

BRIEF REPORT

The Relative Impact of Socioeconomic Status and Childhood Trauma on Black-White Differences in Paranoid Personality Disorder Symptoms

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The current study examines mechanisms of racial differences in symptoms of paranoid personality disorder (PPD) in a sample of adults ages 55–64 from the St. Louis, MO area. Socioeconomic status (SES) and childhood trauma were tested as intervening variables in the association between race and PPD symptoms using structural equation modeling. PPD symptoms were modeled as a latent variable composed of items from the PPD scales of the Multi-Source Assessment of Personality Pathology self and informant reports and the Structured Interview for the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (*DSM-IV*) Personality. Childhood trauma was measured using the Traumatic Life Events Questionnaire, and SES was a composite of parent education, participant education, and annual household income. Blacks exhibited higher levels of PPD symptoms across the 3 personality measures, reported significantly lower SES, and reported greater childhood trauma. The proposed model was a good fit to the data, and the effect of race on PPD symptoms operated mainly through SES. The indirect effect through SES was stronger for males. Findings suggest that racial differences in PPD symptoms are partly explained by problems more commonly experienced by Black individuals.

Keywords: paranoid personality disorder, paranoia, race, socioeconomic status, trauma

Symptoms of paranoid personality disorder (PPD), characterized by maladaptive suspiciousness and distrust in the absence of psychotic symptoms, are associated with role impairment, substance dependence, and poor social functioning (Grant et al., 2004; Trull, Jahng, Tomko, Wood, & Sher, 2010). Higher levels of paranoid symptoms in Blacks as compared with Whites are widely reported (Brown, Schulberg, & Madonia, 1996; Cohen, Magai, Yaffee, & Walcott-Brown, 2004; Jarvis, Toniolo, Ryder, Sessa, & Cremonese, 2011), but racial differences in PPD symptoms are understudied. The National Epidemiologic Survey on Alcohol and Related Conditions found greater prevalence of PPD among Blacks as compared with Whites (Grant et al., 2004). Another study found that African-American college students endorsed more PPD symptoms than White students (Combs, Penn, & Fenig-

stein, 2002). Further research is needed to bolster the assertion that there are Black-White differences in the prevalence of PPD symptoms and to identify contributing factors.

Researchers hypothesize that compared with psychotic forms of paranoia, nonpsychotic forms, which would presumably include PPD symptoms, are more strongly related to cultural and environmental influences (Ridley, 1984; Whaley, 1997, 1998). Specifically, environments that foster feelings of victimization, alienation, and lack of control are posited to contribute to the development of paranoid symptoms (Whaley, 1998). Evidence suggests that Blacks are more likely than Whites to experience victimization and to report interpersonal mistrust and cultural mistrust (i.e., mistrust in the dominant White culture) (Hatch & Dohrenwend, 2007; Roberts, Gilman, Breslau, Breslau, & Koenen, 2011; Whaley, 1998). Thus, it is possible that Black-White differences in the prevalence of PPD symptoms are due in part to the increased frequency of disadvantage in the Black community.

To date, studies have only examined environmental correlates of racial differences in constructs related to PPD symptoms, such as paranoia and mistrust. Other studies have identified risk factors for PPD symptoms without attention to racial differences. In considering both literatures, two types of disadvantage are found to be associated with PPD symptoms or related constructs: socioeconomic status (SES) and childhood trauma. Low SES and some traumatic events occur more frequently among Blacks as compared with Whites (Elliott & Urquiza, 2006; Hatch & Dohrenwend, 2007; Hussey, Chang, & Kotch, 2006; Roberts et al., 2011) and thus may explain racial differences in PPD symptoms.

Individuals of lower SES are more likely to experience discrimination and unequal access to social and economic resources that

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provide opportunities to achieve life goals (Demaris & Yang, 1994). Blacks are more likely than Whites to live in poverty and to be victims of socioeconomic and racial discrimination. These experiences may cause individuals to feel powerless and to perceive the dominant culture as threatening and hostile, leading to suspiciousness, mistrust, and paranoia. As such, low SES may partly explain elevated symptoms of PPD among Blacks.

