

**The influence of stress on unhealthy behaviors and depressive symptoms of black and white young adults**

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## **Abstract**

This paper uses data from Waves I-III of the National Longitudinal Study of Adolescent Health (Add Health) to examine the mental health of non-Hispanic black and white young adults. We use latent growth curve modeling to characterize the typical stress trajectories experienced by black and white young adults spanning the bulk of their lives. We identify the following four stress trajectories: 1) relatively stress free; 2) stress peak at age 15 and a subsequent decline; 3) stress peak at age 17 and a subsequent decline; and 4) a moderately high and persistent profile that we call “chronic stress”. Results indicate that black adolescents have significantly higher risk of being in all three of the stressful classes compared to white adolescents. Stress exposure is strongly associated with depression and the race differences in stress profiles account for a modest amount of the observed race differences in mental health. We do not observe any race differences in behavioral responses to stressors; black youth are no more likely than white youth to engage in unhealthy behaviors (e.g., smoking, drinking, or obesity) in response to stress. We provide tentative support for the notion that unhealthy behaviors partially reduce the association between stress and depression for blacks but not whites. These findings contribute to unresolved issues regarding mental and physical health disparities among blacks and whites.

## **Introduction**

Persons exposed to a relatively large number of stressors tend to report worse mental health compared to those who have experienced very few of these events in their lives (Kessler, 1997). Although most of this research has focused on adults, high levels of uncontrollable stressful events are also associated with depressive symptoms among adolescents (Ge, 1994). Because social statuses such as class, gender, and race regularly predict stress exposure (Turner and Lloyd, 1999), social scientists have deemed stress exposure to be a “socially modifiable contingency” (Turner, 2003). In particular, racial and ethnic minorities are consistently shown to be exposed to a greater number of chronic, ambient, and acute stressors and are more likely to face consistent stressful life events throughout their lives (Williams and Collins, 1995; Schulz et al., 2000).

Interestingly, although mental health is linked with stress exposure, and black Americans face a larger number of stressors compared to whites, clear evidence for increased rates of depression among blacks compared to whites remains somewhat tenuous. Recent evidence suggests that black adults face an increased risk of depression (8.0%) compared to non-Hispanic white adults (4.8%) [Pratt and Brody, 2008] but others have shown that this is not always the case (Kessler et al., 1999; Haralson et al., 2002). This relationship is further complicated by age and the timing of stressful life events (George and Lynch, 2003).

Some have argued that the somewhat contradictory relationship between stress and depression among black and white adults can be resolved by accounting for group differences in unhealthy behaviors including drinking and smoking (Jackson et al., 2010). Specifically, moderate use of alcohol and nicotine denote relatively cheap and effective

mechanisms to mitigate the adverse physiological effects of stress; research regularly demonstrates that feelings of anxiety can be reduced and a sense of well-being can be increased with the intake of alcohol and tobacco (Benowitz, 1996; Peele and Brodsky, 2000). The same has been shown for the consumption of fatty and high caloric foods (Dallman et al., 2003b). The use of substances in light of stress exposure is regularly shown among adults (Boardman et al., 2001) and a growing body of work has linked substance use among adolescents with a myriad of stressful events including abuse, work intensity, academic pressure (high school and/or college), relationship and sexuality issues, exposure to violence, and delinquent behavior (Paschall et al., 2004; Hyucksun et al., 2009; Dauber et al., 2009).

The strongest evidence to date for the moderating influence of unhealthy behaviors on the stress-mental health relationship is the recent study by Jackson et al (2010). The research team used data from the Americans' Changing Lives Survey to predict a DSM-III measure of depression as a function of nine stressful events within the past three years. They show that increasing numbers of stressful life events increases the risk of depression among both black and white adults. However, they also show that this association is conditional upon the number of unhealthy behaviors in which individuals are engaged. For whites, the association between stressors and mental health is worse (albeit only moderately significant) for those engaging in more unhealthy behaviors. *Importantly, for blacks, the association between stress and depression is strongest for those who do not engage in any unhealthy behaviors and stressors have virtually no influence on mental health for blacks who engage in several unhealthy coping behaviors.*

This is an important finding because the bulk of research suggests that substance use, specifically alcohol use, exacerbates the stress-depression relationship. One exception to the majority of the research is Lipton (1994) who shows that light-moderate and moderate drinkers (e.g., those who consume moderate amounts infrequently and those who consume small amounts regularly) demonstrate significantly less sensitivity to chronic and acute stressors than do heavy drinkers (e.g., those who regularly consume a large amount of alcohol). As with Jackson et al. (2010) this paper also shows that abstinence is a risk factor because those who never consumed alcohol showed greater vulnerability to the effects of stress on depression compared to the moderate drinkers.

Very few studies have made efforts to characterize the role of racial identity as structuring stress exposure, social norms about health-related behaviors, and subsequent mental health sequelae, and none have specifically looked at younger populations. The emphasis on younger populations is important because a recent study suggests that compared to non-Hispanic whites, minorities experience higher levels of depressive symptoms in early life (Adkins et al., 2009). The purpose of this paper is to test the possibility that coping strategies to deal with excess stress exposure among black adults are initiated early in life. It is possible that the salutary influence of unhealthy behaviors on the mental health of minorities is unique to older adults who have engaged in these behaviors over a substantially longer period of time. However, it is important to demonstrate a similar association with a younger cohort. If the social mechanisms behind stress-coping responses begin early in life then this denotes an important opportunity to elaborate upon this topic and a potentially important intervention mechanism for effective health policy.

