

Ethnic diversity and depression within Black America:
Identifying and understanding within-group differences

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Abstract

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While the literature on Black-white differences in major depressive disorder (MDD) and depressive symptoms is robust, less robust is the literature on how these outcomes are patterned within the US Black population and why differences exist. Given increasing numbers of first-generation immigrants from the Caribbean, sub-Saharan Africa, Latin America, among other regions of the world, as well as increasing numbers of second- and third-generation immigrants, continued aggregation has the potential to mask intra-racial differences between these ethnic-immigrant groups and Black Americans with more distant ancestral ties to Africa (i.e., African Americans). Among these subgroups, the extremely limited data disaggregating the US Black population suggest the following patterns. First, foreign-born Black immigrants have lower levels of MDD and related symptoms relative to US-born Black Americans, a finding which is consistent with theories of foreign-born health advantage. Second, among the US-born, Caribbean adults have higher levels of MDD and related symptoms relative to all other Black Americans, a finding which is inconsistent with theories related to intergenerational declines in health toward convergence to native-born levels. Lastly, and contrary to results among adults, first- and second-generation Caribbeans have lower levels of depressive symptoms relative to all other Black youth. This dissertation sought to better understand how depression and its related symptoms are patterned within the US Black population, as well as how mechanisms causing these outcomes may vary across subgroups defined by domains related to immigration.

Chapter 1 was a systematic review, which comprehensively synthesized depression and related symptoms within the US Black population across these domains, including a summary of mechanisms proposed toward explaining intra-racial variation. Using longitudinal data, Chapter 2 examined whether, and if so when, growth curve models of depressive symptoms varied by immigrant generation contrasts among a representative sample of Black youth followed into adulthood. And using representative data from the largest study of Black mental health, Chapter 3 examined whether the relationship between racial identity, a presumed protective factor against depression and related symptoms, and MDD varied between US-born Caribbeans and all other US-born Black Americans.

The systematic review of Chapter 1 revealed substantial variation in the prevalence of depression and its related symptoms within the US Black population by nativity, region of birth, age at immigration, and Caribbean ethnic origin. Results additionally confirmed that much of what is known about intra-racial heterogeneity comes from a single data source, the National Study of American Life (NSAL). Using longitudinal data of youth followed into adulthood, Chapter 2 found evidence of diverging depressive symptoms trajectories among Black respondents by immigrant generation (first/second-generation compared with third and higher generations); notably, contrasts among Black respondents varied from those of other racial/ethnic groups (Asian, Hispanic/Latinx, non-Hispanic white). Lastly, results from Chapter 3 suggest aspects of racial identity may not be protective for US-born Caribbeans, pointing to variations in racialization experiences as a distal cause. Additional research using larger sample sizes, more diverse subgroups of Black ethnic immigrants, as well as longitudinal data, is needed to further understand patterns of and additional sources underlying heterogeneity of depression and its related symptoms within the US Black population.

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Dedication

To my parents, Francisca and Darlington Esie.

Introduction

“I think I’ve gone through all these phases ... I remember sitting there trying to pick my label. Am I Nigerian? Am I African-American? Am I black? ... [W]hen it really comes down to it – I mean it matters who’s looking at you. For some people I’m black, for others I’m African-American. I just know that I do have a love for Nigeria that was instilled in me growing up. But I am in America so there’s no way I can just label myself one thing and try to live the lifestyle of that label.”

– Andrea, 21-year-old second-generation Nigerian (in Balogun, 2011)

Rising numbers of Black immigrants from around the globe, since immigration reforms of the 1960s, underlie the changing portrait of Black America.¹ While a large portion of Black Americans descend from enslaved African people — an ethnic group which I refer to in this dissertation as African American — a growing portion are recent immigrants who have largely emigrated from the Caribbean and sub-Saharan Africa.² Black immigrants (first-generation immigrants), their children (second-generation immigrants), as well as their grandchildren (third-generation immigrants) contribute not only to the growing ethnic diversity of Black Americans, but also to the destabilization of what it means to be Black in America. In public health research, and the public discourse broadly, the ethnic and cultural diversity within the US Black population is largely ignored. Rather, the terms “African American” and “Black” are used interchangeably.

For immigrants of color broadly, “becoming American” involves becoming racialized and contending with the stigma associated with being a minority.³ For Black immigrants and their children, this process has been documented as simultaneously contending with Black racial, Black ethnic, and immigrant identities, as well as dealing with negative effects associated with being Black in America. Poor mental health consequences of race-related stressors due to structural racism — particularly depression — are well-established.⁴⁻⁹ Yet, little is known about whether the mental health effects of structural racism vary between Black Americans with more

distant ancestral ties to Africa (i.e., African Americans) and Black Americans with more recent immigrant origins (e.g., first-, and second-generation immigrants), given historical and sociocultural factors that appear to underlie variations in racialization processes.

Within mental health research particularly, the literature on racial differences between Black and white Americans is far more robust than the literature on heterogeneity within the US Black population. For instance, numerous studies across various national data sources have documented and sought to explain the purported paradoxical finding that Black people have lower levels of major depressive disorder (MDD) relative to white people.¹⁰⁻¹⁵ This apparent racial “paradox” in MDD is based on expectations of social stress theory, the predominant framework linking social inequities to depression risk, which posits that Black people, relative to white people, should have worse mental health due to a heightened exposures to social stressors (such as racial discrimination) and socioeconomic deprivation.¹⁶⁻¹⁸ This “paradox” appears to only operate for MDD, and not across closely related measures of depressive symptoms or psychological distress.

Within the US Black population, much less is documented regarding variation in depression by immigration-related domains like nativity (foreign-born vs. US-born), ethnic origin (e.g., African American, Caribbean), and immigrant generation. And of the extremely limited data that do exist, investigations have focused largely on patterns between Caribbeans compared with all other (i.e., non-Caribbean) Black Americans. These data, which come from the National Survey of American Life (NSAL), have revealed most of what is known about how depression may be patterned within the Black population — patterns that form the basis for this dissertation. Specifically, MDD, depressive symptoms, and psychological distress were patterned such that foreign-born Caribbeans (first-generation immigrants) had the lowest levels, followed

by non-Caribbeans,* and US-born Caribbeans (second- and third-generation immigrants) had the highest levels of these three groups.¹⁹⁻²¹ Of note, US-born Caribbeans had nearly *twice* the prevalence of lifetime MDD than non-Caribbeans. The magnitude of this heightened level among US-born Caribbeans is surprising for at least two reasons. First, this pattern is inconsistent with theories of intergenerational health deterioration, which suggest the prevalence of health outcomes follow a stepwise pattern where first-generation immigrants fare better than second-generation immigrants, who then fare better than their third- and higher-generation counterparts.²² Rather, as overwhelmingly belonging to the “third- and higher-generation” group, non-Caribbeans in the NSAL have a much lower prevalence of MDD than second- and third-generation (US-born) Caribbeans. And second, foreign-born Caribbeans have a lower prevalence of MDD than their US-born co-ethnics, which mirrors those of other immigrant groups, as in Asian and Latinx groups.²³⁻²⁵ However, the decline in MDD from the foreign-born to the US-born appears more pronounced among Caribbeans than among Asian and Latinx groups.²⁶ These two patterns suggest there may be factors associated with the intersection of race and immigration that uniquely affect the mental health of Caribbeans, and could potentially reflect Black ethnic-immigrant groups broadly.

Another complexity concerns patterns among adolescents, which NSAL data have also captured. For depressive symptoms, Caribbean youth had lower levels relative to non-Caribbean youth,^{27,28} which is inconsistent with the heightened levels among US-born Caribbean adults

* In the NSAL, Caribbeans were defined as those who (1) self-identified as Black and (2) were either born in the Caribbean (foreign-born Caribbean) or had parents or grandparents born in the Caribbean (US-born Caribbean). “African Americans” were defined as those who self-identified as Black and did *not* identify recent Caribbean origin. Subsequently, this characterization of “African American” includes those with more recent immigrant origins in the US (i.e., in the latter half of the 20th century through today). Following Caribbeans, these individuals potentially differ from African Americans terms of cultural identification and socioeconomic factors. Towards recognizing the ethnic diversity within the US Black population and to avoid further imprecision, albeit slight, I refer to these individuals as non-Caribbean.

relative to non-Caribbean adults. If US-born Caribbean adults are comparable to Caribbean youth in the NSAL, these patterns could suggest divergent trajectories of depressive symptoms across the life course whereby Caribbeans begin to have higher levels of symptoms relative to non-Caribbeans at some point during the life course.

These findings from NSAL data left me with unanswered questions, and the few data sources that do disaggregate the US Black population by immigration-related domains point to obvious gaps in the literature. First, I wanted to better understand how depression and its related symptoms were patterned within the US Black population across data sources outside of the NSAL. Despite having been conducted 20 years ago between 2001-2003, the NSAL continues to be a popular data source used in studies of depression heterogeneity within the US Black population. Questions that arose included: Do more recent data exist? Do patterns in depression and its related symptoms differ within foreign-born Black immigrants by ethnicity or region of origin (e.g., Caribbean-born versus African-born)? Have second-generation immigrants who are not Caribbean been captured in the literature? What mechanisms have been proposed toward explaining observed heterogeneity?

Second, I wanted to assess whether trajectories of depressive symptoms from youth to adulthood varied within the US Black population. An appropriate data source to address this gap needs to be longitudinal, contain a measure of depressive symptoms, and contain enough respondents to disaggregate by an identified immigration-related variable. I identified just one — the National Longitudinal Study of Adolescent to Adult Health (Add Health).

To hypothesize variation in trajectories of depressive symptoms implies variation in mechanisms underlying depressive symptoms. One such mechanism could be related to ethnic differences in processes like racial socialization and racial identity developed during childhood.

For children of color broadly, parental racial socialization — typically referred to as the messages parents transmit to their children about race and coping with racialized stressors (vis-à-vis racism) — promotes the development of a strong racial identity.²⁹ Racial identity, in turn, is recognized as a protective factor against depression, as much of the published work has focused on depressive symptoms and distress.³⁰⁻³⁵ Much of what we know about the relationship between racial identity and depression within the US Black population comes from studies of African Americans. An increasing body of ethnographic research, however, finds immigration adds complexity to racial socialization experiences and racial identity development of Black children of immigrants.³⁶⁻⁴⁶ For example, Black immigrant parents may instill both a sense of ethnic pride and a wariness towards African Americans to their children, the latter being a practice that actively draws within-racial ethnic distinctions. But without an accent, these children of immigrants are often seen by others as African American, or through the prism of race alone. It seems likely, then, that these ethnic variations in racial socialization and racial identity development could result in differences in the meaning of race to one's identity and how to cope with racialized stressors, which may then imply differences in the effects of racial identity on depression. Since US-born Caribbean adults in the NSAL had nearly twice the prevalence of MDD relative to non-Caribbean adults, does the protective function of Black racial identity against depression depend on factors related to ethnic origin? This question forms the basis for a third gap I sought to fill — using NSAL data, I wanted to examine whether the relationship between racial identity and MDD varied within the US-born Black population by ethnic origin.

To summarize, this dissertation sought to highlight the ethnic diversity within the US Black population while leveraging the limited data that do exist toward better understanding within-group differences in depression and its related symptoms. This dissertation consists of

three chapters to do so. Chapter 1 is a systematic review of the literature on depression within the US Black population. Here, I identified studies that disaggregated the US Black population by immigration- and ethnicity-related domains and synthesized mechanisms toward explaining within-racial differences. Chapter 2 is an examination of whether longitudinal trajectories of depressive symptoms from adolescence to adulthood varied between Black Americans by immigrant generation contrasts. And Chapter 3 is an examination of whether the relationship between racial identity and MDD varies between US-born Caribbeans and all other US-born Black Americans.

Chapter 1: Depression and related symptoms within the US Black population — a systematic review of patterns and mechanisms

1.1 Introduction

Nationally representative epidemiologic studies of diagnostic psychiatric disorders consistently show that the prevalence of major depressive disorder (MDD) is markedly lower among Black Americans relative to white Americans.¹⁰⁻¹⁴ Such a finding is inconsistent with observed racial patterns in self-reported measures of depressive symptoms and psychological distress.⁴⁷ Between-racial patterns of MDD are also inconsistent with theories of social stress, the predominant framework linking social status to mental health, which posits individuals with disadvantaged social statuses are exposed to more stressors than those of advantaged social statuses, and such stress exposure will lead to worse mental health.¹⁶⁻¹⁸ As a result, numerous investigations have sought to explain this apparent paradoxical between-racial pattern.^{15,47-51} While the literature on between-group Black-white differences in depression and related self-reported symptoms is robust, fewer investigations exist that explore potential heterogeneity *within* the US Black population and explanations underlying any such observed heterogeneity.

Aggregating the US Black population is standard practice in health research, despite increased within-racial ethnic diversity due to rising numbers of Black immigrant populations. This practice of aggregation masks variations in immigration- and ethnicity-related characteristics, like nativity, immigrant generation, and ethnic origin,⁵² which may be important social factors related to more proximate determinants of depression risk. Ethnicity and race, particularly within the US Black population, are rarely treated as conceptually distinct social factors, and are instead often conflated.

Ethnicity refers to groups in which people share characteristics including geographical origins and cultural identification.⁵³ Ethnicity is separate from one's race, although they may co-occur; for instance, individuals of sub-Saharan African ethnicities are almost exclusively Black, yet the race of Hispanic/Latinx individuals in the US is more varied.⁵⁴ In both the US public discourse and in health research, "African American" is often used interchangeably with Black, yet the former refers to an ethnic group and the latter a racial category. "African American" typically refers to Black Americans with distant ancestral ties (i.e., more than three generations removed) to Africa; nearly all being descendants of enslaved Africans forced into the US. Culturally and historically, such people are distinct from the growing US Black population with more recent immigrant origins.⁵²

As of 2019, an estimated record 4.6 million Black immigrants live in the US — nearly doubling since 2000.⁵⁵ While Black immigrants come from several regions, nearly half were born in the Caribbean and about 42% were born in an African nation.⁵⁵ By 2060, projections suggest that one out of six Black people in the US will be foreign-born (i.e., first-generation immigrant).⁵⁶ As of 2019, 12% of the Black population were foreign-born immigrants, and an additional 9% were second-generation — or, those with at least one foreign-born parent.² These growing numbers of both first-generation and second-generation immigrants, originating largely from the Caribbean and Africa, signify increasing ethnic diversity within the US Black population. Yet despite this ethnic diversity, Black ethnicities, nativity, and immigrant generation status are rarely studied in health research.⁵⁷⁻⁶¹ Few data sources contain enough statistical power to disaggregate Black respondents, and subsequently, there are few investigations of prevalence patterns in mental health outcomes within the US Black population and causes underlying potential within-racial differences.

Diagnostic depression outcomes such as MDD are conceptually distinct from self-reported symptoms of depression or distress,⁶² yet these constructs are overlapping. The presence of diagnostic depression is defined by the Diagnostic and Statistical Manual of Mental Disorders (DSM) and is based on the presence of a cluster of symptoms for at least two weeks, causing clinically significant distress.⁶³ Measures of self-reported depressive symptoms, such as the Center for Epidemiological Studies Depression (CES-D) and the Patient Health Questionnaire (PHQ), have been validated as screening scales for diagnostic depression.⁶⁴⁻⁶⁶ Psychological distress is characterized by symptoms of depressed mood, in addition to symptoms of anxiety,⁶⁷ it has also been described as a normal emotional reaction to stressors that does not necessarily rise to the level of disorder.⁶⁸ Together, measures of depressive symptoms and psychological distress fall short of meeting DSM criteria for depression.⁴⁷ Although it follows that these self-reported symptom scales ought to be positively correlated with MDD, data have shown Black Americans have a lower prevalence of MDD relative to white Americans, yet higher levels of both self-reported measures of depressive symptoms and psychological distress across a range of nationally representative studies.⁴⁷ These discrepant patterns may suggest etiologies of diagnostic depression and related self-reported symptoms (i.e., depressive symptoms and psychological distress) vary for Black Americans. A better understanding of how diagnostic depression and these related symptoms are patterned within the US Black population could provide insight into the extent to which etiologies underlying these constructs may vary.

To my knowledge, only one review exists that characterizes the prevalence of depression within the US Black population across immigration-related domains and identifies potential mechanisms underlying differences.⁶⁹ The authors cited studies of diagnostic depression that both disaggregated by nativity and drew from one of two large, nationally representative studies

— the National Study of American Life (NSAL) or the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). NSAL and NESARC data showed foreign-born Black immigrants generally had a lower prevalence of MDD (both past year and lifetime) compared to the US-born. Comparisons within the US-born were limited to NSAL data, which showed those of recent Caribbean origin had a substantially higher prevalence of MDD compared with African Americans. And while the authors included a discussion of mechanisms that may protect the foreign-born, there was no discussion of potential mechanisms underlying differences within the US-born.⁶⁹ A synthesis of prevalence patterns and mechanisms across a wider range of immigration-related domains, like region of birth, immigrant generation or ethnic origin beyond Caribbean, does not exist to my knowledge.

The present study sought to provide a comprehensive synthesis of depression and related symptoms within the US Black population, disaggregating by immigration- and ethnicity-related domains. This review focused on adults (aged 18 or older) and youth (younger than 18 years of age) separately. This review also sought to summarize mechanisms proposed to explain any within-racial differences. The present review was not restricted to nationally representative studies, which may provide additional insight into prevalence patterns beyond nativity or Caribbean origin and mechanisms beyond nativity.

1.2 Methods

Search strategy

In October 2021, I searched Web of Science, PsychINFO, and PubMed databases without date restrictions using the following search string across all fields: (“Black” OR “Africa*” OR “Caribbean”) AND (“Immigra*” OR “Migrant” OR “Nativity” OR “Foreign”) AND

(“Depress*” OR “Distress” OR “Mood disorder”). The search was limited to studies written in English.

Eligibility criteria, study selection, and data extraction

Studies were included in the systematic review if they provided quantitative data on the prevalence of diagnostic measures of depression (major depressive disorder, major depressive episode, dysthymia) or related self-reported symptoms (e.g., depressive symptoms, psychological distress) among Black subgroups in the US disaggregated by a domain related to ethnic or immigrant origin (e.g., Caribbean ethnicity, nativity, immigrant generation). Studies were excluded due to the following reasons: data on major depression or related symptoms were not provided, data were not disaggregated by domains of interest, or the study sample was from a non-Black or non-US population. Unpublished manuscripts, conference abstracts, case reports, and studies without quantitative data were ineligible for inclusion.

Search results were deposited into the reference management program, Endnote, where duplicate records were deleted. Then, titles and abstracts of the remaining records were screened to exclude irrelevant studies. Next, full text articles were retrieved for screening. Finally, the reference lists of retained articles were screened to identify additional articles. The following information was extracted from the final set of articles, separately for adults (those aged 18 and older) and youth (those under age 18): first author and publication year, data source and collection period, depression measure, recall period, instrument, cutoff score or description of depression measure, Black subgroups identified (i.e., by ethnicity, nativity, or immigrant generation), non-Black subgroups identified (if any), and a summary of findings (i.e., the prevalence or mean by subgroup). In addition, I scanned the introduction and discussion sections

of included articles to identify proposed mechanisms underlying reported differences within Black subgroups, and whether the basis of the proposed mechanism was *a priori* or *a posteriori*. A mechanism was classified as *a priori* if it was a clear hypothesis of a given study, raised in the introduction section. A mechanism was classified as *a posteriori* if it was raised only in the discussion section. Extracting mechanisms is more ambiguous than extracting other study characteristics, as mechanisms may not be clearly identifiable; in these cases, mechanisms were categorized as such.

1.3 Results

Study selection

Figure 1 summarizes the study selection. The initial search yielded 1,404 articles across three databases and 389 duplicates were removed. Next, 1,015 titles and abstracts were screened, and 937 additional records were removed as these were deemed ineligible. The full text of the remaining 78 records were sought for retrieval; of these, three were unavailable. The full text was assessed on the remaining 75 articles, where 44 were excluded due to failure to disaggregate by the domains under study (n=28) or failure to provide the distribution of depression or related symptoms (n=16). Eight additional records were identified from reference lists. Based on eligibility criteria, 39 studies were included in the systematic review.

Characteristics of studies included

Table 1 provides characteristics of studies included in the systematic review. Of the 39 studies included in the systematic review, 32 used adult samples and 7 used youth samples. Additionally, 30 drew from national samples of the general (non-institutionalized) US population

(24 adults, six youth). Among adults, these national surveys were the first wave of the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), the National Health Interview Survey (NHIS), mothers interviewed in the Early Childhood Longitudinal Study – Birth Cohort (ECLS-B), and the Collaborative Psychiatric Epidemiology Surveys (CPES). The CPES combined data from three sister studies conducted during the same period (2001-2003): the National Comorbidity Survey-Replication (NCS-R), the National Latino and Asian American Study (NLAAS), and the National Survey of American Life (NSAL). The NSAL is the largest study to date of Black mental health and the majority of studies on adults (19 out of 32, including those labeled as CPES) drew from it. Estimates of the US Black population derived from the CPES combined NSAL and NCS-R data. Among youth, all but one study was nationally representative (6 out of 7); these national surveys were the National Longitudinal Study of Adolescent to Adult Health (Add Health), the adolescent supplement of the NCS-R (NCS-A), and the adolescent supplement of the NSAL (NSAL-A).

Of the 32 studies using adult samples, 15 used fully structured interviews to measure Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) outcomes; 11 included a measure of MDD alone (lifetime or past-year recall), two included MDD in a measure of mood disorders broadly (lifetime recall), and two used only a measure of major depressive episode (MDE) or dysthymia (lifetime or past-year recall). All studies using DSM-IV criteria drew from either the NESARC or the CPES (including NSAL studies). The remaining 21 studies on adults used instruments that captured self-reported depressive symptoms or psychological distress, such as the Center for Epidemiologic Studies Depression Scale (CES-D), the Patient Health Questionnaire-2 (PHQ-2), and the Kessler Psychological Distress Scale (K6). Of the seven studies using youth samples, three used structured interviews to measure lifetime or past-year

DSM-IV MDD. Of these three, two measured MDD alone and used the NSAL-A, while one included MDD in a measure of mood disorders broadly using the NCS-A. The remaining four studies used instruments that captured depressive symptoms (e.g., CES-D, Mood and Feelings Questionnaire).

Among studies estimating diagnostic measures of depression, these data were collected from surveys conducted between 2001-2003. Among studies estimating levels of self-reported depressive symptoms, these corresponding studies took place between 2001-2015. Lastly, studies estimating psychological distress collected data between 1998-2018.

Subgroups identified

Subgroups identified in studies using adult samples were defined by US nativity alone (US-born, foreign-born; n=8), region of birth (e.g., US-born, Caribbean-born, African-born; n=3), or age at immigration (US-born, immigrated at younger than 13 years, immigrated at 13 years or older; n=1), and most studies characterized subgroups by Caribbean origin (e.g., Caribbean, non-Caribbean; n=20), with some additionally disaggregating Caribbeans by nativity (foreign-born Caribbean, US-born Caribbean, non-Caribbean). All studies disaggregating by Caribbean origin drew from the NSAL (including using the broader CPES) or NESARC.

Regarding identifying subgroups by Caribbean origin, several studies categorized Black non-Caribbeans as “African American.” One consequence of such categorization is that Black immigrants who arrived in the US more recently (i.e., during the 20th and 21st centuries) and who are not Caribbean origin would thus be categorized as “African American.” Likewise, the second- (and third-, to an extent) generation of these immigrants would also be incorrectly labeled as “African American.” It should be noted that, as an ethnic group with several

generations in the US, African Americans are a non-immigrant ethnic group. And while Caribbeans are the largest Black immigrant ethnic group, collapsing all non-Caribbeans as “African American” renders all other Black immigrant groups (e.g., of African origin) invisible. To avoid further imprecision, the present review avoids “African American” in favor of “non-Caribbean” in referencing studies that dichotomize Caribbean origin.

