The impact of residential instability and serious psychological distress on substance use disorders: Testing exposure and vulnerability hypotheses for Non-Hispanic White, African American, and Latinx populations

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Matthew E. Archibald [1]
College of Mount Saint Vincent
6301 Riverdale Ave., Riverdale NY 10471, USA
Matthew.archibald@mountsaintvincent.edu

Pamela Behrman
College of Mount Saint Vincent
6301 Riverdale Ave., Riverdale NY 10471, USA

Jordan Yakoby
College of Mount Saint Vincent
6301 Riverdale Ave., Riverdale NY 10471, USA

John McCullagh
College of Mount Saint Vincent
6301 Riverdale Ave., Riverdale NY 10471, USA

Aydasara Ortega Torres
College of Mount Saint Vincent
6301 Riverdale Ave., Riverdale NY 10471, USA

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[1] Corresponding author

Extended abstract

Objectives/ Summary:

The aim of this study is to extend the social stress model, testing whether and how substance use disorder is related to stress and its precursors, using the National Survey of Drug Use and Health, a well known source of national behavioral health estimates. We examine two foundational hypotheses underlying the model: 1) that greater exposure to stressors (i.e., residential instability) among racial/ethnic minority populations results in higher levels of stress (i.e., serious psychological distress), which in turn increases the likelihood of substance use disorder; 2) that greater vulnerability among minority populations to stressors such as residential instability exacerbates the impact of these conditions on stress as well as the impact of stress on substance use disorder. Using NSDUH population estimates of residential instability, serious psychological distress and substance use disorder, for selected sample years 2005-2014 (replicated in 2015-2019), we constructed and analyzed several models, the results of which provided mixed support for the vulnerability (moderator or conditional process) hypothesis, but not for the exposure (mediation) hypothesis. For example, while the direct effects of residential instability on serious psychological distress are greatest for Blacks, these effects are not translated via serious psychological disorder into racial/ethnic differentials in substance use disorder, due largely to the fact that Whites report higher levels of stress relative to Blacks and Latinx. Ongoing development of the paper will involve specifying and analyzing reasons underpinning these results. The study's key contributions are: 1) extend the social stress framework by including residential instability as stressors and substance use disorders as outcomes (Aneshensel and Mitchell, 2014), in order to: 2) test exposure and vulnerability hypotheses in minority populations (Wheaton, 2010); 3) develop and test the causal linkages in the hypothesized processes, based on innovations in general structural equation models which foster examination of the simultaneous nature of direct and indirect effects, and the dual role the mediator (stress) plays as both a cause for the health outcome (substance use disorder- see e.g., Rajita 2008) and an effect of stressors (residential instability) (See Figures 1 and 2, below), and lastly; 4) generate national population estimates of these linkages which are understudied in this kind of causal framework.

Background:

The social stress model provides an explanatory framework for understanding differences, if not disparities, in mental health outcomes between majority and vulnerable populations (Botha and Frost, 2020; Meyer 2003). In our study, stressors are expected to impact mental health outcomes, defined as serious psychological "stress" which in turn will impact behavioral health, defined as substance use

disorder. There is a considerable literature on how stress impacts substance misuse to which we refer the reader (see for review e.g., Ruisoto and Contador 2019; also, Rajita 2008; SAMHSA 2020).

The central premise of the social stress model is that systems of stratification (e.g., race/ethnicity, socioeconomic class, gender, age), and social institutions underpinning those systems place a greater psychological and emotional burden on some groups, those with fewer socioeconomic and cultural resources relative to others, with dire consequences. The racial/ethnic- minority stress variant of the framework emphasizes the unique importance of race and ethnicity in social hierarchies, documenting the corroding effects of stigma and discrimination as well as socioeconomic disadvantage on mental health (Hatzenbuehler, Phelan and Link, 2013). Naturally, the intersection of race, health, SES, and social class is complex. Research shows multiple pathways from SES and race/ethnicity to health; one such pathway is through differential exposure to chronic stress and its resulting biochemical toll. (Adler and Rehkopf, 2008). Vulnerable groups (e.g., racial and ethnic minorities) are at greater risk of exposure to disruptive life events, situations and conditions such as trauma, income strain, discrimination which, given socioeconomic and cultural disadvantage, increase the likelihood of psychological stress, and in its manifestation, "severe distress," and its various mental health disorders, such as anxiety and depression (Luo et al., 2012; Wang et al., 2020; Pascoe et al., 2009; Williams and Mohammed, 2009; Cronholm et al., 2015; Wade et al., 2016; Institute for Safe Families, 2013).

