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Racial/ethnic differences in the longitudinal progression of co-occurring negative affect and cigarette use: From adolescence to young adulthood

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Abstract

Aims—This study examined the longitudinal progression of the co-occurrence of cigarette use and negative affect among the general population of U.S. adolescents and young adults and between racial/ethnic groups.

Methods—Data for this study consisted of Waves 4, 6, and 8 of the NLSY97 longitudinal study containing a nationally representative sample of U.S. adolescents and young adults. A total of 7979 adolescents (Mean age at Wave 4=17.98, SD=1.44, 49% female) were included in the analyses. To investigate the co-morbidity between negative affect and cigarette use, a latent factor of negative affect and single indicator of cigarette consumption were examined at each wave. A three wave Bivariate Autoregressive Cross-Lagged Effect Model was estimated to test the conjoint trajectory of negative affect and smoking.

Results—For all racial/ethnic groups prior negative affect status influenced future negative affect between waves and prior negative affect was positively related to increases in smoking in subsequent waves. The longitudinal trajectory of negative affect for the three racial/ethnic groups was the same, but racial/ethnic group differences were observed in the strength of the longitudinal relationship between previous and future cigarette use. Specifically, the following racial/ethnic differences were observed, even after controlling for the effect of SES; White young adults were found to exhibit the strongest association between cigarette use in the first two waves, followed by Hispanic individuals and lastly by African Americans. In the last two waves, African American young adults were found to have the strongest association between cigarette use at the latter two waves, followed by White individuals.

Conclusions—Both negative affect and cigarette consumption influence each other during the transition between late adolescence and young adulthood but the magnitude of the associations between cigarettes use across waves differed between racial/ethnic groups. Implications for prevention and treatment programs include considering both cigarette use and negative affect as two factors that jointly impact each other and that should be targeted simultaneously.

Keywords

Tobacco use; Adolescence; Comorbidity; Negative affect; Young adulthood

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1. Introduction

The co-occurrence between mood disorders with substance use problems is widespread in the U.S. (Conway, Compton, Stinson, & Grant, 2006) with the prevalence of being a current smoker substantially higher among individuals who have a diagnosed mental disorder than among those who do not (Breslau, Kilbey, & Andreski, 1994). Results of the National Comorbidity Survey (NCS) suggest that 15% of the U.S. adult population with a diagnosis of 12-month drug abuse and 28.4% of those with a diagnosis of 12-month drug dependence had experienced at least one episode of major depression (Kessler et al., 1996). Although it has been estimated that about a third of lifetime smokers have a mood disorder (Griesler, Hu, Schaffran, & Kandel, 2011) and that mood disorders are associated with the development of nicotine dependent use (Swendsen et al., 2010), the extent to which the association between mood disorders and smoking changes longitudinally and differs by racial/ethnic group is less clear (Mineur & Picciotto, 2009).

1.1. Negative affect

Due to the overlap between anxiety and depressive disorders, some researchers have argued that the two disorders are related features of a common underlying disorder (Brady & Kendall, 1992; Hale, Raaijmakers, Muris, van Hoof, & Meeus, 2009; Laurent & Ettelson, 2001). In fact, neurobiological studies have implicated dysfunction of the hypothalamic–pituitary–adrenal (HPA) axis, involved in coordinating stress reactions, in both mood and anxiety disorders (Gallagher, Reid, & Ferrier, 2009; Kaufman & Charney, 2001; Risbrough & Stein, 2006). The controversy surrounding the relationship of these two disorders is highly salient among adolescent and young adult populations because of the implications for the development of persisting problems into adulthood (Ajdacic-Gross et al., 2009; Copeland, Shanahan, Costello, & Angold, 2009). An explanation for the co-occurrence of these disorders is that they share characteristics that are explained by the overall role that negative affect plays in these emotional disorders (Watson, 2005). Negative affect encompasses a range of negative emotions such as fear, anger, sadness, guilt, and disgust and is thought to reflect stable individual differences in emotionality (Clark, Watson, & Mineka, 1994; Miller, Vachon, & Lynam, 2009; Watson & Clark, 1984).

1.2. The relationship between negative affect and smoking

The examination of the co-morbidity between negative affect and the initiation of smoking is especially relevant during late adolescence and young adulthood because these are periods when smoking rates are among the highest across the lifespan (CDC, 2006; Flay, Phil, Hu, & Richardson, 1998). Further, young adulthood is a time when over a third of individuals have been found to initiate smoking and about a quarter of individuals progress from being experimental smokers to regular smokers (Chassin, Presson, Pitts, & Sherman, 2000; Choi, Harris, Okuyemi, & Ahluwalia, 2003).