Among youth samples, these subgroups identified were defined by immigrant generation (first, second, third+; n=2), parent nativity (at least one foreign-born parent, two US-born parents n=1), and Caribbean origin (n=3). Of the three studies disaggregating by Caribbean origin, all drew from the NSAL-A, and one further disaggregated by parent nativity (non-Caribbean, Caribbean with at least one foreign-born parent, Caribbean with two US-born parents).

Depression and related symptoms among Black adults

The following results pertain to the 32 included studies using samples of Black adults (Table 2).

Nativity

In all eight studies where subgroups were defined by US nativity alone,⁷⁰⁻⁷⁷ instruments used captured symptoms related to diagnostic depression. Additionally, two nationally representative studies were captured (ELCS-B, NHIS). Three studies indicated that symptoms were higher in the US-born compared with the foreign-born,⁷⁰⁻⁷² here, both studies using national datasets were captured as well as a third study drawing from a random sample of enrollees in Minnesota health care programs. Four studies indicated that estimates were roughly equivalent between the two groups,⁷³⁻⁷⁶ and one indicated symptoms were lower among the US-born.⁷⁷ In

Zick et al. (2019), the authors found that among Black women in their study, African-born women had a higher mean level of depressive symptoms relative to US-born women.

Region of birth

Three studies defined subgroups by region of birth;⁷⁸⁻⁸⁰ two used community-based samples and assessed probable depression using depressive symptom scales,^{78,79} while the other study assessed psychological distress in a national sample through the NHIS.⁸⁰ In two studies, regions of birth were characterized as follows: the US, Africa, and the Caribbean.^{78,79} One study used the following five regions of birth: US, Mexico/Central America/Caribbean, South America, Europe, and Africa.⁸⁰ While two found evidence of the US-born having higher levels of depression-related symptoms than foreign-born, all additionally displayed heterogeneity within the foreign-born population. Using a community-based sample of low-income mothers Miranda et al (2005) found that while the prevalence of probable depression was highest among US-born mothers (10.5%), Caribbean-born mothers (4.8%) had a higher prevalence than African-born mothers (3.9%). Consistent with these foreign nativity patterns, Elo et al. (2010) found Caribbean-born women had a higher prevalence of probable depression compared to African-born women (27.4% vs 22.6%) in their study using a community sample of pregnant women, yet US-born women had the lowest prevalence (21.7%). Finally, Marquez-Velarde et al. (2021) used 19 years of data from the NHIS to disaggregate Black foreign-born adults into regions of origin around the globe. The authors found considerable heterogeneity of moderate to severe depression across subgroups —Black European immigrants had the highest prevalence (15.6%) followed by Black Mexicans/Central Americans/Caribbean immigrants (14.9%), US-born Black Americans (14.3%), Black African immigrants (13.2%) and Black South American

immigrants (9.1%). In adjusted models where the US-born was the referent group, only Black European immigrants displayed a higher (non-significant at $p < 0.05$) odds of moderate/severe distress (OR=1.034); all other immigrant groups displayed a significantly lower odds of moderate/severe distress.

Age at immigration

One study defined subgroups by age at immigration.⁸¹ Here, authors used NESARC data and measured the prevalence of any lifetime mood disorder (MDD, dysthymia, or bipolar disorder) and found this was highest among the US-born (16.28%), followed by those who immigrated at < 13 years old (12.67%) and lowest among those who immigrated at ≥ 13 years old (7.72%); in models adjusted for age and sex there was no difference between those who immigrated at < 13 years old compared to the US-born (OR=0.98; $p \geq 0.05$) and there was a substantially lower odds of lifetime mood disorder for those who immigrated at ≥ 13 years old compared to the US-born (OR=0.43; $p < 0.05$).

Caribbean ethnic origin

All 19 studies drawing from the NSAL (including three drawing from the broader CPES), defined subgroups by Caribbean ethnic origin, either alone or in combination with nativity.^{19,20,24,82-97} In addition, one NESARC study defined subgroups by Caribbean origin.⁹⁸ Comparisons between Caribbeans versus non-Caribbeans yielded mixed findings. Twelve studies disaggregated Black respondents into subgroups by Caribbean ethnic origin alone (i.e., Caribbean vs non-Caribbean).^{83,84,87-89,91,93-98} Of these, eight used DSM-IV criteria depression measures and yielded mixed findings.^{83,84,89,94-98} Specifically, four studies using NSAL data

found Caribbeans had either a slightly higher (yet non-statistically significant) prevalence of depression relative to non-Caribbeans or estimates roughly equivalent.^{83,84,94,95} However, using NSARC data, Gibbs et al. (2013) found lifetime MDD was significantly lower among Caribbeans relative to non-Caribbeans (4.27% vs 9.10%; adjusted OR=0.57). Lastly, three studies drawing from a subset of older adults in the NSAL and CPES (age ≥ 50 , ≥ 55 , and ≥ 60 respectively) found Caribbeans had a higher prevalence of MDD relative to non-Caribbeans, and these results were robust to adjustment.^{89,96,97} In Woodward et al. (2012), using the CPES, the authors found older Caribbeans had over three times the odds of MDD or dysthymia compared to older non-Caribbean Black adults (OR=3.17). The other four studies used depression measures capturing symptoms, and each found levels were slightly lower among Caribbeans relative to non-Caribbeans.^{87,88,91,93}

Ten studies additionally disaggregated participants by nativity (including two previously mentioned).^{19,20,24,82,85,86,89,90,92,95} All used NSAL data (including two that used the broader CPES data) and all but one used the following Caribbean ethnicity and nativity categorizations: US-born Caribbean, foreign-born Caribbean, and non-Caribbean (one study additionally disaggregated non-Caribbeans by nativity). In all studies that used diagnostic measures of depression, relative to non-Caribbeans, US-born Caribbeans had a substantially higher prevalence of depression and foreign-born Caribbeans had a lower prevalence; patterns were similar for depression-related symptoms. For example, while Williams et al. (2007) found minimal differences between Caribbeans and non-Caribbeans (12.9% vs 10.4%), disaggregating by ethnicity alone obscured the heightened prevalence among US-born (19.8%) and the lowered prevalence among foreign-born Caribbeans (8.9%). Likewise for depressive symptoms and psychological distress, these studies also found US-born Caribbeans had the highest levels,

followed by non-Caribbeans, and foreign-born Caribbeans had the lowest levels.^{19,20} Finally, Brewton-Tiayon (2015) et al. assessed patterns in lifetime MDD, lifetime MDE, and depressive symptoms in the past seven days by Caribbean ethnicity and nativity across age groups. The authors here found differences in age-related trends across subgroups that were largely consistent across each depression measure; first, US-born Caribbeans displayed the most consistent pattern of heightened prevalence and second, foreign-born Caribbeans displayed an increased prevalence at older ages.⁸⁵

Potential mechanisms among adults

In 18 (out of 32) studies using samples of Black adults, authors clearly proposed mechanisms underlying within-racial group differences in either the *prevalence of* or *processes causing* depression and its levels of related symptoms across subgroups. Regarding mechanisms underlying prevalence variation, these were either artefactual or etiologic. All proposed mechanisms underlying within-racial group differences in processes causing depression or its related symptoms were etiologic.

Artefactual explanations

Measurement error. Four studies attributed findings to measurement error, with two primary reasons cited.^{71,77,97,98} First, although the US-born had higher levels of depressive symptoms in Henning-Smith et al. (2013) and lower levels in Zick et al. (2019) compared to foreign-born participants, both studies cited the possibility of cultural differences in the way questions were answered. Relatedly, Woodward et al. (2012) attributed the lower prevalence of MDD among older non-Caribbeans (relative to older Caribbeans) to these individuals being more

likely to minimize symptoms of depression and thus answer questions differently. Second, Gibbs et al. (2013) produced estimates of the prevalence of MDD by Caribbean origin using NESARC data, which differed from NSAL data that were collected during the same period. The authors attributed these differences to either the NESARC underestimating or the NSAL overestimating the prevalence of MDD among Caribbean and non-Caribbean Black people in the US.

Healthy immigrant effect. While not a mechanism itself, the “healthy immigrant effect” refers to the phenomenon that upon arrival to the US, immigrants have better health profiles than their US-born counterparts.²² The “healthy immigrant effect” may be an artifact of selection bias, as research explicating this phenomenon has attributed it to selective migration.⁹⁹⁻¹⁰¹ That is, people who are in better health appear more likely to emigrate, thus leading to the appearance of a health advantage among immigrants. The present review identified four studies where authors attributed the pattern of lower levels of depression or related symptoms among the foreign-born (relative to the US-born) to the “healthy immigrant effect.”^{24,70,90,91} One study measured psychological distress and disaggregated participants by nativity,⁷⁰ two measured lifetime MDD and disaggregated by Caribbean origin and nativity,^{24,90} and the fourth study measured depressive symptoms and disaggregated by Caribbean ethnicity alone.⁹¹ In the latter study which disaggregated by Caribbean ethnicity alone, authors characterized depressive symptoms using a two-class latent profile model — a high symptoms class, and a low symptoms class. Here, more recent foreign-born Caribbeans (those who have resided in the US for 10 years or less) were more likely to be in the low symptoms class than all US-born respondents, as well as all other Caribbeans.⁹¹

Etiologic explanations

While the above studies focused on artefactual explanations for within-racial heterogeneity, the following studies provided etiologic explanations for heterogeneity.

Acculturation and childhood socialization in the US. There is evidence that suggests the initial immigrant health advantage declines over time, converging to levels similar to US-born counterparts due (at least in part) to acculturation, which refers to immigrants adopting American values and behaviors with — in this case — important health implications.²² Although acculturation scales exist, studies in the health research literature often approximate it through variables such as nativity, generational status, and length of stay in the US.¹⁰² In the present review, two studies, both using NSAL data, attributed findings to acculturation *a posteriori* — in Brewton-Tiayon (2015) pertaining to the unique pattern of higher prevalence of MDD, MDE, and depressive symptoms among foreign-born Caribbeans at older ages relative to younger ages; and in Doyle et al. (2012) regarding the higher prevalence of MDD among US-born Caribbean fathers relative to foreign-born Caribbean fathers.

Consistent with the idea that acculturation among immigrants is detrimental for mental health, results from a third study using NESARC data found the lower prevalence of lifetime mood disorders among immigrants (compared to the US-born) was limited to those who immigrated at 13 years of age or older.⁸¹ As hypothesized (*a priori*), the authors found no significant difference in the prevalence of lifetime mood disorder between immigrants who arrived at younger than 13 years of age and the US-born, yet a lower prevalence among immigrants who arrived at 13 years of age or older, and attributed findings to detrimental mental health effects of childhood socialization in the US. Of note, this finding of variation within

immigrants was not limited to Black participants but was also observed for those of Mexican origin and Eastern European origin in the same study, as well as Asian Americans in a separate study conducted by the same first author.^{81,103} However, there was no formal evaluation of childhood socialization in the US beyond a descriptive assessment of subgroup differences.

Religiosity. Only one study invoked religiosity as a potential mechanism in explaining within-racial differences *a posteriori*.⁹⁷ Specifically, Woodward et al. (2012) postulated that the lower prevalence of mood disorders among non-Caribbeans in their study of older adults (aged ≥ 50 years) in the CPES may be due to increased religious involvement, which may be linked to increased levels of social support, which then may be linked to lower levels of mood disorders.

Racial context hypothesis. In one study, the authors disaggregated Black adult participants in the US by region of birth and found variation in levels of psychological distress, consistent with their *a priori* hypothesis.⁸⁰ The purpose of the study was to test the “racial context hypothesis,” which suggests those from contexts where Black people are a racial minority would have worse mental health outcomes relative to those from contexts where Black people are a racial majority, in part due to higher exposures to racism and its detrimental effects on mental health.¹⁰⁴ Indeed, Black European immigrants were the only foreign-born group without a significantly lower odds of moderate or severe distress compared to the US-born.⁸⁰ The authors here, however, did not evaluate “racial context hypothesis” beyond a descriptive assessment.

Subgroup variation in causal mechanisms. Six studies found evidence of subgroup variation in *a priori* hypothesized causal mechanisms leading to depression via effect modification, and each used NSAL data.^{20,82,87,88,93,94} In four of these studies, authors further postulated that ethnic differences in childhood socialization between Black participants with and without recent immigrant origins underlie the significant group differences in main effects estimates.^{82,88,93,94} For instance, while Ida and Christie-Mizell (2012) found no difference in levels of depressive symptoms between Caribbean and non-Caribbeans, the authors found the protective function of racial identity on depressive symptoms was markedly weaker for Caribbeans than for non-Caribbeans, attributing these findings to differences in racial socialization. Similarly, Molina and James (2016) found no substantive difference between Caribbeans and non-Caribbeans in past-year MDD yet “internalized racism,” a measure that other NSAL studies have operationalized in its inverse as a racial identity measure indicating the extent to which one positively evaluates Black people in the US,^{88,105,106} was associated with a lower prevalence of MDD for Caribbeans alone. In other words, as the inverse of this internalized racism measure indicates racial identity, results suggest higher levels of racial identity are associated with a higher prevalence of MDD for Caribbeans alone.

Depression and related symptoms among Black youth

The following results pertain to the seven studies using samples of Black youth (Table 3).

Immigrant generation & parent nativity

Two studies defined groups by immigrant generation similarly found third+ generation youth had a heightened prevalence of depression or levels of related symptoms, relative to first-

generation youth.^{107,108} Using NCS-R data, Georgiades et al. (2018) found the prevalence of any lifetime mood or anxiety disorder was heightened for third+ generation youth (36.76%) and second-generation youth (37.16%), relative to first-generation youth (30.71%). And Harker (2001) found, using the second wave of Add Health data, differences depressive symptoms such that levels were highest among third+ generation youth, followed by similar levels between second-generation and first-generation youth. Yet in a third study using parent nativity, the authors drew from a sample of sixth-grade students in Seattle and found no substantive difference in levels of depressive symptoms between those with at least one foreign-born parent (i.e., first- and second-generation youth combined) and those with two US-born parents (i.e., third+ generation youth).¹⁰⁹

Caribbean ethnic origin

As with studies using NSAL data among adults, all four drawing from the NSAL-A used subgroups defined by Caribbean origin either alone^{27,110,111} or with parent nativity.²⁸ In studies using Caribbean origin alone, all three found similar results under their respective measures — there was no substantive difference between Caribbean and non-Caribbean youth in lifetime or past-year MDD^{110,111} nor depressive symptoms in the past seven days.²⁷ In Smith (2020), Caribbean origin was additionally disaggregated by parent nativity; the author found statistically significant group differences in levels of depressive symptoms such that non-Caribbeans had the highest mean, followed by Caribbean youth with two US-born parents, and the lowest mean was among Caribbean youth with at least one foreign-born parent.

Potential mechanisms among youth

In three of the seven studies on Black youth, proposed mechanisms were each related to subgroup differences in *a priori* hypothesized causal mechanisms leading to depression through analyses of effect modification, each using NSAL data.^{27,28,111} In two, the authors examined whether subgroup membership modified the relationship between perceived discrimination and depressive symptoms. Seaton et al. (2008) found this relationship was stronger for Caribbean youth compared to non-Caribbean youth. Smith (2020) expanded upon this work by additionally disaggregating by parent nativity. The author found that although Caribbean youth with foreign-born parents had the lowest levels of depressive symptoms (compared to both Caribbean youth with US-born parents and non-Caribbean youth), the relationship between perceived discrimination and depressive symptoms was strongest for this group. In both studies, authors further attribute their findings of effect modification to ethnic differences in racial socialization.

1.4 Discussion

This systematic review drew from data sources ranging from nationally representative studies to community-based samples and found substantial variation in the prevalence of depression and its related symptoms within the US Black population. This review also found variation in how researchers disaggregated the US Black population. Among studies disaggregating by nativity, the pattern of heightened levels of depression or related symptoms among the US-born relative to the foreign-born was most consistent in nationally representative samples. Studies disaggregating by region of birth found foreign-born African immigrants consistently had the lowest levels of depression-related symptoms relative to foreign-born Black immigrants from other regions. This review also found one study suggesting the apparent protective function of being foreign-born is limited to those who immigrated at 13 years or older.

Additionally, most studies drew from the NSAL and thus, Caribbean ethnic origin was the most used disaggregation domain. NSAL data revealed that comparisons between Caribbeans and non-Caribbeans alone obscured both the heightened prevalence of depression among US-born Caribbeans and the lower prevalence among foreign-born Caribbeans, which was consistent across both diagnostic measures and measures of related symptoms.

Through categorizing studies by domains of disaggregation, this study was also able to identify Black subgroups that have received no attention. Namely, no study among adults disaggregated by immigrant generation, so the patterning of depression among second-generation immigrant adults is unclear. The closest approximation identified was US-born Caribbeans from NSAL studies, who are comprised of both second- and third-generation immigrants. To determine whether results among US-born Caribbeans apply to second-generation immigrants broadly, future research is needed.

The NSAL is the only nationally representative epidemiologic survey on psychiatric disorders that is designed to disaggregate the Black US population.¹¹² Though it was conducted 20 years ago from 2001-2003, the NSAL remains the largest study of Black mental health. While NSAL data can be disaggregated by nativity (foreign-born, US-born) the survey focused on two ethnic subgroupings of the Black population — those of recent Caribbean origin (first-, second-, and third-generation immigrants) and those without, the latter being categorized as African American. NSAL data are limited, in that they are unable to address whether patterns within Caribbeans extend to other Black immigrant ethnicities (e.g., African origin, Latin American origin, etc.). And since the NSAL has not been replicated, it is unclear whether these patterns hold using more recent data collected at a similar scale. Similarly, the other data source captured in this review that estimated diagnostic depression, wave 1 of the NESARC, was conducted from

2001-2002. While the most recent version of NESARC was conducted from 2012 to 2013, no study using these data were captured. Given recent increases in the Black immigrant populations, collecting more recent data is an important area of future research.

Also of note, no study identified in this review further disaggregated by Hispanic/Latinx ethnicity, thus, it is possible that there is further variation, particularly in MDD within Black Americans of Caribbean origin. One quarter of the US Hispanic/Latinx population identifies as “Afro-Latino, Afro-Caribbean or of African descent with roots in Latin America.”¹¹³ Consistent with this statistic, about a quarter of the total population of Latin America are people of African descent, with the largest numbers residing in Brazil, followed by Venezuela, and the Dominican Republic.¹¹⁴ To illustrate the possibility of additional variation, studies using NSAL data in the present review included Black Hispanic/Latinx Caribbeans, whereas Gibbs et al. (2013) used NESARC data collected during the same period and excluded Black Hispanic/Latinx Caribbeans. If MDD is heightened among Black Hispanic/Latinx Caribbeans, this may explain discrepant findings by Caribbean ethnicity between NSAL and NESARC data; namely, that relative to non-Caribbeans, Caribbeans in the NSAL had a similar prevalence of MDD and Caribbeans in the NESARC had a lower prevalence of MDD. Indeed, one NSAL study suggests at least among Black Caribbean women, those originating from Spanish-speaking countries had a heightened odds of any mood disorder relative to those originating from English-speaking countries.¹¹⁵

This systematic review also identified proposed mechanisms underlying within-racial differences, including both artefactual and etiological explanations. Nativity differences have been attributed to artefactual explanations — the healthy immigrant effect (i.e., selective migration) and measurement error. Regarding differences across region of birth, only one study tested the “racial context hypothesis,” whereby those born in a context where Black is a racial

minority would have the highest levels of depressive symptoms due to increased exposures to racism and its detrimental effects on mental health. Differences across age at immigration were attributed to childhood socialization in the US functioning as a causal factor for depression. And among studies disaggregating by Caribbean origin, some proposed and evaluated subgroup differences in causal mechanisms related to differences in racial socialization. Relatedly, while studies drawing from youth samples found no substantial variation, some did evaluate subgroup differences in causal mechanisms, and attributed these to differences in racial socialization. Notably, etiologic explanations point to the notion that there exists within-group heterogeneity in factors related to racialization, or the process of how subgroups under study are identified as Black, ultimately leading to variation in depression and related symptoms.

By summarizing potential mechanisms, this review reveals at least two promising areas of future research accounting for ethnic differences in outcomes under study. The first concerns the importance of racial context toward explaining variation by region of origin. Under this mechanism, selective migration may be at play where healthier immigrants are more likely to migrate from majority Black countries. Alternatively, variation may be explained by the long-term negative mental health effects of racism and discrimination due to being born and raised as a member of a racialized minority.²⁹

A second, related promising area of future work involves exploring variation in the relationship between racial socialization experiences on depression and its related outcomes. Numerous studies support the notion that parental racial socialization messages lead to the development of strong racial identities among children of color, as evidenced by a recent literature review.²⁹ Among Black respondents, other studies find support for racial identity leading either directly to lower levels of poor mental health outcomes or functioning as a buffer

against the negative effects of discrimination on poor mental health outcomes.³⁰⁻³⁵ The present review found that while studies of youth using NSAL data found no substantive differences in depression and its related symptoms by Caribbean ethnic origin, these data showed the relationship between discrimination and depression (or its related symptoms) was stronger for Caribbeans. Likewise, among adults in the NSAL, studies revealed variation in the effects of racial identity such that racial identity is either not protective or not as protective for Caribbeans relative to non-Caribbeans. Consistent with the idea that racial identity may have varying effects on depression, an increasing body of ethnographic research suggests racial socialization experiences during adolescence relevant to mental health, such as racial identity formation, do differ between Black youth with and without foreign-born parents.³⁶⁻⁴⁶ Additional research is needed to clarify how such differences in racial socialization and racial identity formation impact depression within the US Black population. In addition, future research addressing whether racial identity functions as a protective factor for Black Americans without recent immigrant origins may help elucidate the apparent paradoxical Black-white racial patterns in the prevalence of MDD.

Immigration is an important sociocultural factor associated with variations in depression and related symptoms within the US Black population. This review contributes to the scant literature recognizing Black ethnic diversity, providing additional insight into patterns of depression and its related symptoms within the US Black population, as well as mechanisms that may explain subgroup differences. Though data are limited, extant evidence suggests substantial heterogeneity across immigration- and ethnicity-related domains of disaggregation. Given the heterogeneity identified in this review, it is important to determine whether these patterns are consistent using more recent data. Future surveillance efforts that fail to disaggregate Black

respondents have the potential to conceal important differences in depression prevalence and hinder progress toward understanding mechanisms unique to the Black population. Furthermore, results from this review suggest treatment efforts may benefit from more tailored approaches that acknowledge the unique lived experiences associated with the intersection of race and immigration within the Black population.