This heuristic is part of a broad social-determinants-of-health research framework which investigates mechanisms by which social disadvantage (and its related vulnerabilities) creates psychosocial stress, in turn, shaping health outcomes (for discussion see e.g., Stuber, Meyer and Link 2008; Williams and Williams-Morris, 2000). While social stress/ minority stress/ social determinants research frameworks overlap and share an intuitive appeal, the nexus of social status, its stressors, stress and health outcomes remains open to theoretical refinement and empirical investigation, especially with regard to racial/ethnic disparities in behavioral health outcomes.

For example, although a number of studies have shown that racial and ethnic minorities have higher rates of exposure to some stressors (Boardman, 2011), other research (e.g., Schieman and Reid 2009) argues that social advantage carries its own share of stressors. Differences in health outcomes for the two, in the face of ubiquitous stress, are explained by disparities in resource access: those who function at higher ends of the social hierarchy have more resources to meet the demands of their stressors (e.g. Epel et al., 2018), and are better positioned to manage their stress, with fewer detrimental consequences (e.g. Sapolsky, 1994; Marmot et al., 1991). The question, in a narrow sense, then, hinges on the extent to

which types of social conditions constitute a "stressor," and which resources impact their effects, in order to determine the scope of relevant so-called exposure hypotheses (see e.g., discussion of exposure in Turner et al., 1995). In our study, residential instability, a key economic determinant of health is expected to have a strong negative impact on individual mental health, measured by serious psychological distress, and substance use disorder (Jones 2004; Office of Disease Prevention, 2022; Park and Seo 2020).

In contrast, a vulnerability hypothesis suggests that it is not simply exposure to certain social conditions which determines variation in stress between populations, but the extent to which some groups are more susceptible to the debilitating potential of certain classes of stressors. In both processes, exposure and vulnerability, the underlying assumption posits material and cultural resource advantage which functions as a buffer between stressors and their detrimental impact on mental health, and the consequences of that impact. Presumably, resource advantages also mitigate the effects of the stress response on other health outcomes, including mental health and medical co-morbidities, such as substance misuse, so that those at a disadvantage, with fewer resources, are at greater risk to experience the damaging impact of stress on their health and wellbeing.

Before examining resource interventions, however, we argue for a fuller examination of the question of stressors-stress-outcomes. To do so, this study analyzes racial/ethnic variation at the nexus of residential instability, its impact on serious psychological distress and substance use disorder. We advocate the use of the National Survey of Drug Use and Health, because it is the chief source of SAMHSA estimates of behavioral health in the US. With these data, based on prior theory and empirical findings, we develop and test minority stress -exposure and -vulnerability hypotheses.

Prior research has shown that racial/ethnic minority populations are 1.5 to 2.0 times more likely than Whites to have most of the major chronic diseases (Adler and Rehkopf, 2008; Almanac of Chronic Diseases, 2008), and also more likely to be at greater risk to experience some forms of distress relative to other groups (Williams, 2018). Although research shows chronic illness is associated with higher levels of stress (e.g., Swartz and Jantz, 2014), it remains for the current study to clarify how race/ethnicity structure the relationship. Alternatively, in light of the failure of early stress research to fully support an exposure hypothesis (Aneshensel and Mitchell 2014), the vulnerability hypothesis remains a viable explanation for (some) observed population differences in health outcomes. The claim underlying the vulnerability model is that exposure per se does not necessarily generate population differences in stress responses because stress is a ubiquitous social experience. Rather, some populations are more vulnerable than others to the impact of stressors which emerge as variation between groups in stress responses. In

terms of modeling, the exposure hypothesis suggests a mediation model in which stressors explain differences in racial/ethnic variation in stress which then account for racial/ethnic variation in health outcomes. The vulnerability hypothesis suggests a model in which race/ethnicity exacerbates these relationships. The classic form of this model is to statistically assess the degree to which race/ethnic moderate these relationships.