## **METHODS**

### **Data**

All data in these analyses are drawn from Add Health, a study that examines health and health-related behaviors among a nationally representative sample of adolescents in seventh through twelfth grades. In 1994, roughly 90,000 adolescents from 134 schools completed questionnaires about their daily activities, health-related behaviors, and basic social and demographic characteristics. Respondents were then followed up with three in-home interviews (Waves I-III) with more detailed questions across a number of important domains. Our study includes only non-Hispanic White and non-Hispanic Black respondents. All data are weighted to reflect the complex sampling design of the Add Health study (Chantala and Tabor, 1999).

### **Measures**

Control variables used in this study included sex (1 if male, 0 if female), age at wave 3, region (1 if living in south, 0 otherwise), and in college (1 if in college at wave 3, 0 otherwise). Socioeconomic status at wave 1 was also used and was a composite of three census tract variables (proportion of tract age 25+ without high school diploma or equivalent; proportion of tract age 15+ with at least a college degree; and median household income), household income, and highest education level of a parent ( $\alpha=.81$ ). The key measures in our study include depression, stressful life events, and the unhealthy behaviors of regular drinking and smoking.

*Depression:* Our study makes use of the nine item version of the Center for Epidemiologic Studies depression scale (CES-D). Respondents were asked how often during the last seven days (0=never or rarely to 3=most of the time or all of the time) they

felt the following: you were bothered by things that usually don't bother you; you could not shake off the blues, even with help from your family and your friends; you felt that you were just as good as other people (reverse); you had trouble keeping your mind on what you were doing; you were depressed; you were too tired to do things; you enjoyed life (reverse); you were sad; you felt that people disliked you ( $\alpha = .80$ ). Depression is measured at Waves I and III. Based on previous research (Schulz et al., 2000) we used a conservative threshold and identified those with score of 10 or higher as having depression.

*Unhealthy behaviors:* We followed the approach of Jackson et al. (2010) and identified three unhealthy behaviors in this study. Regular drinking was characterized by the consumption alcohol at least 1-2 times per week over the past year. Regular smoking was characterized as smoking at least one cigarette per day for 30 consecutive days. Finally, obesity was measured as a function of respondents measured height and weight from the Wave III study. Those with a body mass index of 30 or higher were characterized as obese. As with Jackson et al., unhealthy behaviors is the sum of these three behaviors (ranges from 0 to 3).

*Stressful life events:* We analyzed all three waves of Add Health and identified for each respondent the age at which various stressful life events (SLE) occurred. As a guideline we used the stressful life events list found in Adkins et al. (2009) and identified the following occurrences of stress: ran away from home, was expelled from school, unwanted pregnancy, abortion, gave baby up for adoption, cohabitation ended romantic relationship ended, marriage ended, non-romantic sexual relationship ended, diagnosed with STD, attempted suicide, threatened someone, shot or stabbed someone, injured

someone in a fight, discharged from the military, entered the military, evicted from home, utility service cut off, receiving welfare, involuntarily cut from welfare, juvenile conviction or detention, adult conviction, adult jailtime, miscarriage, death of biological father, death of biological mother, death of parental figure, death of a romantic relationship, death of a spouse, death of a baby, suicide of friend or family member, baby had medical problems, relationship abuse (threatening, insulting, swearing, throwing things, pushing), was jumped, saw violence, was shot or stabbed, had sex for money, was threatened, mother or father on public assistance, was raped, was injured in a fight, skipped needed health care.

We used information about the age at which each of the events occurred to calculate the number of SLEs experienced by each individual at each age between 11 and 21. We then conducted latent class trajectory analysis using the *mmlcr* package (Buyske 2006) in R 2.9.0 (R Development Core Team 2009) for the sum number of stressors across this age range. This procedure uses mixed-mode latent class regression to describe intra-individual change over time as a function of group membership ( $C_i$ ) where group membership in the  $K^{\text{th}}$  class is a post-hoc determination based on similarity in initial levels of stress (intercept) and change in stress over time (slope). The general model is specified below.

$$f(y) = \sum_{k=1}^K \Pr(C = k) \Pr(Y = y | C = k) = \sum_{k=1}^K p_k f(y, \lambda_k)$$

In this model, the dependent variable is the observed number of stressors reported at each age during the study. The AIC and BIC are used to characterize the total number of stress trajectories in the population. The main parameter estimate of this model is  $p_k$  which is the probability of belonging to class  $k$  which can also vary as a function of the parameters



$\lambda_k$  which change differentially over time. Because of the count nature of the dependent variable, we use a Poisson link in this general trajectory model to predict the average number of stressors that individuals in class  $k$  are expected to have at each age. We allow for cubic age terms in our estimation of growth trajectories.

[Figure 1 about here]

Using these methods, we identify four stress trajectories which are presented in Figure 1. In total 53.9% were minimally exposed to stress. This group showed a slight increase in average stress exposure but at no point did the predicted average number of stressors/year exceed one-half. Two of the remaining three trajectories were similar in that they contained a sharp increase in the number of SLEs but this increase quickly subsided over the following two years. These two trajectories differ by the estimated peak which was early (age 15) for the first group and somewhat later for the second group (age 17). The “peak at 17” group was more common (18.7%) than as the “peak at 15” group” (11.4%). Finally, we describe the fourth group as “chronic” because important SLEs occurred as young as 12 and this group demonstrates a fairly chronic stress profile throughout the study. This group comprised the remaining 15.9% of the sample.