1.5 Tables and Figures

Table 1.1: Characteristics of included studies in systematic review

| | Total n=39 studies | Adults n=32 studies | Youth n=7 studies |
|--|------------------------------|-------------------------------|-----------------------------|
| Data sources | | | |
| Nationally representative | 30 | 24 | 6 |
| CPES/NSAL | 19 | 19 | 0 |
| ECLS-B | 1 | 1 | 0 |
| NESARC | 2 | 2 | 0 |
| NHIS | 2 | 2 | 0 |
| Add Health | 1 | 0 | 1 |
| NCS-A | 1 | 0 | 1 |
| NSAL-A | 4 | 0 | 4 |
| Non-nationally representative | 9 | 8 | 1 |
| Depression measures | | | |
| Diagnostic depression | 18 | 15 | 3 |
| MDD ¹ | 13 | 11 | 2 |
| MDE or dysthymia | 2 | 2 | 0 |
| Mood disorder ² | 3 | 2 | 1 |
| Self-reported symptoms only ³ | 21 | 17 | 4 |
| Subgroups | | | |
| US Nativity | 8 | 8 | 0 |
| Region of birth | 3 | 3 | 0 |
| Age at immigration | 1 | 1 | 0 |
| Immigrant generation | 2 | 0 | 2 |
| Parent nativity | 1 | 0 | 1 |
| Caribbean ethnic origin ⁴ | 24 | 20 | 4 |

CPES = Collaborative Psychiatric Epidemiological Studies; ECLS-B: Early Childhood Longitudinal Survey – Birth Cohort; NCS-A: National Comorbidity Survey-Adolescent supplement; NHIS = National Health Interview Survey; NSAL = National Survey of American Life; NSAL-A = National Survey of American Life-Adolescent supplement

MDD = major depressive disorder; MDE = major depressive episode

1. Refers to all studies with a separate measure of MDD. Studies that measured MDD and other depression measures separately are included.
2. Refers to a broader measure that includes MDD.
3. Refers to measures of depressive symptoms or psychological distress
4. Caribbean ethnic origin either alone or in combination with nativity

Table 1.2: Summary of studies included in systematic review, Black adults

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|------------------------------------|--|--------------------------------|--------------|-----------------------------|--|--|--|---|---------------------------------------|----------------------------------|
| Domain: Nativity (n=8) | | | | | | | | | | |
| Dey (2006) ⁷⁰ | NHIS (1998-2003) | SPD | Past 30 days | K6 | ≥ 13 | US-born (NR) Foreign-born (NR) | White, Latinx, Asian; each were additionally disaggregated by nativity | <i>Prevalence</i> US-born: 3.3% Foreign-born: 1.9% | Healthy immigrant effect ³ | <i>a posteriori</i> |
| Doe (2017) ⁷³ | Community sample of mothers, NYC (2015) | Probable postpartum depression | NR | EPDS | ≥ 9 | US-born (NR) Foreign-born (NR) | US-born Latinx, foreign-born Latinx | <i>Prevalence</i> US-born: 22.81% Foreign-born: 23.81% | None proposed | N/A |
| Donovan (2013) ⁷⁴ | Multi-site study of college students (2008-2009) | Depressive symptoms | Past 7 days | 20 items adapted from CES-D | Continuous; range: 1-100 | US-born (NR) Foreign-born (NR) <i>Total n=896</i> | Asian, Latinx, and white; each were additionally disaggregated by nativity | <i>Mean (n.s.)</i> US-born: 51.81 Foreign-born: 49.81 | None proposed | N/A |
| Henning-Smith (2013) ⁷¹ | Random sample of enrollees in Minnesota Health Care Programs (2008) | Probable depression | Past 2 weeks | PHQ-2 | ≥ 3 | US-born (242) Somali-born (288) | White | <i>Prevalence*</i> US-born: 31.6% Somali-born: 9.1% | Measurement error | <i>a posteriori</i> |
| Huang (2007) ⁷² | ELCS-B; mothers (2001-2002) | Depressive symptoms | NR | 12 items adapted from CES-D | Categorical; severe: ≥15 moderate: 10-14 mild: 5-9 no depression: 0-4 | US-born (NR) Foreign-born (NR) | White, Latinx, Asian, Pacific Islander, Native American; each were additionally disaggregated by nativity, where available | <i>Severe depression prevalence</i> US-born: 10.7% Foreign-born: 4.9% <i>Moderate depression prevalence</i> US-born: 15.2% Foreign-born: 16.9% <i>Mild depression prevalence</i> US-born: 30.6% Foreign-born: 20.7% | No clear mechanism proposed | N/A |
| Kreiger (2011) ⁷⁵ | Community sample, Boston (2003-2004) | SPD | Past 30 days | K6 | ≥ 13 | US-born (193) Foreign-born (275) | None | <i>Prevalence</i> US-born: 14.2% Foreign-born: 16.2% | None proposed | N/A |
| | | Psychological distress | Past 30 days | K6 | Continuous; range: 0-24 | “ | “ | <i>Mean</i> US-born: 6.6 Foreign-born: 8.3 | “ | “ |
| Schwarz (2012) ⁷⁶ | NYC Community Health Survey; women only (2002, 2003, 2005, 2006, 2008) | Probable depression | Past 30 days | K6 | ≥ 7 | US-born (NR) Foreign-born (NR) <i>Total n=7655</i> | White, Latinx; each were additionally disaggregated by nativity | <i>Prevalence (n.s.)</i> US-born: 24.2% Foreign-born: 23.6% <i>Adjusted OR</i> US-born: ref Foreign-born: 1.01 (n.s.) | None proposed | N/A |

Table 1.2: Summary of studies included in systematic review, Black adults (cont.)

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|--------------------------------------|---|------------------------|--------------|------------|---|---|---|---|------------------------------------|----------------------------------|
| Zick (2019) ⁷⁷ | Community sample of women, Utah (2012-2015) | Depressive symptoms | Past 2 weeks | PHQ-2 | Continuous; range: 0-6 | US-born (96) Foreign-born African (79) | Latina, Native Hawaiian/Pacific Islander, American Indian/Alaskan Native, and white | <i>Mean</i> US-born: 1.04 Foreign-born African: 2.02 | Measurement error | N/A |
| Domain: Region of birth (n=3) | | | | | | | | | | |
| Elo (2010) ⁷⁸ | Community sample of pregnant women, Philadelphia (1999-2004) | Probable depression | NR | CES-D | ≥ 23 | US-born (2816) Foreign-born African (106) Foreign-born Caribbean (179) | None | <i>Prevalence*</i> US-born: 21.7% Foreign-born: 25.6% - Foreign-born African: 22.6% - Foreign-born Caribbean: 27.4% <i>Adjusted ORs</i> US-born: ref Foreign-born African: 1.52 (n.s.) Foreign-born Caribbean: 1.61* | No clear mechanism proposed | N/A |
| Marquez-Velarde (2021) ⁸⁰ | NHIS (2000-2018) | Psychological distress | Past 30 days | K6 | Categorical; severe: ≥13 moderate: 5-12 low or no distress: 0-4 | US-born (216,538) Foreign-born Mexican/Central American/Caribbean (14,725) Foreign-born South American (1386) Foreign-born European (857) Foreign-born African (7537) | None | <i>Moderate/severe distress prevalence</i> US-born: 14.3% Foreign-born Mexican/Central American/Caribbean: 14.9% Foreign-born South American: 9.1% Foreign-born European: 15.6% Foreign-born African: 13.2% <i>Moderate/severe distress ORs</i> US-born: ref Foreign-born Mexican/Central American/Caribbean: 0.795* Foreign-born South American: 0.499* Foreign-born European: 1.034 Foreign-born African: 0.810* | Racial context hypothesis | <i>a priori</i> |
| Miranda (2005) ⁷⁹ | Community sample of low-income mothers; DC-Maryland-Virginia area (1997-2001) | Probable depression | Past month | PRIME-MD | NR | US-born (7965) Foreign-born African (913) Foreign-born Caribbean (273) | None | <i>Prevalence*</i> US-born: 10.5% Foreign-born African: 3.9% Foreign-born Caribbean: 4.8% <i>Adjusted ORs</i> US-born: ref Foreign-born African: 0.34* Foreign-born Caribbean: 0.42* | No clear mechanism proposed | N/A |

Table 1.2: Summary of studies included in systematic review, Black adults (cont.)

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|---|---------------------------------|------------------------------------|-------------|-----------------------------|-----------------------------|--|--|--|------------------------------------|----------------------------------|
| Domain: Age at immigration (n=1) | | | | | | | | | | |
| Breslau (2009) ⁸¹ | NESARC (2001-2002) | MDD, dysthymia or bipolar disorder | Lifetime | AUDADIS DSM-IV version | DSM-IV criteria | US-born (7541) Immigrated at < 13 years of age (153) Immigrated ≥13 years of age (613) | Latinx: Mexico, Cuba, Puerto Rico, Central/South America; White: Western Europe, Eastern Europe. Each were additionally disaggregated by age at immigration | <i>Prevalence*</i> US-born: 16.28% Immigrated at < 13 years of age: 12.67% Immigrated ≥13 years of age: 7.72% <i>Adjusted ORs</i> US-born: ref Immigrated at < 13 years of age: 0.98 Immigrated ≥13 years of age: 0.43* | Childhood socialization in the US | <i>a priori</i> |
| Domain: Caribbean ethnic origin (n=20) | | | | | | | | | | |
| Anglin (2014) ⁸² | NSAL (2001-2003) | MDD | Past year | WMH-CIDI | DSM-IV criteria | Non-Caribbean (3464) US-born Caribbeans (373) Foreign-born Caribbeans (1114) | None | <i>Prevalence*</i> US-born Non-Caribbean: 5.3% US-born Caribbean: 13.4% Foreign-born Caribbean: 5.6% | Subgroup variation in main effects | <i>a priori</i> |
| Assari (2014) ⁸³ | NSAL (2001-2003) | MDD | Past year | WMH-CIDI | DSM-IV criteria | Non-Caribbean (3570) Caribbean (1621) | None | <i>Prevalence</i> Non-Caribbean: 5.4% Caribbean: 7.8% | No clear mechanism proposed | N/A |
| Boyd (2011) ⁸⁴ | NSAL; mothers (2001-2003) | MDE | Lifetime | WMH-CIDI | DSM-IV criteria | Non-Caribbean (2019) Caribbean (799) | White | <i>Prevalence</i> Non-Caribbean: 14.51% Caribbean: 15.22% | No clear mechanism proposed | N/A |
| | | MDE | Past year | WMH-CIDI | DSM-IV criteria | | | <i>Prevalence</i> Non-Caribbean: 7.95% Caribbean: 6.77% | " | " |
| Brewton-Tiayon (2015) ⁸⁵ | NSAL (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | Non-Caribbean (3434) US-born Caribbeans (432) Foreign-born Caribbeans (1141) | None | US-born Caribbeans had the most consistent pattern of heightened prevalence of each measure of depression across age groups (18-34, 35-54, 55-64, 65-74, ≥75), relative to both foreign-born Caribbeans and non-Caribbeans, with exceptions at older ages (65-74, ≥75) | Acculturation | <i>a posteriori</i> |
| | | MDE | Lifetime | WMH-CIDI | DSM-IV criteria | Non-Caribbean (3433) US-born Caribbeans (432) Foreign-born Caribbeans (1141) | " | | " | |
| | | Depressive symptoms | Past 7 days | 12 items adapted from CES-D | Continuous; range: 0-36 | Non-Caribbean (3578) US-born Caribbeans (432) Foreign-born Caribbeans (1141) | " | | " | |

Table 1.2: Summary of studies included in systematic review, Black adults (cont.)

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|-------------------------------|------------------------------------|---------------------|-------------|-----------------------------|-----------------------------|---|--|--|---------------------------------------|----------------------------------|
| Doyle (2012) ⁸⁶ | NSAL; fathers (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | Non-Caribbean (1254) US-born Caribbean (175) Foreign-born Caribbean (458) | None | <i>Prevalence*</i> Non-Caribbean: 6.9% US-born Caribbean: 20.2% Foreign-born Caribbean: 8.3% | Acculturation | <i>a posteriori</i> |
| Erving (2021) ⁸⁷ | NSAL; adults ≥40 years (2001-2003) | Depressive symptoms | Past 7 days | 12 items adapted from CES-D | Continuous; range: 0-36 | Non-Caribbean (1616) Caribbean (601) | None | <i>Mean (n.s.)</i> Non-Caribbean: 6.17 Caribbean: 5.90 | Subgroup variation in main effects | <i>a priori</i> |
| Gibbs (2013) ⁹⁸ | NESARC (2001-2002) | MDD | Lifetime | AUDADIS DSM-IV version | DSM-IV criteria | Non-Caribbean (7529) Caribbean (469) | White | <i>Prevalence</i> Non-Caribbean: 9.10% Caribbean: 4.27% <i>Adjusted OR</i> Non-Caribbean: ref Caribbean: 0.57* | Measurement error | N/A |
| | | MDD | Past year | AUDADIS DSM-IV version | DSM-IV criteria | | | <i>Prevalence</i> Non-Caribbean: 4.58% Caribbean: 2.22% <i>Adjusted OR</i> Non-Caribbean: ref Caribbean: 0.58 (n.s.) | " | " |
| González (2010) ²⁴ | CPES (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | Foreign-born Caribbean (66.6%) US-born Caribbean (33.4%) <i>Caribbean total=1476</i> | Chinese, Filipino, Vietnamese, Cuban, Mexican, Puerto Rican, and white; each were additionally disaggregated by nativity | <i>Prevalence</i> Foreign-born Caribbean: 9.1% US-born Caribbean: 24.1% Foreign-born Non-Caribbean: 13.4% US-born non-Caribbean: 13.1% | Healthy immigrant effect ³ | N/A |
| | | MDD | Past year | WMH-CIDI | DSM-IV criteria | Foreign-born non-Caribbean (2.3%) US-born non-Caribbean (97.1%) <i>non-Caribbean total=4249</i> | | <i>Prevalence</i> Foreign-born Caribbean: 4.8% US-born Caribbean: 13.4% Foreign-born Non-Caribbean: 8.0% US-born non-Caribbean: 7.2% | " | " |

Table 1.2: Summary of studies included in systematic review, Black adults (cont.)

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|-------------------------------|------------------------------------|----------------------------------|--------------|-----------------------------|-----------------------------|--|--|---|---------------------------------------|----------------------------------|
| Griffith (2011) ¹⁹ | NSAL (2001-2003) | Depressive symptoms ⁴ | Past 7 days | 12 items adapted from CES-D | Continuous; range: 0-36 | Non-Caribbean (3570) US-born Caribbean (440) Foreign-born Caribbean (1166) | None | <i>Mean</i> non-Caribbeans: 6.70 US-born Caribbeans: 7.45 Foreign-born Caribbeans: 5.81 | None proposed | N/A |
| Head (2017) ²⁰ | NSAL (2001-2003) | Psychological distress | Past 30 days | K6 | Continuous; range: 0-4 | Non-Caribbean (3414) US-born Caribbean (446) Foreign-born Caribbean (1114) | None | <i>Mean*</i> non-Caribbeans: 0.521 US-born Caribbeans: 0.635 Foreign-born Caribbeans: 0.417 | Subgroup variation in main effects | <i>a priori</i> |
| Ida (2012) ⁸⁸ | NSAL (2001-2003) | Depressive symptoms | Past 7 days | 12 items adapted from CES-D | Continuous; range: 0-36 | Non-Caribbean (2953) Caribbean (1140) | None | <i>Mean*</i> Non-Caribbean: 6.60 Caribbean: 6.25 | Subgroup variation in main effects | <i>a priori</i> |
| Jimenez (2010) ⁸⁹ | CPES; adults ≥60 years (2001-2003) | MDE, dysthymia | Lifetime | WMH-CIDI | DSM-IV criteria | non-Caribbean (671) Caribbean (163) | White, Latinx, Asian; each were additionally disaggregated by nativity | <i>Prevalence</i> Non-Caribbean: 5.4% Caribbean: 8.1% | None proposed | N/A |
| | | MDE, dysthymia | Past year | WMH-CIDI | DSM-IV criteria | <i>within Caribbean ethnicity</i> US-born Caribbean (34) Foreign-born Caribbean (159) | | <i>Prevalence</i> Non-Caribbean: 2.3% Caribbean: 4.6% | | |
| Jones (2020) ⁹⁰ | NSAL; women only (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | <i>Nativity and Caribbean ethnicity</i> US-born non-Caribbean (2242) US-born Caribbean (264) Foreign-born Caribbean (705) | None | <i>Prevalence*</i> US-born non-Caribbean: 14.4% US-born Caribbean: 22.6% Foreign-born Caribbean: 11.2% | Healthy immigrant effect ³ | <i>a posteriori</i> |
| | | MDD | Past year | WMH-CIDI | DSM-IV criteria | | | <i>Prevalence</i> US-born Non-Caribbean: 8.2% US-born Caribbean: 7.5% Foreign-born Caribbean: 7.0% | | |
| Lincoln (2007) ⁹¹ | NSAL (2001-2003) | Depressive symptoms | Past 7 days | 12 items adapted from CES-D | Continuous; range: 0-36 | Non-Caribbean (3361) Caribbean (1554) | None | <i>Mean</i> Non-Caribbean: 6.78 Caribbean: 6.07 | Healthy immigrant effect ³ | <i>a posteriori</i> |

Table 1.2: Summary of studies included in systematic review, Black adults (cont.)

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|-------------------------------|------------------------------------|---------------------|-------------|-----------------------------|-----------------------------|---|----------------------|--|------------------------------------|----------------------------------|
| Mays (2018) ⁹² | NSAL; men only (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | US-born non-Caribbean (1222) US-born Caribbean (176) Foreign-born Caribbean (461) | None | <i>Prevalence*</i> US-born Non-Caribbean: 8.8% US-born Caribbean: 21.1% Foreign-born Caribbean: 8.9% | None proposed | N/A |
| | | MDD | Past year | WMH-CIDI | DSM-IV criteria | | | <i>Prevalence*</i> US-born Non-Caribbean: 4.6% US-born Caribbean: 16.4% Foreign-born Caribbean: 4.9% | " | " |
| Mereish (2016) ⁹³ | NSAL; men (2001-2003) | Depressive symptoms | Past 7 days | 12 items adapted from CES-D | Continuous; range: 0-3 | Non-Caribbean (1201) Caribbean (545) | None | <i>Mean</i> Non-Caribbean: 0.51 Caribbean: 0.49 | Subgroup variation in main effects | <i>a priori</i> |
| Molina (2016) ⁹⁴ | NSAL (2001-2003) | MDD | Past year | WMH-CIDI | DSM-IV criteria | Non-Caribbean (3570) Caribbean (1418) | None | <i>Prevalence (n.s.)</i> Non-Caribbean: 6.76% Caribbean: 6.70% <i>Adjusted OR</i> Non-Caribbean: ref Caribbean: 1.58 (n.s.) | Subgroup variation in main effects | <i>a priori</i> |
| Williams (2007) ⁹⁵ | NSAL (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | Non-Caribbean (3570) Caribbean (1621) | White | <i>Prevalence (n.s.)</i> Non-Caribbean: 10.4% Caribbean: 12.9% <i>within Caribbean ethnicity*</i> US-born Caribbean: 19.8% Foreign-born Caribbean: 8.9% | No clear mechanism proposed | N/A |
| | | MDD | Past year | WMH-CIDI | DSM-IV criteria | <i>within Caribbean ethnicity</i> US-born Caribbean (440) Foreign-born Caribbean (1166) | | <i>Prevalence (n.s.)</i> Non-Caribbean: 5.9% Caribbean: 7.2% <i>within Caribbean ethnicity (n.s.)</i> US-born Caribbean: 10.9% Foreign-born Caribbean: 2.0% | " | " |
| Woodward (2012) ⁹⁷ | CPES; adults ≥55 years (2001-2003) | MDD or dysthymia | Lifetime | WMH-CIDI | DSM-IV criteria | Non-Caribbeans ≥55 years (780) Caribbeans ≥55 years (262) | White, Latinx, Asian | <i>Prevalence</i> non-Caribbean ≥55 years: 5.4% Caribbean ≥55 years: 11.2% <i>Adjusted OR</i> non-Caribbean ≥55 years: ref Caribbean ≥55 years: 3.17* | Measurement error, Religiosity | <i>a posteriori</i> |

Table 1.2: Summary of studies included in systematic review, Black adults (cont.)

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|-------------------------------|------------------------------------|--------------------|-----------|------------|-----------------------------|---|---------------------|--|------------------------------------|----------------------------------|
| Woodward (2013) ⁹⁶ | NSAL; adults ≥50 years (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | Non-Caribbean ≥50 years (1074) Caribbean ≥50 years (415) | White | <i>Prevalence*</i> non-Caribbean ≥50 years: 8.8% Caribbean ≥50 years: 11.2% | No clear mechanism proposed | N/A |
| | | MDD | Past year | WMH-CIDI | DSM-IV criteria | | | <i>Prevalence (n.s.)</i> non-Caribbean ≥50 years: 4.0% Caribbean ≥50 years: 8.1% | " | " |

CPES = Collaborative Psychiatric Epidemiological Studies; ECLS-B: Early Childhood Longitudinal Survey – Birth Cohort; NHIS = National Health Interview Survey; NSAL = National Survey of American Life
MDD = major depressive disorder; MDE = major depressive episode; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, fourth edition; WMH-CIDI = World Mental Health version of the Composite International Diagnostic Interview
NR = Not reported

* Statistically significant (p<0.05); n.s. = non-significant

1. Proposed mechanism(s) refers to within-Black racial differences

2. Mechanism basis reasoning to whether the listed mechanism was a clear hypothesis in the given study raised in the introduction section (*a priori*) or if it was raised in the discussion section alone

3. The healthy immigrant effect is not technically a mechanism, but rather an observed pattern of better health outcomes among the foreign-born compared to the US-born

4. Depression measure was treated as a covariate, rather than as an outcome

Table 1.3: Summary of studies included in systematic review, Black youth

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|---|---|---------------------------------------|---|--|---|--|--|--|------------------------------------|----------------------------------|
| Domain: Immigrant generation (n=2) | | | | | | | | | | |
| Georgiades (2018) ¹⁰⁷ | NCS-A (2001-2004) | Mood or anxiety disorder ³ | Lifetime | WMH-CIDI | DSM-IV criteria | First-generation (33) Second-generation (57) Third+ generation (999) | White, Latinx, Asian; each disaggregated by immigrant generation | <i>Prevalence</i> First-generation: 30.71% Second-generation: 37.16% Third+ generation: 36.76% | None reported | N/A |
| Harker (2001) ¹⁰⁸ | Add Health (wave 2, 1996) | Depressive symptoms | Past week or past year (varied by item) | 23 items adapted from the CES-D and Beck inventory | Continuous summary index reflecting mean item score; range: 1-4 | First-generation (59) Second-generation (135) Third+ generation (2582) | Mexican, Cuban, Central/South American, Puerto Rican, Chinese, Filipino, Other Asian/Pacific Islander, white; each disaggregated by immigrant generation | <i>Mean</i> First-generation: 1.54 Second-generation: 1.55 Third+ generation: 1.61 | None reported | N/A |
| Domain: Parent nativity (n=1) | | | | | | | | | | |
| Kim (2018) ¹⁰⁹ | Mental health screening of sixth grade students in Seattle public schools (2001-2004) | Depressive symptoms (continuous) | NR | 30 items from MFQ | Continuous; range: NR | At least one foreign-born parent (145) Two US-born parents (320) | Asian/Pacific Islander, white, Latinx; each disaggregated by parent nativity | <i>Mean (n.s.)</i> At least one foreign-born parent: 11.0 Two US-born parents: 10.0 | None reported | N/A |
| Domain: Caribbean ethnicity (n=4) | | | | | | | | | | |
| Assari (2017) ¹¹¹ | NSAL-A (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | non-Caribbean (810) Caribbean (360) | None | <i>Prevalence (n.s.)</i> Non-Caribbean: 6.25% Caribbean: 6.63% <i>Adjusted OR</i> Non-Caribbean: ref Caribbean: 0.86 (n.s.) | Subgroup variation in main effects | <i>a priori</i> |
| Patcher (2017) ¹¹⁰ | NSAL-A (2001-2003) | MDD | Lifetime | WMH-CIDI | DSM-IV criteria | non-Caribbean (810) Caribbean (360) | None | <i>Prevalence (n.s.)</i> Non-Caribbean: 6.3% Caribbean: 6.6% | None reported | N/A |
| | | MDD | Past year | WMH-CIDI | DSM-IV criteria | | | <i>Prevalence (n.s.)</i> Non-Caribbean: 4.2% Caribbean: 5.2% | " | " |

Table 1.3: Summary of studies included in systematic review, Black youth (cont.)