Previous research on the relationship between negative affect and smoking has put forth several explanatory mechanisms. One suggested mechanism, the self-medication hypothesis, posits that deficits in mood predispose individuals to initiate smoking to alleviate the distressing symptoms they experience (Khantzian, 1985, 1997). Consistently studies have shown that negative affect exhibits a positive association with increases in smoking (Breslau, Kilbey, & Andreski, 1991; Escobedo, Reddy, & Giovino, 1998; McLeish, Zvolensky, Marshall, & Leyro, 2009; Pomerleau, Zucker, & Stewart, 2003). There is evidence that negative affect at age 16 is a predictor of smoking behavior two years later (Mason, Hitch, & Spoth, 2009). Using data from the National Longitudinal Study of Adolescent Health, Munafò, Hitsman, Rende, Metcalfe, and Niaura (2008) found that among individuals who had never smoked at baseline, increases in depressive symptoms lead to

smoking initiation but not to becoming regular smokers (Munafò et al., 2008). Neurochemical changes in the brain caused by depression, specifically in the cholinergic system (Dagyte, Den Boer, & Trentani, 2011), may create a vulnerability to seek the rewarding effects of smoking (Lerman et al., 1998).

A second mechanism proposes that the use of cigarettes is a precursor to the development of mood disorders. Several studies have found that smoking increases the likelihood of developing mood disorders such as depression and anxiety (Brook, Cohen, & Brook, 1998), even after controlling for prior levels of depression (Steuber & Danner, 2006). Studies have shown that nicotine use changes neurochemical systems in the brain associated with the onset of mood disorders, specifically depression (Dagyte et al., 2011), via cholinergic receptors (Pomerleau, 1986) and the neuronal nicotinic acetylcholine receptors (Tuesta, Fowler, & Kenny, 2011). In addition, the effect of nicotine on dopamine involves increases in attentional bias towards negative affect stimuli (Rzetelny et al., 2008) and further, among smokers who tend to use a ruminative coping style, nicotine appears to heighten the salience of negative thoughts which in turn increase negative mood (Richmond, Spring, Sommerfeld, & McChargue, 2001).

A third mechanism suggests that a third factor may be involved in the development of these co-occurring problems. Support for this mechanism comes from studies providing evidence that depressive disorders and cigarette use share similar, if not the same, environmental and genetic precursors (Fergusson, Boden, & Horwood, 2011; Hu, Davies, & Kandel, 2006; Mykletun, Overland, Aaro, Liabo, & Stewart, 2008; Rohde, Lewinsohn, Brown, Gau, & Kahler, 2003). Furthermore, exploration of the mechanisms responsible for the comorbidity between negative affect and smoking suggests that once one of the two manifest itself, the relationship between them may be bidirectional (Berlin, Covey, & Glassman, 2009). For example, high levels of negative affect during mid-adolescence have been found to predict higher levels of smoking four years later (Audrain-McGovern, Rodriguez, & Kassel, 2009) while daily smokers, compared to individuals who have never smoked, have been found to be 1.8 times more likely to have a mood disorder (John, Meyer, Rumpf, & Hapke, 2004; Kendler et al., 1993).

As the extant literature shows, extensive research exists describing the ways by which negative affect and cigarette use may be co-occurring. However, considerably less is known about how the relationship between these two potentially long-term problems may differ among racial/ethnic groups particularly when these are examined longitudinally.

1.3. Racial/ethnic differences in smoking and negative affect among U.S. populations

Racial/ethnic differences in measures of psychological distress among adolescents suggest that racial/ethnic minorities experience higher rates of psychological distress (Anderson & Mayes, 2010). In particular, several studies have found that Hispanic adolescents report higher depressive symptoms than Whites or African American youth (Adkins, Wang, Dupre, van den Oord, & Elder, 2009; McLaughlin, Hilt, & Nolen-Hoeksema, 2007; Roberts, Chen, & Roberts, 1997; Roberts, Roberts, & Chen, 1997; Schraedley, Gotlib, & Hayward, 1999; Twenge & Nolen-Hoeksema, 2002).

During adolescence the prevalence of cigarettes smoking has been found to be higher among Whites than Hispanics or African Americans (Johnston, O'Malley, Bachman, & Schulenberg, 2009). Although some studies suggest that White adolescents are more likely to persist in smoking after the initiation stage and to become dependent on nicotine than Hispanic or African American adolescents (Gritz et al., 1998), others have found that Hispanic adolescents initiate smoking earlier (Kandel, Kiros, Schaffran, & Hu, 2004), and have higher rates of lifetime cigarette use than White adolescents (Shih, Miles, Tucker,

Zhou, & D'Amico, 2010). Other studies have found that Hispanic adolescents have higher rates of smoking than African American adolescents (Brook, Pahl, & Ning, 2006) but lower than Whites (Griesler, Kandel, & Davies, 2002; Kandel & Chen, 2000). By the time adolescents are in 8th grade, Hispanic and White youth are smoking at twice the rate as their African American peers (Kelder et al., 2003).

