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Evaluating hypothesized explanations for the Black-white Depression Paradox: A critical review of the extant evidence

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Abstract

The Black-white Depression paradox, the lower prevalence of major depression among non-Hispanic Black (relative to non-Hispanic white) individuals despite their greater exposure to major life stressors, is a phenomenon that remains unexplained. Despite a decade plus of research, there is little clarity as to whether the paradoxical observations are an invalid finding, spuriously produced by selection bias, information bias, or confounding, or are a valid finding, representative of a true racial patterning of depression in the population. Though both artefactual and etiologic mechanisms have been tested, a lack of synthesis of the extant evidence has contributed towards an unclear picture of the validity of the paradox and produced challenges for researchers in determining which proposed mechanisms show promise, which have been debunked, and which require further study. The objective of this critical review is to assess the state of the literature regarding explanations for the Black-white depression paradox by examining some of the more prominent hypothesized explanatory mechanisms that have been proposed and assessing the state of the evidence in support of them. Included mechanisms were selected for their perceived dominance in the literature and the existence of at least one, direct empirical test using DSM major depression as the outcome. This review highlights the very limited evidence in support of any of the extant putative mechanisms, suggesting that investigators should redirect efforts towards identifying novel mechanisms, and/or empirically testing those which show promise but to date have been relatively understudied. We conclude with a discussion of the broader implications of the evidence for well-accepted social theories and raise questions regarding the use of DSM major depression to assess mental health burden in Black communities.

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Introduction

Despite greater exposure to major life stressors (Dohrenwend, 2000; Hammen, 2005; Kessler, 1997; Kessler et al., 1999; R. Williams & Williams-Morris, 2000), Black people in the United States (U.S.) relative to white people, experience comparable or lower prevalence of unipolar major depression (operationalized in accordance with DSM-III-R, IV, and V diagnostic criteria for major depressive episode and disorder) (Breslau et al., 2006; Breslau et al., 2005; Hasin et al., 2018; Kessler et al., 1994; Somervell et al., 1989). This phenomenon is referred to as the Black-white depression paradox due to the well documented relationship between major life stressors and depression and has been a topic of study for decades. Lifetime prevalence of major depressive disorder is 2% – 8% lower among Black Americans (Barnes & Bates, 2017; Hasin et al., 2018). However, investigators have yet to reach a consensus on whether these racial patterns are valid, and the underlying drivers of the paradox remain unknown.

The paradox has important implications for how we think of depression in communities, though these depend on the validity of the estimates. Invalid estimates would imply the presence of artefactual mechanisms (i.e., selection bias, information bias, confounding). This would suggest that the burden of depression within Black communities is being systematically underestimated, and that more resources should be devoted towards addressing an already stark treatment disparity (Simpson et al., 2007). Conversely, valid estimates would be the product of etiologic mechanisms producing a true lower burden of depression for Black people relative to white people, challenging our understanding of the factors that denote a group as high-risk for depression, relative to another.

The question of what drives the observed racial patterns in depression has yet to be resolved. One impediment to this resolution has been inconsistent use of the relevant operationalization of depression. Despite having a lower burden of diagnosed depression, Black people often have higher levels of depressive symptoms and non-specific psychological distress, relative to their white counterparts (Kessler & Neighbors, 1986; Mehta et al., 2015; Vega & Rumbaut, 1991; Weissman et al., 2015; Wellman, 1993). This means that the paradoxical racial pattern observed for diagnosed depression does not extend to depressive symptomology, making symptomology an inappropriate proxy measure when evaluating the paradox.

Another critical impediment is that the extant empirical evidence has yet to be synthesized and critically evaluated, despite many mechanisms being proposed and tested across different disciplines (e.g., epidemiology, sociology, social psychology). As a result, it is difficult to draw conclusions about the degree of empirical support for the proposed mechanisms (i.e., which mechanisms have found support, which have been falsified or lack evidence, which have not been empirically tested). This lack of synthesis is especially

problematic for investigators when determining whether to devote effort towards testing existing hypothesized mechanisms or towards developing new ones.