| Author (year) | Data source (collection period) | Depression measure | Recall | Instrument | Cutoff score or description | Black subgroups (n) | Non-Black subgroups | Summary of findings | Proposed mechanism(s) ¹ | Mechanism reasoning ² |
|-----------------------------|---------------------------------|---------------------|-------------|-----------------------------|-----------------------------|--|---------------------|--|------------------------------------|----------------------------------|
| Seaton (2008) ²⁷ | NSAL-A (2001-2003) | Depressive symptoms | Past 7 days | 12 items adapted from CES-D | Continuous; range: 0-36 | non-Caribbean (810) Caribbean (360) | None | <i>Mean (n.s.)</i> Non-Caribbean: 9.08 Caribbean: 8.29 | Subgroup variation in main effects | <i>a priori</i> |
| Smith (2020) ²⁸ | NSAL-A (2001-2003) | Depressive symptoms | Past 7 days | 12 items adapted from CES-D | Continuous; range: 0-36 | non-Caribbean (783) Caribbean w/US-born parents (216) Caribbean w/foreign-born parents (144) | None | <i>Mean (n.s.)</i> Non-Caribbean: 9.09 Caribbean w/US-born parents: 8.80 Caribbean w/foreign-born parents: 7.36 | Subgroup variation in main effects | <i>a priori</i> |

Add Health = National Longitudinal Study of Adolescent to Adult Health; NCS-A = National Comorbidity Study (Replication) – Adolescent Supplement; NSAL-A = National Survey of American Life – Adolescent Supplement
MDD = major depressive disorder; MFQ = Mood and Feelings Questionnaire; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, fourth edition; WMH-CIDI = World Mental Health version of the Composite International Diagnostic Interview
NR = Not reported

* Statistically significant (p<0.05); n.s. = non-significant

1. Refers to within-Black racial differences

2. Mechanism reasoning refers to whether the listed mechanism was a clear hypothesis in the given study raised in the introduction section (*a priori*) or if it was raised in the discussion section alone

3. Mood disorders: major depressive disorder, dysthymia, or bipolar disorder; anxiety disorders: panic disorder, agoraphobia, social phobia, specific phobia, generalized anxiety disorder, posttraumatic stress disorder, separation anxiety disorder

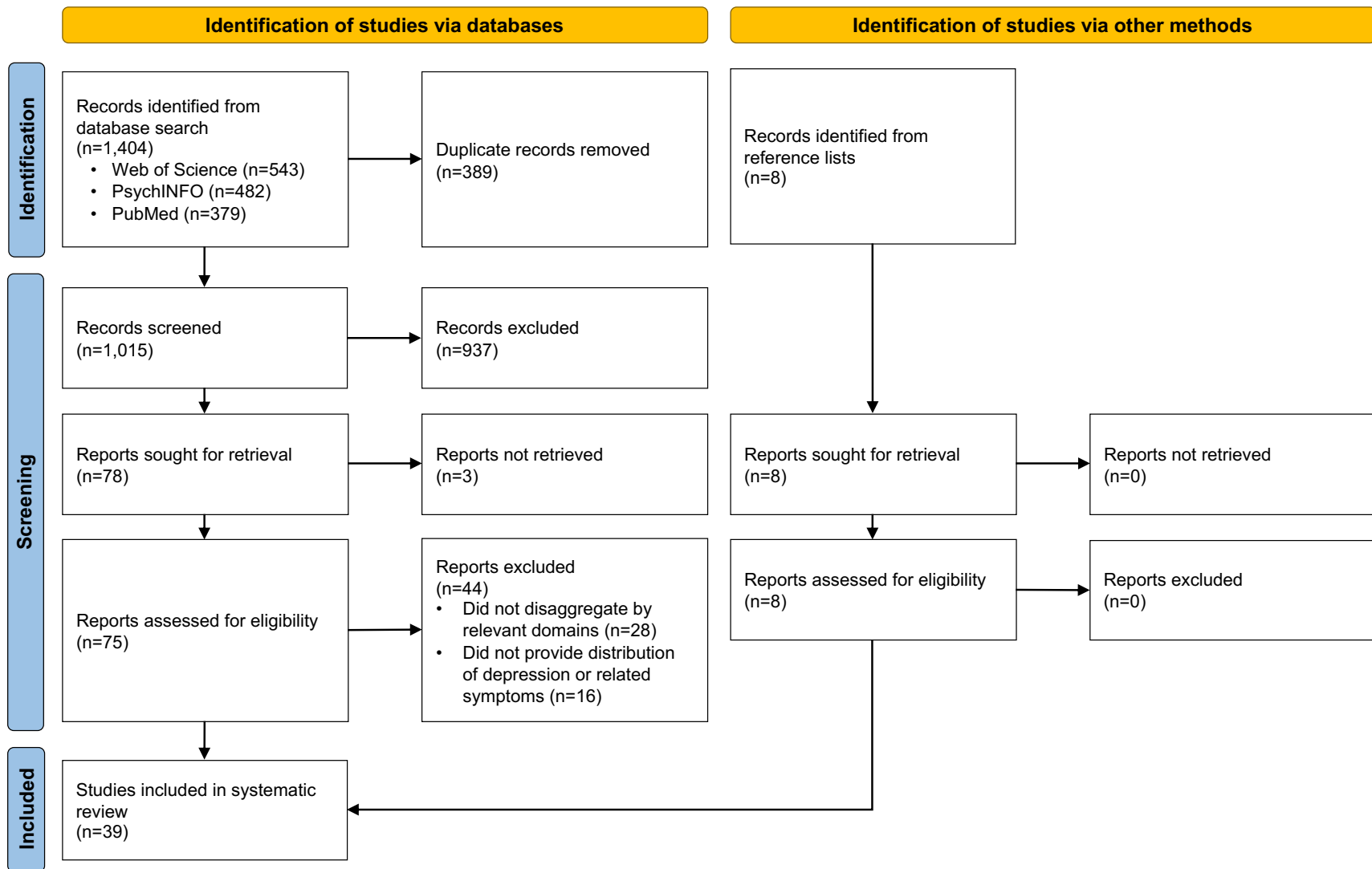


Figure 1.1: PRISMA flowchart for systematic review

Chapter 2: Trajectories of depressive symptoms from adolescence to adulthood — exploring variation by immigrant generation among Black Americans

2.1 Introduction

Despite having a lower prevalence of major depressive disorder, national data have shown non-Hispanic Black Americans experience higher levels of depressive symptoms than non-Hispanic white Americans among adults.⁴⁷ These patterns in depressive symptoms are consistent among youth.¹¹⁶ This racial variation in depressive symptoms has largely been attributed to Black Americans facing greater social stressors, namely racial discrimination.^{4,7,117} Less research has been conducted within the US Black population of how depressive symptoms may be patterned across domains like immigrant generation, despite both a rapid growth in the Black immigrant population in recent years,^{55,56} as well as recent calls to disaggregate the Black population in health research.⁵⁷⁻⁶¹ Although data that disaggregate the US Black population are extremely limited, what does exist suggests within-racial variation among adults¹¹⁸ that appears inconsistent with patterns among youth.¹¹⁹ Examining longitudinal trends of depressive symptoms within the US Black population may provide additional insight into the nature and robustness of this apparent variation.

Shifts in Black immigrant patterns to the US since the passage of the Immigration and Nationality Act of 1965 have resulted in ever increasing ethnic diversity to the Black population.¹ Roughly 47 million people in the US identify as Black; of these, about one-in-ten are foreign-born (first-generation immigrants), and a comparable share are US-born with at least one foreign-born parent (second-generation immigrants).² The number of first-generation immigrants has nearly doubled since 2000 and by 2060, one-in-six Black people in the US are

projected to be foreign-born.^{55,56} Black immigrants come from regions across the globe, but nearly half come from the Caribbean and about 42% come from African nations.⁵⁵

The US Black population may be crudely divided into two ethnic groupings — those with and those without recent immigrant origins, the latter which is often referred to as “African American.” African Americans typically refer to those with distant ancestral ties (i.e., more than three generations removed) to Africa, and nearly all are the descendants of enslaved Africans forced into the US. These Black Americans are distinct from those with recent immigrant origins in the 20th century through today in terms of migration history, culture, and language.⁵² African Americans are also distinct from those with recent immigrant origins in terms of health outcomes. For example, first-generation Black immigrant adults appear to have a health advantage compared to their Black US-born counterparts across a range of physical health outcomes.¹²⁰⁻¹²⁵ This pattern of a foreign-born health advantage is consistent among other racial/ethnic groups, and commonly referred to as the “healthy immigrant effect.”¹²⁶ Although comparisons between African Americans and US-born groups of recent immigrant origins (i.e., second-generation immigrants, third-generation immigrants) are less researched than comparisons by nativity (foreign-born vs. US-born), there also appears to be a stepwise intergenerational decline in physical health outcomes, converging to levels comparable to African Americans by the third generation.¹²⁷⁻¹²⁹

Regarding depression-related outcomes, foreign-born (versus US-born) Black adults also appear to have lower levels across depressive symptoms,¹¹⁸ psychological distress,⁸⁰ and major depressive disorder (MDD).²¹ However, there is limited knowledge of whether patterns in these outcomes follow a stepwise intergenerational decline and convergence to the African Americans risk. To date, no study of depression disaggregates the US Black population such that one can

compare estimates across immigrant generation, while simultaneously comparing estimates from African Americans. Data from the National Survey of American Life (NSAL), the largest study of Black mental health in the US, however, may be used to examine whether such a stepwise intergenerational decline towards convergence occurs for Black immigrant groups.

NSAL data allow for disaggregating the US Black population by Caribbean origin, the largest subgroup of Black immigrants. Caribbean origin was ascertained by asking participants whether they were born in the Caribbean (first-generation immigrants) or whether they have a parent or grandparent who was born in the Caribbean (second- and third-generation immigrants, respectively). All other respondents were categorized as “African American,” which subsumed estimates for non-Caribbean immigrant groups (e.g., African-origin, Latin American-origin). Although numbers of non-Caribbean immigrant groups in the NSAL are likely too small to appreciably impact estimates for African Americans, nevertheless, this study will refer to this group as non-Caribbean to avoid further imprecision. Studies using NSAL data have reported prevalence patterns for foreign-born Caribbeans (i.e., first-generation immigrants), US-born Caribbeans (i.e., second- and third-generation immigrants), and non-Caribbeans (nearly all being African American). These data show US-born Caribbeans had higher levels of depressive symptoms and MDD relative to both foreign-born Caribbeans and non-Caribbeans,^{21,24,118} suggesting a convergence does not occur but rather levels among second- and third-generation Caribbean immigrants exceed that of African Americans.

Among youth, however, patterns appear to differ. Data from the adolescent supplement of the NSAL (NSAL-A) drew from a sample of youth aged 13 to 17 years who resided in the same households as adults sampled in the NSAL. One study using these data showed Caribbean youth with at least one foreign-born parent (i.e., first- and second-generation immigrants

combined) had significantly lower levels of depressive symptoms compared with non-Caribbean youth; levels between Caribbean youth with two US-born parents (third-generation immigrants) and non-Caribbeans did not differ substantially.¹¹⁹ That is, these results among youth suggest an intergenerational increase in depressive symptoms toward convergence to African Americans.

Because Caribbeans constitute the largest share of Black immigrants, it is plausible that these observed patterns among Caribbean youth and adults may apply to first- and second-generation Black immigrants broadly. Yet, less is known about how generalizable this hypothesis is to other Black ethnic-immigrant groups separately, likely because data with large enough sample sizes to disaggregate Black immigrant groups by ethnic or regional origin do not exist. It should also be noted that the NSAL-A did not ascertain nativity of the index child, so it is unclear what proportion of youth captured in these data were foreign-born and which were US-born with at least one foreign-born parent. This introduces another challenge in drawing conclusions about trajectories of depressive symptoms from NSAL-A and NSAL data — we must also assume first- and second-generation youth were similar enough in terms of risk factors for depressive symptoms. While a foreign-born health advantage is well-documented, research also suggests such an advantage might be limited to those who immigrated at older ages. In studies of mental health outcomes, specifically, those who immigrated to the US at younger than 13 years of age — often referred to as “1.5-generation” immigrants — had levels of mood disorders that were not appreciably different from levels among US-born counterparts,^{115,130} providing evidence of some comparability between a subset of the foreign-born and the US-born.

These contrasting patterns between youth and adults in the NSAL-A and NSAL suggest that while Caribbean youth had similar or lower levels of depressive symptoms compared with non-Caribbean youth, at some point in the life course there was an inflection point in trajectories

whereby Caribbean adults began to have higher levels than their non-Caribbean counterparts, barring cohort effects. Additionally, the above trends appear to suggest adolescence or early adulthood could mark a critical period of development resulting in a lasting effect on depression and related symptoms. Longitudinal data, however, are needed to both mitigate cohort effects and to identify whether, and if so when in the life course, this inflection point occurs.

For many Black youth, adolescence is a stage marked by increased exposures to and awareness of racial discrimination through spending more time outside of the home (e.g. schools, restaurants, stores),¹³¹ and racial discrimination is generally viewed a social stressor that causes depression.^{7,9,132} Racial socialization and racial identity have been proposed as protective psychosocial factors against depressive symptoms for Black Americans.³⁰⁻³⁵ Racial identity is largely considered an outcome of the racial socialization process, where it is formed based on messages parents transmit to their children about race and how to cope in a racialized society.^{29,133,134} For instance, research suggests messaging on instilling racial pride or preparation for racial bias leads to a strong racial identity.²⁹ A growing body of research suggests substantial variation in racial socialization messages exist between Black Americans with and without recent immigrant origins.^{37,38,40,44,45} Specifically, those with immigrant parents (i.e., first-generation immigrants who were socialized in the US, and second-generation immigrants) may receive fewer messages instilling *racial* pride but rather messages instilling *ethnic* pride, as well as messages situating African Americans as out-group members. Such messages may therefore lead to variation in the meaning and salience of race to one's identity, affecting the extent to which racial identity is protective among first- and second-generation Black immigrants. It follows that late adolescence or emerging adulthood may be a period marked by a trajectory of heightened depressive symptoms for first/second-generation immigrants.

To my knowledge, no study has examined whether trajectories of depressive symptoms from adolescence to adulthood vary between Black Americans by ethnic origin (e.g., Caribbean vs. non-Caribbean), or immigrant generation. Using nationally representative longitudinal data, the purpose of this study is, first, to examine whether, and if so how, trajectories of depressive symptoms from adolescence into adulthood vary across subgroups of Black Americans defined by immigrant generation. The second purpose of this study is to examine trajectories by parent nativity contrasts for other racial/ethnic groups (Asian, Hispanic/Latinx, non-Hispanic white) as a point of comparison to identify the influence of Black racial group membership. That is, the hypothesized divergent trajectories within Black respondents could be due to parental nativity alone if contrasts are similar across these four racial/ethnic groups. However, if parental nativity contrasts vary across the racial/ethnic groups, this could indicate variation in the effects of racialization processes on depressive symptoms exist.

2.2 Methods

Data

Data were drawn from the National Longitudinal Study of Adolescent to Adult Health (Add Health). Add Health is an ongoing, nationally representative study of health and health-related behaviors of adolescents who have been followed into adulthood. Add Health used a school-based stratified sampling design and captured a representative sample of 132 middle and high schools.¹³⁵ Data for the present study were drawn from Waves I-IV. Respondents were in grades 7-12 at baseline in 1994-95 (Wave I) and grades 8-12 in 1996 (Wave II). As adults, respondents were aged 18-26 in 2001-02 (Wave III) and 24-32 in 2008-09 (Wave IV). Response rates across these four waves ranged from 77.4% to 88.6%.¹³⁵ The present study focused on self-

identified non-Hispanic Black (hereafter, Black), Asian, Hispanic/Latinx, and non-Hispanic white (hereafter, white) respondents who were interviewed at each of these four waves; this reflected 7,752 observations across 1,938 unique Black respondents, 2,432 observations across 608 unique Asian respondents, 5,792 observations across 1,448 unique Hispanic/Latinx respondents, and 21,272 observations across 5,318 unique white respondents. While Waves I-IV captured respondents aged 12-35, analyses were limited to observations between the ages of 13-32, as the number of participants aged 12 and 33 to 35 were too small relative to all other ages. This secondary analysis of Add Health data was approved by the Institutional Review Board at Columbia University (New York, NY).

Measures

Depressive symptoms. Add Health measured depressive symptoms based on the 20-item Center for Epidemiologic Studies – Depression (CES-D) scale over the past seven days, ascertained using the following scores: 0 = never/rarely, 1 = sometimes, 2 = a lot of the time, 3 = most of the time/all the time.⁶⁴ In Waves I and II, respondents were asked 19 questions. The number of questions asked in each subsequent wave was much smaller (nine in Wave III, 10 in Wave IV). Supplemental Table 1 lists questions asked across waves, compared with both the original CES-D and items assessed in the NSAL. The present study used only the following nine items that were consistently asked across waves: (1) felt bothered by things that don't usually bother them, (2) could not shake off the blues even with help from family or friends, (3) felt just as good as other people (reverse coded), (4) had trouble keeping their mind on what they were doing, (5) felt depressed, (6) enjoyed life (reverse coded), (7) felt sad, (8) felt that people disliked them, and (9) felt too tired to do things. Accordingly, the theoretical range of depressive

symptoms per wave is 0 to 27. This nine-item version of depressive symptoms has strong internal validity, as Cronbach's α ranged from 0.77 to 0.82 across Waves I-IV,¹³⁶ and psychometric analyses suggest this measure has good construct validity among adolescents across race/ethnicity and immigrant generation.¹³⁷ If an observation was missing one of nine items, that item was imputed with the mean of all other items; observations missing two or more items were excluded.

Immigrant generation. Adolescents who reported they were not born in the US and not born a US citizen abroad were categorized as first-generation immigrants. Adolescents who were born in the US or born a US citizen but reported at least one parent was foreign-born were categorized as second-generation immigrants. All other adolescents born in the US were assumed to have two US-born parents and were categorized as "third+ generation." Among Black respondents, it is assumed nearly all categorized as third+ generation are African American, given that the influx of Black immigrants to the US is a relatively recent phenomenon. First-generation and second-generation respondents were then collapsed into a single category for two reasons. First, it was assumed that these respondents shared similar parental socialization experiences due to having at least one foreign-born parent. And second, analyzing these groups separately, particularly among Black respondents, would be underpowered.

Sociodemographic variables. Following previous studies using Add Health to model within-person changes in depressive symptoms,^{136,138-140} the following sociodemographic variables measured at wave I were included as covariates in adjusted models: sex (male,

female), family structure (two biological parents, two parents [at least one non-biological], single parent, all others), highest level of parent education (less than high school, high school/GED, some college, college or higher), and annual household income in 1994. And while not theoretical confounders, these variables were included to determine whether potential variations in trajectories by immigrant generation persist beyond distributional variations in these variables.

Statistical analyses

Growth curve models (GCMs) were used to estimate age trajectories of depressive symptoms. These models were operationalized as two-level linear mixed effects models with random intercepts and random slopes, as multiple observations (i.e., across each wave) were nested within individuals. Age centered at 13 (the youngest age in the present study) was treated as the time variable. GCMs estimate individual trajectories for each person in the sample, and thus allow for assessing between-person differences (level 2) in within-person patterns of change (level 1).^{141,142} Under this multilevel framework, repeated observations per respondent reflect level 1 units and respondents reflect level 2 units. Thus, time-varying variables are level 1 units (i.e., age) and time-invariant variables are level 2 units (i.e., confounders measured at Wave I). Previous studies using Add Health have used linear GCMs to document age-based changes in outcomes such as depressive symptoms,^{136,138-140,143} substance use,¹⁴⁴⁻¹⁴⁸ alcohol consumption,¹⁴⁹ BMI,¹⁵⁰ and weight gain.¹⁵¹ The present analysis assessed the extent to which depressive symptoms trajectories varied by immigrant generation (first/second vs. third+).

GCMs were estimated separately for each racial/ethnic group. First, to determine the optimal functional form of age, preliminary analyses evaluated unconditional models using linear and quadratic terms for age. Models failed to converge under a cubic age pattern. Maximum

likelihood estimation was used to appropriately compare models. Fit indices suggested a baseline model with a quadratic age pattern best fit the data for each racial/ethnic group (Supplemental Table 2). Second, to evaluate whether age trajectories of depressive symptoms varied by immigrant generation, the baseline model additionally included interaction terms between each age term and immigrant generation (Model 1). A second model additionally adjusted for covariates (Model 2). These two models were estimated using restricted maximum likelihood (REML) to improve precision of random effects estimates. Two covariates contained missing values — highest level of parent education and annual household income in 1994 — e.g., accounting for 513 (26.5%) unique Black respondents with missing covariate information. To maximize sample size, Model 2 used multilevel multiple imputation with chained equations (MICE) to impute these level 2 covariates. Here, 30 imputed datasets were generated using the predictive mean matching method for two-level data.¹⁵² Imputed datasets were separately analyzed, and the final Model 2 estimates were pooled based on rules developed by Rubin (1987).¹⁵³ Lastly, to visualize findings, predicted growth curves for each immigrant generation category were plotted, based on model estimates. As a point of comparison to identify the influence of race, analyses for Asian, Hispanic/Latinx, and white respondents are discussed in terms of how they differ from models of Black respondents.

Analyses incorporated longitudinal survey weights created by Add Health, to account for both unequal probability of selection and attrition over waves.¹⁵⁴ All analyses were conducted in R (version 4.1.1) using the following packages: *nlme*¹⁵⁵ for multilevel (growth curve) models, *mice*¹⁵⁶ for multiple imputation, *ggplot2*¹⁵⁷ for visualizations, along with other packages which contained helper functions.^{158,159} Survey design weights were incorporated using two methods. For sample characteristics, the *survey* package¹⁶⁰ was used to estimate distributions and standard

errors. And in GCMs, survey weights were rescaled to account for the grouping structure of multilevel models.¹⁶¹⁻¹⁶³

2.3 Results

A total of 7,726 observations among 1,983 self-identified Black respondents from Waves I-IV had outcome information and were included in analyses. This included 137 first/second-generation immigrants and 1,801 identified as third+ generation. Weighted sample characteristics at Wave I, stratified by both race/ethnicity and immigrant generation, are summarized in Table 1. Compared with the third+ generation, first/second-generation immigrants reported higher household incomes in 1994 and a higher proportion of parents with a college degree. At Wave I, there were no substantial differences between both groups in depressive symptoms. Weighted sample characteristics for Asian, Hispanic/Latinx, and white respondents are presented in Table 2. Analyses included the following with outcome information: 608 unique Asian respondents (2,424 total observations), 1,448 unique Hispanic/Latinx respondents (5,772 total observations), and 5,318 white respondents (21,243 total observations).

Unadjusted GCM results among Black respondents (Model 1, Table 3) revealed evidence of varying trajectories of depressive symptoms by immigrant generation. Multiply imputed estimates adjusted for covariates (Model 2) did not substantively differ. Model 2 results showed that at age 13, depressive symptoms were elevated among first/second-generation immigrants (mean = 9.314) relative to those in the third+ generation (mean = 8.452), although differences were characterized by imprecision ($\beta = 0.862$; 95% CI: -0.096, 1.820). Cross-level interactions between immigrant generation and age showed considerable variation in depressive symptoms trajectories across age. The negative coefficient for linear age ($\beta = -0.064$; 95% CI: -0.131,

0.004) and positive coefficient for quadratic age ($\beta = 0.004$; 95% CI: 0.001, 0.008) support a U-shaped pattern for the third+ generation, where depressive symptoms showed a decrease from adolescence into the early 20s, followed by an increase into the early 30s. The interaction term between immigrant generation and linear age was negative ($\beta = -0.276$; 95% CI: -0.511, -0.041), and the corresponding interaction term for quadratic age was positive ($\beta = 0.013$; 95% CI: 0.001, 0.025); for the first/second-generation, this indicated the estimated U-shaped trajectory has a steeper decline, followed by a steeper increase, relative to the third+ generation.