Among adults in the United States, Whites report higher rates of negative affect and depressive disorders (Anderson & Mayes, 2010; Riolo, Nguyen, Greden, & King, 2005) and African Americans have lower rates of depression than White adults (Mezuk et al., 2010). For adults, the positive relationship between negative affect and smoking has been found for Whites but not for African American or Hispanics (Kiviniemi, Orom, & Giovino, 2011). Given the differences in rates of smoking and in psychological distress experienced by adolescents and young adults of different racial and ethnic groups, in the present study differences were expected in the longitudinal relationship between negative affect and smoking behavior among racial and ethnic groups.

1.4. The present study

This study examined the co-occurrence of cigarette use and negative affect longitudinally. Longitudinal modeling allows researchers to learn about patterns of co-variation among variables by studying their behavior over time. Modeling data from multiple time points allows for examining the possible directions of causality that are more difficult to test in cross-sectional designs. The purpose of the current study was to understand the longitudinal co-variation between and within these problems over time in U.S. adolescents as they age into young adulthood.

1.4.1. Study hypotheses—Given prior research on the racial/ethnic differences in rates of negative affect problems and rates of cigarette use, it was first hypothesized that there would be racial/ethnic differences in the association between negative affect at Wave 4 and negative affect at the two subsequent waves. Secondly, it was hypothesized that racial/ethnic differences would be observed in the association between cigarette use at Wave 4 and cigarette use at later waves. The third hypothesis stated that there would be racial/ethnic differences in the association between negative affect and cigarette use across waves and similarly that there would be racial/ethnic differences in how cigarette use would be related to negative affect over time.

2. Methods

Data for this study came from the National Longitudinal Survey of Youth 1997 (NLSY97). The NLSY97 is a nationally representative sample of U.S. residents who were born between 1980 and 1984. Adolescents were first sampled and interviewed in 1997 and then were assessed yearly until 2008. The survey assesses adolescents' development in several areas; educational, labor market experiences, mental health, and drug use. The questions about mental health that relate to negative affect were included in Waves 4, 6, and 8, that is, in years 2000, 2002, and 2004. Thus, the analyses for the present study are based on these three waves of data. A total of 7979 adolescents (Mean age at wave 4=17.98, SD=1.44, 49% female) were included in the analyses.

2.1. Measures

To investigate the co-occurrence between negative affect and cigarette use, latent factors of negative affect and a composite item indicator of cigarette consumption were constructed at each wave.

2.1.1. Socioeconomic status—An index of socioeconomic status was constructed using three indicators assessed from the first wave of the NLSY (Wave 1). These indicators were parental income, mother's highest year of education completed, and father's highest year of education completed. A linear combination of the three indicators was created as a measure of the SES index using the loadings from a principal components analysis. The first component explained more than 60% of the total variation among the three indicators. The SES scale index is presented in standard deviation units by ethnic group in Table 1.

2.1.2. Negative affect—Negative affect at Waves 4, 6, and 8 were measured as a latent factor indicated by self-reports of feeling down, depressed, and nervous. Participants were asked the following three questions; "How much of the time during the last month have you felt downhearted and blue?", "How much of the time during the last month have you been a very nervous person?", and "How much of the time during the last month have you felt so down in the dumps that nothing could cheer you up?". Response options for each question were *All of the time* (1), *Most of the time* (2), *Some of the time* (3), and *None of the time* (4). Due to the distribution of the variable for each of the three questions, the categories *All of the time* and *Most of the time* were collapsed into one. Table 2 illustrates the percentage of participants at Wave 4 who indicated they had experienced the above emotions some of the time or more frequently. Higher scores represent greater negative affect.

2.1.3. Cigarette use—The cigarette use indicator at each wave was based on two items assessing whether participants had smoked since the prior interview and the number of days in the previous 30 they had smoked. In the NLSY, to assess whether participants smoked they were asked "Have you smoked a cigarette since the last interview on [date of last interview]?" Response options were *Yes* (1) or *No* (0). Then, participants were asked "During the past 30 days, on how many days did you smoke a cigarette?" For each wave of data, the variable measuring the number of days that participants smoked was recoded such that participants who reported not smoking were coded as having zero days of smoking. The days of smoking were recoded into three additional categories: smoked between one day and 7 days, smoked between 7 days and 21 days, and smoked more than 21 days in the past 30. The percentage of participants, at each wave, who reported smoking more than 21 days in the previous month is presented in Table 2.

2.1.4. Race/ethnicity—The race and ethnicity of participants in the NLSY97 are assessed by two separate questions. One question asked respondents to indicate their race (e.g., White, Black, and Asian) and the other asked about Hispanic ancestry. Participants who identified as Asian (n=160) and American Indian (n=61), were excluded because the number of cases were too low compromising the statistical power of the analyses. Based on these two questions we constructed a race/ethnicity indicator with three categories: African American (n=2192), White (n=4090) and Hispanic (n=1697).