The objective of this critical review is to assess the state of the literature regarding explanations for the Black-white depression paradox by examining some of the more prominently proposed explanatory mechanisms and assessing the state of the evidence in support of them. To do so, this review first summarizes and critically reviews the state of the evidence regarding 7 frequently discussed explanations for the Black-white depression paradox. These include 4 artefactual mechanisms (selection based on institutionalized or residential status, differential misclassification by diagnostic instrument, somatization, and clinician bias) and 3 etiologic mechanisms (racial socialization, social support, and the Environmental Affordances Model). Each was selected by the authors based on the mechanisms' prominence in the literature and the existence of at least one, direct empirical test using DSM major depression as the outcome. We then discuss the implications of these findings for future research, especially regarding decisions to prioritize testing existing hypotheses versus developing alternative ones. Finally, we discuss the broader implications of the evidence for dominant social theory and raise questions regarding the use of DSM major depression to assess mental health burdens in Black communities.

Artefactual Mechanisms

In this section, we review artefactual mechanisms that have been proposed to explain the Black-white Depression Paradox. Each mechanism, if present, would suggest that the "paradox" is a product of invalid estimates of racial patterns in depression. One of the proposed mechanisms, selection based on institutionalized or residential status, is a form of selection bias, while the other three mechanisms, differential misclassification by diagnostic instrument, somatization, and clinician diagnostic bias, are all forms of measurement error. A summary of the four mechanisms is presented below, along with an evaluation of the empirical support for each.

Selection based on institutionalized or residential status

A limitation of the epidemiologic studies in which the paradox has been documented is that they are all comprised of samples of non-institutionalized and stably housed populations, meaning the depression paradox could be a product of selection based on institutionalized or residential status (Barnes et al., 2013; C. L. Keyes, 2009; K. M. Keyes et al., 2011; Schwartz & Meyer, 2010). People who are homeless, living on military bases, or incarcerated, are all absent from studies of community-based populations. All three groups have higher rates of depression than the general population (Bassuk et al., 1998; Diamond et al., 2001; Gadermann et al., 2012; North et al., 1998; Ramsawh et al., 2014; Ritchey et al., 1990; Smith et al., 1993; Teplin et al., 1996), and are disproportionately comprised of Black individuals (Bronson, 2019; Nunez & Fox, 1999; OneSource, 2017). Failing to account for the prevalence of depression among these populations in which Black people are disproportionately represented could mask a true higher prevalence of depression among Black people relative to whites. To test this hypothesis, Barnes et al. used data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), the

National Comorbidity Survey Replication, and the National Survey of American Life (NSAL), to analyze Black-white patterns in depression prevalence across strata of gender, age, and educational attainment. If selection on institutionalized or residential status is driving the depression paradox, then the paradox should be less present among the subgroups less likely to be institutionalized or unstably housed (e.g., highly educated older women). Across datasets, the finding of lower depression prevalence among Black individuals remained stable across strata, suggesting that the observed racial patterns in depression are not artificially produced by selection bias (Barnes et al., 2013).

Differential Misclassification by Diagnostic Instrument

Some investigators have suggested that the paradox may be produced by differentially performing structured assessment tools (Barnes & Bates, 2019; Barnes et al., 2013; Breslau et al., 2008; K. M. Keyes et al., 2011; Mezuk et al., 2010; Mouzon, 2014). Black and white individuals could systematically respond differently to items in structured diagnostic instruments (because of cultural significance of wording or differences in symptom experiences), especially considering that many of the tools were developed and tested in primarily all-white samples (Andreasen et al., 1981; Robins et al., 1981; Rogler, 1999). Under this hypothesis, Black individuals with depression would be misclassified as not having depression, such that the prevalence would be artificially low. Breslau et al. tested this mechanism by using data from the National Comorbidity Survey (NCS) to assess differential item functioning (DIF) (group-based differences in the probability of endorsement of items on an assessment tool) by race, of items in the Composite International Diagnostic Interview (CIDI) (Breslau et al., 2008; Kessler et al., 1998). Though there were small differences between groups, they did not account for the lower prevalence of depression among Black individuals in the sample. Furthermore, the higher prevalence of depression among white individuals remained even after recalculating the prevalence excluding the questions with DIF between racial groups (Breslau et al., 2008).