Past research supports this contention. Lower educational attainment and social status are related to greater odds of meeting diagnostic criteria for PPD (Coid, Yang, Tyrer, Roberts, & Ullrich, 2006; Torgersen, Kringlen, & Cramer, 2001), and SES indicators (i.e., income and education) are associated with greater paranoid ideation (Bazargan, Bazargan, & King, 2001) and mistrust (Whaley, 1998) within Black samples. Whaley (1998) found that when education and income were accounted for, Black and White adults no longer significantly differed on a measure of mistrust. In addition, Whaley (1998) found that Black adults of lower social status (i.e., psychiatric patients and high school dropouts) reported higher levels of cultural mistrust than Black college graduates. The role of SES in specifically explaining racial differences in PPD symptoms has not been studied.

Childhood trauma may also account for racial differences in PPD symptoms. Traumatic experiences early in life may make it difficult for individuals to see others as trustworthy and safe, leading to the development of PPD symptoms. Blacks may be more likely than Whites to develop PPD symptoms in part because they are more likely to experience childhood trauma (Elliott & Urquiza, 2006; Hatch & Dohrenwend, 2007; Hussey et al., 2006; Roberts et al., 2011).

Studies show that a history of childhood physical, emotional, and sexual abuse is associated with PPD symptoms in adolescents and adults (Afifi et al., 2011; Lobbestael, Arntz, & Bernstein, 2010; Tyrka, Wyche, Kelly, Price, & Carpenter, 2009), and these forms of abuse are prospective risk factors for PPD symptoms in adulthood (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999; Natsuaki, Cicchetti, & Rogosch, 2009). These associations are found to persist when controlling for race. One study found that, in a large, urban community-based sample of adults over age 55, lifetime traumatic events were associated with paranoid ideation in Blacks but not Whites (Cohen et al., 2004). The role of trauma in explaining racial differences in PPD symptoms has not been explored.

Although previous studies have shown that SES and trauma are associated with racial differences in paranoid symptoms, none have done so directly using appropriate analytical methods, and very few have focused on PPD symptoms. For example, techniques such as mediation analysis may be better suited to answering questions about mechanisms of racial differences than comparing separate regression analyses for Whites and Blacks (e.g., Cohen et al., 2004). Structural equation modeling (SEM) tests whether a conceptual model adequately represents the pattern of relationships within the observed data. SEM allows for the creation of latent variables, which aggregates multiple measures of a single underlying construct while adjusting for measurement error (Ullman & Bentler, 2012). In addition, SEM can compare nested models (i.e., in which one or more parameters are constrained), enabling researchers to test competing statistical models.

Personality measurement issues present another challenge in research concerned with personality pathology. Multimethod as-

essment enhances the robustness and meaningfulness of conclusions (Galione & Oltmanns, *in press*; Huprich, Bornstein, & Schmitt, 2011). However, most studies have relied on self-report questionnaires or diagnostic interviews to assess paranoid symptoms without including informant-report measures, although informants provide an important perspective on personality problems (Oltmanns, Turkheimer, & Strauss, 1998).

An additional consideration for this research is the potential contribution of gender. The prevalence of paranoid symptoms is found to differ between males and females, although findings conflict as to which gender reports elevated symptoms and under what conditions (Freeman et al., 2011). No studies to date have investigated whether gender influences racial differences in PPD symptoms.

The current analyses use SEM to examine the potential mediating roles of SES and trauma in the association between race and PPD symptoms. We hypothesize that both SES and trauma will explain racial differences in PPD symptoms. In addition, we are interested in whether trauma or SES is a stronger mediator of this association and whether gender moderates the indirect effect of race on PPD symptoms.

Methods

Design

Data are from a community-based sample of adults ages 55–64 participating in a longitudinal study of personality, aging, and health (St. Louis Personality and Aging Network – SPAN; Oltmanns, Rodrigues, Weinstein, & Gleason, *in press*). Participants were recruited using listed phone numbers that were crossed with census data to identify households with at least one member in the target age range. Figure 1 shows the rate of participation from initial contact to follow-up 5 (FU5), 2.5 years after baseline.