2.2. Analytic approach

To study the longitudinal co-occurrence between negative affect and smoking, a multi group bivariate autoregressive cross-lagged model was fit. The analyses were carried out in three steps. The first step of the analyses involved estimating the measurement model; in the second, the lagged and crossed paths were estimated; and the third step was used to estimate the ethnic differences among White, African American and Hispanic. For the measurement model, three parts were required. In the first part, the factor loadings for negative affect were estimated. An examination of the factor loadings suggested that the confirmatory factor model was adequate. The measurement model also assumed the presence of non-invariance in the factor structure of both factors, which was verified by the robust chi-square difference test with mean and variance adjusted test statistics. The chi-square test for difference testing

was 194.475 with 34 degrees of freedom, $p < 0.001$. The significance of the test supported the assumption of non-invariance across the three ethnic groups.

The second part of the measurement model examined the error structure of the categorical indicators across waves which were assumed to be correlated. Third, the longitudinal structure of the factor loadings was also assumed to vary across groups but was fixed across time. In other words, longitudinal measurement invariance was assumed. The chi-square test for difference testing favored the longitudinal measurement invariant model ($\chi^2_4 = 8.070$, $p = 0.089$).

In the second step of the analyses, a total of eleven paths and correlations of errors were added in the measurement model; eight crossed and lagged paths between cigarette and negative affect. The other three estimates were the correlations of the disturbance terms in Waves 4, 6 and 8 between smoking and negative affect. These crossed, lagged paths coefficients, and correlations were estimated in two parts of the multi-group analysis. In these two parts, the measurement model was identical in all models to accurately explore racial/ethnic differences only in the relationship between smoking and negative affect. First, the invariant model was estimated constraining all lagged, crossed and disturbances to be equal across groups (Model 1). Second, the models were estimated separately by each group (one model for African Americans, another for Whites and another for Hispanics) (Model 2). Based on these preliminary results there were four differences tested between groups; (a) differences in the crossed effect from smoking in Wave 4 to negative affect in Wave 6 (Model 3); (b) differences in the crossed effect from smoking in Wave 6 to negative affect in Wave 8 (Model 4); (c) third difference in the lagged effect from smoking at Wave 4 to Wave 6 (Model 5); and (d) differences in the lagged effect of smoking at Wave 6 to Wave 8 (Model 8). Then, Model 7 was estimated by combining the resulting relationships found in Models 5 and 6.

In the third step of the analyses, the results from the final set of models comparing racial/ethnic groups were adjusted by socioeconomic status. In order to test that there were still racial/ethnic differences after controlling by SES, two additional models were tested. Model 8 is the comparison model where no racial/ethnic differences are assumed and SES was added as a control variable. The final model (Model 9) tested racial/ethnic differences in the lagged coefficients from smoking in Wave 4 to smoking in Wave 6, and from smoking in Wave 6 to smoking in Wave 8. Fig. 1 displays the results of this final model that shows the coefficients after controlling by SES.

For all the analyses, the estimator used was WLSMV, a weighted least square parameter estimate using a diagonal weight matrix with standard errors and mean- and variance-adjusted chi-square test statistic that uses a full weight matrix. All models were estimated using MPLUS Version 6.1 (Muthén & Muthén, 2010). The analyses were weighted to account for non-response, different probability of selection, and for oversampling. Missing data were treated using full information matrix.

3. Results

The correlation matrix by racial/ethnic group is presented in Table 3. Overall, the correlation matrix does not suggest the presence of any strong correlation that potentially would cause multicollinearity problems. Despite the fact that some variables presented in Table 3 have high correlations across time, it is exactly the relationship between these variables that this study seeks to model.

In order to test for racial/ethnic differences in the co-occurrence between negative affect and smoking, six models were tested. The results of these tests are displayed in Table 4. The

reference model (Model 1) is the partial invariant model (see Table 4, Model 1) in which the paths in the model are the same across racial/ethnic groups and the measurement of negative affect construct is partly different by group.

Model 1 is the partial invariant model with lagged and crossed paths and it is also the reference model to test for ethnic differences. Model 2 tested for racial/ethnic differences in all lagged and crossed paths from each wave to the subsequent waves. To determine whether this model fit the data better than the reference model in which all lagged or cross paths were the same across groups, the fit indices of both models were examined and used the robust chi-square difference test to compare nested models. The robust chi-square difference test was significant at level $p < 0.05$, suggesting that Model 2 fits the data better. However, the fit indices (CFI, and RMSEA) for the partial invariant model (Model 1) indicate a better fit to the data. According to Muthén, in the presence of a discrepancy between the robust chi-square test and the fit indices, the chi-square test is preferred (Muthén, 2011). In addition, the discrepancies between the fit indices and the chi-square test might also suggest that racial/ethnic differences may not exist in *all* eleven paths at the same time, just in some of them. This means that the full multi-group model in which all paths are different is not the best model.

