. This study provides an important step in clarifying when neighborhoods matter for health, yielding insight that can be leveraged by policy makers and practitioners alike. Still, several important directions for future developmentally oriented neighborhood effects research remain. These include 1) the development and use of longitudinal datasets that address current methodological shortcomings in neighborhoods and health

research, 2) studies that move beyond socioeconomic status to investigate associations between other environmental features of the neighborhood (e.g., built environment, physical environment) and health from a developmental lens, and 3) the incorporation of upstream, structural factors that shape the neighborhood context into research. Such evidence will demonstrate not only whether, but *when* and *how* neighborhoods shape health throughout development, the findings from which may be useful for promoting health neighborhoods for all.

References

Anderson, S., Johnston, W., & Leventhal, T. (2019). When neighborhoods matter: Developmental timing and youth reading achievement and problem behaviors. *Social Science Research*, *81*(Aber, J.L., Gephart, M., Brooks-Gunn, J., Connell, J.P., Spencer, M. (1997). Neighborhood, family, and individual processes as they influence child and adolescent outcomes. Neighborhood Poverty: Context and Consequences for Children, vol I. New York: Russell Sage Foundation.), 1–11. https://doi.org/10.1016/j.ssresearch.2019.02.010

Anderson, S., Leventhal, T., Newman, S., & Dupéré, V. (2014). Residential mobility among children: A framework for child and family policy. *Cityscape*, *16*(1), 5–36.

Andresen, E. M., Malmgren, J. A., Carter, W. B., & Patrick, D. L. (1994). Screening for depression in well older adults: Evaluation of a short form of the CES-D. *American Journal of Preventive Medicine*, *10*(2), 77–84.

Arcaya, M. C., Tucker-Seeley, R. D., Kim, R., Schnake-Mahl, A., So, M., & Subramanian, S. V. (2016). Research on neighborhood effects on health in the United States: A systematic review of study characteristics. *Social Science & Medicine*, *168*, 16–29. https://doi.org/10.1016/j.socscimed.2016.08.047

Arnett, J. J. (2007). Emerging Adulthood: What Is It, and What Is It Good For? *Child Development Perspectives*, *1*(2), 68–73. https://doi.org/10.1111/j.1750-8606.2007.00016.x

Askari, M. S., Belsky, D. W., Olfson, M., Breslau, J., Mojtabai, R., Kajeepeta, S., Bruzelius, E., & Keyes, K. M. (2023). An integrative literature review of birth

cohort and time period trends in adolescent depression in the United States. *Social Psychiatry and Psychiatric Epidemiology*. https://doi.org/10.1007/s00127-023-02527-8

Assari, S., & Caldwell, C. H. (2017). Neighborhood Safety and Major Depressive Disorder in a National Sample of Black Youth; Gender by Ethnic Differences. *Children*, *4*(2). https://doi.org/10.3390/children4020014

Assari, S., Smith, J. R., Caldwell, C. H., & Zimmerman, M. A. (2015). Gender Differences in Longitudinal Links between Neighborhood Fear, Parental Support, and Depression among African American Emerging Adults. *Societies*, *5*(1), 151–170. https://doi.org/10.3390/soc5010151

Barr, P. B. (2018a). Early neighborhood conditions and trajectories of depressive symptoms across adolescence and into adulthood. *Advances in Life Course Research*, *35*, 57–68. https://doi.org/10.1016/j.alcr.2018.01.005

Barr, P. B. (2018b). Neighborhood conditions and trajectories of alcohol use and misuse across the early life course. *Health & Place*, *51*, 36–44. https://doi.org/10.1016/j.healthplace.2018.02.007

Björgvinsson, T., Kertz, S. J., Bigda-Peyton, J. S., McCoy, K. L., & Aderka, I. M. (2013). Psychometric properties of the CES-D-10 in a psychiatric sample. *Assessment*, *20*(4), 429–436.