Although there is an observable dip during mid to late adolescence, there is a return to a fairly regular exposure to SLEs in young adulthood.

The remainder of the analysis is conducted using STATA 10.0. We construct appropriate weights by race/ethnicity and incorporated the characteristics of the sample design to ensure unbiased estimates and standard errors using the *SVY* commands in STATA (Chantala & Tabor 1999). Bivariate analyses were conducted to examine race/ethnicity differences in the sample with respect to control variables and key

measures of depression, drinking, smoking, obesity, and stress trajectory. These results are reported in Tables 1 and 2. Following these bivariate models, we use logistic, ordered logistic, and multinomial logistic regression models to examine specific questions regarding race, stress, depression, and unhealthy behaviors. These results from these models are described in detail below.

## RESULTS

[Table 1 about here]

Table 1 shows the stress profiles, control measures, depression, and unhealthy behaviors by race. Three important findings emerge from this table. First, Non-Hispanic blacks are significantly more likely than whites to have an elevated stress profile throughout their teens and into their twenties. Whereas 56% of whites were considered to have minimal stress profiles, only 45% of blacks experienced this relatively low stress exposure throughout adolescence and into early adulthood. Similarly, blacks are more likely to have the two peaked stress profiles as well as a chronically stressed adolescence. The design based estimate suggests that the observed differences in the distribution of stress trajectories is significant ( $p < .006$ ) for blacks and whites. Relative risk ratios (RRR) were calculated from multinomial logistic regression models and indicate that the observed differences are statistically significant for each elevated stress level compared to the minimal trajectory. Specifically, the RRR estimates (and 95% confidence intervals) for blacks compared to whites are 1.54(1.22, 1.94), 1.48(1.14, 1.91), and 1.56(1.11, 2.18) for the peak 15, peak 17, and chronic stress trajectories, respectively.

Second, the prevalence of depression is significantly higher for blacks ( $p_d = .146$ ) compared to whites ( $p_d = .101$ ). As with the results of other studies (Adkins, 2009), this

information is important because it contributes to a fairly large body of work that has, to date, provided fairly inconclusive results regarding depression as a health disparity. Finally, the prevalence of smoking and drinking are significantly higher for whites compared to blacks but blacks are more likely than whites to have an increased risk of obesity. Overall, whites have a significantly worse health behavior profile ( $\bar{B}_w = .861$ ) compared to blacks ( $\bar{B}_b = .660$ ) and this difference is statistically significant ( $p < .000$ ). These results are comparable to Jackson et al. (2010) who also showed that blacks had lower rates of unhealthy behaviors compared to whites.

[Table 2 about here]

Table 2 presents the prevalence of depression by stress trajectory for non-Hispanic black and whites separately. These results provide strong evidence that SLEs are strongly associated with depression for young adults regardless of the stress trajectory. However, they also indicate that the prevalence of depression is significantly higher for those who have been exposed to stressors for the longest period of time. The chronic stress group displays the highest risk of depression for both groups and nearly one in five blacks in this category have symptom counts of depression that exceed the criteria for depressive diagnosis. Equally important, blacks display an elevated risk of depression at each stress trajectory. In ancillary analyses (results not shown) we estimated a logistic regression model predicting depression as a function of stress trajectory, race, and race\*trajectory interaction terms and none of these interaction terms were statistically significant suggesting that the effects of stress on depression are comparable for whites and blacks.

[Table 3 about here]

The results from Tables 1 and 2 support findings from previous research showing an increased risk of depression among blacks compared to whites. We also show that blacks face an increased risk of SLEs across their adolescence and that stress is strongly associated with an increased risk of depression with chronic stress as the most risk stress trajectory. These results pose an important question that is addressed in Table 3. Specifically, to what extent is the increased prevalence of depression among blacks compared to whites due to greater exposure to stress among blacks? Model 1 in Table 3 provides an adjusted baseline risk for blacks compared to whites (OR = 1.411) that is slightly attenuated (OR = 1.332) with the controls for stress (Model 2). This suggests that roughly 17% of the increased risk of depression among blacks is due to the increased average stress levels among blacks compared to whites. Importantly, the strong associations between the two early onset stress categories (e.g., Chronic and Peak 15) remain statistically significant despite controlling for social and demographic background characteristics. Those in the chronic stress trajectory are more than 2 times as likely as those in the minimal stress trajectory to be characterized as depressed in Wave III.

The third model in Table 3 provides an initial look at the complex association between health behaviors, stress, depression, and race. These results indicate that unhealthy behaviors are positively and significantly associated with a risk of depression; compared to those who do not engage in any unhealthy behaviors, each successive deleterious behavior is associated with a 23% increase in the risk of depression. Importantly, these controls act as a moderate suppressor for the race coefficient which increases back to its level prior to the controls for stressors. This provides some (albeit weak) support for the notion that health behaviors may actually buffer the deleterious

effects of stress on depression for blacks. Further support for this idea can be seen from the final model in Table 3 which introduces an interaction term between unhealthy behaviors and race. The interaction is only moderately significant ( $p < .072$ , two-tailed) but the direction of the term suggests that the effect of unhealthy behaviors on depression is weaker for blacks compared to whites. Importantly, it also suggests that if one considers the effects of race with health behaviors removed (e.g., unhealthy behaviors = 0) that blacks actually face an even stronger risk of depression compared to whites (OR = 1.68).