To get a better understanding of the racial/ethnic differences in these relationships, we examined models that tested differences in specific crossed or lagged paths. Five models were compared to Model 1. Model 3 tested race/ethnic differences in the crossed paths from smoking at Wave 4 to negative affect at Wave 6. This model fit the data worse than Model 1 (test of difference was not significant), implying that racial/ethnic groups have the same crossed paths from smoking to negative affect across waves. Following the same logic, Model 4 tested racial/ethnic differences in the crossed paths from smoking at Wave 6 to negative affect at Wave 8. According to the fit indices and the chi-square test Model 4 does not fit the data better than Model 1.

In Model 5 we examined racial/ethnic differences in the lagged paths for smoking between Waves 4 and 6. Model 6 tested the lagged paths for smoking from Waves 6 to 8. For both Models 5 and 6 the robust chi square test was significant suggesting that Models 5 and 6 fit the data slightly better than Model 1. This indicates that the racial/ethnic group differences might manifest themselves in how smoking changes across time. The last model (Model 7) combined the results of Models 5 and 6 into one model to compare with Model 1. The fit indices of the combined model and chi-square test of difference suggest a better fit to the data than Model 1. The results suggest that there are racial/ethnic differences in the lagged paths of smoking across time (the robust chi square tests is significant at $p < 0.001$). Model 7, depicted in Fig. 1, presents the lagged coefficients of negative affect across waves. In addition, Fig. 1 shows the lagged path coefficients for smoking across time by each group. Lastly, the crossed path coefficients are presented. By all criteria, the fit of this model was excellent (RMSEA=0.024; RMSEA 90% CI: 0.021–0.027; CFI=0.995; and TLI=0.992). A similar pattern of results as that found in Model 7 is found in the model (Model 9) that adjusts the relationships by SES. The resulting model fit for Model 9 is excellent (RMSEA=0.025; RMSEA 90% CI: 0.022–0.028; CFI=0.994; TLI=0.991). In both Model 7 and Model 9, the CFI and TLI exceeded the 0.95 proposed by Bentler (1990) and colleagues (Hu & Bentler, 1999). In order to test for racial/ethnic differences in the smoking lag that remains after controlling by SES, Model 9 was compared to Model 8 (same as Model 1 but adjusted by SES) and the resulting chi-square test of difference is 22.365 ($p > 0.001$), indicating that the restrictions in Model 8 worsen the fit of the model. Therefore, the restrictions posed by Model 8 can be rejected and Model 9, which shows racial/ethnic differences in the cigarette lagged paths across the three waves controlling by SES, is the better fit to the data. The results of this final model are displayed in Fig. 1 below.

For all racial/ethnic groups, negative affect at previous waves influenced negative affect at subsequent waves. Also, cigarette use in prior waves predicted increased cigarette consumption. Racial/ethnic differences, however, were found in how strongly smoking in prior waves is related to smoking in subsequent waves. For instance, for White participants the lag coefficient for smoking between Waves 4 and 6 is 0.685 ($p < 0.001$), while for African American participants it is 0.861 ($p < 0.001$), and for Hispanic participants it is 0.757 ($p < 0.001$). Although the three racial/ethnic groups exhibit the same pattern of positive association for smoking between Waves 4 and 6, these results suggest that Hispanic youth who smoke at Wave 4 are less likely than White or African American youth to smoke at Wave 6. The lagged coefficient for smoking between Waves 6 and 8 was 1.386 ($p < 0.001$) for White participants, 1.232 ($p < 0.001$) for African American participants, and -0.955 ($p < 0.001$) for Hispanic participants. Similar to the lag association for smoking between Waves 4 and 6, Hispanic youth who smoked in Wave 6 are less likely to smoke at Wave 8 than the other two racial/ethnic groups. These results imply that the strength of the relationship of smoking across time is stronger for African American and White adolescents suggesting that they are more likely to maintain their smoking behavior compared to Hispanic young adults.

Also depicted in Fig. 1 is the effect of SES on both negative affect and smoking. There are two interesting results to notice. First, the effect of SES disappears in Wave 8. The fact that SES is measured at Wave 1 of data collection might explain that after 8 years the SES effect becomes weaker. This trend can also be seen in the way the coefficients become smaller across waves.

Second, the effect of SES has a clear pattern in the case of smoking but not in the case of negative affect. The effect of SES is negative on smoking for African American and for White youth but slightly positive for Hispanics. In other words, the higher the SES levels in the household that White and African American adolescents are raised in, the less likely they are to smoke. However, the effect of SES tends to be zero for the Hispanic group. These results are explained in part because the SES distribution is not the same across the three groups. In the case of African American adolescents, the distribution is more leptokurtic centered below the overall mean and skewed to lower levels of SES. In the case of White adolescents the distribution is less leptokurtic than the African American distribution but more centered above the overall mean and more skewed to higher values of SES. However, the SES distribution for Hispanics has three distinctive features: a larger range covering almost all the distribution, a significant higher density in the lower levels of the distribution, a tendency to a bimodal distribution with one mode centered on lower levels of SES and the other centered on the overall SES mean. The difference in the Hispanic SES distribution might make the SES effect among the Hispanic individuals be closer to zero and not showing the expected negative effect.