Bornstein, M. H. (1989). Sensitive periods in development: Structural characteristics and causal interpretations. *Psychological Bulletin*, *105*(2), 179–197. https://doi.org/10.1037/0033-2909.105.2.179

Brady, K. T., & Randall, C. L. (1999). Gender differences in substance use disorders. *Psychiatric Clinics of North America*, *22*(2), 241–252. https://doi.org/10.1016/S0193-953X(05)70074-5

Bronfenbrenner, U. (1979). *The ecology of human development: Experiments by nature and design*. Harvard university press.

Crowder, K., & South, S. J. (2011). Spatial and temporal dimensions of neighborhood effects on high school graduation. *Social Science Research*, *40*(1), 87–106. https://doi.org/10.1016/j.ssresearch.2010.04.013

Diez Roux, A. (2018a). Foreword. In *Neighborhoods and health* (2nd ed.). Oxford University Press.

Diez Roux, A. (2018b). Foreword. In *Neighborhoods and health* (2nd ed.). Oxford University Press.

Ding, D., Sallis, J. F., Kerr, J., Lee, S., & Rosenberg, D. E. (2011). Neighborhood Environment and Physical Activity Among Youth: A Review. *American Journal of Preventive Medicine*, *41*(4), 442–455. https://doi.org/10.1016/j.amepre.2011.06.036

Dong, Y., & Peng, C.-Y. J. (2013). Principled missing data methods for researchers. *SpringerPlus*, *2*(1), 222. https://doi.org/10.1186/2193-1801-2-222

Duncan, D. T., & Kawachi, I. (2018). *Neighborhoods and health*. Oxford University Press.

Duncan, G. J., Ziol-Guest, K. M., & Kalil, A. (2010). Early-Childhood Poverty and Adult Attainment, Behavior, and Health. *Child Development*, *81*(1), 306–325. https://doi.org/10.1111/j.1467-8624.2009.01396.x

Dupere, V., Leventhal, T., Crosnoe, R., & Dion, E. (2010). Understanding the positive role of neighborhood socioeconomic advantage in achievement: The contribution of the home, child care, and school environments. *Developmental Psychology*, *46*(5), 1227–1244. https://doi.org/10.1037/a0020211

Estrada-Martinez, L. M., Lee, H., & Shapiro, E. (2019). Trajectories of depressive symptoms from adolescence to adulthood among multiple Latino subgroups. *Journal of Latinx Psychology*, *7*(4), 322–338. https://doi.org/10.1037/lat0000130

Farrell, C. (2020). Exploring the overlap between sexual victimization and offending among young women across neighborhoods: Does the type of force and type of offending matter? *Journal of Interpersonal Violence*, *35*(3–4), 571–599. https://doi.org/10.1177/0886260516689778

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American Journal of Preventive Medicine*, *14*(4), 245–258.

Gard, A. M., Maxwell, A. M., Shaw, D. S., Mitchell, C., Brooks-Gunn, J., McLanahan, S. S., Forbes, E. E., Monk, C. S., & Hyde, L. W. (2021). Beyond family-level adversities: Exploring the developmental timing of neighborhood disadvantage

effects on the brain. *Developmental Science*, *24*(1). https://doi.org/10.1111/desc.12985

Geolytics. (2021). *Neighborhood change database [NCDB] tract data from 1970 from 1970-2010* [Dataset]. https://geolytics.com/products/normalized-data/neighborhood-change-database

Gliem, J. A., & Gliem, R. R. (2003). *Calculating, interpreting, and reporting Cronbach's alpha reliability coefficient for Likert-type scales*.

Gregory, I. N., & Ell, P. S. (2005). Breaking the Boundaries: Geographical Approaches to Integrating 200 Years of the Census. *Journal of the Royal Statistical Society Series A: Statistics in Society*, *168*(2), 419–437. https://doi.org/10.1111/j.1467-985X.2005.00356.x

Gresham, B., & Karatekin, C. (n.d.). A scoping review of neighborhood effects and health among children and adolescents: Measurement and design characteristics. *Under Revision*.