While Aneshensel and Mitchell (2014) call for research to more fully examine mediating and moderating models, their discussion of these two types of models focuses on resources as mediators and moderators, rather than the simpler model we propose as a necessary first step in developing more complicated resource-focused frameworks. As support for taking this remedial step, we turn to Williams (2018) and others who have observed that mental health outcomes, whether stress related or not, are not very robust with respect to race and ethnicity. Moreover, research on substance use disorders typically shows varying SUD/AUD rates among different racial and ethnic groups, depending on substance (Center for Behavioral Health Statistics and Quality 2021, based on NSDUH 2015-2019). This study tries to help clarify those relationships before moving in the direction of more complicated models.

Methods:

Data and study population

To understand how minority stress functions as a consequence of residential instability and as a precursor to substance use disorder among non-majority racial and ethnic populations, we examine socioeconomic and mental health data from the NSDUH (2005-2014, as well as 2015-2019)[2], a nationally representative sample comprising the US population's behavioral health information (SAMHSA 2019). NSDUH data serve as a preeminent source of yearly US incidence and prevalence estimates of behavioral health, including measures of major depression, anxiety, schizophrenia, substance use disorders and serious psychological distress. The data cover a variety of health conditions as well as socioeconomic and demographic characteristics. Our sample for 2005-2014 consists of 348,901 adult respondents. Following previous studies, we operationalized racial and ethnic group membership based on self-identified race-ethnicity: Latino/a/Hispanic, White (non-Latino/ non-Hispanic), and Black (non-Latino/ Non-Hispanic). Since we cannot adequately theorize about stress and substance use disorder for other racial-ethnic groups such as Asian Americans, Native Americans, and mixed racial-ethnic groups, and because their sample sizes diminish rapidly, these populations have been excluded from analyses.

[2] We discuss the reasons for focusing on these particular early years in a more extended version of this document.

Measures

Residential instability. Prior research indicates that residential instability is related to stress (Office of Disease Prevention, 2022) and substance use disorders (Park and Seo 2022). NSDUH asks respondents to indicate how many times they have moved in the past year. Because only a small proportion of the population moves more than several times in a year, NSDUH set the upper limit on this measure to six moves or more. In the general structural equation models we examine groups falling into all seven categories from no moves (0) to six of more (6). In the decomposition analyses, we analyze differences between those who did not move (0), moved once (1) or moved more than once (2).

Serious psychological distress. While stress is a physiological and psychological response (with positive or negative valence) to internal or external stressors, affecting biochemical and psychological systems and influencing how people feel and behave, distress is delineated as severe negative affect and physiological reactivity, sometimes conflated with mental illness (Goldberg 2000). The Kessler 10 and Kessler 6 scales were developed to assess an individual's emotional state with respect to with this affect. As described in detail in Kessler et al. (2003), the scales were designed to be sensitive around the threshold for the clinically significant range of nonspecific distress in an effort to discriminate cases of serious mental illness (gleaned from other measures). The NSDUH uses a version of the K6 scales, asking respondents to imagine their worst month during the year, then describe how often they felt- restless, nervous, hopeless, no good, burdened by effort, and couldn't be cheered up. The SPD scale ranges from 0 (none of the items, none of the time) to 24 (all of the items, all of the time).

Substance use disorder. The NSDUH surveys assess substance use disorders based on the diagnostic guidelines for substance dependence and substance abuse found in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). The DSM-IV provides a classification system for clinicians, insurance providers, researchers, and policymakers to use in matters related to diagnosing, researching, and treating behavior health conditions including Sustance use disorders . In the NSDUH, survey respondents answer detailed questions about substance use behavior, and, for these meeting DSM criteria, the answers to which are categorized as indicating substance dependence or abuse for each of the following substances: alcohol; marijuana; cocaine (including crack); heroin; hallucinogens; inhalants; and prescription pain relievers, stimulants, sedatives, and tranquilizers. In 2013 measurement of substance use disorders was changed based on changes to the DSM-5. The DSM-5 revision contained changes in organization and changes to the diagnostic criteria for nearly every DSM-IV disorder, including those for substance use disorders . These changes prompted a revision process to redesign and update NSDUH to provide high-quality data on Substance use disorders that reflect the DSM-5 criteria. As a consequence, substance dependence and abuse are no longer continuous pre-2014 and post-2014 when the new criteria were instituted. We

therefore selected our pooled sample, and primary outcome measures based on this consideration. Further, for the initial set of analyses, we focused on alcohol and drug dependence rather than abuse because withdrawal demarcates a physical dysfunction and its measure may be less prone to interpretative error on respondents' part. In later analyses, we examine both alcohol and drug abuse measures.