Finally, the effect of negative affect on smoking becomes non-significant from Wave 6 to Wave 8. Further analysis showed that adolescents who dropped out of the study in Wave 8 had higher rates of smoking at Waves 4 and 6 and also had slightly higher proportions in the indicators of negative affect than youth that remained in the study. This attrition underestimates the relationship between negative affect and smoking.

4. Discussion

The purpose of this study was to explore the longitudinal relationship between negative affect and smoking between adolescence and young adulthood for various racial/ethnic groups. The hypothesis that there would be racial/ethnic differences in the longitudinal trajectory of negative affect was not supported. That is, the association of negative affect with smoking at the subsequent wave was the same for each racial/ethnic group. Similarly,

the association between smoking and negative affect at a subsequent wave did not differ by racial/ethnic group. In particular, the findings indicated that between Waves 4 and 6 smoking was associated with positive increases in the level of negative affect reported by youth from all racial/ethnic groups and that negative affect was associated with increases in smoking. Between the last two waves, it was found that increases in smoking were related to future increases in negative affect for all racial/ethnic groups.

One explanation for the finding that there was no crossed effect from negative affect at Wave 6 to smoking at Wave 8 might be related to the compatibility of smoking and adult roles. At Wave 8, participants in this study were about 22 years of age. Some researchers have suggested that when young adults begin to assume adult roles and responsibilities, such as attending college (McDade et al., 2011), getting married and becoming parents (Oesterle, Hawkins, & Hill, 2011), their levels of smoking decrease because these roles are not compatible with risky and unhealthy behaviors. Further, we suspect that the finding that there was no crossed effect might be due to the fact that attrition at Wave 8 occurred among those individuals who tended to smoke more in prior waves which might have resulted in an underestimation of the crossed coefficient.

The hypothesis regarding the presence of racial/ethnic differences in the longitudinal trajectory of cigarette use was supported. Examining the longitudinal trajectories of cigarette use in this sample reveals that although White young adults are more likely to continue using cigarettes between the first two waves than the other two racial/ethnic groups, they are less likely than African American young adults to use cigarettes between the last two waves. The longitudinal trajectories of cigarette use among the Hispanic young adults reveal a weak relationship between the earlier waves and no significant relationship in the last two waves. We suspect that other factors such as sociocultural variables not modeled in this study may account for the pattern of racial/ethnic differences found (Baler & Volkow, 2011).

4.1. Limitations

The study findings need to be interpreted within the context of three limitations. First, due to the design of the study from which these data were drawn, it was not possible to control for prior level of negative affect or smoking. Second, in this study the only covariate included in the models was SES. There may in fact be other important and common predictors of both cigarette use and negative affect, such as self-efficacy (Engels, Hale, Noom, & De Vries, 2005; Minnix, Blalock, Marani, Prokhorov, & Cinciripini, 2011) and neurological vulnerabilities (Rao, Hammen, London, & Poland, 2009), that were not assessed by the NLSY survey and thus not available for modeling. The third limitation has to do with the time interval between assessments. Due to the design of the NLSY survey which included assessments for negative affect two years apart, the cross effect of negative affect on cigarette use, and vice versa, may in fact be underestimated. Notwithstanding these limitations, the study, using large national representative samples of White, Hispanic, and African American youth, do help explain that negative affect and smoking are interdependent behaviors that influence each other over time and that are correlated at each wave.

4.2. Conclusions and implications

The study findings shed light on an important debate regarding the nature of the relationship between smoking cigarettes or negative affect and suggest that the relationship is bidirectional and interdependent among various racial/ethnic groups, even after accounting for the effect of SES. The findings from this study show that over time, both negative affect and cigarette use are correlated with each other and that, in the early waves, increases in one behavior increase the other. Consistent with prior work on the role that negative affective

states play in the maintenance of cigarette (McLeish et al., 2009), this study's findings further illustrates how these two behaviors increase over time during young adulthood.

The implications of these results for the prevention and treatment of either behavior center on focusing attention on both problem behaviors simultaneously, rather than in isolation. In fact, efforts at incorporating treatment for mood disorders, specifically depression, in smoking cessation programs are underway (Brown et al., 2007; Weinberger, George, & McKee, 2011) and show some promise (Kapson & Haaga, 2010; Strong et al., 2009). In addition, there is some evidence that smoking cessation programs are beginning to examine the effect of quitting on mood disorders. For example, quitting smoking cigarettes has been recently found to not increase the risk on developing mood disorders such as depression (Bolam, West, & Gunnell, 2011). Further, early detection and early prevention of both problems are important given the continuity observed in the results of this study. Early prevention efforts need to continue exploring of the factors that lead adolescents with mood disorders to perceive the risks in smoking (Lawn et al., 2011) and to enhance the use of adaptive coping during negative mood states to prevent cigarette use (Cohen, McChargue, & Morrell, 2007).