Gresham, B., Thyden, N. H., Gailey, S., & Osypuk, T. L. (2024). Effect of a Randomized Controlled Trial of Housing Vouchers on Adolescent Risky Sexual Behavior Over a 15-Year Period. *Archives of Sexual Behavior*, *53*(2), 457–469. https://doi.org/10.1007/s10508-023-02736-x

Hackman, D. A., Cserbik, D., Chen, J.-C., Berhane, K., Minaravesh, B., McConnell, R., & Herting, M. M. (2021). Association of Local Variation in Neighborhood Disadvantage in Metropolitan Areas With Youth Neurocognition and Brain

Structure. *JAMA Pediatrics*, *175*(8), e210426. https://doi.org/10.1001/jamapediatrics.2021.0426

Harris, K. M. (2013a). *The add health study: Design and accomplishments*. https://doi.org/10.17615/C6TW87

Harris, K. M. (2013b). *The add health study: Design and accomplishments*. https://doi.org/10.17615/C6TW87

Harris, K. M., Halpern, C. T., Whitsel, E. A., Hussey, J. M., Killeya-Jones, L. A., Tabor, J., & Dean, S. C. (2019). Cohort Profile: The National Longitudinal Study of Adolescent to Adult Health (Add Health). *International Journal of Epidemiology*, *48*(5), 1415–1415k. https://doi.org/10.1093/ije/dyz115

Horn, L., Peter, K., & Rooney, K. (2002). *Profile of undergraduates in U.S. postsecondary institutions: 1999-2000* (Nos. 2002–168). U.S. Department of Education, National Center for Education Statistics. https://nces.ed.gov/pubs2002/2002168.pdf

Huang, Y., & Sparks, P. J. (2023). Longitudinal exposure to neighborhood poverty and obesity risk in emerging adulthood. *Social Science Research*, *111*, 102796. https://doi.org/10.1016/j.ssresearch.2022.102796

Iceland, J., & Hernandez, E. (2017). Understanding trends in concentrated poverty: 1980–2014. *Social Science Research*, *62*, 75–95. https://doi.org/10.1016/j.ssresearch.2016.09.001

Jackson, S., & Goossens, L. (2020). *Handbook of adolescent development*.

Jivraj, S., Nicholas, O., Murray, E. T., & Norman, P. (2021). Life Course Neighbourhood Deprivation and Self-Rated Health: Does It Matter Where You Lived in Adolescence and Do Neighbourhood Effects Build Up over Life?. *International Journal of Environmental Research and Public Health*, *18*(19). https://doi.org/10.3390/ijerph181910311

Jones-Webb, R., & Karriker-Jaffe, K. J. (2013). Neighborhood Disadvantage, High Alcohol Content Beverage Consumption, Drinking Norms, and Drinking Consequences: A Mediation Analysis. *Journal of Urban Health*, *90*(4), 667–684. https://doi.org/10.1007/s11524-013-9786-y

Karatekin, C., & Hill, M. (2019). Expanding the Original Definition of Adverse Childhood Experiences (ACEs). *Journal of Child & Adolescent Trauma*, *12*(3), 289–306. https://doi.org/10.1007/s40653-018-0237-5

Karriker-Jaffe, K. J., Au, V., Frendo, M., & Mericle, A. A. (2017). Offsetting the Effects of Neighborhood Disadvantage on Problem Drinking. *Journal of Community Psychology*, *45*(5), 678–684. https://doi.org/10.1002/jcop.21881

Kim, H., Schmidt, N. M., Osypuk, T. L., Thyden, N., & Rehkopf, D. (2023). Effects of housing vouchers on the long-term exposure to neighbourhood opportunity among low-income families: The moving to opportunity experiment. *Housing Studies*, *38*(1), 128–151. https://doi.org/10.1080/02673037.2022.2112154

Kim, J., Liu, J., Colabianchi, N., & Pate, R. R. (2010). The Effect of Perceived and Structural Neighborhood Conditions on Adolescents' Physical Activity and

Sedentary Behaviors. *Archives of Pediatrics & Adolescent Medicine*, *164*(10), 935–942. https://doi.org/10.1001/archpediatrics.2010.167

King, C., Huang, X., & Dewan, N. A. (2022). Continuity and change in neighborhood disadvantage and adolescent depression and anxiety. *Health & Place*, *73*, 102724. https://doi.org/10.1016/j.healthplace.2021.102724

Lanza, S. T., & Linden-Carmichael, A. N. (2021). *Time-varying effect modeling for the behavioral, social, and health sciences*. Springer.