To determine the extent to which trends may be driven by factors specific to Black respondents, the above analyses were replicated for Asian, Hispanic/Latinx, and white respondents (Table 4). Likewise, models adjusted for covariates were not substantially different from corresponding unadjusted models for each of these racial/ethnic groups. Here, the pattern of first/second-generation immigrants exhibiting a steeper decrease in depressive symptoms in adolescence followed by a steeper increase towards heightened levels in adulthood compared with the third+ generation held exclusively for Black respondents. For Asian, Hispanic/Latinx, and white respondents, levels of depressive symptoms were not substantively different between immigrant generation groups across ages 13-32. Figure 1 visualizes these adjusted results for Black, Asian, Hispanic/Latinx, and white respondents separately.

2.4 Discussion

Using nationally representative, longitudinal data, this study found evidence suggesting unique variation in depressive symptom trajectories among Black Americans by immigrant generation that differed from patterns found among Asian, Hispanic/Latinx, and white respondents. Models showed trajectories of depressive symptoms for both first/second-

generation immigrants and the third+ generation that were U-shaped, where symptoms were lowest during the early 20s for both groups and lower for the first/second-generation compared with the third+ generation during this period. In addition, first/second-generation immigrants exhibited a steeper decrease in symptoms from adolescence into the early 20s, followed by a steeper increase into the early 30s. This pattern was consistent in sensitivity analyses restricting observations to those who were interviewed at all four waves.

Findings were not consistent with cross-sectional patterns of depressive symptoms between Caribbean youth in the NSAL-A and Caribbean adults in the NSAL. While first/second-generation Caribbean youth exhibited lower levels of symptoms compared with non-Caribbean (and largely African American) youth in the NSAL-A, the present results did not indicate such a pattern. Rather, models suggested heightened levels for first/second generation immigrants between ages 13 to 15, relative to the third+ generation, and similar levels between these groups at ages 16 and 17. Among adults, NSAL data suggested higher levels of depressive symptoms for US-born Caribbeans relative to non-Caribbeans, yet the present results support lower levels for first/second generation immigrants compared with the third+ generation from age 18 to the early 30s. Extrapolating beyond the early 30s suggests depressive symptoms among first/second-generation immigrants would exceed levels compared with the third+ generation; however, such a conclusion assumes the observed trend fits data beyond the age range used to estimate the model, which cannot be confirmed. Future work using a larger age range of adults is needed to assess whether the present trends extend beyond the early 30s.

Inconsistencies between the present analysis and NSAL data could also be due to variations in how depressive symptoms were ascertained. The present study used nine items adapted from the CES-D, while NSAL studies used 12 items from the CES-D.^{118,119} Only five

items were shared across these studies (Supplemental Table 1). While it is possible that non-overlapping items may, at least in part, drive the observed differences between Add Health and NSAL, future work is needed to address the extent to which individual items may vary across immigrant generation. A second possible source of inconsistency could be due to variations in how target populations were defined. The present study did not restrict the immigrant group to Caribbeans, which raises the question of whether additional variation exists within Black ethnic-immigrant groups. Power limitations, however, undermine such an analysis using Add Health data. And a third possible source of inconsistency concerns differences in when the data were collected. The NSAL is a cross-sectional study conducted from 2001-2003. However, Add Health data used in the present study were collected between 1994-2009, which included more recent data than what the NSAL provides.

Notably, only among Black respondents did trajectories indicate evidence of a steeper increase into adulthood for first/second-generation immigrants compared with the third+ generation. These results suggest unique variation in depressive symptom trajectories exist among Black respondents; these could point to differences by immigrant generation groups in the effects of race-related social stressors and protective factors relevant to mental health. For Black Americans, specifically, these could be social stressors like racial discrimination and protective factors like racial socialization and racial identity. To illustrate, NSAL-A data showed the association between perceived discrimination and depressive symptoms was stronger for Caribbean youth — and especially first/second-generation Caribbean youth (i.e., those with foreign-born parents) — than non-Caribbean youth.^{27,28} Thus, the effects of racial discrimination may be more detrimental on mental health for first/second-generation youth immigrants than the third+ generation. Such a conclusion is consistent with the hypothesis that there exists variation in the

protective effects of racial socialization and racial identity. Future work, however, is needed to disentangle how mechanisms like racial socialization and racial identity may operate differentially by immigrant generation and across the life course among Black Americans.

It should be noted that the present study used self-reported depressive symptoms, rather than a clinical diagnosis of depression like major depressive disorder (MDD). National epidemiologic data consistently show the prevalence of MDD is lower for Black Americans,¹⁰⁻¹⁴ who are largely made up of those without recent immigrant origins (i.e., African Americans). Researchers have characterized this pattern as the “Black-white depression paradox” due to the expectation that increased social stressors among Black Americans should translate into a higher prevalence of MDD.^{15,47} In contrast, Black Americans also show higher levels of depressive symptoms relative to white Americans, despite depressive symptoms being correlated with MDD.⁴⁷ Thus, the relationship between diagnostic depression and depressive symptoms appears to function differently for Black Americans. While the present results are specific to depressive symptoms from adolescence to adulthood within Black Americans, a similar pattern of diverging trajectories may also exist for MDD. Likewise with depressive symptoms, NSAL-A data reveal minimal differences in lifetime MDD between Caribbean youth and non-Caribbean youth, while NSAL data among adults show among the US-born, Caribbeans have a prevalence of MDD almost twice as high as their non-Caribbean counterparts. Future studies using longitudinal data are needed to confirm whether analogous diverging trajectories exist between US-born Caribbean compared with US-born non-Caribbeans, and by immigrant generation (i.e., first/second versus third+) broadly.

Limitations of the present study include the use of four waves of Add Health data, despite the availability of Wave V. These Wave V data were collected from 2016 to 2018 and would

have additionally included data from ages up to 42 years. However, the measure of depressive symptoms was less robust, as it only included three consistent items across waves — (1) could not shake off the blues even with help from family or friends, (2) felt depressed, and (3) felt sad — in contrast to the nine consistent items from Waves I to IV. Second, this analysis was unable to further disaggregate of Black first/second-generation immigrants by regional or ethnic origin (e.g., Caribbean origin, Africa origin) due to prohibitively small sample sizes. Thus, it is unclear whether the trajectory among first/second-generation immigrants applies to all regardless of ethnic origin, or if results are driven by the those of Caribbean origin — who make up the largest share of Black immigrants. Third, and relatedly, it is unclear whether further variation in trajectories exists within first/second-generation immigrants, as the present study was unable to disentangle individual trajectories of the first-generation and the second-generation separately due to small sample sizes. Future longitudinal studies with more granular information on immigrant generation, including a “1.5-generation” distinction, would provide additional insight into this possible source of variation. Lastly, the first wave of Add Health began in 1994. Changes in the composition of the US Black population, as well as recent overall rises in depressive symptoms among adolescents,¹⁶⁴ may limit transportability of results to younger cohorts of Black Americans.

Despite these limitations, the present study is the first examination, to my knowledge, of whether longitudinal trajectories of depressive symptoms differ between first/second-generation immigrants and the third+ generation among a representative sample of Black Americans. Results support unique trajectories among Black respondents; while trajectories followed a U-shape for both subgroups, symptoms from adolescence to the early 20s decreased faster for first/second-generation immigrants, followed by a faster increase into adulthood. Given observed

heterogeneity in depressive symptoms, the present study adds to the limited literature disaggregating the US Black population. To echo previous researchers, additional studies are needed to investigate how sociocultural factors, such as those distinguishing first/second generation-immigrants from the third+ generation immigration, lead to the observed variation in trajectories depressive symptoms.⁵⁷⁻⁶¹ Future mental health surveillance studies that do not include information on immigrant generation could inadvertently mask meaningful variations across this domain and subsequently hinder progress toward better understanding the source of this variation within the US Black population. Results also suggest treatment strategies for first/second-generation immigrants that account for sociocultural correlates of immigrant generation may be more effective than those that do not. For example, results appear consistent with the notion that racial discrimination differentially affects these subgroups of Black Americans; however, additional work is needed to clarify how this mechanism may operate across the life course.

2.5 Tables and Figures

Table 2.1: Sample characteristics of Black respondents^a by immigrant generation, Wave I of the National Longitudinal Study of Adolescent to Adult Health

| | N | First/second (n=137) | Third+ (n=1790) |
|--|------|-------------------------|--------------------|
| Depressive symptoms, M (SE) | 1927 | 8.54 (0.15) | 7.97 (0.49) |
| Age, M (SE) | 1927 | 15.71 (0.19) | 16.28 (0.52) |
| Sex, % (SE) | | | |
| Male | 785 | 48.99 (1.96) | 42.24 (7.30) |
| Female | 1142 | 51.01 (1.96) | 57.76 (7.30) |
| Annual household income (thousands, \$), M (SE) ^b | 1424 | 28.86 (1.85) | 35.83 (3.76) |
| Highest parent education, % (SE) ^b | | | |
| Less than high school | 225 | 18.61 (2.53) | 15.80 (5.17) |
| High school or equivalent | 555 | 44.39 (2.58) | 19.51 (6.13) |
| Some college | 366 | 17.30 (1.58) | 21.07 (5.08) |
| College or higher | 511 | 19.71 (2.71) | 43.62 (6.94) |
| Family structure, % (SE) | | | |
| Two biological parents | 678 | 30.27 (1.96) | 38.79 (7.14) |
| One biological & one non-biological parent | 265 | 12.74 (1.25) | 15.99 (4.25) |
| Single parent | 829 | 46.63 (1.97) | 41.17 (8.12) |
| Other | 155 | 10.37 (1.11) | 4.05 (2.23) |

^a Excludes 11 respondents with missing depressive symptoms scores at wave 1

^b Excludes missing observations

Table 2.2: Sample characteristics of Asian, Hispanic/Latinx, and white respondents by immigrant generation, Wave I of the National Longitudinal Study of Adolescent to Adult Health

| | Asian ^a | | | Hispanic/Latinx ^b | | | White ^c | | |
|--|--------------------|--------------------------|-------------------|------------------------------|--------------------------|-------------------|--------------------|-------------------------|--------------------|
| | N | First/second (n=1185) | Third+ (n=176) | N | First/second (n=2220) | Third+ (n=987) | N | First/second (n=648) | Third+ (n=9334) |
| Depressive symptoms, M (SE) | 605 | 8.09 (0.24) | 7.84 (0.34) | 1439 | 8.00 (0.16) | 8.01 (0.19) | 5305 | 7.90 (0.23) | 7.93 (0.07) |
| Age, M (SE) | 605 | 15.87 (0.27) | 14.72 (0.22) | 1439 | 15.77 (0.27) | 15.37 (0.19) | 5305 | 15.32 (0.19) | 15.47 (0.13) |
| Sex, % (SE) | | | | | | | | | |
| Male | 315 | 55.77 (3.52) | 52.31 (5.30) | 684 | 48.85 (1.93) | 54.41 (3.02) | 2438 | 50.57 (3.32) | 49.86 (0.92) |
| Female | 290 | 44.23 (3.52) | 47.69 (5.30) | 755 | 51.15 (1.93) | 45.59 (3.02) | 2867 | 49.43 (3.32) | 50.14 (0.92) |
| Annual household income (thousands, \$), M (SE) ^d | 374 | 47.28 (3.90) | 57.77 (3.56) | 1071 | 30.33 (2.34) | 44.50 (4.14) | 4416 | 56.39 (5.35) | 50.96 (1.99) |
| Highest parent education, % (SE) ^d | | | | | | | | | |
| Less than high school | 28 | 10.86 (3.25) | 2.86 (2.88) | 466 | 48.20 (4.67) | 17.86 (2.95) | 286 | 6.54 (2.16) | 6.10 (0.84) |
| High school or equivalent | 89 | 17.85 (3.55) | 28.15 (8.68) | 367 | 24.21 (2.65) | 38.17 (3.42) | 1720 | 22.21 (3.63) | 37.29 (1.71) |
| Some college | 73 | 17.70 (3.99) | 22.67 (5.26) | 209 | 13.93 (2.35) | 20.79 (2.97) | 1040 | 23.23 (3.68) | 20.93 (0.93) |
| College or higher | 240 | 53.59 (5.22) | 46.31 (9.59) | 206 | 13.67 (2.64) | 23.18 (3.50) | 1834 | 48.02 (4.88) | 35.69 (2.12) |
| Family structure, % (SE) | | | | | | | | | |
| Two biological parents | 437 | 71.72 (4.02) | 71.94 (4.11) | 833 | 61.20 (3.88) | 51.76 (3.51) | 3251 | 58.42 (3.73) | 62.53 (1.27) |
| One biological & one non-biological parent | 56 | 9.69 (2.28) | 7.54 (2.98) | 228 | 15.10 (1.90) | 16.85 (2.79) | 989 | 19.89 (2.86) | 16.96 (0.65) |
| Single parent | 80 | 13.71 (2.63) | 19.19 (3.91) | 310 | 19.12 (2.66) | 28.28 (2.69) | 917 | 18.70 (2.54) | 17.73 (0.89) |
| Other | 32 | 4.88 (1.30) | 1.32 (0.64) | 68 | 4.58 (1.35) | 3.11 (0.98) | 148 | 2.99 (1.16) | 2.79 (0.35) |

^a Excludes 3 respondents with missing depressive symptoms scores at wave 1

^b Excludes 9 respondents with missing depressive symptoms scores at wave 1

^c Excludes 13 respondents with missing depressive symptoms scores at wave 1

^d Excludes missing observations

Table 2.3: Growth curve models of depressive symptoms among Black respondents, Waves I-IV of the National Longitudinal Study of Adolescent to Adult Health

| | Model 1 | | Model 2 ^a | |
|---|------------|----------------|----------------------|----------------|
| | Estimate | (95% CI) | Estimate | (95% CI) |
| Fixed effects | | | | |
| Intercept | 8.710 | 8.439, 8.980 | 8.452 | 7.978, 8.927 |
| Immigrant generation | | | | |
| First/second | 0.787 | -0.176, 1.749 | 0.862 | -0.096, 1.820 |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Age | -0.062 | -0.130, 0.005 | -0.064 | -0.131, 0.004 |
| Age ² | 0.004 | 0.001, 0.008 | 0.004 | 0.001, 0.008 |
| Immigrant generation x Age | | | | |
| First/second | -0.273 | -0.508, -0.038 | -0.276 | -0.511, -0.041 |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Immigrant generation x Age ² | | | | |
| First/second | 0.013 | 0.001, 0.025 | 0.013 | 0.001, 0.025 |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Random effects (variances) | | | | |
| Residual (within, level 1) | 13.668 | | 13.631 | |
| Intercept (between, level 2) | 14.751 | | 14.513 | |
| Age slope | 0.702 | | 0.702 | |
| Age ² slope | 0.002 | | 0.002 | |
| N observations | 7726 | | 7726 | |
| N respondents | 1938 | | 1938 | |

^a Adjusts for the following covariates collected at Wave I: sex, annual household income, highest parent education, and family structure

Table 2.4: Growth curve models of depressive symptoms among Asian, Hispanic/Latinx, and white respondents, Waves I-IV of the National Longitudinal Study of Adolescent to Adult Health

| Asian respondents | | | | |
|---|-----------------|-----------------|----------------------------|-----------------|
| | Model 1 | | Model 2^a | |
| | Estimate | (95% CI) | Estimate | (95% CI) |
| Fixed effects | | | | |
| Intercept | 7.888 | (6.532, 9.244) | 7.703 | (6.085, 9.320) |
| Immigrant generation | | | | |
| First/second | 0.762 | (-0.722, 2.245) | 0.776 | (-0.705, 2.256) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Age | -0.030 | (-0.357, 0.297) | -0.031 | (-0.358, 0.296) |
| Age ² | 0.000 | (-0.016, 0.016) | 0.000 | (-0.016, 0.016) |
| Immigrant generation x Age | | | | |
| First/second | -0.115 | (-0.470, 0.240) | -0.114 | (-0.468, 0.241) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Immigrant generation x Age ² | | | | |
| First/second | 0.007 | (-0.011, 0.024) | 0.007 | (-0.011, 0.024) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Random effects (variances) | | | | |
| Residual (within, level 1) | 12.589 | | 12.589 | |
| Intercept (between, level 2) | 23.695 | | 23.616 | |
| Age slope | 1.129 | | 1.13 | |
| Age ² slope | 0.002 | | 0.002 | |
| N observations | 2424 | | 2424 | |
| N respondents | 608 | | 608 | |
| Hispanic/Latinx respondents | | | | |
| | Model 1 | | Model 2^a | |
| | Estimate | (95% CI) | Estimate | (95% CI) |
| Fixed effects | | | | |
| Intercept | 8.776 | (8.191, 9.361) | 8.246 | (7.597, 8.895) |
| Immigrant generation | | | | |
| First/second | -0.447 | (-1.160, 0.265) | -0.409 | (-1.121, 0.303) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Age | -0.127 | (-0.273, 0.019) | -0.125 | (-0.271, 0.020) |
| Age ² | 0.006 | (-0.001, 0.013) | 0.006 | (-0.002, 0.013) |
| Immigrant generation x Age | | | | |
| First/second | 0.077 | (-0.100, 0.253) | 0.075 | (-0.101, 0.251) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Immigrant generation x Age ² | | | | |
| First/second | -0.004 | (-0.012, 0.005) | -0.004 | (-0.012, 0.005) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Random effects (variances) | | | | |
| Residual (within, level 1) | 14.829 | | 14.794 | |
| Intercept (between, level 2) | 18.821 | | 18.431 | |
| Age slope | 1.015 | | 1.016 | |
| Age ² slope | 0.002 | | 0.002 | |
| N observations | 5772 | | 5772 | |
| N respondents | 1448 | | 1448 | |

| White respondents | | | | |
|---|-----------------|-----------------|----------------------------|-----------------|
| | Model 1 | | Model 2^a | |
| | Estimate | (95% CI) | Estimate | (95% CI) |
| Fixed effects | | | | |
| Intercept | 8.010 | (7.867, 8.153) | 8.021 | (7.741, 8.301) |
| Immigrant generation | <i>ref</i> | | <i>ref</i> | |
| First/second | -0.286 | (-0.886, 0.314) | -0.264 | (-0.860, 0.333) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Age | 0.012 | (-0.024, 0.048) | 0.011 | (-0.025, 0.047) |
| Age ² | 0.000 | (-0.002, 0.001) | 0.000 | (-0.002, 0.001) |
| Immigrant generation x Age | | | | |
| First/second | 0.131 | (-0.021, 0.282) | 0.130 | (-0.021, 0.281) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Immigrant generation x Age ² | | | | |
| First/second | -0.007 | (-0.015, 0.001) | -0.007 | (-0.015, 0.001) |
| Third+ | <i>ref</i> | | <i>ref</i> | |
| Random effects (variances) | | | | |
| Residual (within, level 1) | 7.819 | | 7.797 | |
| Intercept (between, level 2) | 11.232 | | 10.996 | |
| Age slope | 0.561 | | 0.562 | |
| Age ² slope | 0.001 | | 0.001 | |
| N observations | 21243 | | 21243 | |
| N respondents | 5318 | | 5318 | |

^a Adjusts for the following covariates collected at Wave I: sex, annual household income, highest parent education, and family structure

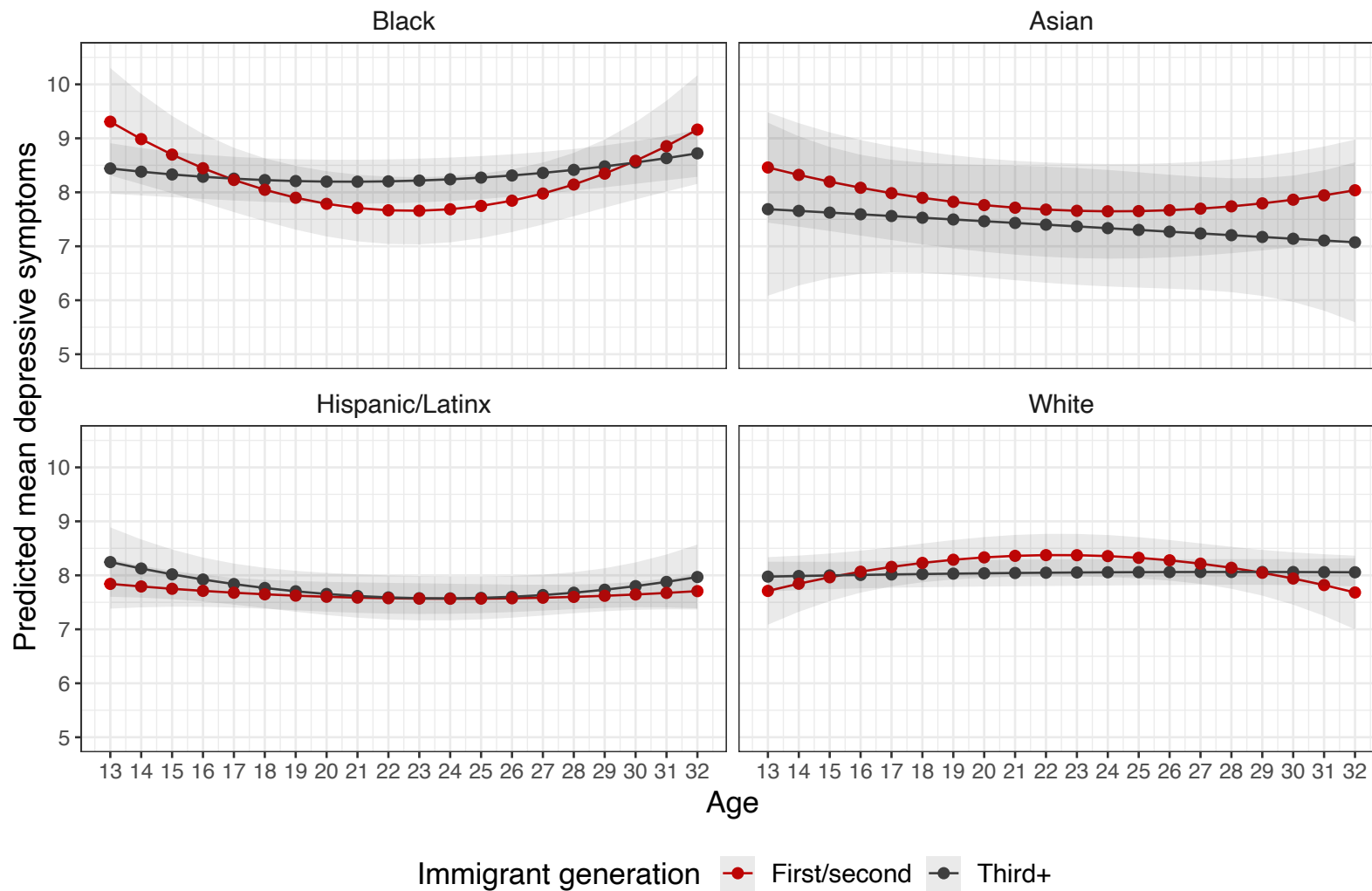


Figure 2.1: Predicted depressive symptoms trajectories by racial/ethnic group and immigrant generation, Waves I-IV of the National Longitudinal Study of Adolescent to Adult Health

Note: Models adjust for the following covariates collected at Wave I: sex, annual household income, highest parent education, and family structure

2.6 Supplemental Material

Supplemental Table 2.1: Depressive symptoms items across four waves of Add Health compared with the CES-D and items assessed in the NSAL

| Item | CES-D | NSAL | Add Health | | | |
|---|-------------|-------------|-------------|-------------|-------------|-------------|
| | | | I | II | III | IV |
| 1. I was bothered by things that don't usually bother me | X | -- | X | X | X | X |
| 2. I did not feel like eating; my appetite was poor | X | -- | X | X | -- | -- |
| 3. I felt that I could not shake off the blues even with help from my family or friends | X | -- | X | X | X | X |
| 4. I felt I was just as good as other people | X | X | X | X | X | X |
| 5. I had trouble keeping my mind on what I was doing | X | X | X | X | X | X |
| 6. I felt depressed | X | X | X | X | X | X |
| 7. I felt that everything I did was an effort | X | X | -- | -- | -- | -- |
| 8. I felt hopeful about the future* | X | X | X | X | -- | -- |
| 9. I thought my life had been a failure | X | -- | X | X | -- | -- |
| 10. I felt fearful | X | -- | X | X | -- | -- |
| 11. My sleep was restless | X | X | -- | -- | -- | -- |
| 12. I was happy* | X | X | X | X | -- | X |
| 13. I talked less than usual | X | -- | X | X | -- | -- |
| 14. I felt lonely | X | -- | X | X | -- | -- |
| 15. People were unfriendly | X | X | X | X | -- | -- |
| 16. I enjoyed life* | X | X | X | X | X | X |
| 17. I had crying spells | X | X | -- | -- | -- | -- |
| 18. I felt sad | X | -- | X | X | X | X |
| 19. I felt that people dislike me | X | X | X | X | X | X |
| 20. I could not get "going" | X | X | -- | -- | -- | -- |
| 21. You felt that you were too tired to do things | -- | -- | X | X | X | X |
| 22. It was hard to get started doing things | -- | -- | X | X | -- | -- |
| 23. You felt life was not worth living | -- | -- | X | X | -- | -- |
| Total items | 20 | 12 | 19 | 19 | 9 | 10 |
| Theoretical range | 0-60 | 0-36 | 0-57 | 0-57 | 0-27 | 0-30 |

CES-D = Center for Epidemiologic Studies – Depression Scale⁶⁴

NSAL = National Survey of American Life¹¹²

Add Health = National Longitudinal Study of Adolescent to Adult Health¹³⁵

* reverse-coded

Note: NSAL was a cross-sectional study of mental health among Black Americans with and without Caribbean origins; data were collected from 2001-2003 for youth (aged 13-17) and adults (aged 18 or older) separately. Add Health data included in the present study were collected over four waves: 1994-1995 (Wave I), 1996 (Wave II), 2001-2002 (Wave III) and 2008-2009 (Wave IV).