Covariates. We include in our models a number of covariates that may influence the relationships between race/ethnicity, stressors, stress and substance use disorder. These are: age, gender, SES, and marital status. Although the tables below do not show estimates for these covariates, all models have been adjusted for them.

Analytic Strategy

A review of the statistical methods commonly used to analyze the relationships identified in the minority stress framework shows a mix of linear models as well as structural/ simultaneous equation models (SEMs). Researchers use SEMs because the former can be problematic with regard to establishing cause-effect relationships due to endogeneity, multi-collinearity among explanatory variables, and erroneous handling of non-normal and non-continuous distributions of response variables. Except for multi-collinearity, our data share all of these challenges. Therefore, our approach relies on the principles underlying SEM. Moreover, NSDUH data are culled from the population through a complex stratified sampling scheme further taxing the underlying assumption of normality on which most least squares models depend (see discussion NSDUH Methodological Resource Book 2018). To meet these various conditions, we propose an approach based on modification of SEMs for non-normal variables (see Muthen 1984 for discussion of non-normality in SEMs). These are generalized structural equation models (GSEM). GSEMs combine the power and flexibility of both SEM and linear models. The variables in the following analyses are observed, not latent, and therefore the standard simultaneous equation reduces to an econometric-type path model. That is, there are several variables the serve as predictors of some variables, yet are predicted by others. This holds for both the mediation and moderation models. The simultaneous mediation model constructed for GSEM analysis can be described by:

$$x' = a'_0 + a'x'' + \Sigma_k e_k m_k \qquad \text{[path a']}$$

$$z = a''_0 + a''x' + \Sigma_k e_k m_k \qquad \text{[path a'']}$$

$$y = b'_0 + bz + \Sigma_k e_k m_k \qquad \text{[path b]}$$

$$y = b''_0 + c'x'' + b_1x' + b_2z + b_3y + \Sigma_k e_k m_k \qquad \text{[path c']}$$

where each path, a', a'', b and c' are linked to coefficient estimates (b'', c', b_1, b_2, b_3) based on the specific type of distribution for each x'', x', z, y, (i.e., Gaussian, Bernoulli and Bernoulli, respectively).

The $\Sigma_k e_k m_k$ are the covariates and error terms. We ran two versions of the model: a constrained version and an unconstrained one. A potential causal (indirect) mediation effect was then estimated using the product of coefficients method (MacKinnon, Fairchild, and Fritz, 2007). A bootstrap analysis with 1,000 replications was applied to estimate the average causal mediation effects without requiring the assumption of normality (Preacher & Hayes, 2008). With a bias-corrected bootstrap technique, the total, direct and indirect (mediation) effects and their 95% CIs were estimated.
We ran the same model for the moderation analysis, except that instead of x'' (race-ethnicity), the first term in the model was x', chronic illness (stressor) and the model varied by subpopulation. Again, we examined direct and indirect effects for each of the subpopulations and tested whether or not the paths, a, b and c' differed significantly between our sub groups. Note that there are two outcomes we test with this model. One version is for alcohol dependence, the other is for drug dependence.

The program we use to estimate the equations is Stata 17 (further details will be incorporated in later version- not shown).

Results:
Figures 1 presents the exposure/ mediation model. We tested two versions of the model. The first is a constrained model in which race and ethnicity are expected to influence substance use disorder by way of residential instability which impact serious stress. The broken line between race/ethnicity and substance use disorder indicates that the direct effect of minority group membership is constrained to operate through residential instability (results not shown). In the unconstrained model, race and ethnicity are freed to impact all three factors in the model. Estimates of the direct and indirect effects shown in Table 2.

The advantage of structural models is that they estimate parameters simultaneously rather than piecemeal. Moreover, in this system of equations, the size of direct and indirect effects of the parameters can be estimated. Overall, while the system of relationships between residential instability->stress->substance use disorder was supported with these data for the entire sample, its function explaining why racial and ethnic minorities have (slightly) higher rates of substance use disorder (dependence) was not supported.