Lanza, S. T., Vasilenko, S. A., & Russell, M. A. (2016a). Time-varying effect modeling to address new questions in behavioral research: Examples in marijuana use. *Psychology of Addictive Behaviors*, *30*(8), 939–954. https://doi.org/10.1037/adb0000208

Lanza, S. T., Vasilenko, S. A., & Russell, M. A. (2016b). Time-varying effect modeling to address new questions in behavioral research: Examples in marijuana use. *Psychology of Addictive Behaviors*, *30*(8), 939–954. https://doi.org/10.1037/adb0000208

Lawrence, W. R., Kucharska-Newton, A. M., Magnani, J. W., Brewer, L. C., Shiels, M. S., George, K. M., Lutsey, P. L., Jenkins, B. D., Sullivan, K. J., Carson, A. P., & Freedman, N. D. (2024). Neighborhood Socioeconomic Disadvantage Across the Life Course and Premature Mortality. *JAMA Network Open*, *7*(8), e2426243–e2426243. https://doi.org/10.1001/jamanetworkopen.2024.26243

Lee, H., & Estrada-Martinez, L. M. (2020). Trajectories of Depressive Symptoms and Neighborhood Changes from Adolescence to Adulthood: Latent Class Growth

Analysis and Multilevel Growth Curve Models. *International Journal of Environmental Research and Public Health*, *17*(6). https://doi.org/10.3390/ijerph17061829

Leventhal, T. (2018). Neighborhood context and children's development: When do neighborhoods matter most? *Child Development Perspectives*, *12*(4), 258–263.

Leventhal, T., Dupéré, V., & A. Shuey, E. (2015). Children in Neighborhoods. In *Handbook of Child Psychology and Developmental Science* (pp. 1–41). https://doi.org/10.1002/9781118963418.childpsy413

Leventhal, T., Dupéré, V., & Brooks-Gunn, J. (2009). Neighborhood influences on adolescent development. In *Handbook of adolescent psychology: Contextual influences on adolescent development, Vol. 2, 3rd ed.* (pp. 411–443). John Wiley & Sons, Inc.

MacKinnon, A. L., Tomfohr-Madsen, L., & Tough, S. (2021). Neighborhood socio-economic factors and associations with infant sleep health. *Behavioral Sleep Medicine*, *19*(4), 458–470. https://doi.org/10.1080/15402002.2020.1778478

Madley-Dowd, P., Hughes, R., Tilling, K., & Heron, J. (2019). The proportion of missing data should not be used to guide decisions on multiple imputation. *Journal of Clinical Epidemiology*, *110*, 63–73. https://doi.org/10.1016/j.jclinepi.2019.02.016

Mair, C., Roux, A. V. D., & Galea, S. (2008). Are neighbourhood characteristics associated with depressive symptoms? A review of evidence. *Journal of Epidemiology and Community Health*, *62*(11), 940. https://doi.org/10.1136/jech.2007.066605

Manson, S., Schroeder, J., Van Riper, D., Knowles, K., Kugler, T., Roberts, F., & Ruggles, S. (2024). *IPUMS National Historical Geographic Information System: Version 19.0* [Dataset]. https://doi.org/10.18128/D050.V19.0

Matheson, F. I., White, H. L., Moineddin, R., Dunn, J. R., & Glazier, R. H. (2012). Drinking in context: The influence of gender and neighbourhood deprivation on alcohol consumption. *Journal of Epidemiology and Community Health*, *66*(6), e4. https://doi.org/10.1136/jech.2010.112441

Minh, A., Muhajarine, N., Janus, M., Brownell, M., & Guhn, M. (2017a). A review of neighborhood effects and early child development: How, where, and for whom, do neighborhoods matter?. *Health & Place*, *46*(din, 9510067), 155–174. https://doi.org/10.1016/j.healthplace.2017.04.012

Minh, A., Muhajarine, N., Janus, M., Brownell, M., & Guhn, M. (2017b). A review of neighborhood effects and early child development: How, where, and for whom, do neighborhoods matter?. *Health & Place*, *46*(din, 9510067), 155–174. https://doi.org/10.1016/j.healthplace.2017.04.012