Initially, 31% of Black participants were males, compared with 46% of White participants. Thus, additional measures were taken to ensure that Black males were adequately represented in our sample. We sent slightly modified letters to homes in zip codes in which more than 90% of residents were Black and for which a phone number was listed under a male's name. Further detail on

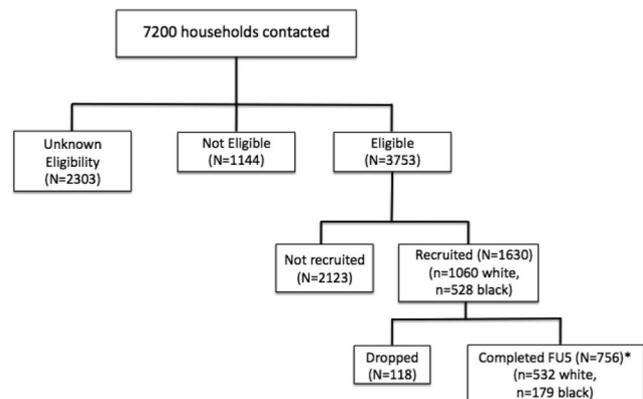


Figure 1. Recruitment flowchart. * Only those with complete personality data were included in analyses ($N = 711$).

this process can be found elsewhere (Spence & Oltmanns, 2011). At the end of baseline recruitment, males made up 43% of the Black sample.

Each participant completed a 3-h baseline assessment, which included a semistructured diagnostic interview for personality disorders and several self-report questionnaires. All participants were also asked to complete an additional 3-h in-person assessment at FU5. Participants were compensated \$20 per hour of participation, and informed consent was obtained before the baseline assessment. All data utilized in the current analyses were collected at baseline, except for data on childhood traumatic experiences, which were collected at FU5. Given that these events occurred before the onset of the study, the timing of their assessment would likely not bias analyses beyond what would be expected with cross-sectional retrospective reports.

Participants

Data are from White and Black participants who completed FU5 by July 2012, when funding was interrupted ($N = 711$, 25% Black, 75% White). The racial breakdown of our sample is similar to that of the St. Louis metropolitan area (76.9% White, 18.1% Black; St. Louis Regional Chamber, 2013). Total attrition at this time was 7% ($n = 118$; 37% Black, 58% White).

Demographic information, broken down by race, is presented in Table 1. Most participants were female and had some postsecondary education. The median annual household income was \$60,000–80,000, slightly higher than that reported for the greater St. Louis area (St. Louis Regional Chamber, 2013), likely due in

part to exclusion criteria (e.g., lack of permanent residence and lower than sixth-grade reading level).

All participants provided informants of their choosing (65.2% female). Approximately 25% of informants were Black and 73% were White, with the remainder being Latino, South Asian, Native American, biracial, or “other.” Ninety-six percent of White participants and 95% of Black participants matched their informant in race. Participants knew their informants for an average of 30 years. Most White participants’ informants were significant others (55.1%) whereas most Black participants’ informants were family members (42.5%). Black and White participants did not differ significantly in terms of how well they knew their informant.

Instruments

Multi-Source Assessment of Personality Pathology. Self- and informant-report PPD symptoms were measured using the Multi-Source Assessment of Personality Pathology (MAPP) (Okada & Oltmanns, 2009; Oltmanns et al., 1998). The MAPP consisted of 88 items reflecting each criterion of the 10 *DSM-IV* personality disorders. Responses were rated on a 5-point scale ranging from 0 (*I/he or she is never like this*) to 4 (*I/he or she is always like this*). The internal consistency of the self-report items is .66 ($\alpha = .67$ for Blacks, .65 for Whites). Internal consistency of the informant-report items is .78 ($\alpha = .79$ for Blacks, .78 for Whites).

Structured Interview for DSM-IV Personality. Interviewer-rated PPD symptoms were measured using the Structured Interview for the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition (*DSM-IV*) Personality (SIDP; Pfohl, Blum, & Zimmerman, 1997). The SIDP is a semistructured diagnostic interview composed of 101 questions corresponding to the criteria for the 10 *DSM-IV* personality disorders. Trained bachelors- and masters-level staff and graduate students in clinical psychology administered the interviews. Responses are rated on a 4-point scale ranging from 0 (*not present or limited to isolated examples in the past 5 years*) to 3 (*strongly present in the past 5 years*). The internal consistency of interviewer-rated PPD items is .70 for the whole sample ($\alpha = .71$ for Blacks, .64 for Whites).