Supplemental Table 2.2: Model fit statistics for growth curve models, Waves I-IV of the National Longitudinal Study of Adolescent to Adult Health

| Black respondents | | | | | | | | | |
|------------------------------|-------------------|-----------------|----------|----------------------------|-----------------|----------|-------------------------------|------------------|----------|
| | Null model | | | Linear growth model | | | Quadratic growth model | | |
| | Estimate | (95% CI) | P | Estimate | (95% CI) | P | Estimate | (95% CI) | P |
| Fixed effects | | | | | | | | | |
| Intercept | 8.511 | (8.404, 8.619) | <0.001 | 8.404 | (8.236, 8.572) | <0.001 | 8.775 | (8.515, 9.035) | <0.001 |
| Age | | | | 0.017 | (0.003, 0.032) | 0.020 | -0.085 | (-0.149, -0.020) | 0.010 |
| Age ² | | | | | | | 0.005 | (0.002, 0.009) | 0.003 |
| Variance components | | | | | | | | | |
| Residual (within, level 1) | 21.821 | | | 17.474 | | | 13.658 | | |
| Intercept (between, level 2) | 2.777 | | | 6.710 | | | 14.766 | | |
| Age slope | | | | 0.032 | | | 0.707 | | |
| Age ² slope | | | | | | | 0.002 | | |
| Diagnostics | | | | | | | | | |
| AIC | 42594.87 | | | 41938.82 | | | 41503.29 | | |
| BIC | 42615.72 | | | 41980.54 | | | 41572.82 | | |
| Log likelihood | -21294.43 | | | -20963.41 | | | -20741.65 | | |
| Degrees of freedom | 3 | | | 6 | | | 10 | | |
| Likelihood ratio test | — | | | p<0.001 ^a | | | p<0.001 ^b | | |
| N observations | 7726 | | | 7726 | | | 7726 | | |
| N groups | 1938 | | | 1938 | | | 1938 | | |
| Asian respondents | | | | | | | | | |
| | Null model | | | Linear growth model | | | Quadratic growth model | | |
| | Estimate | (95% CI) | P | Estimate | (95% CI) | P | Estimate | (95% CI) | P |
| Fixed effects | | | | | | | | | |
| Intercept | 7.949 | (7.774, 8.124) | <0.001 | 8.061 | (7.776, 8.345) | <0.001 | 8.531 | (7.987, 9.076) | <0.001 |
| Age | | | | -0.011 | (-0.034, 0.012) | 0.356 | -0.127 | (-0.254, 0.000) | 0.049 |
| Age ² | | | | | | | 0.006 | (0.000, 0.012) | 0.071 |
| Variance components | | | | | | | | | |
| Residual (within, level 1) | 21.137 | | | 18.037 | | | 12.594 | | |
| Intercept (between, level 2) | 2.286 | | | 5.652 | | | 23.591 | | |
| Age slope | | | | 0.024 | | | 1.122 | | |
| Age ² slope | | | | | | | 0.002 | | |
| Diagnostics | | | | | | | | | |
| AIC | 13162.36 | | | 13055.35 | | | 12871.33 | | |
| BIC | 13179.74 | | | 13090.11 | | | 12929.26 | | |
| Log likelihood | -6578.178 | | | -6521.676 | | | -6425.665 | | |
| Degrees of freedom | 3 | | | 6 | | | 10 | | |
| Likelihood ratio test | — | | | p<0.001 ^a | | | p<0.001 ^b | | |
| N observations | 2424 | | | 2424 | | | 2424 | | |
| N groups | 608 | | | 608 | | | 608 | | |

| Hispanic/Latinx respondents | | | | | | | | | |
|------------------------------|------------|----------------|--------|----------------------|----------------|--------|------------------------|------------------|--------|
| | Null model | | | Linear growth model | | | Quadratic growth model | | |
| | Estimate | (95% CI) | P | Estimate | (95% CI) | P | Estimate | (95% CI) | P |
| Fixed effects | | | | | | | | | |
| Intercept | 8.149 | (8.027, 8.270) | <0.001 | 8.170 | (8.145, 8.387) | <0.001 | 8.474 | (8.436, 8.825) | <0.001 |
| Age | | | | -0.001 | (0.012, 0.034) | 0.870 | -0.075 | (-0.130, -0.031) | 0.071 |
| Age ² | | | | | | | 0.003 | (0.003, 0.008) | 0.099 |
| Variance components | | | | | | | | | |
| Residual (within, level 1) | 25.617 | | | 21.704 | | | 14.822 | | |
| Intercept (between, level 2) | 2.519 | | | 5.524 | | | 18.819 | | |
| Age slope | | | | 0.027 | | | 1.013 | | |
| Age ² slope | | | | | | | 0.002 | | |
| Diagnostics | | | | | | | | | |
| AIC | 32173.62 | | | 31866.01 | | | 31175.82 | | |
| BIC | 32193.6 | | | 31905.98 | | | 31242.43 | | |
| Log likelihood | -16083.81 | | | -15927.01 | | | -15577.91 | | |
| Degrees of freedom | 3 | | | 6 | | | 10 | | |
| Likelihood ratio test | — | | | p<0.001 ^a | | | p<0.001 ^b | | |
| N observations | 5772 | | | 5772 | | | 5772 | | |
| N groups | 1448 | | | 1448 | | | 1448 | | |
| White respondents | | | | | | | | | |
| | Null model | | | Linear growth model | | | Quadratic growth model | | |
| | Estimate | (95% CI) | P | Estimate | (95% CI) | P | Estimate | (95% CI) | P |
| Fixed effects | | | | | | | | | |
| Intercept | 8.041 | (7.985, 8.097) | <0.001 | 7.967 | (7.882, 8.052) | <0.001 | 7.993 | (7.854, 8.132) | <0.001 |
| Age | | | | 0.012 | (0.004, 0.020) | 0.004 | 0.019 | (-0.016, 0.054) | 0.279 |
| Age ² | | | | | | | -0.001 | (-0.003, 0.001) | 0.382 |
| Variance components | | | | | | | | | |
| Residual (within, level 1) | 12.653 | | | 10.332 | | | 7.819 | | |
| Intercept (between, level 2) | 1.842 | | | 4.012 | | | 11.229 | | |
| Age slope | | | | 0.023 | | | 0.561 | | |
| Age ² slope | | | | | | | 0.001 | | |
| Diagnostics | | | | | | | | | |
| AIC | 112379.8 | | | 110740.8 | | | 109065.2 | | |
| BIC | 112403.7 | | | 110788.6 | | | 109144.8 | | |
| Log likelihood | -56186.89 | | | -55364.41 | | | -54522.59 | | |
| Degrees of freedom | 3 | | | 6 | | | 10 | | |
| Likelihood ratio test | — | | | p<0.001 ^a | | | p<0.001 ^b | | |
| N observations | 21243 | | | 13055 | | | 13055 | | |
| N groups | 5318 | | | 4058 | | | 4058 | | |

^a Compares linear growth model to null model

^b Compares quadratic growth model to linear model

Chapter 3: Dismantling the monolith — ethnic origin, racial identity, and major depression among US-born Black Americans

3.1 Introduction

Differences between non-Hispanic Black and non-Hispanic white Americans in the prevalence of major depressive disorder (MDD) have been characterized as the “Black-white depression paradox,” wherein Black adults have a lower lifetime prevalence of MDD and an equivalent or lower past-year prevalence of MDD compared to white adults, despite increased exposure to social stressors as a result of anti-Black systemic racism.⁴⁷ This “paradox” has been documented extensively using multiple national datasets¹⁰⁻¹⁴ and has been the focus of substantial research seeking to explain it, as illustrated in a recent review.¹⁵ But far less documented and explored, in part due to limited data, are marked differences in the prevalence of MDD within the US Black population by immigration-related domains like ethnic origin and immigrant generation.

In the US, the Black population as a racial group is ethnically heterogeneous, as it includes African Americans — an ethnic group whose members largely trace their origins in the US to chattel slavery — as well as immigrants from the Caribbean, African nations, Latin America, among other regions of the world. The US Black population is also characterized by increasing numbers of second- and third-generation immigrants, i.e. those with foreign-born parents and grandparents, respectively; while 12% of the US Black population are first-generation immigrants, an additional 9% are estimated to be second-generation immigrants.²

This ethnic diversity within the US Black population is also patterned by differences in sociodemographic characteristics between immigrant groups and non-immigrant Black Americans.⁵⁸ For instance, in 2012, 40% of Black second-generation immigrants aged 25 or

older had a bachelor's degree, higher than both first-generation Black immigrants (31%) and all other US-born Black Americans (20%).¹⁶⁵ Related, one study found Black second-generation immigrants not only attained higher socioeconomic status than their foreign-born parents, but also reached parity with the intergenerational mobility of the white mainstream across education, income, and occupation indices.¹⁶⁶ Cultural differences between Black Americans who descend from enslaved people and Black Americans with recent immigrant origins (i.e., first-, second-, and third-generation immigrants) stem from at least two sources: the historical legacy of American slavery and socialization of first-generation immigrants in majority-Black countries.¹ Despite these ethnic, socioeconomic, and sociocultural differences, the US Black population is often treated as a monolithic group in health research. These sociocultural differences may have important implications for explaining differences in the prevalence of depression within the US Black population.

In response to the lack of studies disaggregating the Black US population, the National Survey of American Life (NSAL), conducted from 2001-2003, was designed to examine within-racial differences in DSM-IV psychiatric disorders in a national sample, allowing for investigations that distinguish the Black population by nativity (foreign-born vs US-born) and Caribbean origin (first-, second-, and third-generation Caribbean immigrants vs non-Caribbeans).¹¹² It should be noted that Caribbeans are the largest subgroup of Black immigrants in the US, and in the NSAL, African Americans almost exclusively comprise the “non-Caribbean” subgroup. These data reveal the prevalence of MDD among first-generation Caribbean immigrants was considerably lower than all US-born Black subgroups.²¹ This finding is consistent with evidence of the “healthy immigrant effect,” where foreign-born populations display more favorable health outcomes compared to their US-born counterparts.¹⁰¹ A surprising

finding was that NSAL data revealed second- and third-generation Caribbean immigrants (i.e., US-born Caribbeans) have a prevalence of MDD nearly twice as high as US-born non-Caribbeans (e.g., lifetime MDD: 19.8% vs 10.4%, respectively).²¹ It should also be noted that these latter two US-born Black subgroups are presumed to share similar social stress exposure levels.

Patterns in mental health outcomes for Black Caribbeans by nativity in the NSAL — the foreign-born had a lower prevalence than their US-born co-ethnics — mirrored those of other immigrant groups, like Asian and Hispanic/Latinx Americans.²³⁻²⁵ For Caribbeans, however, the intergenerational decline in mental health appeared more pronounced than in those of Asian and Latinx American origin.²⁶ For example, the lifetime prevalence of MDD for foreign-born Caribbeans using NSAL data was 8.9%, which is similar to foreign-born Asians (8.0%) and lower than foreign-born Latinx adults (11.8%) in the National Latino and Asian American Study of Mental Health (NLAAS), a sister study of the NSAL; yet among the US-born counterparts, these lifetime prevalence estimates of MDD were 19.8% for Caribbean, 13.0% for Asian, and 15.9% for Latinx people.^{167,168} Earl et al. (2011) describes a similar pattern for lifetime prevalence of psychiatric disorders broadly. These intergenerational patterns in MDD prevalence add another complexity to the literature — not only have NSAL data shown US-born Caribbeans had the highest within-race MDD prevalence, NSAL and NLAAS data have collectively shown US-born Caribbeans also had the highest MDD prevalence within US-born, non-white ethno-racial groups of immigrant origin. These patterns both add complexity to the “Black-white depression paradox” and suggest Black immigrant groups face a faster rate of intergenerational mental health decline compared to other racial/ethnic immigrant groups.

Comparable estimates of patterns in health outcomes by immigrant generation among both white Americans and Black Americans (when including African Americans) are complicated by the relative predominance among those labeled as “third+ generation,” typically identified as having two US-born parents. “Third+ generation” is usually the highest generation level discernible in population health data, but at least among white and Black Americans, these individuals are actually at least fourth-generation Americans (i.e., all four *grandparents* are US-born). This overrepresentation by the “fourth+ generation” among white and Black Americans is due to longer settlement histories in the US, a function of historic patterns of migration — voluntary or involuntary. Such individuals are much less likely to be connected culturally to their immigrant histories or countries of origin, which is especially true for African Americans, whose ethnic heritage prior to enslavement was systematically erased. That said, the available data suggest that among adults, the pattern of successively worse mental health with each subsequent generation deviates for Black adults alone.

Treating Black Americans as a monolithic group not only masks differences in the prevalence of MDD, but it also implies that mechanisms causing MDD are the same across subgroups defined by ethnic origin. Yet because NSAL data reveal US-born Caribbeans have a markedly higher prevalence of MDD compared to (US-born) non-Caribbeans, this may suggest mechanisms causing MDD vary. For Black Americans, and people of color broadly, racial identity is generally considered to be a protective factor against poor mental health outcomes like MDD. Racial identity functioning as such is consistent with the theory proposed by Crocker and Major (1989) of how stigma may be self-protective.¹⁶⁹ One mechanism through which group identification may be protective against MDD among members of stigmatized or oppressed groups is by attributing one’s negative outcomes to the prejudiced attitudes of others.¹⁶⁹ For

instance, attributing negative outcomes caused by discrimination to racism, rather than personal inadequacies, may be a successful coping strategy; one could also say the process of making racial attributions is a function of a strong racial identity. In fact, towards explaining the “Black-white depression paradox,” some have attributed Black racial identity as a source of mental health resiliency.^{170,171}

While much of the published work on racial identity and depression within communities of color has focused on depressive symptoms, racial identity appears to function as a protective factor against both diagnostic depression and its related symptoms. Reviews and meta-analyses of racial identity and depressive symptoms among racial minorities support the hypothesis that aspects of racial identity are protective, through either an inverse main effect or as a buffer (i.e., negative interaction term) on the relationship between discrimination and depressive symptoms.³⁰⁻³⁵ Among the US Black population in the NSAL, results from previous studies also support racial identity as a protective factor against not only depressive symptoms^{88,172} but also psychological distress¹⁷³ and MDD.⁹⁴ Further, NSAL data support racial identity functioning as a buffer on the association between discrimination and distress.¹⁷³ However, only two studies to my knowledge have explored whether the main effect of racial identity on depression may differ *within* the US Black population; results from both, also using NSAL data, suggested racial identity was more protective for non-Caribbeans than Caribbeans on depressive symptoms⁸⁸ and MDD.⁹⁴ Yet despite this evidence of ethnic variation in the effects of racial identity, no study has examined the extent to which this variation explains differences in MDD seen between *US-born* Caribbean and non-Caribbean Black adults. For racial identity to have varying effects within the US Black population points to variation in the processes underlying racial identity formation.

Racial identity (and by extension, ethnic identity) is largely considered an outcome of the ethnic-racial socialization (ERS) process,^{29,133,134} which refers to the ways parents teach their children about race, ethnicity, and how to cope in a racialized society. Historically, research on ERS among Black samples came from scholars' efforts to understand how parental childrearing strategies influenced Black children to maintain high self-esteem, given racialized barriers such as discrimination.¹³³ Qualitative research has found differences in the content of ERS messages between Black Americans with and without immigrant origins, which may imply there exist differences in the salience of race to one's identity. For instance, studies suggest Black children of immigrants may receive messages situating non-immigrant Black Americans as out-group members — i.e., promoting social distancing from and mistrust towards non-immigrant Black Americans.^{38,40,45} To illustrate with a quote from a study on ERS messages among second-generation Haitian immigrants, one participant remarked: “[my] *father wouldn't allow us to play with any of the Black American children.*”⁴⁰ Black children of immigrants may also receive fewer messages promoting *racial* pride but rather messages promoting *ethnic* or *cultural* pride.^{37,40,44} These differences in ERS messages may be attributed to immigrant parents not holding race as central to their identity from (largely) being socialized in majority Black countries, and thus being unaware of US racial hierarchies that situate the US Black population as the most disadvantaged racialized group.

Qualitative studies have also revealed the added complexity of immigration in the racial identities of Black Americans who are the children of immigrants. For instance, in interviews of West Indian children of immigrants in New York City, Waters (1994) identified three distinct identities: (1) those with an immigrant or national identity identified most with their immigrant nationality (i.e., being Jamaican), (2) those with a hyphenated-American identity who stressed an

ethnic/immigrant identity (e.g., being Jamaican-American) over a racial identity, and (3) those with a Black American identity, who stressed a racial identity over an ethnic/immigrant identity.³⁶ More recent research finds support that as young adults, the children of Black immigrants may not necessarily see a conflict between a Black racial identity and an ethnic identity; rather these individuals may embrace both a racial identity and an ethnic identity simultaneously.^{39,41-43,46} Further, qualitative studies have explored ways in which second-generation Black immigrants develop a racial identity independent of parental socialization. For example, one study found evidence that children of Black immigrants may reject their parents' anti-Black American socialization messages.⁴⁵ Here, participants cited a range of reasons, including their own personal experiences being racialized as Black, and having an understanding of how structural racism negatively impacts all Black people in America regardless of immigrant origin. Because immigration adds complexity in the process of racial identity formation for Black Americans of immigrant origin, it appears that the meaning of “being Black” differs between this group and non-immigrant Black Americans. And if the meaning of “being Black” differs, then one might expect effects of racial identity on mental health should also differ between these two groups, thus potentially explaining MDD differences.

To summarize, NSAL data from 2001-2003 have shown US-born Black Americans with recent immigrant origins had a higher prevalence of MDD relative to their US-born non-immigrant counterparts. Having a strong sense of racial identity is generally considered protective against negative mental health outcomes like MDD, but less is known about how it operates in preventing MDD. Furthermore, racial identity processes appear to differ across Black ethnicities — specifically between those with and without immigrant origins. As such, how racial identity functions as protective mechanism against MDD may also differ between US-born

Black Americans with and without immigrant origins, thus explaining the higher prevalence of MDD among those with recent immigrant origins, as seen in NSAL data. I focus on US-born Black Americans because racial identity as a protective function may be less relevant for first-generation immigrants, who were largely not socialized in the US.^{3,174,175} As such, the objective of this study is to assess the extent to which differential effects of racial identity on MDD by ethnic origin explain the elevated prevalence among US-born Caribbeans relative to all other US-born Black Americans in the NSAL. Hypotheses are two-fold. First, I hypothesize that racial identity and MDD will be inversely associated in the overall sample. And second, I hypothesize that, due to differences in racialization processes between US-born Black Americans with and without recent immigrant origins (i.e., US-born Caribbeans vs non-Caribbeans), the nature and magnitude of racial identity as a protective factor will vary between these two groups. Specifically, I hypothesize racial identity will be less protective for US-born Caribbeans compared to non-Caribbeans (i.e., a two-way interaction between racial identity and Caribbean origin).

3.2 Methods

Data source

This study used secondary data from the NSAL. To date, it remains the largest study of Black mental health in the US and is one of the only health data sources designed to be disaggregated by ethnicity, specifically Caribbean vs all other Black Americans. The NSAL contains 3,570 respondents categorized as “African American” and 1,621 self-identified Caribbean respondents. The NSAL includes sample weights to correct for nonresponse and population representation. Additional details of the survey can be found elsewhere.^{21,112}

Measures

Major depressive disorder. The main outcome in this analysis is lifetime DSM-IV MDD, evaluated using a fully structured, modified version of the World Mental Health Composite International Diagnostic Interview (CIDI) administered by trained lay interviewers.¹⁷⁶ The CIDI has been previously shown to have good concordance with DSM-IV MDD diagnoses made by clinicians in clinical reappraisal studies.^{177,178}

Ethnic origin. The NSAL defines Caribbeans as those who self-identified as having been born in the Caribbean or had parents or grandparents born in the Caribbean. The NSAL defines “African American” as those who self-identified as Black but who did not identify having ancestral ties to the Caribbean.²¹ However, dichotomizing Black ethnicities as “Caribbean” or “African American” is subject to imprecision. It should be noted that “African American” is generally understood as an ethnic category whose members are largely descendants of enslaved Africans brought to the US primarily between the 17th and 19th centuries. These Black Americans potentially differ from those with more recent, immigrant origins in the US (i.e., in the latter half of the 20th century through today) in terms of cultural identification and socioeconomic factors, and these differences are often ignored in both the public discourse and in population health research. Thus, by essentially dichotomizing Black ethnicities, it is possible the NSAL definition of “African American” includes Black Americans of immigrant origin (i.e., such as first- or second-generation African immigrants). In fact, the NSAL categorized 64 (1.8% of 3,570) respondents born in Africa — first-generation immigrants — incorrectly as “African American.” Because this study seeks to examine the extent to which variation in the effect of racial identity may explain differences in the prevalence of MDD between among US-born Black

Americans, analyses will only include US-born respondents. Furthermore, dichotomizing Black ethnicities precludes researchers from appropriately identifying US-born Black Americans of immigrant origin who are not Caribbean, as these respondents are subsumed under the “African American” designation. However, this source of measurement error will likely not meaningfully affect results since the number of foreign-born Africans was relatively low. Moving forward, this study will distinguish US-born Black Americans in the NSAL in terms of Caribbean ethnic origin — Caribbean or non-Caribbean.