Musliner, K. L., Munk-Olsen, T., Eaton, W. W., & Zandi, P. P. (2016). Heterogeneity in long-term trajectories of depressive symptoms: Patterns, predictors and outcomes. *Journal of Affective Disorders*, *192*, 199–211. https://doi.org/10.1016/j.jad.2015.12.030

National Center for Education Statistics. (2024). *College enrollment rates*. U.S. Department of Education, Institute of Education Sciences. https://nces.ed.gov/programs/coe/indicator/cpb/college-enrollment-rate

Nelson, S. E., Van Ryzin, M. J., & Dishion, T. J. (2015). Alcohol, marijuana, and tobacco use trajectories from age 12 to 24 years: Demographic correlates and young adult substance use problems. *Development and Psychopathology*, *27*(1), 253–277.

Oakes, J. M. (2004). The (mis)estimation of neighborhood effects: Causal inference for a practicable social epidemiology. *Social Science & Medicine*, *58*(10), 1929–1952. https://doi.org/10.1016/j.socscimed.2003.08.004

Oakes, J. M., Andrade, K. E., Biyoow, I. M., & Cowan, L. T. (2015). Twenty Years of Neighborhood Effect Research: An Assessment. *Current Epidemiology Reports*, *2*(1), 80–87. https://doi.org/10.1007/s40471-015-0035-7

Openshaw, S. (1984). The modifiable areal unit problem. *Concepts and Techniques in Modern Geography*.

R Core Team. (2023). *R: A language and environment for statistical computing* [Computer software]. https://www.r-project.org.

Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*(3), 385–401. http://dx.doi.org.ezproxy3.lhl.uab.edu/10.1177/014662167700100306

Reboussin, B. A., Furr-Holden, D. M., Green, K. M., Ialongo, N. S., Rabinowitz, J. A., Matson, P. A., Maher, B., Nelson, V., & Milam, A. J. (2019). Social Influences on Drinking Trajectories From Adolescence to Young Adulthood in an Urban Minority Sample. *Journal of Studies on Alcohol and Drugs*, *80*(2), 186–195. https://doi.org/10.15288/jsad.2019.80.186

Renalds, A., Smith, T. H., & Hale, P. J. (2010). A Systematic Review of Built Environment and Health. *Family & Community Health*, *33*(1). https://journals.lww.com/familyandcommunityhealth/fulltext/2010/01000/a_systematic_review_of_built_environment_and.9.aspx

Richardson, R., Westley, T., Gariépy, G., Austin, N., & Nandi, A. (2015). Neighborhood socioeconomic conditions and depression: A systematic review and meta-analysis. *Social Psychiatry and Psychiatric Epidemiology*, *50*(11), 1641–1656.

Romer, D. (2010). Adolescent risk taking, impulsivity, and brain development: Implications for prevention. *Developmental Psychobiology*, *52*(3), 263–276. https://doi.org/10.1002/dev.20442

Schmidt, N. M., Glymour, M. M., & Osypuk, T. L. (2017). Adolescence Is a Sensitive Period for Housing Mobility to Influence Risky Behaviors: An Experimental Design. *Journal of Adolescent Health*, *60*(4), 431–437. https://doi.org/10.1016/j.jadohealth.2016.10.022

Schubert, K. O., Clark, S. R., Van, L. K., Collinson, J. L., & Baune, B. T. (2017). Depressive symptom trajectories in late adolescence and early adulthood: A systematic review. *Australian & New Zealand Journal of Psychiatry*, *51*(5), 477–499. https://doi.org/10.1177/0004867417700274

Schuler, M. S., Vasilenko, S. A., & Lanza, S. T. (2015). Age-varying associations between substance use behaviors and depressive symptoms during adolescence and young adulthood. *Drug and Alcohol Dependence*, *157*, 75–82. https://doi.org/10.1016/j.drugalcdep.2015.10.005

Shorey, S., Ng, E. D., & Wong, C. H. J. (2022). Global prevalence of depression and elevated depressive symptoms among adolescents: A systematic review and meta-analysis. *British Journal of Clinical Psychology*, *61*(2), 287–305. https://doi.org/10.1111/bjc.12333

Shuey, E. A., & Leventhal, T. (2019). Neighborhoods and parenting. In *Handbook of parenting* (pp. 371–399). Routledge.