[Table 4 about here]

To examine this possibility, Table 4 presents estimates from two ordered logistic regression models in which the sum of unhealthy behaviors (0-3) is regressed on stress and the full set of controls for blacks and whites separately. The first finding to emerge is that stress leads to an increased risk of unhealthy behaviors. For both groups, there are strong and positive links between each stress trajectory and an increased likelihood of engaging in unhealthy behaviors. However, if, as Jackson et al. (2010) hypothesize, blacks are more likely than whites to turn to unhealthy behaviors in the face of stress, then we should expect a larger regression coefficient for blacks for each of the stress profiles. The results provide strong evidence that this is not the case. The parameter estimates for the two regression models are virtually identical and in the case of chronic stressors, whites are slightly more likely to engage in unhealthy behaviors compared to whites.

[Table 5 about here]

As described above, it is still possible that the combined effects of chronic strain and unhealthy behaviors on the risk of depression are still different for blacks and whites. To examine models comparable to the Jackson et al. (2010) paper, we estimate the main and interactive effects of chronic stress and unhealthy behaviors for blacks and whites separately. Because we have four stress categories and three health behaviors, we limited our analysis of stress to chronic stress at this stage of the analysis in order to reduce the number of interactions and to simplify the focus. We chose chronic stress because of the evidence linking this measure of stress to depression and its strong association with unhealthy behaviors. Table 5 presents results from a logistic regression model in which depression at Wave III is regressed on the full set of controls but now we include an interaction term between stress and behaviors. In the Jackson et al. (2010) study, the interaction was positive (but not statistically significant) for whites and negative (and statistically significant) for blacks. Thus suggesting that an increasing number of unhealthy behaviors buffers the negative effects of strain on depression for blacks and slightly exacerbates these effects for whites. As shown in Table 5, we show relatively weak evidence for buffering effects among blacks and no moderating effects for whites. The effect for blacks ( $OR=.733$ ) is only moderately significant at the one-tail level but it remains in the direction suggested by Jackson et al. (2010). To better illustrate these associations, predicted probabilities for depression are plotted in Figure 2 by race, unhealthy behaviors, and chronic stress status. The shaded bars denote those who are chronically stressed and the non-shaded bars are others. As with Jackson et al. (2010), we show that the greatest mental health disparity occurs for those who are exposed to chronic stress and who do not engage in any unhealthy behaviors; blacks exposed to chronic

stress who lead the most healthy lives have the highest estimated probability of depression. There is virtually no disparity among stress exposed blacks and whites who are engaged in at least two unhealthy behaviors. We believe this highlights the importance of characterizing the long-term stress profiles to clarify the role of race and unhealthy behaviors related to mental health.

## **DISCUSSION AND CONCLUSION**

Exposure to chronic stressors is regularly shown to both increase depressive symptoms and to increase the risk of unhealthy behaviors including alcohol use, smoking, and obesity (Adkins et al., 2009; Mason et al., 2008; Meadows et al., 2004; Dallman et al., 2003a; Dallman et al., 2003b; Ge et al., 1994). While some studies have simultaneously investigated the complex relationships between these three factors (stress, unhealthy behaviors, and depression) only a limited number have focused explicitly on adolescents (Chaiton et al., 2009; Lipton, 1994). Further, very little work has examined the ways in which race structures this complex web of social stressors, unhealthy coping behaviors, and depressive symptoms. This study examined the complex relationship between stress trajectories unhealthy behaviors, and depressive symptoms among non-Hispanic Black and non-Hispanic White adolescents and young adults. Although there is agreement that stress is composed of both operant and cumulative stress (Turner et al. 1995) measurement strategies typically involve summing the total number of stressors across longer periods of time. A distinguishing feature of our study was the use of stress trajectories rather than simple counts of stressful life events. This allowed us to differentiate not only between low and high stress but between those who faced relatively high stress at younger ages and those who had more typical peaks or minimal stress

through adolescence. Others have made a strong case for stress-related trajectories (George and Lynch, 2003), but little work has attempted to characterize the typical stress trajectories that exist in a population of young adults. Our analyses confirmed that non-Hispanic blacks were more likely than non-Hispanic whites to be exposed to early stress and to begin to see increasing stressful life events entering young adulthood. Given that stress and depressive symptoms are typically associated, it was not surprising to find that the rate of depressive symptoms was also higher for non-Hispanic blacks.

This paper also contributes to ongoing discussions regarding the complex relationships between stress, coping behaviors, and mental health among members of different racial and ethnic groups. The most intriguing finding to emerge from our analysis was that although we found support (although relatively weak) for findings presented by Jackson et al. (2010), we were unable to find evidence of the purported mechanism through which these disparities were operating. Specifically, we show that unhealthy behaviors moderate the association between stress and depression for blacks but we do not find any evidence that blacks are more likely than whites to use unhealthy behaviors to cope with stress. This may simply be due to the age differences in the two studies; our study examines youth while the Jackson et al. study examines older adults. Specifically, cigarettes are very expensive and the limited resources of most youth may preclude them from smoking when they may otherwise choose to do so. We used a composite indicator of unhealthy behaviors in order to follow the approach of Jackson et al. (2010), however, it is possible that much of the association is being driven by obesity because the real costs associated with consuming fatty foods may be so much cheaper than cigarettes or alcohol; people have to eat and cheap food that is readily available may be



‘costless’ compared to additional fees paid for alcohol. To explore this possibility we examined the association between chronic stress and depression for obese and non-obese respondents (full results are not presented). For both blacks and whites, the effects of chronic stress on the risk of depression were significantly higher for non-obese persons ( $OR_{black} = 1.81, p < .023$ ;  $OR_{white} = 2.10, p < .000$ ) compared to obese persons ( $OR_{black} = 1.44, n.s.$ ;  $OR_{white} = 1.19, n.s.$ ). Although we show a moderating effect of obesity, again, this effect is not in the direction specified by the stress-coping model.