Somatization

There is suggestive yet inconsistent evidence that Black Americans are more likely to report depression in terms of somatic versus psychological symptoms (Blazer et al., 1998; Brown et al., 1996; Cole et al., 2000). This could result in a lower depression prevalence among Black individuals, as the DSM diagnostic algorithm is preferentially weighted towards psychological symptoms (5 psychological symptoms compared to 4 somatic; both depression screener symptoms are psychological) (American Psychiatric Association, 2013). Two studies empirically assessed this hypothesis using versions of the Alcohol Use Disorders and Associated Disabilities Interview Schedule: one using data from NESARC to assess DIF in somatic symptoms among a number of different race and ethnic groups (Uebelacker et al., 2009), and the other using the National Longitudinal Alcohol Epidemiologic Survey to assess racial differences in reporting somatic symptoms, and its impact on meeting diagnostic criteria for depression (Barnes & Bates, 2019). Both studies found slightly higher somatization among Black individuals, and in both cases these differences were driven by the same somatic symptom, changes in weight/appetite. The study by Uebelacker et al. (2009) observed that with the exception of the weight/appetite variable, one group was not more likely to report somatic symptoms than the others.

Additionally, the small difference in somatization found in the study by Barnes & Bates (2019) did not account for differences in likelihood of endorsing a screener question, meaning endorsement of somatic symptoms had no impact on the likelihood of endorsing one of the psychological symptoms that generally serve as a gatekeeper to the rest of the diagnostic assessment. In sum, neither study found support for somatization as an explanation for the depression paradox.

Clinician Diagnostic Bias

Clinician diagnostic bias, though important for understanding racial patterns in communities, holds little bearing for understanding patterns of depression documented in nationally representative surveys. Unlike clinician examinations which may or may not be based on a structured assessment tool, depression outcomes in community-based surveys are derived from non-clinician-administered structured instruments, and therefore are less likely to be influenced by the implicit biases of the person administering the assessment (Dohrenwend & Dohrenwend, 1982). Nonetheless, diagnostic bias is frequently raised and has been tested empirically as a potential explanation for the paradox.

When evaluating Black patients, clinicians are more likely to misdiagnose depression as a psychotic spectrum disorder than when evaluating white patients (Neighbors et al., 1999; Strakowski et al., 2003). This finding has been referenced as a possible cause of the depression paradox, positing that the misdiagnosing of Black patients due to physician-held biases could artificially lower the observed prevalence of depression among Black individuals (Barnes & Bates, 2019; Barnes et al., 2013; K. M. Keyes et al., 2011; Neighbors et al., 2003). Neighbors et al. (2003) explored this hypothesis by blindly administering semi-structured interviews to a group of Black and white psychiatric inpatients with existing admitting diagnoses (i.e., schizophrenia, schizoaffective disorder, depression, bipolar disorder, “other”) based on unstructured clinician examination. Though there were absolute changes in diagnoses when using the structured instrument, the racial pattern in depression diagnoses remained the same, indicating that the lack of an increased burden of depression for Black people was not driven by clinician bias (Neighbors et al., 2003).

Etiologic Mechanisms

In this section, we review 3 etiologic mechanisms as potential explanations for the paradox. If operative, these mechanisms would mean that the paradox is the product of valid estimates for racial patterns in depression, and therefore is a “true” finding. Below we summarize the 3 mechanisms, racial socialization, social support, and the Environmental Affordances Model, and evaluate the extant empirical evidence for each.