Racial identity. Racial identity was assessed using four separate items, which correspond to either the *centrality* or *private regard* dimensions of the Multidimensional Model of Racial Identity (MMRI), a widely used framework for conceptualizing Black racial identity.¹⁷⁹ Centrality refers to the extent to which a person normatively defines their identity with respect to race, while private regard refers to the extent to which a person feels positively about their race.¹⁷⁹ In the NSAL, there are three centrality proxies, assessing: (1) closeness in ideas and feelings about Black people, (2) the relative importance of race to one’s identity, and (3) whether one believes their fate is linked to that of Black people in the US generally. The proxy for private regard assesses the extent to which one endorses positive stereotypes and opposed negative stereotypes about Black people in the US (“Black group evaluation”). The decision to use these measures as proxies for centrality and private regard dimensions was informed by characterizations in previous studies of racial identity using NSAL data.^{105,180} Racial identity as a multidimensional construct, as opposed to a single composite score, allows for exploring whether the relationship between racial identity and MDD varies across dimensions, which has been captured in previous studies drawing from the MMRI.¹⁸¹⁻¹⁸³ Furthermore, because these measures are not validated scales, the conceptual meaning of a collapsed summary measure may

be unclear. Table 3.1 describes each racial identity measure as proxies for centrality and private regard MMRI dimensions and includes coding schema.

Potential confounders. The following variables were included as confounders, based on prior research indicating these variables are associated with racial identity and MDD and are conceivably common causes of each within the US Black population:^{21,105,184} binary sex (male, female), age (continuous), and achieved markers of SES — years of education (0-11, 12, 13-15, ≥ 16), annual household income (continuous), marital status (married/cohabitating, divorced/separated/widowed, never married), and employment status (employed, unemployed, not in labor force). Sensitivity analyses (described below) additionally included perceived racial discrimination as a confounder. Perceived racial discrimination (“racial discrimination” hereafter) was ascertained using the 10-item Everyday Discrimination Scale.¹⁸⁵ Participants were asked how often in their day-to-day lives have they experienced discriminatory treatment such as being treated with less courtesy than others or being threatened or harassed. Frequency was assessed on a five-point scale (5 = almost every day, 4 = at least once a week, 3 = a few times a month, 2 = a few times a year, 1 = less than once a year, 0 = never). This self-report measure of perceived discrimination has previously been shown to have high internal consistency and validity in samples of Black respondents.^{186,187} Respondents who reported “a few times a year” or more on at least one item were additionally asked to identify the primary reason for such discriminating experiences (e.g., race, gender, height). Racial discrimination was assessed by limiting reports of discrimination to those primarily attributed to race or a similar construct (skin color or ancestry); those who did not make a primary attribution to race or a similar construct were coded to have a value of 0. Although there does not appear to be a gold standard assessment of racial discrimination using this particular NSAL measure of discrimination,

studies using NSAL data have made similar distinctions between racial and non-racial discrimination,^{173,188,189} and at least one reported about 75% of respondents in the NSAL attributed everyday discrimination primarily to race, skin color, or ancestry.¹⁹⁰ About 2% (n=84) of observations had a missing value for at least one of the 10 items, and the majority of these were missing only one item. To preserve these observations in sensitivity analyses, analyses used the average of non-missing responses; thus, the theoretical range of racial discrimination was from 0 to 5 where higher values reflect higher frequency of everyday racial discrimination.

Statistical analyses

First, bivariate analyses were conducted to compare the distribution of MDD, potential confounders, and racial identity measures between Caribbeans and non-Caribbeans. Next, prevalence ratios (PRs) were estimated for each racial identity indicator on MDD using separate multivariable Poisson regression models with robust variance estimation. Then, effect modification was assessed on the multiplicative scale. Effect modification analyses were three-fold. First, interaction terms were included between each racial identity indicator and ethnic origin (i.e., racial identity x ethnic origin) in separate models for each of the four racial identity variables. Second, to further evaluate effect modification, PRs of racial identity on MDD were estimated within each stratum of ethnic origin using indicator variables, based on recommendations in the literature.¹⁹¹ And third, to facilitate interpretation of statistical interactions, marginal predicted probabilities were estimated and graphed. All analyses were performed on available cases and incorporated NSAL complex survey weights. Additionally, all models adjusted for potential confounders. For all effect estimates, 95% confidence intervals

(CIs) were reported. Analyses were performed using R version 4.1.1 using the *survey*¹⁹² and *ggplot2*¹⁵⁷ packages.

Sensitivity analyses

Additional analyses were conducted to assess the robustness of findings. First, racial discrimination may be conceptualized as a mediator or confounder on the causal path between racial identity and depression. That is, although the relationship between discrimination and subsequent poor mental health is well-established,^{5,8,31,193-197} empirical evidence regarding the relationship between racial identity and discrimination is more ambiguous. Some studies support the identification-attribution model where higher levels of racial identity lead to increased perceptions of discrimination,^{182,198} others support the hypothesis that higher perceptions of discrimination lead to either increased or decreased levels of racial identity,^{199,200} and yet longitudinal findings support a bi-directional association between discrimination and racial identity.²⁰¹ Because NSAL data cannot establish temporality, main analyses treated racial discrimination as a mediator by omitting it, and sensitivity analyses included it as a confounder.

And second, analyses were repeated using past-year MDD as the main outcome, rather than lifetime MDD. Using one over the other is a balance between preserving statistical power and limiting reverse causation; focusing on lifetime MDD preserves statistical power that is otherwise lost using past-year MDD, as the prevalence of MDD is higher under “lifetime” versus “past-year” time frames. Yet, focusing on past-year MDD reduces possibility of reverse causation, which may be of concern as the NSAL is a cross-sectional study. If results are similar between these two outcomes, this may provide some evidence that reverse causation did not drive results under the outcome of lifetime MDD.

3.3 Results

Sample characteristics overall and by ethnic origin are displayed in Table 3.2. There were 432 Caribbean and 3340 non-Caribbean respondents with MDD measures; as expected, the prevalence of lifetime MDD among Caribbeans was twice as high as the prevalence among non-Caribbeans (20.9% vs 10.3%; $p=0.023$). Caribbeans were generally younger than non-Caribbeans in this sample (35.2 years vs 42.2 years; $p<0.001$), had more years of education ($p=0.01$), had greater annual household incomes (\$52,300 vs \$36,000; $p=0.02$), and were more likely to be never married ($p<0.001$). Overall, each of the four racial identity measures — closeness to other Black Americans, the relative importance of race to one’s identity, shared fate with other Black Americans, and Black group evaluation — did not differ appreciably between non-Caribbeans and Caribbeans in this sample.

Analyses of the first hypothesis assessed whether racial identity functioned as a protective factor against MDD in the US-born sample. Results showed that higher levels of racial identity were associated with a lower prevalence of lifetime MDD (Table 3.3). Compared to low levels of closeness to Black Americans, high levels were associated with a 34% lower prevalence of MDD (PR=0.66, 95% CI: 0.49, 0.90) and moderate levels were associated with a 30% lower prevalence of MDD (PR=0.70, 95% CI: 0.49, 0.99). Results suggest identifying as “being Black” compared to something else (e.g., American, both Black and American equally) was associated with a lower prevalence of MDD (PR=0.78, 95% CI: 0.56, 1.09). Compared to having relatively low levels of Black group evaluation, having high levels was associated with a 30% lower prevalence of MDD (PR = 0.70, 95% CI: 0.50, 0.99) while having moderate levels was associated with a more modest lower prevalence characterized by imprecision (PR = 0.90, 95%

CI: 0.68, 1.19). Conversely, results suggest shared fate with Black Americans (compared to no shared fate) was associated with a somewhat higher, yet imprecise, prevalence of MDD (PR = 1.17, 95% CI: 0.93, 1.47). These patterns across each racial identity measures were consistent in models which additionally adjusted for racial discrimination, and likewise in models where the outcome was past-year MDD (Supplemental Table 3.1).

Analyses of the second hypothesis assessed whether the effects of racial identity may vary between Caribbean and non-Caribbean Black Americans. Here, effect modification models (cross-product terms in Table 3.4, indicator variables in Table 3.5, and marginal predicted probabilities in Figure 1) suggested the presence and degree of variation in the effect of racial identity by ethnic origin depends on the domain of racial identity. For instance, models where closeness to other Black Americans was the main exposure revealed no substantial effect modification of ethnic origin. However, in models where the relative importance of race to one's identity was the main exposure, effect modification was suggested; although PRs were in opposite directions 95% CIs were characterized by imprecision. Specifically, while results suggest identifying as "being Black" was associated with a lower prevalence of MDD within strata of non-Caribbeans (PR=0.75, 95% CI: 0.52, 1.08), this measure was suggestive of higher prevalence of MDD within strata of Caribbeans (PR=1.29, 95% CI: 0.76, 2.21).

Where shared fate with Black Americans was the main exposure, these models revealed a positive effect modification across strata of ethnic origin on the multiplicative scale (interaction PR=3.05, 95% CI: 1.58, 5.87). Models using indicator variables showed that while results suggest shared fate was associated with a somewhat higher prevalence of MDD within strata of non-Caribbeans (PR = 1.12, 95% CI: 0.89, 1.43), an affirmative response was associated with a substantially higher prevalence of MDD among Caribbeans (PR=3.43, 95% CI: 1.87, 6.27).

Finally, in models where Black group evaluation was the main exposure, effect modification was suggested. Similar to results for the relative importance of race to one's identity, stratified PRs in multiplicative models were also in opposite directions. For instance, while high (vs relatively low) levels of Black group evaluation was associated with a lower prevalence of MDD among non-Caribbeans (PR=0.67, 95% CI: 0.47, 0.96), results suggest the same measure was associated with a higher prevalence of MDD among Caribbeans (high vs low PR=1.66, 95% CI: 0.50, 5.50).

Figure 3.1 visualizes these above effect modification results in the form of marginal predicted probabilities. Additional analyses modeling racial discrimination as a confounder were not substantively different from corresponding effect modification analyses, as well as additional analyses where past-year MDD was modeled as the outcome (supplemental table 3.2); however, confidence intervals were generally wider in these sensitivity analyses.

3.4 Discussion

The goal of this study, using data from the largest survey of Black mental health, was to examine the role of racial identity in explaining the heightened prevalence of MDD among US-born Caribbeans compared to US-born non-Caribbeans. Specifically, this study sought to assess whether the relationship between racial identity and MDD varies by ethnic origin among Black Americans born in the US. The first hypothesis was that racial identity measures would be inversely associated with MDD across the entire US-born sample. This was supported, or at least suggested, for three of the four measures — closeness to other Black Americans, Black group evaluation, and relative importance of race to one's identity.

The second hypothesis was that racial identity would be less protective against MDD for US-born Caribbeans than non-Caribbeans. This was supported for shared fate with Black Americans. For two racial identity measures — relative importance of race to one’s identity and Black group evaluation — effect modification within these models was suggestive as 95% CIs contained null values. While the relationship between shared fate with Black Americans and MDD suggested a weak, positive effect among non-Caribbeans, an affirmative response was associated with a substantially higher prevalence of MDD among Caribbeans. And while analyses suggested the relative importance of race to one’s identity and Black group evaluation were inversely associated with MDD among non-Caribbeans, these racial identity measures were associated with a higher prevalence of MDD among Caribbeans, albeit non-appreciably. These effect modification results for US-born Caribbeans contrast with an established literature supporting racial identity as a protective factor against depression.^{30-35,181-183}

Findings from this study support the notion that whether racial identity functions as a source of mental health resiliency depends on an unmeasured factor related to ethnic origin. That is, ethnic origin (in this study, Caribbean vs non-Caribbean) may function as a proxy for sociocultural factors, like ethnic differences in the racial socialization process, that produce variation in the racial identity formation process, leading to differences in the meaning — and thus effect — of racial identity on MDD. Additional research is needed to elucidate the role of racial socialization on differences in depression across Black ethnicities among the US-born.

It is possible that racial identity may only be protective if there are sufficient levels of social support. Indeed, one study using a sample of Black medical students found higher levels of centrality were associated with increased depressive symptoms; authors attributed this finding to limited in-group interactions, leading to low levels of social support.²⁰² For US-born

Caribbeans, limited in-group interactions could refer to limited interactions with other Black Americans of immigrant origin, who may share similar sociocultural exposures. Further work is needed to clarify the role of social support towards explaining variation in the effect of racial identity on MDD between US-born Caribbeans and non-Caribbeans.

One finding inconsistent with the present pattern of results was that closeness to other Black Americans was the only racial identity measure where increased levels were associated with a lower prevalence of MDD for both US-born Caribbeans and non-Caribbeans. These results suggest ethnic variation in the impact of racial identity may additionally vary across dimensions of racial identity. Additional studies are needed to better understand this potential heterogeneity of racial identity dimensions on the relationship between racial identity and MDD across Black ethnicities.

At least two previous studies using the NSAL have explored whether the main effect of racial identity on depression differs by Caribbean ethnicity. Consistent with the present study, results from both found the proxy for private regard (i.e., Black group evaluation) was less protective for Caribbeans than for non-Caribbeans on depressive symptoms⁸⁸ and was associated with an increased MDD prevalence for Caribbeans alone.⁹⁴ But unlike other studies using NSAL, the present is the first (to my knowledge) that excluded foreign-born Caribbeans and focused on variation within the US-born, for whom racial identity effects may be more relevant in part due to differences in socialization contexts. Studies on ethnic variation in the effect of racial identity on subsequent health outcomes that do not exclude foreign-born participants assume racial identity functions the same across nativity status, despite research suggesting the contrary.^{3,174,175}

In addition to the hypothesized direct protective effect of racial identity, researchers have proposed that racial identity buffers the negative effects of discrimination on mental health.^{30,34,35}

Given that the present results provide some support for ethnic origin as an effect modifier of the relationship between racial identity and MDD, ethnic origin may also modify the two-way interaction between racial identity, discrimination, and MDD (i.e., a three-way interaction). In other words, it is plausible that racial identity may function less as a buffer against poor mental health outcomes in the face of discrimination for US-born Caribbeans vs non-Caribbeans and may even amplify the magnitude of this relationship. However, power limitations due to small sample sizes in the present study, particularly among US-born Caribbeans, undermine such an analysis. Relatedly, racial identity measures may interact with each other such that the inverse relationship between one racial identity dimension and depression could be stronger for those with higher levels of a second racial identity dimension.²⁰³ Likewise, such an interactive model accounting for possible differences across ethnic origin would have been infeasible to perform in the present study.

There are limitations to consider when interpreting the findings of this study. First, although the NSAL is a nationally representative data set, power limitations likely contributed to wide confidence intervals, some of which contained null values. Consequently, estimates where confidence intervals contained null values were interpreted as suggestive. Second, the racial identity measures used in this study may have also contributed to null findings. In contrast to validated measures such as the Multidimensional Inventory of Black Identity,²⁰⁴ the measures used may be weak proxies, resulting in measurement error. Third, data designed to disaggregate Black Americans are limited to the NSAL, which was conducted 20 years ago from 2001-2003 and only considers those of Caribbean origin as recent Black immigrants. Changes in the composition of US-born Black Americans (e.g., by ethnicity), the prevalence of MDD, or the prevalence or effects of racial identity would limit transportability to Black Americans today,

and it is unclear whether patterns in MDD among Caribbeans apply similarly to other Black immigrant populations (e.g., those from sub-Saharan Africa). Despite the potential for transportability and generalizability issues, this study contributes to arguments questioning the homogeneity of Black Americans and may inform future studies and data collection efforts. Lastly, the NSAL is a cross-sectional study and establishing the temporal order of racial identity and MDD is limited. However, conceptual frameworks supported by empirical studies do support racial identity leading to poor subsequent mental health.^{30,35,205} And sensitivity analyses using past-year MDD as the outcome of interest yielded similar results.

This study, using data from NSAL which is the largest survey of Black mental health, is the first empirical investigation to my knowledge seeking to explain why US-born Caribbeans have a heightened prevalence of MDD relative to US-born non-Caribbeans in the NSAL. Findings provide new insight into the role of racial identity on MDD; namely, while racial identity was generally protective among the entire US-born sample, indicators of racial identity were associated with a higher prevalence of MDD for US-born Caribbeans. This study contributes to the scant literature examining within-group heterogeneity among Black populations and responds to recent calls to focus on racialization processes in the study of Black immigrants and their descendants.²⁰⁶⁻²⁰⁸

3.5 Tables and Figures

Table 3.1: Racial identity measures and their relation to the Multidimensional Model of Racial Identity, National Survey of American Life (NSAL)

| Dimension | Measure | Item in NSAL | Coding |
|---|---|--|--|
| Centrality <i>The extent to which a person normatively defines their identity with respect to race</i> | Closeness to Black Americans | Eight-item index, where respondents were asked, “How close do you feel in your ideas and feelings about things to: (1) Black people who are poor, (2) Black people who are religious, (3) young Black people, (4) upper-class Black people, (5) working-class Black people, (6) Black elected officials, (7) Black doctors, lawyers, and other professional people, and (8) older Black people. Responses were ascertained using the following scores: 0 = not close at all, 1 = not too close, 2 = fairly close, and 3 = very close; the theoretical range of values is 0-24. | Values were categorized into the following tertiles: 0-16 = low 17-20 = moderate 21-24 = high |
| | The relative importance of race to one’s identity | A single item asking participants which is more important: being Black, being American, both are equally important, or something else. This item was dichotomized as “being Black” versus all other responses. | 1 = “Black” 0 = “American”, “both equally”, something else |
| | Shared fate with Black Americans | A single, binary item asking respondents, “Do you think what happens generally to Black people in this country will have something to do with what happens in your life?” | 1 = yes 0 = no |
| Private regard <i>The extent to which a person feels positively about their race</i> | Black group evaluation | A six-item index of positive and negative stereotypes, where respondents were asked how true they believed most Black Americans: (1) are intelligent, (2) are lazy, (3) are hard-working, (4) give up easily, (5) are proud of themselves, and (6) are violent. Responses were ascertained using the following scores: 0 = not at all true, 1 = a little true, 2 = somewhat true, and 3 = very true. Negative stereotypes (i.e., items 2, 4, and 6) were re-coded so that higher scores reflect a more favorable group evaluation. | Values were categorized into the following tertiles: 0-11 = low 12-14 = moderate 15-18 = high |

Table 3.2: Sample characteristics of US-born Black Americans by Caribbean ethnic origin, National Survey of American Life (n=3772)

| | N ^a | Overall (n=3772) | Non-Caribbean (n=3340) | Caribbean (n=432) |
|---|----------------|---------------------|---------------------------|----------------------|
| Lifetime MDD, % (SE) | 424 | 10.57 (0.61) | 10.29 (0.60) | 20.87 (5.94) |
| Age (years), M (SE) | 3772 | 41.98 (0.53) | 42.17 (0.54) | 35.17 (1.33) |
| Education, % (SE) | | | | |
| 0-11 years | 931 | 24.40 (1.18) | 24.47 (1.21) | 21.89 (4.35) |
| 12 years | 1394 | 37.39 (1.01) | 37.77 (1.04) | 23.88 (1.57) |
| 13-15 years | 905 | 24.24 (1.00) | 24.07 (1.01) | 30.47 (5.89) |
| ≥ 16 years | 542 | 13.97 (1.09) | 13.70 (1.12) | 23.75 (2.20) |
| Annual household Income (thousands, \$), M (SE) | 3772 | 36.47 (1.34) | 36.04 (1.36) | 52.26 (6.63) |
| Marital status, % (SE) | | | | |
| Married/cohabitating | 1291 | 41.13 (1.05) | 41.23 (1.07) | 37.52 (2.65) |
| Divorced/separated/widowed | 1164 | 26.57 (0.85) | 26.87 (0.86) | 15.67 (3.45) |
| Never married | 1317 | 32.30 (1.33) | 31.90 (1.36) | 46.81 (3.68) |
| Sex, % (SE) | | | | |
| Male | 1344 | 43.71 (0.88) | 43.52 (0.86) | 50.54 (9.44) |
| Female | 2428 | 56.29 (0.88) | 56.48 (0.86) | 49.46 (9.44) |
| Work status, % (SE) | | | | |
| Employed | 2484 | 66.47 (1.07) | 66.29 (1.10) | 72.85 (3.32) |
| Unemployed | 402 | 10.44 (0.75) | 10.46 (0.77) | 9.65 (3.31) |
| Not in labor force | 886 | 23.10 (1.00) | 23.25 (1.02) | 17.50 (1.83) |
| Everyday racial discrimination, M (SE) | 3769 | 1.04 (0.03) | 1.03 (0.03) | 1.34 (0.14) |
| Closeness to Black Americans, % (SE) ^b | | | | |
| High | 1156 | 31.09 (1.10) | 31.34 (1.12) | 21.99 (3.24) |
| Moderate | 1003 | 26.27 (1.09) | 26.40 (1.11) | 21.39 (5.27) |
| Low | 1547 | 42.64 (1.35) | 42.25 (1.38) | 56.61 (6.46) |
| More important to be Black or something else, % (SE) ^b | | | | |
| Being Black | 752 | 19.44 (0.84) | 19.38 (0.85) | 21.72 (3.12) |
| Something else | 3013 | 80.56 (0.84) | 80.62 (0.85) | 78.28 (3.12) |
| Shared fate with Black Americans, % (SE) ^b | | | | |
| Yes | 2339 | 62.43 (1.35) | 62.17 (1.39) | 71.37 (4.59) |
| No | 1364 | 37.57 (1.35) | 37.83 (1.39) | 28.63 (4.59) |
| Black group evaluation, % (SE) ^b | | | | |
| High | 1288 | 34.34 (1.40) | 34.25 (1.44) | 37.44 (3.19) |
| Moderate | 1294 | 34.77 (1.16) | 34.84 (1.18) | 32.41 (4.57) |
| Low | 1147 | 30.89 (1.20) | 30.91 (1.23) | 30.15 (3.73) |

All estimates incorporated complex survey weights.

^a Unweighted.

^b Racial identity measures are missing for some observations.

Table 3.3: Adjusted robust Poisson models estimating prevalence ratios (PRs) of lifetime major depressive disorder, US-born sample of the National Survey of American Life

| | Prevalence (%) | PR (95% CI) ^a |
|--|----------------------|--------------------------|
| Panel 1 (n=3706) | | |
| Closeness to Black Americans | | |
| High | 8.35 (6.65, 10.43) | 0.66 (0.49, 0.90) |
| Moderate | 9.18 (6.62, 12.62) | 0.70 (0.49, 0.99) |
| Low | 12.97 (11.24, 14.93) | <i>ref</i> |
| Panel 2 (n=3765) | | |
| More important to be Black or something else | | |
| Being Black | 8.58 (6.34, 11.53) | 0.78 (0.56, 1.09) |
| Something else | 11.07 (9.71, 12.60) | <i>ref</i> |
| Panel 3 (n=3703) | | |
| Shared fate with Black Americans | | |
| Yes | 11.02 (9.52, 12.72) | 1.17 (0.93, 1.47) |
| No | 9.61 (8.02, 11.46) | <i>ref</i> |
| Panel 4 (n=3729) | | |
| Black group evaluation | | |
| High | 8.82 (6.78, 11.41) | 0.70 (0.50, 0.99) |
| Moderate | 10.92 (9.50, 12.52) | 0.90 (0.68, 1.19) |
| Low | 12.14 (9.73, 15.05) | <i>ref</i> |

Each panel reflects a separate model.

All estimates incorporated complex survey weights.

Ns are unweighted.