In structural equation models, PPD symptoms were modeled as a latent variable with seven indicators (i.e., the average of the self, informant, and SIDP item corresponding to each of the seven *DSM-IV* PPD criteria). The measurement model fit the data well (comparative fit index [CFI] = .95, root mean square error of approximation [RMSEA] = .08), indicating that the three sources assess overlapping characteristics of PPD. The factor loadings of criteria 5 and 7 were nonsignificant, and a nested model excluding them did not differ from the larger model, $\chi^2[2] = 0.79, p > .10$. The more parsimonious model was used in subsequent analyses. This model was invariant when factor loadings were constrained to be equal between Blacks and Whites, $\chi^2[4] = 4.02, p > .10$, suggesting that any differences between Blacks and Whites is not due to the measurement of PPD symptoms.

SES. SES was modeled as a composite variable (i.e., mean of parent’s education, participant education, and annual household income). Missing data were imputed for household income ($n = 36$) and parent education ($n = 24$; see *Analytic Plan*).

Traumatic events. Trauma was measured using the Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000). The TLEQ

Table 1
Demographic Characteristics

Characteristic	Black ($n = 179$)	White ($n = 532$)
Gender		
Male	71 (39.7)	242 (45.5)
Female	108 (60.3)	290 (54.5)
Mother’s education		
Less than high school	74 (47.4)	95 (18.4)
Completed high school/GED	63 (40.4)	263 (50.9)
Postsecondary education	16 (10.2)	133 (25.8)
Graduate education	3 (1.9)	26 (5)
Father’s education		
Less than high school	79 (55.6)	97 (18.9)
Completed high school/GED	49 (34.5)	218 (42.3)
Postsecondary education	10 (7)	122 (23.8)
Graduate education	4 (2.8)	78 (15.2)
Education		
Less than high school	8 (4.5)	4 (0.8)
Completed high school/GED	60 (33.6)	92 (17.3)
Postsecondary education	83 (46.4)	226 (42.5)
Graduate education	28 (15.6)	210 (39.5)
Annual household income		
Under \$20,000	40 (22.3)	22 (4.1)
\$20,000–39,999	53 (29.7)	76 (14.3)
\$40,000–59,999	39 (22)	114 (21.5)
\$60,000–79,999	16 (8.9)	76 (14.3)
\$80,000–99,999	13 (7.3)	73 (13.9)
Above \$100,000	18 (10)	171 (32.2)
Childhood trauma		
No events	110 (61.5)	431 (81)
One or more events	69 (38.5)	101 (19)

Note. Data presented as n (%). GED = General Educational Development.

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is a 23-item self-report measure of exposure and response to 22 types of potentially traumatic events. The TLEQ has shown convergent validity when compared with a structured trauma interview and *DSM-IV* posttraumatic stress disorder diagnoses (Kubany et al., 2000). For current analyses, the “fear, helplessness, or horror” item was used to measure trauma related to childhood maltreatment (i.e., physical abuse, witnessing family violence, sexual abuse by an adult or child before age 13, sexual abuse between age 13 and 18). Responses were summed to create a score between 0 and 5.

Analytic Plan

Structural equation models were tested with Amos v.21 for SPSS. All other analyses were performed in SPSS. Racial differences were examined using independent sample *t* tests and χ^2 tests. SEM analyses used the maximum likelihood estimator to report standardized path estimates. Model fit was evaluated using the CFI and the RMSEA (Ullman & Bentler, 2012). The significance of indirect effects, the contrast between indirect effects, and conditional indirect effects were tested using bootstrapping with 95% confidence intervals (CIs). All variables were standardized in SEM analyses. Missing data for predictor variables were handled in Amos using regression imputation.

Results

Prevalence of PPD Symptoms

On the basis of the SIDP, 13.6% (*n* = 97) of participants endorsed one or more symptoms, 4.2% (*n* = 30) endorsed two or more, 1.8% (*n* = 13) endorsed three or more, and 0.7% (*n* = 5) endorsed four or more symptoms. On the basis of the self-MAPP, 25.6% (*n* = 182) endorsed one or more symptoms, 7.4% (*n* = 53) endorsed two or more, 2.5% (*n* = 18) endorsed three or more, and 0.7% (*n* = 5) endorsed four or more. On the basis of the informant-MAPP, 36.4% (*n* = 259) endorsed one or more symptoms, 17.4% (*n* = 124) endorsed two or more, 7% (*n* = 50) endorsed three or more, and 3.5% (*n* = 25) endorsed four or more.