Racial Socialization

Racial socialization refers to the process in which people of varying racial groups (in this case, Black people) are implicitly and explicitly primed for the realities that they will face as a result of their place in a racialized society (Hughes et al., 2006; Reynolds & Gonzales-Backen, 2017; Stevenson Jr, 1995). For Black people, this socialization process often includes messages of racial pride, instilling expectations of having to work harder than their

white peers to be successful, and priming for potentially dangerous encounters with the police and other experiences of discrimination (Hughes et al., 2006; Reynolds & Gonzales-Backen, 2017). This socialization process has been hypothesized to prime Black individuals for future experiences with stressors and other hardships in a way that may lessen their impact on mental health and depression specifically (Barnes & Bates, 2019; Barnes et al., 2013; K. M. Keyes et al., 2011; Mouzon, 2017; Reynolds & Gonzales-Backen, 2017; Rosenfield, 2012). A study by Rosenfield tested the racial socialization mechanism using self-salience schemas as a proxy for racial socialization. Self-salience is a sociological construct that refers to the importance of one's self relative to others in a social relationship (Rosenfield et al., 2005). Schemas of self-salience that elevate the importance of 'others' at the expense of the self, raise the risk of internalizing problems, such as depression. Conversely, self-schemas that elevate the importance of the self are hypothesized to reduce risk of internalizing problems (Rosenfield et al., 2005). In this study, Rosenfield (2012) posited that racial socialization protects Black people from depression in the face of socioeconomic stressors by providing messaging that leads to improved schemas of self-salience that prioritize the self(Rosenfield, 2012). Using data from the NCS, Rosenfield (2012) found that Black women had a reduced odds of depression relative to white women (OR: 0.65, 95% CI: 0.43, 0.98), and that the magnitude of the effect attenuated (though not fully) when adjusting for self-salience (OR: 0.89, 95% CI: 0.53, 1.48).

There are two major limitations of the Rosenfield study. Firstly, racial socialization was not directly measured, and secondly, the analysis was restricted to women, though the paradox holds for men as well. The lack of direct measurement of racial socialization speaks to two broader challenges of assessing racial socialization as an explanation for between-racial-group differences. A standard, validated measure of racial socialization has yet to be developed and widely used in the literature. Secondly, if such a measure did exist, there would be questions regarding its utility in white samples, as the construct is experienced very differently within white families (Hughes et al., 2006).

One study has interrogated the role of racial socialization in racial patterns of depression without restricting the analyses by gender; however, the results are not applicable for the paradox as the outcome was measured using the Center for Epidemiologic Studies Depression Scale (CESD), a frequently used screener tool that assesses depressive symptomatology (DeAngelis, 2020) (Radloff, 1977).

Limitations aside, though the findings of the Rosenfield study are supportive of a partial role of racial socialization in producing the depression paradox, more evidence is needed. In particular, new approaches to measuring the construct of racial socialization that can be applied to between-racial group comparisons are critical to further investigation of racial socialization as an explanatory mechanism for the depression paradox.

Social support

Social support has also been posited as a potential cause of the depression paradox. Black Americans have been theorized to develop stronger familial and non-familial support systems, in part, to cope with frequent life stressors such as social and economic exclusion (e.g. unemployment, heavy debt, etc.), and injustice at the hands of dominant groups (e.g.

trouble with the law, violence victimization, etc.) (Kiecolt et al., 2008). Additionally, greater social support (in terms of both number and quality of relationships) is associated with decreased prevalence of mood disorders in general, and among Black Americans specifically (Dressler, 1991; Gray & Keith, 2003; Thoits, 1992; Turner & Marino, 1994). Accordingly, social support could account for the lack of an increased burden of depression among Black people.

Using data from the NCS, Kiecolt et al. aimed to test whether racial differences in social support could explain the depression paradox. The investigators found inconsistent findings regarding racial patterns in social support, depending on the operationalization of the construct; Black people were more likely to have a non-spousal confidant than whites, but were less likely to be married. If married, Black people were less likely to report seeing their spouse as a confidant, and reported less support from friends (Kiecolt et al., 2008). These mixed findings challenge the narrative that lower prevalence of depression among Black people could be due to their higher levels of social support.