As Table 2 indicates, the exposure model did not find support in these data. Blacks and Latinx were no more likely than Whites to have an alcohol dependence disorder (OR 1.01 p<.910, OR 1.06 p<.095, respectively), although Blacks were more likely than Whites to have a drug dependence disorder (OR 1.26 p<.000), and Latinx least likely (OR .779 p<.000). To explain the latter differential with an exposure model, psychological distress would have to be higher for Blacks, as would residential instability, as the

exogenous precursor to stress. However, contrary to expectations, Blacks and Latinx were less likely to experiences stress than Whites (OR .456 and .411, respectively, p<.000), and, similarly, Blacks and Latinx were not significantly more likely than Whites to experience residential instability, adjusting for other factors (OR .982 and 1.02, respectively, p<.096 and p<.133). As the stress literature indicates, there may be a number of reasons that higher status groups are more likely to experience stress or perhaps to express their stress reactions (see e.g., Schieman and Reid 2009). Further exploration of these relationships is now underway with these data in order to determine why a workable exposure model failed to show racial/ethnic differences is stressors and stress. [3]

Figure 2 presents the vulnerability/moderation model. The basic idea is that to the extent that race and ethnicity structure the relationships in the system, they should reveal a greater vulnerability to the effects of residential instability (even if they are no more likely than Whites to move) and stress (even if their level of stress is lower than Whites).

Table 3 provides two sets of statistics to assess this model. We examine whether the parameter estimates of factor effects are significant for each of the three groups. Then we constrain the parameters and evaluate whether they are significantly different (greater or less) than one another. We can also decompose those effects into direct and indirect effects in order to determine whether the model operates the same for each group. Unlike the mediation hypothesis, there is some support for expectations of vulnerability, although the pattern of findings is complicated.

In brief, with regard to the impact of stress on substance use disorder, based on Wald adjusted F tests, Whites had greater odds of having a stress-related drug dependence disorder— but not alcohol— than Blacks (OR 1.09 p<.000) and Latinx (OR 1.07 p<.017). Latinx themselves were less likely than Blacks to see their stress result in a substance use (OR 1.07 Pp<.048). Moving back in the model, Latinx were least likely to experience stress as a result of residential instability than Blacks (OR 2.77 versus 3.55 p<.000) and Whites (OR 3.00). The difference between Blacks and Whites was not significant. Here the vulnerability hypothesis finds some support although why the link between Black residential instability and stress does not translate into higher greater substance use disorder in not apparent.

With respect to the path from residential instability to substance use disorder, there were no differences between racial and ethnic groups. That is, Whites were no more likely than Blacks or Latinx to see their

[3] Explicit two-way interactions between race-ethnicity and age, SES did not alter the findings.

residential instability directly translated into either alcohol dependence or drug dependence (although we know that for the entire sample, residential instability does impact both alcohol and drug dependence.

As for decomposing the effects of each path across the three populations, percentages in Table 3 indicate how much of the total impact of chronic illness on substance use disorder operates via serious psychological distress. The pattern is not at all similar for Whites and Blacks and Latinx depending on the number of residential moves. For example, for Whites with no moves (relative to more than 1), 42.3 percent of the effect of race operates through serious psychological stress whereas for Blacks it is almost 70 percent (69.8 percent), yet for Latinx it is almost 21 percent (20.7 percent). This ratio is the same for Whites but drops to below 50 percent for Blacks with one move – relative to none-- (48.3 percent) or many (43.9 percent) which is about the same size as the indirect path for Latinx (36.9 percent for one move, 40.7 percent for more than one move), indicating that moving seems to have more of a direct effect on substance use disorders for blacks but not through psychological distress.

Again, as with the exposure model, analyses are currently underway to expand our understanding of these relationships, including tests of three -way interactions for example, between race-ethnicity, stress and age.