Lastly, the results further indicate that racial and ethnic groups differ in how their smoking behavior changes over time. Racial/ethnic differences in cigarette consumption reported by previous studies (Trinidad, Perez-Stable, White, Emery, & Messer, 2011) are paralleled in this study by differences in longitudinal changes in smoking. Future research may consider exploring the patterns of smoking trajectories by racial and ethnic groups to better understand the stressor specific to each racial group that might be related in the maintenance of interdependent relationship between negative affect and smoking.

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Fig. 1.

Bivariate Autoregressive Cross Lagged model of negative affect and smoking adjusted by SES (N=7979). This Bivariate Autoregressive Cross Lagged model accounts for the longitudinal relationship between negative affect (down, depressed and nervous in the past month) and smoking (number of days smoked in the past month). The measurement part of negative affect has fixed loading factors across time and correlated errors across time. For the multi-group analyses, the residual variances of nervous, down, and depressed were fixed to one allowing free estimation of thresholds for identification purposes. All factor loads are significant at $p < 0.001$. The model takes into account the correlated disturbances of endogenous measurements between negative affect and smoking. All crossed and lagged coefficients are constrained to be equal across group except for the lagged path of cigarettes and the effects of SES on negative affect and smoking. Group differences are presented according to the following font styles: **Bold italic: African American (on top)**; *Italic: white (in the middle)*; **Bold: Hispanic (on the bottom)**; and normal font meaning no ethnic differences. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$, dotted lines represent non-significant paths (coefficients not shown), and all factor loadings and residual variances are significant at $p < 0.001$.

Table 1

Sample demographic characteristics by racial/ethnic group

	<u>African American</u>	<u>White</u>	<u>Hispanic</u>
Demographics	%	%	%
Female	49.9	48.1	48.6
Age			
16	18.6	20.4	20.3
17	20.5	20.8	20.5
18	21.3	20.3	20.9
19	19.3	20.2	19.6
20	20.3	18.3	18.7

	Mean	Std. dev.	Mean	Std. dev.	Mean	Std. dev.
SES	-0.168	(0.62)	0.371	(0.935)	-0.709	(1.083)

Note: SES has a mean of zero and standard deviation of 1, higher values indicate higher SES level.

Table 2

Percentage of individuals who endorsed study variable categories by racial/ethnic group.

Negative affect	African American			White			Hispanic		
	None of the time	Some of the time	Most/all of the time	None of the time	Some of the time	Most/all of the time	None of the time	Some of the time	Most/all of the time
Feel depressed (w4)	62.1	27.8	10.1	66.2	28.9	5.0	58.9	32.0	9.1
Feel depressed (w6)	59.8	30.3	9.9	67.1	27.9	5.0	61.0	32.0	7.0
Feel depressed (w8)	65.0	27.4	7.6	71.5	24.8	3.8	66.7	26.7	6.6
Feel nervous (w4)	40.8	47.9	11.4	31.9	57.3	10.8	35.1	51.7	13.2
Feel nervous (w6)	31.2	54.8	13.9	24.9	61.3	13.8	26.4	57.4	16.2
Feel nervous (w8)	48.4	41.5	10.1	35.6	54.4	10.0	39.8	49.3	10.8
Feel down (w4)	33.3	49.8	16.9	28.8	59.2	12.0	31.2	51.6	17.2
Feel down (w6)	29.5	53.7	16.9	27.7	60.3	12.0	30.2	55.7	14.1
Feel down (w8)	34.7	51.3	13.9	32.8	57.4	9.8	36.4	51.1	12.6
Smoking	Not smoke	Less than a week	More than a week	Not smoke	Less than a week	More than a week	Not smoke	Less than a week	More than a week
Smoke w4	78.1	7.0	14.9	60.0	8.9	31.1	72.0	11.0	17.0
Smoke w6	70.9	7.7	21.4	55.7	8.4	35.9	67.9	11.0	21.1
Smoke w8	71.4	5.4	23.3	54.8	8.0	37.2	69.4	9.8	20.8

Table 3

Correlation matrix by racial/ethnic group of variables modeled in Fig. 1.