Subsequently, Shim et al. conducted another test of the hypothesis using data from the NSAL to estimate the relationship between race/ethnicity and depression, adjusting for varying measures of social support (i.e., closeness felt towards family members, closeness felt towards friends, frequency family members help, frequency friends help) (Shim et al., 2012). Shim et al. found that Black Americans had a lower odds of depression relative to non-Hispanic whites (OR: 0.52, 95% CI: 0.44, 0.61) and that the association between racial group membership and depression did not change when adjusting for social support measures (aOR: 0.51, 95% CI: 0.43, 0.60), suggesting that social support was not a driver of the depression paradox in the sample (Shim et al., 2012). Using data from the NSAL, Mouzon conducted similar analyses and assessed whether the relationship between race and any DSM mood or anxiety disorder was mediated by family-based social support (Mouzon, 2013), or friend-based social support (Mouzon, 2014). In regards to family-based support, Black individuals did not have greater levels of social support than white individuals in most of the measures assessed; the few measures that were higher for Black people however did not account for the lower prevalence of depression relative to whites (Mouzon, 2013). Similarly, none of the 7 friend-based social support measures that were assessed attenuated the relationship between race and DSM mood or anxiety disorders (Mouzon, 2014).

None of the above-mentioned studies found support for social support as a driver of the depression paradox. However, social support is a complex construct as it has many dimensions that need to be considered: type of support (material support vs emotional support), the quantification of the support (number of supportive ties versus depth of ties), and the source of support (family support, friend support, etc.). As shown in Table 1, operationalizations of social support in these studies varied substantially. Given that it is not clear which (if any) of these dimensions is the most important for protection from depression, it cannot be said whether the appropriate dimension(s) were captured by these studies, or even if they are appropriately racially patterned in a way that would explain the paradox. Ultimately, refinement of the social support hypothesis is needed in order to determine what facet of social support would be the most salient for the paradox; at which point the state of the evidence could be more appropriately assessed.

Environmental Affordances Model

The Environmental Affordances (EA) Model is one of the more frequently referenced putative explanations for the depression paradox (Barnes & Bates, 2019; Barnes et al., 2013; Anonymous, 2011; Boardman & Alexander, 2011; Hoggard et al., 2019; Jackson et al., 2010; Jamal, 2016; K. M. Keyes et al., 2011; Mezuk et al., 2013; Mezuk et al., 2010; Mezuk et al., 2017; Mouzon, 2013, 2014, 2017; Rodriquez et al., 2017; Rodriquez et al., 2018). First introduced in 2010 by Jackson et al. (2010), and named in 2013 by Mezuk et al. (2013), the EA Model posits that Black individuals, in response to heightened exposure to major life stressors and discrimination, engage in unhealthy coping behaviors (i.e., consumption of alcohol, cigarette smoking, eating of calorie-dense foods) which simultaneously protect them from the negative mental health consequences of stressor exposure while increasing their risk for chronic physical illness. The first empirical test of the theory, conducted by Jackson et al., (2010) used data from the Americans' Changing Lives Survey to assess a 2-way interaction between stressor exposure and unhealthy behaviors on the odds of depression, stratified by race. The investigators noted that among Black individuals, the relationship between stressor exposure and depression was modified such that the relationship was muted among those with greater engagement with unhealthy behaviors; this patter however was not observed among white people, as the odds of depression increased with stressor exposure regardless of engagement with unhealthy behaviors (Jackson et al., 2010). This same pattern was observed in the study by Mezuk et al., (2010) using data from the Epidemiologic Catchment Area study. Though neither study demonstrated that the use of unhealthy behaviors accounted for observed racial patterns in depression, the findings provided evidence suggesting the plausibility of the mechanism.