Conclusions:

This study explored two simplified hypotheses related to racial and ethnic minority health outcomes: greater exposure to stressors and stress leads to substance use disorders, and, greater vulnerability to stress exacerbates the impact of stress on substance use disorders. The underlying premise of the stress process framework is that stress is a major social determinant of health, with direct and indirect effects on it. The direct relationship between stress and health outcomes is the effect of stress on human physiology. The long-term stress hormone, cortisol, is believed to be the key driver in this relationship. Chronic stress is significantly associated with chronic low-grade inflammation, slower wound healing, increased susceptibility to infections, and poorer responses to vaccines (Gouin 2011; Miller, Chen, Zhou, 2007). Stress also has an indirect effect on substance use disorders, by way of strain on material and psychological resources. As we have shown, serious psychological distress increases the odds of substance use disorder. While any number of traumatic life events may create the conditions for a substance use disorder, in this study we examined residential moves and their impact on stress. Individuals with more moves were expected to experience greater levels of stress when compared to those

with greater stability. Our expectation that this mediation model would hold for non-majority racial and ethnic groups, relative to Whites was not supported by our results (so far). Blacks and Whites and Latinx all reported similar patterns of residential moves. As for greater levels of minority stress, Whites had greater odds of endorsing the experience of it than Blacks and Latinx, although they also had significantly lower odds of having a drug dependence disorder. This suggested, alternatively, that perhaps the explanation for greater minority substance use disorder is that minority populations are more vulnerable to stressors themselves. Moderation analysis showed that this was in part the case. Although stress was less likely to translate into substance use disorders for Blacks and Latinx relative to Whites, Blacks who had prior residential moves were at significantly greater risk to experience serious psychological distress (although this did not seem to translate into a substance use disorder).

Discussion:

There are a number of limitations that may motivate future research. We are in the process of addressing these as we revise this study. Resources and access to care are two central processes linking the elements of the stress model. To what extent does caregiver or close family support for minority respondents mitigate the stress response? In addition, to what extent does the stigma – especially in minority communities – associated with mental health issues cause underreporting of stress? We posited that perhaps the higher minority drug dependence disorder is due to higher vulnerability but we are not sure from which factors this vulnerability originated. Our results indicate that Blacks and Latinx experience less stress compared to Whites, yet are more vulnerable to substance use disorder. These results are paradoxical. It is likely there are differences in risk/protective factors among Blacks and Latinx when compared to Whites. Our next step is to focus on the unique factors among Blacks and Latinx that impact the relationship between chronic stress and substance use disorder. Factors such as coping style and social support have been found to be important variables in the context of health and stress (Cwikel 2010; Cohen and Garth, 1984). Additional analyses with these data will help bring a greater understanding of Blacks and Latinx resilience and coping. There are multiple conditions which might lead to greater vulnerability to substance use disorder (Lustig and Strauser, 2007), and our study highlights the need for further study examining those that are behind the greater vulnerability to substance use disorder among Black and Latinx groups (for some specific types of disorder). Our goal is to provide as thorough an explanation to these findings. Some of these analyses (of resources primarily) are being developed currently for the next version of this paper.

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Figure 1: Minority stress mediation model (constrained)

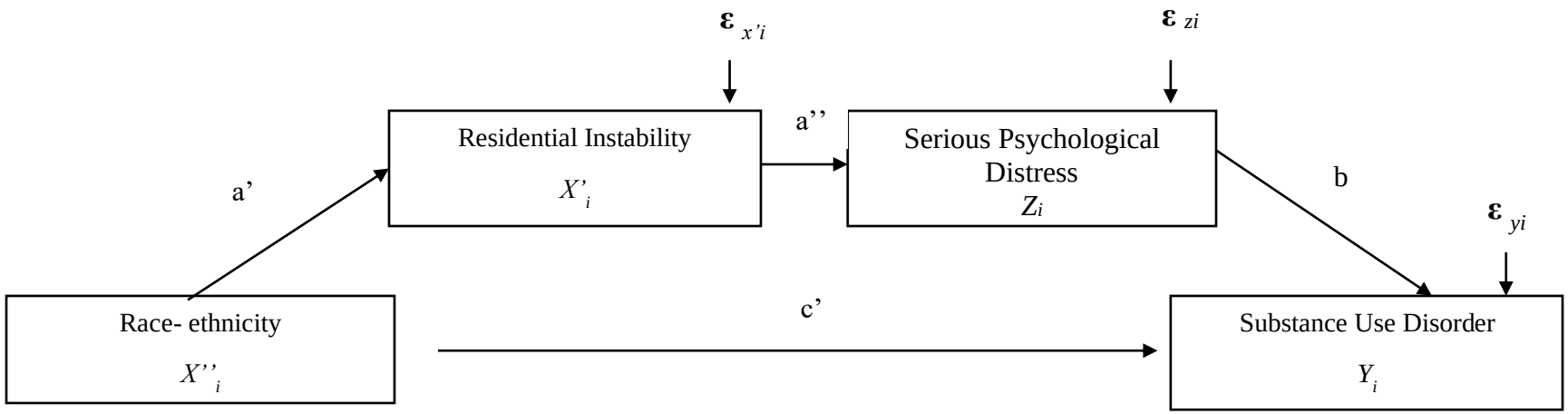

Figure 2: Minority stress moderation model (conditional process)