This still leaves us with an interesting social dynamic without a clear explanation; the stress-coping model anticipates the associations described in Table 5 and Figure 2 but the results presented in Table 4 suggest that differential social responses (e.g., norms) to social stressors may not be the cause for younger adults. There are several possible explanations and future research may consider examining the following. First, it may be that the types of stressors faced by persons who are not engaged in any unhealthy behaviors are very different from the types of stressors faced by persons who smoke, drink, and/or are obese. Persons who are more likely to engage in unhealthy behaviors may also select in to particular types of stressful environments. Therefore, the number of total stressors at any particular time might not be as informative as the types of stressor to which individuals are exposed. This is particularly relevant because this same concern may be further confounded by race such that the specific components of the typical stress profile of a black woman who neither smokes, drinks, or is obese may be very different from a comparable woman from a comparable stressful background who is white. For example, others have shown that family stress is one of the strongest predictors of depression, especially among women (Hammen, 2005). It may be that black women with

the healthiest lifestyles may have a stress profile that is lower on ambient, external, acute, or other chronic indicators but tend to face much more family-related stressors compared to comparable white women. If this is the case, then the moderating effects shown above may have to do with stress composition rather than stress response.

Second, it may be that stress does not cause depression, per se; rather, they may simply co-occur because of unobserved factors. Silberg (1999) and others have argued that genetic factors that make an individual vulnerable to symptoms of depression may also increase their risk of greater exposure to stressful life events. For example, Kendler et al. (1999) compare lifetime measures of stressful events and measures of depression among sibling and twin pairs and suggest that one-third of the association between stress and depression is due to common genes that cause both stressful life events and depression. They show that this association is particularly strong for stressors in which individuals have some control and virtually non-existent for stressful life events that are independent of individual autonomy. This is important because environmental factors have been shown to significantly modify the relative influence of specific genotypes on complex phenotypes like mental health (Caspi et al., 2003). If a sizable portion of the correlation between stress and depression is due to common genetic factors that predispose persons to both outcomes, then this association should be the largest for those in which the environment does not suppress genetic sources of comorbidity. For blacks and whites it appears that the association between stress and depression is the weakest for those who are engaged in the most number of unhealthy behaviors (see Figure 2). This may have more to do with the immediate social environments in which persons who engage in many unhealthy behaviors reside rather than the behaviors themselves. In this

case, the environments of people (and more so for blacks than whites) who are engaged in at least two unhealthy behaviors may control, suppress, or overwhelm genetic tendencies that may otherwise link these two phenotypes. Raine (2001) argues that social environments characterized by disorganization in which people are uncertain about their roles, cause genetic factors to be less relevant because there is so much background noise. But when this noise is reduced by social institutions, organizations, norms, and behavioral expectations, then it allows for “biology to shine through” (Raine, 2002:14). Therefore, there may be environmental factors that are unique to those who do not smoke, do not drink, and are not obese that enhance otherwise socially neutral genetic tendencies. Most importantly, the social selection mechanism may be stronger for blacks vis-à-vis their behaviors, compared to whites.

### *Limitations*

There are several limitations with our current study that should be considered when interpreting the results. First, while the trajectory based stress assessment provides new and useful information for stress-related research and health-disparities research in general, we limited our items to comparable measures used in other research (Adkins et al. 2009). To be sure, there are other known sources of stress and strain that could be obtained from the Add Health data that are not included in our analyses. For example, we make no efforts to include ambient stressors related to the physical or built environment. These factors have been linked to psychological well-being (Cohen and Weinstein 1981) and are believed to be an important contributor to mental health disparities (Downey and Van Willigen 2005). Moreover, our stress measure didn't differentiate between the different sources of stress. In other words, these stressors may be linked to social, health,

residential, work-related, or familial issues which may have very different consequences with respect to mental health and unhealthy coping behaviors. Second, we emphasized race in this paper but others have shown that “race effects” are complicated by other identities such as gender, work status, and family status (Boardman et al., 2008). These researchers show that living near heavy industrial activity is seen as stressful by most persons but working class men without families actually derive mental health gains from this otherwise noxious activity. The authors argue that manufacturing activity symbolizes the proximity to work which is beneficial to working class men. Taken together, future work should attempt to differentiate between the different stress domains and to include additional measures of stress. Further, more efforts should be made to characterize stressful life events that are unique and socially meaningful to particular groups at particular points in their lives.

### *Conclusion*

We encourage future researchers to replicate the stress-coping model described by Jackson et al. (2010) and shown here, but we also encourage future research to provide evidence for the mechanism through which these subtle social forces may (or may not) be operating. This research speaks directly to the health disparities, social stress, and life course perspective research. We encourage others to use a trajectory based stress exposure model that is used here and to make efforts to distill the specific domains and items that are meaningful for different groups at different periods of their lives.