However, a study conducted by Keyes et al. (2011) using data from the NESARC failed to replicate the findings of the two previous studies. Among Black individuals in the study, the odds of depression increased with stressor exposure and the relationship was not modified by unhealthy behavior engagement. Specifically, for those with 3 or more stressors, the predicted probability of depression was lower for Black individuals than white individuals at every level of unhealthy behavior engagement, demonstrating that the EA Model could not explain the paradox in this large, nationally representative sample (K. M. Keyes et al., 2011). Furthermore, Keyes et al. (2011) discussed how an analytic approach (operationalizing stressor exposure as a mean-centered continuous variable) used by both, Jackson et al. (2010) and Mezuk et al. (2010) was inappropriate due to its violation of linearity assumptions. Importantly, when using the improper operationalization, Keyes et al. (2011) found results consistent with those of the earlier studies, suggesting that the evidence in support of the EA Model may be driven by a mis-specified model. Keyes et al. (2011) raised additional logical concerns regarding the EA Model that have also been discussed elsewhere (Anonymous, 2011). For example, alcohol and cigarette use are not more prevalent among Black individuals (Ellickson et al., 2004; Johnston et al., 2006; K. M. Keyes et al., 2015; Pacek et al., 2012; Anonymous, 2019), and substance use is highly correlated with depression (Hasin et al., 2005; Hasin et al., 2007; Regier et al., 1990), both of which challenge the notion that increased substance use among Black people could be the cause of their decreased depression risk.

Of note, other studies have tested the EA Model; however, their use of depressive symptomology as an outcome precludes them from contributing to the evidence base regarding explanations for the paradox (Boardman & Alexander, 2011; Hoggard et al., 2019; Rodriquez et al., 2017; Rodriquez et al., 2018). Taken together, these results suggest that the promise of the EA Model as an explanation for the depression paradox is likely overstated, and the continued attention it receives may not be warranted given the collective state of the evidence across studies.

Discussion

To date, considerable efforts have been made to explain the presence of the Black-white depression paradox. Proposed explanatory mechanisms range from the artefactual, suggesting observed estimates of racial patterns of depression are invalid, to etiologic mechanisms indicating that the observed pattern is “real”. This review aimed to critically examine the evidence regarding the most prominent proposed explanations in the literature.

None of the 4 artefactual mechanisms reviewed currently have compelling empirical support, though it is worth noting that for 3 of these mechanisms (clinician bias, differential misclassification by diagnostic instrument, selection based on institutionalized or residential status), the available evidence consisted of only one study each. Regarding the proposed etiologic mechanisms, multiple studies tested the EA Model and social support as explanations for the paradox, but neither was compellingly supported by the sum of the evidence. Moderate support was found for racial socialization, but the evidence is derived from a study with considerable limitations; additional evidence is needed.

One issue highlighted by this review is the discordance between the prominence with which some hypothesized mechanisms are discussed in the literature and the strength of the empirical support regarding those mechanisms. For example, the EA Model is one of the more prominent in the literature, between the empirical tests of the model (Anonymous, 2011; Boardman & Alexander, 2011; Hoggard et al., 2019; Jackson et al., 2010; K. M. Keyes et al., 2011; Mezuk et al., 2010; Rodriquez et al., 2017; Rodriquez et al., 2018), and references to it as a possible alternative explanation in articles testing other mechanisms (Barnes & Bates, 2019; Barnes et al., 2013; Mouzon, 2013, 2014, 2017). However, despite the model’s popularity in the literature, the presence of a robust non-confirming empirical test, observations at odds with the articulated theory (e.g., racial patterns in substance use and correlations between substance use and depression), and the identification of analytic flaws that may have driven the supportive results, should dampen enthusiasm for this proposed mechanism, and suggest that attention be directed elsewhere.