Smith, D. J., Kyle, S., Forty, L., Cooper, C., Walters, J., Russell, E., Caesar, S., Farmer, A., McGuffin, P., Jones, I., Jones, L., & Craddock, N. (2008). Differences in depressive symptom profile between males and females. *Journal of Affective Disorders*, *108*(3), 279–284. https://doi.org/10.1016/j.jad.2007.10.001

Smith, K. E., & Pollak, S. D. (2021). Rethinking Concepts and Categories for Understanding the Neurodevelopmental Effects of Childhood Adversity. *Perspectives on Psychological Science*, *16*(1), 67–93. https://doi.org/10.1177/1745691620920725

Snow, J. (1856). On the mode of communication of cholera. *Edinburgh Medical Journal*, *1*(7), 668.

Solar, O., & Irwin, A. (2010). *A conceptual framework for action on the social determinants of health*.

Substance Use and Mental Health Services Administration. (2022). *Key substance use and mental health indicators in the United States: Results from the 2021 National Survey on Drug Use and Health* (No. HHS Publication No. PEP22-07-01-005, NSDUH Series H-57). Center for Behavioral Health Statistics and Quality,

Subtance Use, and Mental Health Services Administration. https://www.samhsa.gov/data/report/2021-nsduh-annual-national-report

Taylor, R. L., Cooper, S. R., Jackson, J. J., & Barch, D. M. (2020). Assessment of Neighborhood Poverty, Cognitive Function, and Prefrontal and Hippocampal Volumes in Children. *JAMA Network Open*, *3*(11), e2023774. https://doi.org/10.1001/jamanetworkopen.2020.23774

Vasilenko, S. A., Piper, M. E., Lanza, S. T., Liu, X., Yang, J., & Li, R. (2014). Time-Varying Processes Involved in Smoking Lapse in a Randomized Trial of Smoking Cessation Therapies. *Nicotine & Tobacco Research*, *16*(Suppl_2), S135–S143. https://doi.org/10.1093/ntr/ntt185

Wodtke, G. T., Elwert, F., & Harding, D. J. (2016). Neighborhood Effect Heterogeneity by Family Income and Developmental Period. *American Journal of Sociology*, *121*(4), 1168–1222. https://doi.org/10.1086/684137

Zhang, L., Zhou, S., & Kwan, M.-P. (2019). A comparative analysis of the impacts of objective versus subjective neighborhood environment on physical, mental, and social health. *Health & Place*, *59*, 102170.

Appendix A

Nine items used to assess depressive symptoms at Add Health Waves I-III.

1. You felt depressed
2. You enjoyed life (reverse coded)
3. You felt that people disliked you
4. You felt that you could not shake off the blues, even with help from your family and friends
5. You felt that you were too tired to do things
6. You felt you were just as good as other people (reverse coded)
7. You had trouble keeping your mind on what you were doing
8. You were bothered by things that don’t usually bother you
9. You felt sad

Appendix B

Equations for TVEM models predicting the age-varying effects of neighborhood concentrated disadvantage on depressive symptoms, as well as moderation by biological sex.

Intercept-Only Model (M0)

$$Depressive\ Symptoms_i = \beta_0(t) + \varepsilon_i$$

Unadjusted Neighborhood Disadvantage Model (M1)

$$Depressive\ Symptoms_i = \beta_0(t) + \beta_1(t)Neighborhood\ Disadvantage_{it} + \varepsilon_i$$

Unadjusted Biological Sex Model (M2)

$$Depressive\ Symptoms_i = \beta_0(t) + \beta_1(t)Sex_i + \varepsilon_i$$

Adjusted Predictor Model, Time-Varying (M3)

$$\begin{aligned} Depressive\ Symptoms_i \\ = \beta_0(t) + \beta_1(t)Neighborhood\ Disadvantage_i + \beta_2(t)Sex_i \\ + \beta_3(t)Caregiver\ Education_i + \beta_4(t)Caregiver\ Employment_i \\ + \beta_5(t)Receipt\ of\ Public\ Assistance_i + \beta_6(t)Participant\ Race_i \\ + \beta_7(t)Ethnicity_i + \varepsilon_i \end{aligned}$$