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**Table 1. Descriptive statistics by self-reported race.**

	NH White	NH Black	Pr (W=B)
Stress Trajectories			
Minimal	.559	.454	<.006
Peak 15	.109	.137	
Peak 17	.180	.217	
Chronic	.152	.192	
Health behavior profile [Wave 3]			
Regular smoker	.491	.244	<.000
Regular drinker	.115	.065	<.000
Obese	.256	.351	<.000
Sum of health behaviors	.861	.660	<.000
Depression [Wave 3]	.101	.146	<.000
Sociodemographic Controls			
Sex [Male]	.470	.441	<.766
Age [years]	21.841	21.858	<.677
Region [South]	.345	.649	<.000
Socioeconomic Status, Wave1	0.142	-.268	<.000
In College, Wave3	.364	.286	<.006

Note: all data come from Waves I and III of the National Longitudinal Study of Adolescent Health (Add Health).

**Table 2.** Prevalence of depression by stress trajectory for black and white adolescents and young adults (Add Health, W3)

Stress Trajectory	Proportion of respondents meeting criteria for depression		
	White	Black	Total
Minimal	0.074	0.091	.076
Peak 15	0.141	0.214	.158
Peak 17	0.110	0.161	.121
Chronic	0.158	0.214	.171
Design based F	17.29	10.06	27.12
d.f.	2.89, 352.16	2.88, 276.23	2.96, 379.46
Pr. <	0.000	0.000	.000

Note: all data come from Waves I and III of the National Longitudinal Study of Adolescent Health (Add Health). All estimates are adjusted for the complex sampling design of the Add Health study using the SVY commands in STATA 10.0.

**Table 3.** The role of stress and unhealthy behaviors in race differences in mental health: Odds ratios for the risk of depression.

	Model 1	Model 2	Model 3	Model 4
Depression (W1)	3.483 (3.007, 4.034)	3.155 (2.724, 3.654)	3.127 (2.697, 3.625)	3.127 (2.697, 3.626)
Sex [Female]				
Male	0.643 (0.548, 0.755)	0.622 (0.529, 0.731)	0.615 (0.522, 0.724)	0.617 (0.524, 0.726)
Age (years)	0.916 (0.877, 0.957)	0.972 (0.921, 1.024)	0.969 (0.919, 1.022)	0.969 (0.919, 1.022)
Region [All others]				
South	0.795 (0.67, 0.943)	0.817 (0.689, 0.969)	0.811 (0.684, 0.962)	0.809 (0.682, 0.96)
Enrolled in college [No]				
Yes	0.691 (0.57, 0.838)	0.745 (0.617, 0.899)	0.778 (0.647, 0.936)	0.78 (0.649, 0.938)
Race [NH White]				
NH Black	1.411 (1.155, 1.725)	1.332 (1.091, 1.628)	1.417 (1.153, 1.742)	1.678 (1.278, 2.205)
Socioeconomic status	0.857 (0.754, 0.974)	0.871 (0.764, 0.993)	0.872 (0.765, 0.994)	0.873 (0.765, 0.995)
Stress Trajectory [Minimal]				
Peak at 15		1.736 (1.318, 2.287)	1.654 (1.254, 2.18)	1.649 (1.251, 2.174)
Peak at 17		1.273 (0.995, 1.628)	1.217 (0.948, 1.562)	1.215 (0.947, 1.56)
Chronic		2.05 (1.577, 2.664)	1.938 (1.482, 2.534)	1.932 (1.478, 2.525)
Sum of unhealthy behaviors (0-3)			1.232 (1.086, 1.397)	1.295 (1.115, 1.504)
Black*Sum of unhealthy behaviors (0-3)				0.809 (0.641, 1.019)
Log Likelihood	-3853.7938	-3821.5791	-3813.4479	-3812.15
-2LL	7707.5876	7643.1582	7626.8958	7624.3
Likelihood Ratio	493.73	64.4294	16.2624	2.5958
DF	7	3	1	1
Pr.	0.000	0.000	0.000	0.107
Pseudo R2	0.060	0.068	0.070	0.070

Note: all data come from Waves I and III of the National Longitudinal Study of Adolescent Health (Add Health). All estimates are adjusted for the complex sampling design of the Add Health study using the SVY commands in STATA 10.0.

**Table 4.** Unhealthy behaviors (Wave 3) as a function of stress for black and white adolescents and young adults: Ordered logistic regression estimates.

	White	Black
Sum of unhealthy behaviors W1 (0-3)	3.899 (3.449, 4.407)	3.16 (2.478, 4.030)
Sex [Female]		
Male	1.243 (1.107, 1.397)	1.354 (1.105, 1.661)
Age (years)	0.939 (0.903, 0.976)	1.021 (0.943, 1.105)
Region [All others]		
South	1.009 (0.863, 1.180)	1.19 (0.840, 1.685)
Enrolled in college [No]		
Yes	0.659 (0.570, 0.761)	0.632 (0.502, 0.794)
Socioeconomic status	0.934 (0.841, 1.037)	1.008 (0.845, 1.203)
Stress Trajectory [Minimal]		
Peak at 15	1.582 (1.308, 1.913)	1.567 (1.086, 2.261)
Peak at 17	1.555 (1.324, 1.827)	1.542 (1.177, 2.021)
Chronic	2.004 (1.699, 2.364)	1.820 (1.418, 2.337)
cut1	-1.348 (-2.20, -0.495)	1.124 (-0.76, 3.009)
cut2	1.14 (0.287, 1.993)	3.403 (1.457, 5.348)
cut3	4.167 (3.304, 5.03)	5.783 (3.849, 7.717)
Log-Likelihood base model	-7650.2429	-2732.2706
Log-Likelihood with stress controls	-7576.1089	-2715.2225
Likelihood ratio	148.268	34.0962
d.f.	3	3
Pr. <	0	0
Pseudo r <sup>2</sup>	0.099	0.085