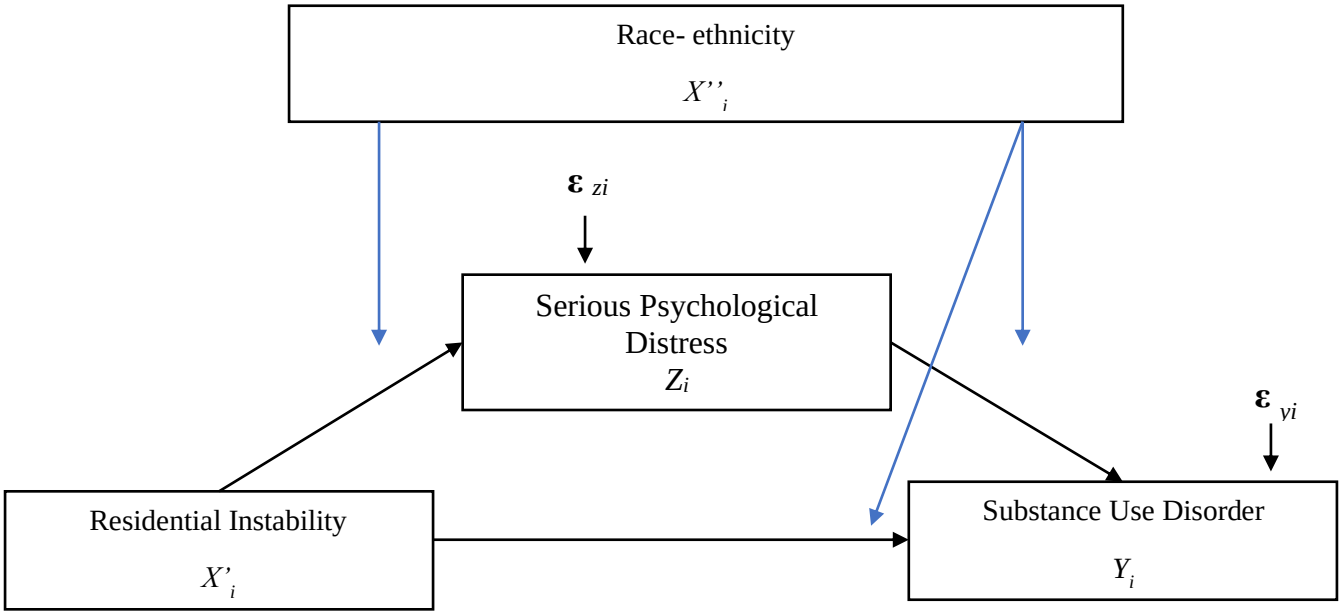

Table 2: Generalized structural equation models; adjusted odds ratios and standard errors [a] for residential instability, serious psychological distress, and substance use disorder; NSDUH 2005-2014 [b]

Unconstrained Mediation Model		SUD- Alcohol dependence		SUD- Drug dependence	
Endogenous	Exogenous	Odds ratio (SE)	Prob	Odds ratio (SE)	Prob
Substance use disorder (0,1)	← Serious psychological distress (0 thru 24) [c]	1.12(.002)	.000	1.14(.003)	.000
	← Residential instability (0 through 6+) [d]	1.13(.008)	.000	1.16(.009)	.000
	← Race/ ethnicity (Wh (ref), Bl, L)				
	Bl	1.01(.044)	.910	1.26(.057)	.000
	L	1.06(.039)	.095	.779(.039)	.000
Serious psychological distress	← Residential instability (0, 1, 2+)	1.85(.019)	.000	1.85(.019)	.000
	← Race/ ethnicity (Wh (ref), Bl, L)				
	Bl	.456(.018)	.000	.456(.018)	.000
	L	.411(.018)	.000	.411(.018)	.000
Residential instability	← Race/ ethnicity (Wh (ref), Bl, L)				
	Bl	,982(.011)	.096	,982(.011)	.096
	L	1.02(.012)	.133	1.02(.012)	.133
Model Fit – Adjusted Wald (3, 108 df)		3908.65	.000	2400.93	.000
Number of Cases (unweighted)		346,304		346,304	