Adjusted Predictor Model, Time-Invariant (M4)

$$\begin{aligned} Depressive\ Symptoms_i \\ = \beta_0(t) + \beta_1(t)Neighborhood\ Disadvantage_i + \beta_2(t)Sex_i \\ + \beta_3 Caregiver\ Education_i + \beta_4 Caregiver\ Employment_i \\ + \beta_5 Receipt\ of\ Public\ Assistance_i + \beta_6 Participant\ Race_i \\ + \beta_7 Ethnicity_i + \varepsilon_i \end{aligned}$$

Two-Way Interaction Model (M5)

$$Depressive\ Symptoms_i = \beta_0(t) + \beta_1(t)Neighborhood\ Disadvantage_{it} + \beta_2(t)Sex_i + \beta_3(t)Neighborhood\ Disadvantage\ x\ Sex_{it} + \beta_4 Caregiver\ Education_i + \beta_5 Caregiver\ Employment_i + \beta_6 Receipt\ of\ Public\ Assistance_i + \beta_7 Participant\ Race_i + \beta_8 Ethnicity_i + \varepsilon_{it}$$

Appendix C

Equations for TVEM models predicting the age-varying effects of neighborhood concentrated disadvantage on binge drinking, as well as moderation by biological sex.

Intercept-Only Model (M0)

$$Binge\ Drinking\ Odds(t) = e^{\beta_0(t)}$$

Unadjusted Neighborhood Disadvantage Model (M1)

$$Binge\ Drinking\ Odds(t) = e^{\beta_0(t)} + e^{\beta_1(t)Neighborhood\ Disadvantage_i}$$

Unadjusted Biological Sex Model (M2)

$$Binge\ Drinking\ Odds(t) = e^{\beta_0(t)} + e^{\beta_1(t)Sex_i}$$

Adjusted Predictor Model, Time-Varying (M3)

$$\begin{aligned} &Binge\ Drinking\ Odds(t) \\ &= e^{\beta_0(t)} + e^{\beta_1(t)Neighborhood\ Disadvantage_i} + e^{\beta_2(t)Sex_i} \\ &+ e^{\beta_3(t)Caregiver\ Education_i} + e^{\beta_4(t)Caregiver\ Employment_i} \\ &+ e^{\beta_5(t)Receipt\ of\ Public\ Assistance_i} + e^{\beta_6(t)Participant\ Race_i} \\ &+ e^{\beta_7(t)Participant\ Ethnicity_i} \end{aligned}$$

Adjusted Predictor Model, Time-Invariant (M4)

$$\begin{aligned} &Binge\ Drinking\ Odds(t) \\ &= e^{\beta_0(t)} + e^{\beta_1(t)Neighborhood\ Disadvantage_i} + e^{\beta_2(t)Sex_i} \\ &+ e^{\beta_3 Caregiver\ Education_i} + e^{\beta_4 Caregiver\ Employment_i} \\ &+ e^{\beta_5 Receipt\ of\ Public\ Assistance_i} + e^{\beta_6 Participant\ Race_i} \\ &+ e^{\beta_7 Participant\ Ethnicity_i} \end{aligned}$$

Two-Way Interaction Model (M5)

$$Binge\ Drinking\ Odds(t)$$

$$= e^{\beta_0(t)} + e^{\beta_1(t)Neighborhood\ Disadvantage_i} + e^{\beta_2(t)Sex_i}$$

$$+ e^{\beta_3(t)Neighborhood\ Disadvantage\ x\ Sex_i} + e^{\beta_4(t)Caregiver\ Education_i}$$

$$+ e^{\beta_5(t)Caregiver\ Employment_i} + e^{\beta_6(t)Receipt\ of\ Public\ Assistance_i}$$

$$+ e^{\beta_7(t)Participant\ Race_i} + e^{\beta_8(t)Participant\ Ethnicity_i}$$