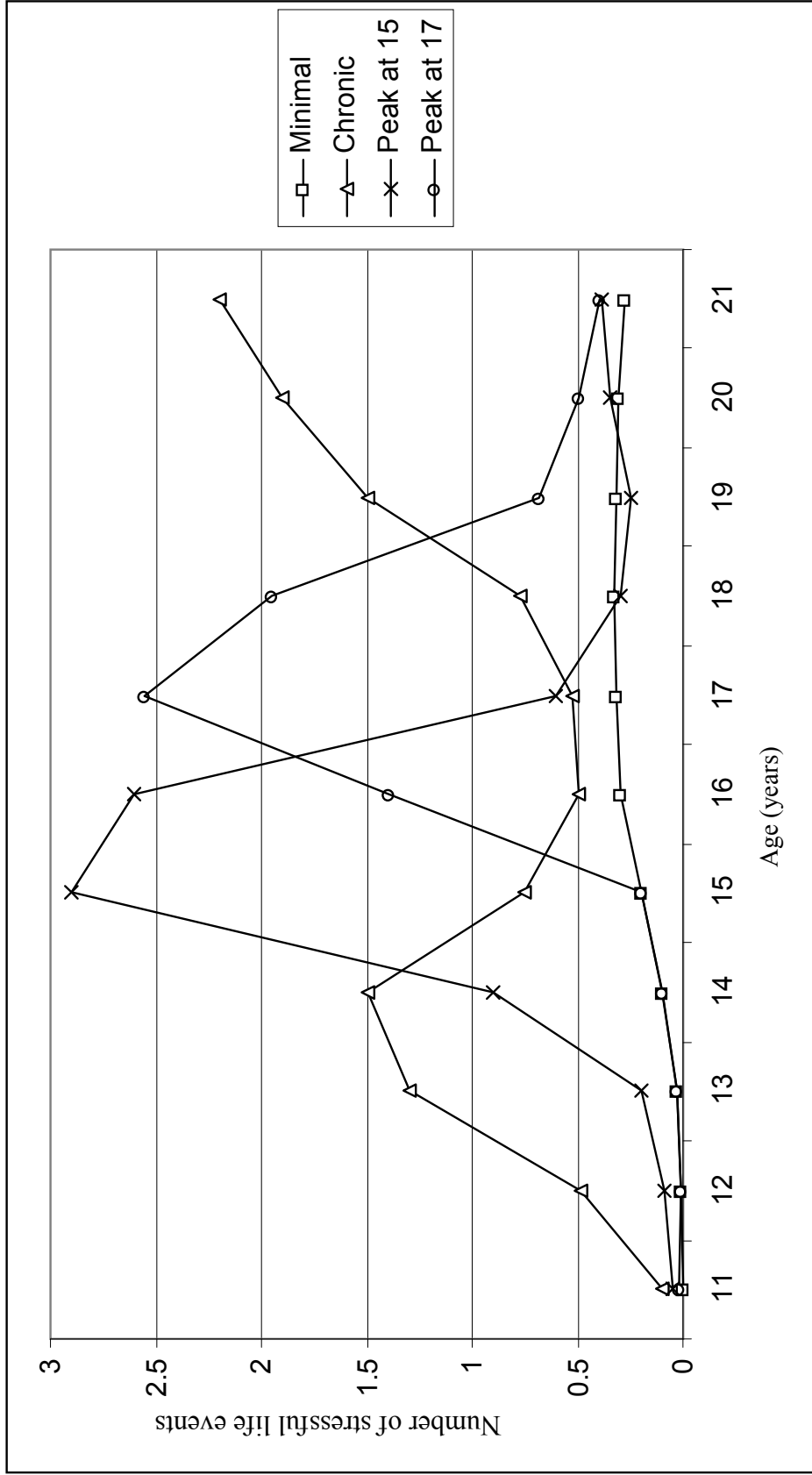
Note: all data come from Waves I - III of the National Longitudinal Study of Adolescent Health (Add Health). All estimates are adjusted for the complex sampling design of the Add Health study using the SVY commands in STATA 10.0.

**Table 5.** The interactive role of stress and unhealthy behaviors as determinants of depression among black and white adolescents and young adults.