[a] Odds ratio adjusted for age, socioeconomic status, marital status, gender. [b] Samples weight- and design- adjusted: see series NSDUH releases 2002-2014, 2015-2019 [c] Kessler 6-item distress instrument. Frequency of condition during worst month time t - past year. Includes feeling nervous, hopeless, restless, no good, burdened by effort, and couldn't be cheered up .[d] Residential instability ranges from 0 moves in the past year to 6 or more.

Table 3: Generalized structural equation models; adjusted odds ratios and standard errors [a] for residential instability, serious psychological distress, and substance use disorder; dependence on alcohol, dependence on drugs. NSDUH 2005-2014 [b]

Vulnerability Model - Alcohol					Race/Ethnicity		
Endogenous	Exogenous	Non Hispanic (Wh)	African American (Bl)	Latinx (L)	Contrast	F test (1, 110)	Prob F
Substance use disorder (0,1)	← Serious psychological distress (0 thru 24) [c]	1.07(.002)	1.06(.004)	1.07(.005)	Wh v Bl	0.17	.680
					Wh v L	0.00	.992
					Bl v L	0.11	.737
	← Residential instability (0 through 6+) [d]	.867(.006)	.860(.017)	.884(.018)	Wh v Bl	0.20	.654
					Wh v L	0.72	.399
					Bl v L	1.22	.272
Serious psychological distress	← Residential instability	3.50(.040)	3.55(.100)	2.77(.077)	Wh v Bl	0.26	.612
					Wh v L	50.88	.000
					Bl v L	44.42	.000
Vulnerability Model - Drugs							
Endogenous	Exogenous	Non Hispanic (Wh)	African American (Bl)	Latinx (L)	Contrast	F test (1, 110)	Prob F
Substance use disorder (0,1)	← Serious psychological distress (0 thru 24) [c]	1.09(.002)	1.07(.005)	1.07(.005)	Wh v Bl	16.53	.000
					Wh v L	4.35	.017
					Bl v L	1.77	.048
	← Residential instability (0 through 6+) [d]	.899(.008)	.960(.020)	.892(.023)	Wh v Bl	7.04	.598
					Wh v L	0.08	.905
					Bl v L	4.40	.835
Serious psychological distress	← Residential instability	3.50(.040)	3.55(.100)	2.77(.077)	Wh v Bl	0.26	.612
					Wh v L	50.88	.000
					Bl v L	44.42	.000

[a] Odds ratio adjusted for age, socioeconomic status, marital status, gender. [b] Samples weight- and design- adjusted: see series NSDUH releases 2002-2014, 2015-2019 [c] Kessler 6-item distress instrument. Frequency of condition during worst month time t - past year. Includes feeling nervous, hopeless, restless, no good, burdened by effort, and couldn't be cheered up.[d] Residential instability ranges from 0 moves in the past year to 6 or more.

Table 4: Decomposition of direct and indirect effects in a moderated-mediation model. Percent of total effect of residential instability on substance use disorder mediated by SPD for each level of residential instability by race/ethnicity. NSDUH 2005-2014 [a]

Alcohol dependence (0,1)<- SPD (0 to 24) <-Residential instability (0,1, more than 1 move).

		Entire Sample	Wh	Bl	L	P>|z|
	None	41.8	42.3	69.8	20.7	.000
	One	41.7	42.1	48.3	36.9	.000
	More than one	41.7	42.1	43.9	40.7	.000

Drug dependence (0,1)<- SPD (0 to 24) <-Residential instability (0,1, more than 1 move).

		Entire Sample	Wh	Bl	L	
	None	47.3	46.7	33.3	37.7 [b]	.000
	One	46.8	48.3	39.4	51.1	.000
	More than one	46.6	49.3	41.9	40.0	.000
Number of	Cases (unweighted)	348,901	242,527	46,896	59,478	

[a] Samples weight- and design- adjusted: see series NSDUH releases 2002-2014, 2015-2019 [c] All contrasts between levels of residential instability are significant at p> .000 except for the impact of first move on drug dependence for Latinx.