	1	2	3	4	5	6	7	8	9	10	11	12
African American (n=2192)												
1 Nervous W4												
2 Down W4	0.452											
3 Depressed W4	0.453	0.599										
4 Nervous W6	0.417	0.256	0.267									
5 Down W6	0.267	0.430	0.370	0.474								
6 Depressed W6	0.287	0.314	0.405	0.457	0.623							
7 Nervous W6	0.424	0.237	0.276	0.459	0.306	0.294						
8 Down W6	0.275	0.386	0.299	0.285	0.404	0.343	0.492					
9 Depressed W8	0.300	0.268	0.400	0.308	0.376	0.467	0.468	0.655				
10 Smoking W4	0.119	0.086	0.046	0.045	0.066	0.086	0.043	0.032	0.086			
11 Smoking W6	0.107	0.078	0.099	0.117	0.139	0.176	0.090	0.077	0.156	0.564		
12 Smoking W8	0.081	0.076	0.092	0.098	0.091	0.122	0.140	0.087	0.163	0.530	0.850	
13 SES	-0.061	-0.033	-0.117	-0.090	-0.065	-0.132	-0.027	-0.014	-0.101	-0.093	-0.113	-0.095
White (n=4090)												
1 Nervous W4												
2 Down W4	0.495											
3 Depressed W4	0.473	0.713										
4 Nervous W6	0.458	0.339	0.294									
5 Down W6	0.316	0.464	0.381	0.534								
6 Depressed W6	0.274	0.363	0.457	0.493	0.682							
7 Nervous W6	0.455	0.333	0.283	0.497	0.345	0.291						
8 Down W6	0.273	0.403	0.318	0.310	0.450	0.321	0.507					
9 Depressed W8	0.308	0.330	0.395	0.321	0.411	0.460	0.541	0.754				
10 Smoking W4	0.102	0.122	0.171	0.081	0.118	0.144	0.082	0.074	0.145			
11 Smoking W6	0.110	0.113	0.187	0.119	0.149	0.182	0.100	0.125	0.204	0.677		
12 Smoking W8	0.092	0.114	0.215	0.114	0.137	0.193	0.149	0.173	0.240	0.635	0.879	
13 SES	-0.018	-0.001	-0.120	0.001	-0.017	-0.114	0.014	-0.016	-0.100	-0.164	-0.204	-0.203
Hispanic (n=1697)												

	1	2	3	4	5	6	7	8	9	10	11	12
1 Nervous W4												
2 Down W4	0.454											
3 Depressed W4	0.447	0.619										
4 Nervous W6	0.439	0.276	0.297									
5 Down W6	0.267	0.430	0.351	0.552								
6 Depressed W6	0.239	0.303	0.439	0.460	0.617							
7 Nervous W6	0.420	0.277	0.262	0.481	0.329	0.278						
8 Down W6	0.291	0.385	0.277	0.370	0.409	0.346	0.527					
9 Depressed W8	0.247	0.295	0.438	0.306	0.350	0.415	0.475	0.665				
10 Smoking W4	0.043	0.083	0.093	0.059	0.076	0.080	0.040	0.066	0.060			
11 Smoking W6	0.029	0.060	0.051	0.129	0.131	0.144	0.094	0.099	0.090	0.593		
12 Smoking W6	0.022	0.094	0.073	0.073	0.044	0.031	0.134	0.154	0.110	0.547	0.756	
13 SES	-0.039	0.049	-0.090	-0.005	0.066	-0.031	0.012	0.002	-0.088	0.081	0.123	0.089

Table 4

Model comparison to test racial/ethnic differences in the co-occurrence of smoking and negative affect (N=7979).

Models ^a	RMSEA	CI RMSEA	CFI	TLI	Chi square	Test of difference		
1. Fixed lag and cross paths	0.025	(0.022, 0.028)	0.994	0.992	382.675	***		
2. Differences in lag and cross paths	0.031	(0.028, 0.034)	0.992	0.988	443.094	***	35.327	*
3. Smoking W4 to negative affect W6	0.027	(0.024, 0.030)	0.993	0.991	417.998	***	3.081	
4. Smoking W6 to negative affect W8	0.026	(0.023, 0.029)	0.994	0.991	409.106	***	1.479	
5. Smoking W4 to W6	0.025	(0.022, 0.028)	0.994	0.992	377.544	***	12.357	**
6. Smoking W6 to W8	0.024	(0.021, 0.027)	0.995	0.993	369.154	***	19.254	***
7. Combined Models 5 and 6	0.024	(0.021, 0.027)	0.995	0.993	355.973	***	24.335	***
8. Fixed lag and cross paths with SES	0.025	(0.022, 0.028)	0.993	0.990	438.756	*		
9. Final model (Model 7 with SES)	0.025	(0.022, 0.028)	0.994	0.991	426.948	*	22.365	***

In the multi-group analyses, the residual variances of nervous, down and depressed were fixed to one allowing free estimation of thresholds for identification purposes. The reference model to test ethnic differences for Models 2 to 7 is the partial invariant model (Model 1). The reference model to test ethnic differences for Model 9 is Model 8. The ethnic differences were tested in the crossed and lagged paths. In Model 2, all crossed and lagged coefficients differ across ethnic groups. In the rest of the models, specific differences were tested. The final model combines the significant differences found after adjusting by SES.

^a All models, in the measurement part of negative affect, have fixed loading factors across time and correlated errors across time.

*
 $p < 0.05$.

**
 $p < 0.01$.

 $p < 0.001$.