	White	Black
Depression (W1)	3.507 (2.921, 4.21)	2.740 (1.992, 3.768)
Sex [Female = 0]		
Male	0.640 (0.525, 0.779)	0.576 (0.415, 0.800)
Age (years)	0.955 (0.899, 1.014)	0.999 (0.911, 1.097)
Region [All others]		
South	0.748 (0.607, 0.921)	0.926 (0.668, 1.283)
College attendance [No]		
Yes	0.810 (0.653, 1.006)	0.619 (0.438, 0.874)
Socioeconomic status	0.824 (0.707, 0.96)	0.995 (0.765, 1.295)
Stressful profile [All others]		
Chronic	1.872 (1.204, 2.912)	2.171 (1.176, 4.006)
Sum of unhealthy behaviors	1.363 (1.147, 1.621)	1.143 (0.924, 1.414)
Chronic stress* Unhealthy behaviors	0.893 (0.652, 1.223)	0.733 (0.499, 1.077)
Log likelihood without interaction	-2508.702	-1315.34
Log likelihood with interaction	-2508.434	-1315.29
df	1	1
-2LL	0.5354	0.1
Pr.	0.464	0.752
Pseudo R2	0.062	0.058

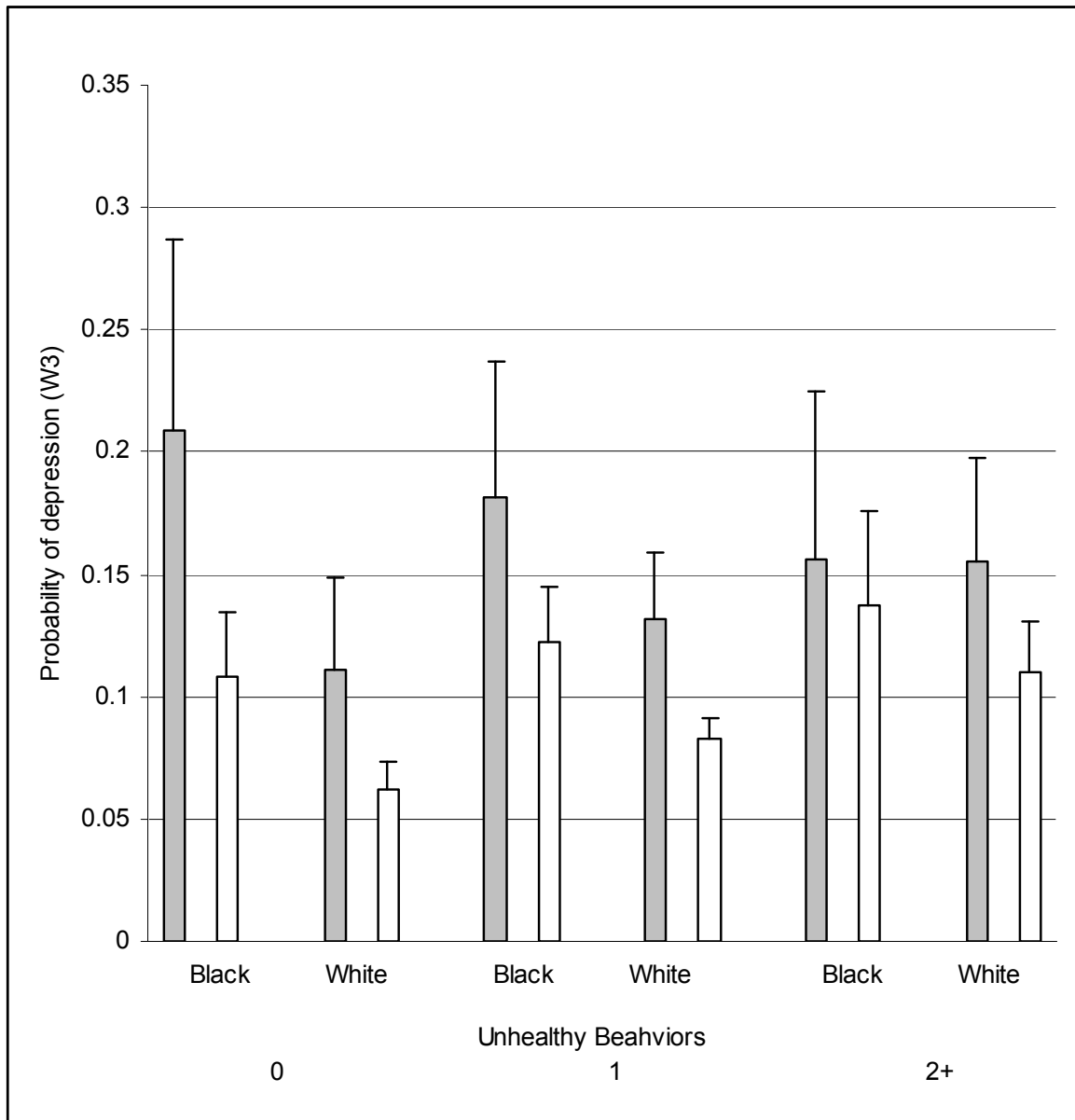
Note: all data come from Waves I- III of the National Longitudinal Study of Adolescent Health (Add Health). All estimates are adjusted for the complex sampling design of the Add Health study using the SVY commands in STATA 10.0.

**Figure 1.** Early life stress exposure among US adolescents: latent growth classes for stressful life events.



Note: all data come from Waves I - III of the National Longitudinal Study of Adolescent Health (Add Health). Values obtained from latent class trajectory modeling using the MMLCR package in R.

**Figure 2.** The estimated effect of chronic stress on depression for adolescents: the moderating role of race and unhealthy behaviors.



Note: all data come from Waves I- III of the National Longitudinal Study of Adolescent Health (Add Health). Values and error estimates derived from the parameter estimates in Table 4 using the *prvalue* command following the design based logistic regression routine in STATA 10.0