^a Models adjusted for ethnic origin, age, sex, marital status, education, annual household income, and employment status

Table 3.4: Effect modification of racial identity on lifetime MDD by Caribbean ethnic origin among US-born Black Americans using cross-product terms

| | PR (95% CI) |
|--|-------------------|
| Panel 1 (n=3706) | |
| Closeness to Black Americans | |
| High | 0.67 (0.49, 0.92) |
| Moderate | 0.70 (0.49, 1.01) |
| Low | <i>ref</i> |
| Ethnic origin | |
| Non-Caribbean | <i>ref</i> |
| Caribbean | 1.94 (0.97, 3.88) |
| Closeness to Black Americans x Ethnic origin | |
| High x Caribbean | 0.65 (0.37, 1.12) |
| Moderate x Caribbean | 1.01 (0.39, 2.62) |
| Low | <i>ref</i> |
| Panel 2 (n=3765) | |
| More important to be Black or something else | |
| Being Black | 0.75 (0.52, 1.08) |
| Something else | <i>ref</i> |
| Ethnic origin | |
| Non-Caribbean | <i>ref</i> |
| Caribbean | 1.68 (1.08, 2.63) |
| More important to be Black or something else x Ethnic origin | |
| Being Black x Caribbean | 1.72 (0.90, 3.29) |
| Something else | <i>ref</i> |
| Panel 3 (n=3703) | |
| Shared fate with Black Americans | |
| Yes | 1.12 (0.89, 1.43) |
| No | <i>ref</i> |
| Ethnic origin | |
| Non-Caribbean | <i>ref</i> |
| Caribbean | 0.75 (0.41, 1.39) |
| Shared fate with Black Americans x Ethnic origin | |
| Yes x Caribbean | 3.05 (1.58, 5.87) |
| No | <i>ref</i> |
| Panel 4 (n=3729) | |
| Black group evaluation | |
| High | 0.67 (0.47, 0.96) |
| Moderate | 0.87 (0.66, 1.16) |
| Low | <i>ref</i> |
| Ethnic origin | |
| Non-Caribbean | <i>ref</i> |
| Caribbean | 1.02 (0.46, 2.25) |

Black group evaluation x Ethnic origin

| | |
|----------------------|-------------------|
| High x Caribbean | 2.47 (0.71, 8.66) |
| Moderate x Caribbean | 2.17 (0.71, 6.64) |
| Low | <i>ref</i> |

Each panel reflects a separate model.

All estimates incorporated complex survey weights.

Ns are unweighted.

Robust Poisson models adjusted for age, sex, marital status, education, annual household income, and employment status.

Table 3.5: Effect modification of racial identity on lifetime MDD by Caribbean ethnic origin among US-born Black Americans using indicator variables

| | Prevalence of MDD | | Prevalence ratios of MDD ^a | |
|--|---------------------------------|-----------------------------|---------------------------------------|-----------------------------|
| | within strata of non-Caribbeans | within strata of Caribbeans | within strata of non-Caribbeans | within strata of Caribbeans |
| | % (95% CI) | % (95% CI) | PR (95% CI) | PR (95% CI) |
| Panel 1 (n=3706) | | | | |
| Closeness to Black Americans | | | | |
| High | 8.30 (6.56, 10.44) | 10.99 (7.74, 15.39) | 0.67 (0.49, 0.92) | 0.43 (0.27, 0.69) |
| Moderate | 8.97 (6.36, 12.50) | 18.84 (6.29, 44.54) | 0.70 (0.49, 1.01) | 0.70 (0.29, 1.70) |
| Low | 12.50 (10.80, 14.43) | 25.63 (12.08, 46.37) | <i>ref</i> | <i>ref</i> |
| Panel 2 (n=3765) | | | | |
| More important to be Black or something else | | | | |
| Being Black | 8.05 (5.83, 11.02) | 25.84 (9.47, 53.72) | 0.75 (0.52, 1.08) | 1.29 (0.76, 2.21) |
| Something else | 10.84 (9.46, 12.40) | 19.65 (11.95, 30.59) | <i>ref</i> | <i>ref</i> |
| Panel 3 (n=3703) | | | | |
| Shared fate with Black Americans | | | | |
| Yes | 10.53 (9.07, 12.20) | 26.19 (13.98, 43.64) | 1.12 (0.89, 1.43) | 3.43 (1.87, 6.27) |
| No | 9.64 (8.01, 11.56) | 7.74 (4.11, 14.10) | <i>ref</i> | <i>ref</i> |
| Panel 4 (n=3729) | | | | |
| Black group evaluation | | | | |
| High | 8.35 (6.34, 10.93) | 24.38 (8.14, 54.00) | 0.67 (0.47, 0.96) | 1.66 (0.50, 5.50) |
| Moderate | 10.56 (9.19, 12.10) | 24.78 (11.42, 45.69) | 0.87 (0.66, 1.16) | 1.90 (0.64, 5.61) |
| Low | 12.13 (9.64, 15.14) | 12.65 (5.53, 26.38) | <i>ref</i> | <i>ref</i> |

Each panel reflects a separate model.

All estimates incorporated complex survey weights.

Ns are unweighted.

^a Robust Poisson models adjusted for age, sex, marital status, education, annual household income, and employment status.

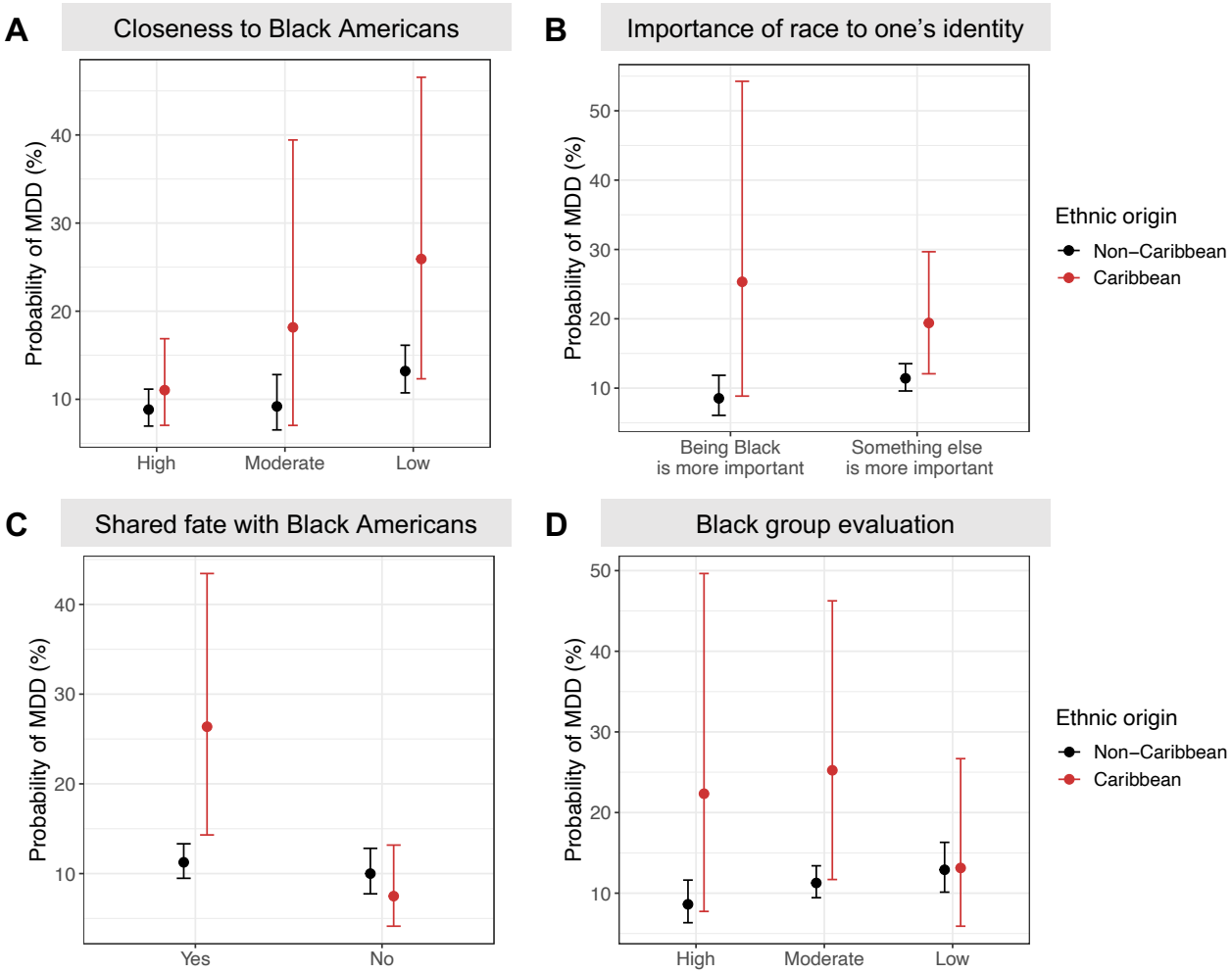


Figure 3.1: Marginal predicted probabilities of lifetime major depressive disorder by levels of Black racial identity and Caribbean ethnic origin using logistic regression models with cross-product interaction terms

Note: Separate models — each adjusting for age, sex, marital status, education, annual household income, and employment status — were generated for each racial identity indicator: (A) closeness to Black Americans, (B) the relative importance of race to one's identity, (C) shared fate with Black Americans, and (D) Black group evaluation. Data were drawn from the US-born sample of the National Survey of American Life.

3.6 Supplemental Material

Supplemental Table 3.1: Sensitivity analyses for adjusted robust Poisson models estimating prevalence ratios (PRs) of lifetime and past-year major depressive disorder, US-born sample of the National Survey of American Life

| | Lifetime MDD ^a | | Past-year MDD | |
|--|---------------------------|-------------------|---------------|-------------------|
| | N | PR (95% CI) | N | PR (95% CI) |
| Panel 1 | 3703 | | 3706 | |
| Closeness to Black Americans | | | | |
| High | | 0.67 (0.50, 0.91) | | 0.71 (0.48, 1.04) |
| Moderate | | 0.69 (0.49, 0.97) | | 0.49 (0.28, 0.88) |
| Low | | <i>ref</i> | | <i>ref</i> |
| Racial discrimination | | 1.29 (1.14, 1.47) | | |
| Panel 2 | 3762 | | 3765 | |
| More important to be Black or something else | | | | |
| Being Black | | 0.75 (0.54, 1.05) | | 0.99 (0.62, 1.60) |
| Something else | | <i>ref</i> | | <i>ref</i> |
| Racial discrimination | | 1.28 (1.13, 1.46) | | |
| Panel 3 | 3701 | | 3703 | |
| Shared fate with Black Americans | | | | |
| Yes | | 1.09 (0.88, 1.36) | | 0.85 (0.60, 1.20) |
| No | | <i>ref</i> | | <i>ref</i> |
| Racial discrimination | | 1.27 (1.11, 1.44) | | |
| Panel 4 | 3728 | | 3729 | |
| Black group evaluation | | | | |
| High | | 0.70 (0.50, 0.99) | | 0.66 (0.38, 1.12) |
| Moderate | | 0.91 (0.69, 1.18) | | 0.81 (0.47, 1.37) |
| Low | | <i>ref</i> | | <i>ref</i> |
| Racial discrimination | | 1.28 (1.12, 1.46) | | |

Each panel reflects a separate model.

All estimates incorporated complex survey weights.

Ns are unweighted.

Models adjusted for age, sex, marital status, education, annual household income, and employment status.

^a Additionally adjusted for racial discrimination

Supplemental Table 3.2: Sensitivity analyses for effect modification of racial identity on lifetime and past-year MDD by Caribbean ethnic origin using cross-product terms, US-born sample of the National Survey of American Life

| | Lifetime MDD ^a | | Past-year MDD | |
|--|---------------------------|-------------------|---------------|--------------------|
| | N | PR (95% CI) | N | PR (95% CI) |
| Panel 1 | 3703 | | 3706 | |
| Closeness to Black Americans | | | | |
| High | | 0.67 (0.49, 0.92) | | 0.75 (0.51, 1.10) |
| Moderate | | 0.68 (0.48, 0.98) | | 0.52 (0.29, 0.94) |
| Low | | <i>ref</i> | | <i>ref</i> |
| Ethnic origin | | | | |
| Non-Caribbean | | <i>ref</i> | | <i>ref</i> |
| Caribbean | | 1.86 (0.91, 3.80) | | 2.62 (1.18, 5.80) |
| Closeness to Black Americans x Ethnic origin | | | | |
| High x Caribbean | | 0.65 (0.35, 1.20) | | 0.19 (0.05, 0.72) |
| Moderate x Caribbean | | 0.97 (0.33, 2.88) | | 0.22 (0.05, 0.97) |
| Low | | <i>ref</i> | | <i>ref</i> |
| Racial discrimination | | 1.29 (1.14, 1.47) | | |
| Panel 2 | 3762 | | 3765 | |
| More important to be Black or something else | | | | |
| Being Black | | 0.72 (0.50, 1.04) | | 0.95 (0.58, 1.58) |
| Something else | | <i>ref</i> | | <i>ref</i> |
| Ethnic origin | | | | |
| Non-Caribbean | | <i>ref</i> | | <i>ref</i> |
| Caribbean | | 1.61 (0.99, 2.62) | | 1.71 (0.78, 3.74) |
| More important to be Black or something else x Ethnic origin | | | | |
| Being Black x Caribbean | | 1.71 (0.93, 3.17) | | 1.79 (0.33, 9.79) |
| Something else | | <i>ref</i> | | <i>ref</i> |
| Racial discrimination | | 1.28 (1.13, 1.46) | | |
| Panel 3 | 3701 | | 3703 | |
| Shared fate with Black Americans | | | | |
| Yes | | 1.05 (0.84, 1.32) | | 0.80 (0.56, 1.15) |
| No | | <i>ref</i> | | <i>ref</i> |
| Ethnic origin | | | | |
| Non-Caribbean | | <i>ref</i> | | <i>ref</i> |
| Caribbean | | 0.73 (0.39, 1.36) | | 0.62 (0.28, 1.38) |
| Shared fate with Black Americans x Ethnic origin | | | | |
| Yes x Caribbean | | 3.05 (1.64, 5.68) | | 4.44 (1.58, 12.50) |
| No | | <i>ref</i> | | <i>ref</i> |
| Racial discrimination | | 1.27 (1.11, 1.44) | | |
| Panel 4 | 3728 | | 3729 | |
| Black group evaluation | | | | |
| High | | 0.68 (0.47, 0.96) | | 0.63 (0.36, 1.11) |
| Moderate | | 0.88 (0.67, 1.15) | | 0.76 (0.44, 1.32) |
| Low | | <i>ref</i> | | <i>ref</i> |
| Ethnic origin | | | | |
| Non-Caribbean | | <i>ref</i> | | <i>ref</i> |
| Caribbean | | 0.98 (0.43, 2.25) | | 0.90 (0.19, 4.41) |

| | | |
|--|-------------------|--------------------|
| Black group evaluation x Ethnic origin | | |
| High x Caribbean | 2.48 (0.67, 9.16) | 2.66 (0.30, 23.52) |
| Moderate x Caribbean | 2.12 (0.69, 6.48) | 3.18 (0.54, 18.89) |
| Low | <i>ref</i> | <i>ref</i> |
| Racial discrimination | 1.28 (1.12, 1.46) | |

Each panel reflects a separate model.

All estimates incorporated complex survey weights.

Ns are unweighted.

Robust Poisson models adjusted for age, sex, marital status, education, annual household income, and employment status.

^a Additionally adjusted for racial discrimination

Conclusion

“One of the things that I think would have helped, maybe, growing up — I just never really remember having conversations with my parents about how to reconcile the two things [being Nigerian and Black]. And now when I think about it, I’m just like, ‘My mom should have helped me out.’ I don’t know that I even approached my mom about something like that. Because when I was at home I was Nigerian . . . I guess my mom didn’t anticipate that I would be going through such a thing.”

– Emeka, 27-year-old second-generation Nigerian (in Awokoya, 2012)

In 1972, Bryce-Laporte described Black immigrants facing a “double invisibility” in US society.²⁰⁹ That is, immigrant narratives rarely include experiences of Black immigrants and Black narratives rarely include the experiences of immigrants. The purpose of this dissertation was to better understand within-group differences in depression and related symptoms, while simultaneously highlighting the “double invisibility” still present, leveraging the scant data sources that disaggregate the US Black population.

A systematic review of the literature (Chapter 1) sought to rigorously characterize the heterogeneity of depression and its related symptoms within the US Black population; not just by nativity contrasts, but also by other immigration-related domains that capture heterogeneity within the foreign-born (e.g., by region of birth), as well as within the US-born (e.g., by ethnic origin, immigrant generation). Analytical aims of this dissertation centered racialization processes towards explaining within-racial differences along immigration-related domains. First (Chapter 2), an indirect test consisted of modeling growth curves of depressive symptoms between first/second-generation immigrants and the third+ generation. And second (Chapter 3), a direct test examined the role of racial identity towards explaining the heightened prevalence of MDD among US-born Caribbeans compared to all other US-born Black Americans.

In Chapter 1, the systematic review characterized the prevalence of depression and its related symptoms separately for adults (aged 18 or older) and youth (younger than 18 years). Among adults, there was evidence of substantial variation in these outcomes by nativity, region of birth, and Caribbean origin. Studies disaggregating by nativity generally showed lower levels of depression or related symptoms among the foreign-born relative to the US-born, which is consistent with patterns characterized by the “healthy immigrant effect.”¹²⁶ By region of birth, African-born immigrants, relative to both US-born Black Americans and foreign-born Black immigrants from other regions, appeared to have the lowest levels of symptoms across studies. Lastly, studies disaggregating by Caribbean origin found minimal differences between Caribbean and non-Caribbean Black Americans across depression and related symptoms. However, studies that further disaggregated Caribbeans by nativity revealed the heightened prevalence of depression among the US-born and the lower prevalence among the foreign-born, relative to non-Caribbeans. Differences among youth were most apparent by immigrant generation and parent nativity, where first-generation youth and youth with at least one foreign-born parent (i.e., first- and second-generation combined) had the lowest levels of the outcomes under study.

Most studies included in the review drew from NSAL data, which focused on two ethnic subgroups of the US Black population — those of recent Caribbean origin (first-, second-, and third-generation immigrants) and those without. Given the limited data that do disaggregate the US Black population, even less is known about patterns among other ethnic-immigrant subgroups. As the ethnic diversity of the Black population continues to increase, it is important that future mental health surveillance studies not only collect data that disaggregate by immigration-related domains, but also include non-Caribbean immigrant populations such as those of recent sub-Saharan African origin and Latin American origin.

Chapter 1 also summarized proposed mechanisms underlying differences in depression and its related symptoms within the US Black population. While artefactual explanations like those stemming from measurement error or selection bias were proposed, other studies proposed etiologic explanations related to subgroup differences in racial socialization. This chapter also proposed two promising areas of future research elucidating within-group differences. The first concerns the racial context hypothesis toward better understanding depression variation by region of origin, particularly within foreign-born immigrants. The second concerns how subgroup variation in racial socialization experiences may explain the heightened prevalence of depression among US-born Caribbeans relative to African Americans.

Chapter 2, the first of two analytical aims, examined whether longitudinal trajectories of depressive symptoms across the life course varied by immigrant generation contrasts (first/second-generation compared with third+ generation) among Black youth followed into adulthood. This aim additionally assessed whether contrasts among Black respondents were consistent with contrasts across other racial/ethnic groups (Asian, Hispanic/Latinx, and white respondents). Using data from the first four waves of Add Health, which included data from respondents aged 13 to 32, Black respondents did exhibit unique variation in depressive symptom trajectories. Trajectories followed a U-shape for both first/second-generation and third+ generation respondents, where symptoms were lowest during the early 20s for both subgroups. First/second-generation immigrants, however, had a steeper decrease from adolescence into the early 20s as well as a steeper increase into the early 30s. U-shaped trajectories were only consistent among other racialized minorities (Asian, Hispanic/Latinx). Additionally, only among first/second-generation immigrants did trajectories indicate a steeper increase into adulthood compared with their third+ generation counterparts. One potential

explanation concerns variation in vulnerability to and resilience against depression; both of which may point to variations in the racial socialization process between first/second-generation immigrants and the third+ generation.

Toward better understanding the heightened prevalence of MDD among US-born Caribbeans relative to all other US-born Black Americans in the NSAL, Chapter 3 assessed whether the relationship between four measures of racial identity and MDD varied between these two groups. This study found evidence of effect modification where racial identity was either less protective for Caribbeans relative to non-Caribbeans or detrimental for Caribbeans only. Findings among Caribbeans contrast with an established literature supporting racial identity as a protective factor against symptoms related to depression.^{30-35,181-183} As in Chapters 1 and 2, variation in racial socialization processes was also proposed as a potential mechanism.

Chapters 2 and 3 were distinct in terms of source populations and outcomes, in part due to limitations in the available data disaggregating Black Americans. Yet despite these distinctions, these chapters offered a fuller picture of how immigration-related constructs lead to variations in depression and its related symptoms. Chapter 2 drew from Add Health, where the source population was Black youth followed into adulthood, disaggregated by immigrant generation. Chapter 3 drew from the NSAL, where the source population was US-born Black adults disaggregated by Caribbean ethnic origin. These chapters also focused on two different outcomes — depressive symptoms in Chapter 2 and MDD in Chapter 3. It should be noted that national epidemiologic studies have found Black Americans (largely made up of African Americans), relative to white Americans, have both a lower prevalence of MDD and higher levels of depressive symptoms and distress.⁴⁷ Together, these chapters provide new insight into

how immigration complicates mechanisms causing depression and its related symptoms within the Black population.

It is possible that there is further variation in MDD and related symptoms within the US Black population by immigration-related domains not captured in this dissertation. For example, this dissertation was not able to isolate second-generation adults, 1.5-generation adults, and Black Hispanic/Latinx populations due to data limitations. Related, this dissertation was also unable to study religion or language as disaggregation domains. Lastly, while NSAL data have shown additional variation by Caribbean ethnicity and demographic characteristics like sex and age,^{85,90,92} prohibitively small sample sizes undermined analyses of further disaggregation.

Drawing from the social stress process, findings from this dissertation suggest social stressors associated with structural disadvantage appear to affect members of the US Black population differently. Among Black Americans specifically, racial socialization and racial identity are purported to promote resiliency in the face of racialized social stressors like racial discrimination.³⁰⁻³⁵ Indeed, some researchers have suggested racial socialization and racial identity formation may explain the lower prevalence of MDD among Black Americans relative to white Americans.^{94,171,210} Yet much of what we know about these sources of resiliency come from predominantly African American samples. While ethnographic research finds variation in these racialization processes between those with and without immigrant parents,^{36-41,43,45,46} few studies have examined how these processes relate to variation in subsequent depression or its related symptoms. This dissertation offered the first empirical tests of whether differences in racialization processes may help explain inter-ethnic differences in depression among Black Americans — indirectly through growth curve modeling in Chapter 2 and directly through effect modification hypotheses of ethnic origin and racial identity in Chapter 3. Given the findings of

this dissertation, and particularly the findings in Chapter 3, future research with richer measures of racial identity are needed to more rigorously examine why ethnic origin appears to modify the relationship between racial identity and MDD.

Findings from this dissertation may also inform future data collection and surveillance efforts. Given the “double invisibility” of Black immigrants in population health literature, it follows that few data sources disaggregating the US Black population exist. Future data collection efforts with large enough sample sizes to disaggregate the US Black population by immigration-related domains like nativity, region of birth, immigrant generation, and ethnic origin could enable a better understanding how these function as sources of depression variation in several ways. First, these data could capture high-risk groups that would otherwise be masked, such as US-born Caribbeans identified in the NSAL. Second, these data would allow researchers to explore whether the observed heterogeneity in this dissertation exists under more recent data. Third, these data would allow researchers to explore whether the observed heterogeneity is consistent across domains studied. For example, a future research question related to Chapter 2 could address whether trajectories of depressive symptoms from youth to adulthood among first/second-generation immigrants are consistent by ethnic origin (e.g., Caribbean-origin, sub-Saharan African-origin, etc.). Similarly, a future research question related to Chapter 3 could examine whether observed positive associations between racial identity and MDD among US-born Caribbeans extend to other US-born Black Americans of recent immigrant origins.

As Black immigrants continue to contribute to both the growth and ethnic diversity of the US Black population, it is increasingly important that future work captures and seeks to understand within-group heterogeneity in depression and related symptoms. Given the evidence this dissertation provides of varying mechanisms underlying depression and related symptoms, it

follows that treatment strategies accounting for sociocultural variation within the US Black population may be more effective than those that do not. However, additional studies that attend to how and why racialization processes may manifest into variations within Black Americans are needed toward better informing interventions. Following this dissertation, these future efforts may continue to shed light on a critical, under-researched dimension of the Black immigrant adaptation experience.

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