Zero-Order Correlations

Table 2 shows zero-order correlations among PPD symptom mean scores, SES indicators, and childhood trauma. All variables were significantly correlated. Correlations of PPD symptom scores among the three measurement sources were all modest and statis-

Table 2
Zero-Order Correlations

	1	2	3	4	5	6	7
1. SIDP		.44^b	.21^b	.08^a	-.12 ^b	-.13 ^b	-.15 ^b
2. Self-MAPP			.24^b	.10^b	-.09 ^a	-.10 ^b	-.10 ^b
3. Informant-MAPP				.14^b	-.11 ^b	-.14 ^b	-.15 ^b
4. Childhood trauma					-.16 ^b	-.14 ^b	-.15 ^b
5. Parent education						.35 ^b	.30 ^b
6. Education							.49 ^b
7. Household income							

Note. Cross-method correlations in bold.
^a *p* < .05. ^b *p* < .01.

Table 3
Racial Differences in PPD Symptoms, Demographics, and Childhood Trauma

	Black, <i>M</i> (<i>SD</i>)	White, <i>M</i> (<i>SD</i>)	<i>d</i>	<i>t</i> (<i>p</i>)
PPD symptoms				
SIDP	.25 (.33)	.13 (.23)	.42	-4.76 (<.001)
Self-MAPP	.80 (.59)	.64 (.44)	.31	-3.49 (<.001)
Informant-MAPP	.95 (.78)	.84 (.65)	.15	-1.78 (.07)
Household income	2.85 (1.68)	4.57 (2.18)	.88	10.96 (<.001)
			ϕ	χ^2 (<i>p</i>)
Parent education	—	—	.51	137.94 (<.001)
Education	—	—	.36	94.20 (<.001)
Childhood trauma	—	—	.20	28.17 (<.001)

tically significant (see also Oltmanns et al., in press). The correlations between childhood trauma and PPD symptom scores were small and significant, as were correlations between SES indicators and PPD symptom scores and SES indicators and childhood trauma. SES indicators were moderately and significantly correlated.

Race and Gender Differences

Racial differences in all variables are shown in Table 3. Blacks had significantly greater PPD mean scores compared with Whites on the basis of self-report and interview, and differences in informant-report scores approached significance. Black participants reported significantly lower levels of education, annual household income, and parent education than Whites. Blacks reported a significantly greater number of traumatic childhood events. Males had significantly higher PPD mean scores than females on the basis of self-report (*t* = 2.04, *d* = 0.15; *p* < .05). Females had lower SES (*t* = 2.89, *d* = 0.22; *p* < .01) and endorsed more childhood trauma (*t* = -2.85, *d* = 0.22; *p* < .01) than males.

Do SES and Childhood Trauma Account for Black-White Differences in PPD Symptoms?

To test the mechanisms responsible for racial differences in PPD symptoms, we predicted PPD symptoms by race (White = 0; Black = 1), in which SES and childhood trauma served as explanatory intervening variables (see Figure 2). Gender was included as a covariate. Overall model fit was good (CFI = .95, RMSEA = .06). As anticipated, race, SES, and childhood trauma were unique predictors of PPD symptoms. The indirect effect of race on PPD symptoms through SES and childhood trauma was significant (.13; 95% CI: .07-.22).

To compare the strength of the separate indirect effects through SES and childhood trauma, a bootstrapping procedure was used to calculate the 95% CI around the contrast between the effects. Both indirect effects independently predicted PPD symptoms (SES .14, 95% CI: .11-.17; trauma .04, 95% CI: .02-.05). The contrast between the indirect effects (SES_{effect} - Trauma_{effect}) was significant and positive (.10, 95% CI: .08-.15), indicating that SES is a stronger mediator than childhood trauma.

To determine if gender moderated each indirect effect, bootstrapping was used to calculate a 95% CI around each conditional

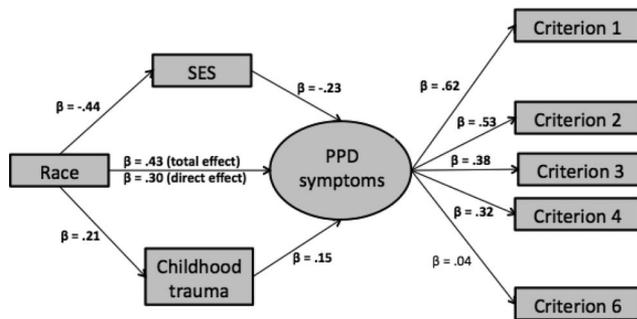


Figure 2. Structural equation model of the indirect effect of race on PPD symptoms through SES and childhood trauma. Gender included as covariate. Estimates in bold significant at $p < .01$.

indirect effect (i.e., the product of the effect of race on the mediator and the two-way interaction between gender and the mediator to predict PPD symptoms; Hayes, 2013). The conditional indirect effect through SES was significant and negative (because of the negative association between race and SES; 95% CI: $-.22$ to $-.14$). The indirect effect was significant for both genders, but it was stronger for males (.16) than females (.13). The conditional indirect effect through trauma was not significant, indicating no difference in the indirect effect between males and females.

Discussion

Up to one third of participants endorsed at least one PPD symptom. Consistent with previous studies, 7–3.5% of participants endorsed four or more symptoms (Lenzenweger, 2008). The sources of personality measurement were significantly, albeit modestly, correlated, suggesting that all sources provide useful information about PPD symptoms. Blacks endorsed significantly elevated PPD symptoms compared with Whites on the basis of self-report and interview, and informant-report differences approached significance, indicating that Black-White differences in PPD symptoms are robust across measurement methods.

We tested models of Black-White differences in PPD symptoms. Lower SES and greater childhood trauma contributed to elevated PPD symptoms among Blacks. However, SES better accounted for Black-White differences than exposure to childhood trauma. These results complement previous research demonstrating that SES partly accounts for racial differences in paranoid symptoms (e.g., Whaley, 1998) and that trauma is associated more strongly with paranoid symptoms among Blacks (Cohen et al., 2004). Findings also revealed that SES better explained racial differences in PPD symptoms for males as compared with females.

Findings suggest that racial differences in PPD symptoms are influenced by social inequality. Individuals from marginalized socioeconomic and racial groups may experience chronic discrimination and prejudice, leading them to become more suspicious, paranoid, and/or distrustful than those from more privileged groups (Demaris & Yang, 1994; Harper, 2011; Whaley, 1998). Although males are a privileged group, the context of low SES may set males at a relative disadvantage to females for various reasons, such as a higher likelihood of being the victim of violent crime and experiencing social surveillance (e.g., being stopped by police; Harper, 2011).

SES and childhood trauma may differ in strength as intervening variables because they are associated with distinct types of mistrust. Childhood trauma may be more robustly associated with interpersonal mistrust and low SES with cultural mistrust (Demaris & Yang, 1994; Whaley, 1998). If so, then the development of PPD symptoms among Blacks may be more strongly related to a lack of trust in the dominant culture than distrust of individuals. These hypotheses can be tested in future studies by including multiple measures of trauma and mistrust. Future research may also examine racial differences in the pathological nature of PPD symptoms by investigating their associations with important life outcomes.

Limitations of the current study should be noted. Participants were adults of a specific generational status residing in the St. Louis area, limiting generalizability to other populations. In addition, other environmental and genetic factors that were not examined are certainly important to understanding racial differences in PPD symptoms and should be included in future studies. For example, perceived discrimination appears to be associated with nonclinical forms of paranoia and is related to race and SES (Combs et al., 2006; Whaley, 1998). Furthermore, the analyses presented herein are cross-sectional, and longitudinal studies are needed to determine whether the current model bears out over time.

The current study adds to a small body of literature on an important racial mental health disparity. Findings indicate that Blacks exhibit higher levels of PPD symptoms as compared with Whites in part due to lower SES and greater childhood trauma. Elevated levels of PPD symptoms among some Blacks may represent a reasonable reaction to cultural and socioeconomic threats. As such, Black-White disparities in PPD symptoms appear not to be caused by race per se, but by problems more commonly experienced by Blacks.

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