Ultimately, none of the proposed mechanisms included in this review are strongly supported by the extant evidence, which translates into a twofold take-home message for the field. Investigators should limit focus on some of the more well-studied proposed mechanisms in order to 1) redirect efforts towards the identification and investigation of new possible mechanisms, and/or 2) direct more attention towards some of the frequently discussed and promising mechanisms that have yet to be sufficiently investigated. As mentioned previously, racial socialization represents one avenue that could benefit from increased

attention as the evidence that it may act as a source of protection against depression for Black people appears promising. However, more studies are needed to determine whether the magnitude of the effect of racial socialization is substantial enough to drive the observed racial patterns in depression and to more concretely establish the relationship between racial socialization and depression in both men and women. A potential challenge of this work is the fact that racial socialization is best represented in white communities by its absence, making it hard to assess directly as a factor to explain between-racial-group differences. Further work in this domain will require exploring ways of investigating the role of racial socialization that can meaningfully inform the study of between-group differences. One potential strategy would be to leverage within-racial group heterogeneity within the U.S. to analyze the potential effects of racial socialization. There is considerable ethnic and cultural heterogeneity within Black people in the U.S. (as with white people); this heterogeneity among Black people would likely be reflected in subgroup differences in the experience of racial socialization that would provide an opportunity to assess its effect on mental health.

Self-esteem and religiosity are two additional mechanisms lacking direct empirical tests that may be worth further exploration. Self-esteem has been discussed broadly as a potential mechanism that could explain the relative lack of mental health burden experienced by Black people despite their on average lower socioeconomic position (Alang, 2014; Barnes & Bates, 2019; Barnes et al., 2013; K. M. Keyes et al., 2011; Kiecolt et al., 2008). Two review articles have documented protective relationships between self-esteem and mental health broadly (though neither looked at DSM depression specifically) (DuBois & Flay, 2004; Sowislo & Orth, 2013). In order for self-esteem to explain the depression paradox, Black people would either need to have higher levels of self-esteem than white people, or the magnitude of the (protective) effect of self-esteem on depression would have to be greater among Black people (Alang, 2014). Evidence regarding racial patterning in self-esteem is inconsistent and varies considerably across studies (Gray-Little & Hafdahl, 2000; Twenge & Crocker, 2002). These inconsistent results may reflect real variation across racial/ethnic groups in how self-esteem is constructed, socially conveyed, and performed, or they may be artefactual, due to variation in the ways in which the construct of self-esteem has been operationalized across studies. Establishing racial patterns in self-esteem across multiple samples using the same measures would be a key first step in investigating a potential role of self-esteem in producing the paradox.

Religiosity has been well-established as an influential component of Black American culture (Frazier & Lincoln, 1974; Lincoln & Mamiya, 1990; Taylor et al., 1987), which is why it has been widely discussed as a potential driver of the depression paradox (Alang, 2014; Barnes & Bates, 2019; Barnes et al., 2013; C. L. Keyes, 2009; K. M. Keyes et al., 2011; Mezuk et al., 2013; Mezuk et al., 2010; Mouzon, 2017; Taylor & Chatters, 2020). If religiosity is protective of mental health and more prominent among Black people, it could result in a lower prevalence of depression among Black people, despite their greater exposure to major life stressors. Black Americans have demonstrated greater levels of religiosity than their white counterparts across a number of study samples and varying dimensions, including frequency of religious service attendance, frequency of engagement with religious texts and broadcasts, likelihood of being a member at a place of worship, rates of daily prayer, likelihood of identifying as “very religious minded”, and endorsing feeling strongly about

their religious beliefs (Alston, 1973; Gallup Jr, 1985; Levin et al., 1994; Taylor et al., 1996; Wuthnow, 1979). Additionally, varying aspects of religiosity have been shown to be protective for a wide array of mental health outcomes, including demoralization, DSM depression, general anxiety disorder, and suicide (Chatters, 2000; Ellison et al., 2001; Ellison & Levin, 1998; Li et al., 2016; Schieman et al., 2013; VanderWeele, 2017). Based on this indirect evidence, religiosity is a promising candidate explanation for the depression paradox; however, direct empirical tests using DSM diagnostic outcomes, are needed. The only study to our knowledge that has to date tested religiosity as an explanation for the paradox (Mouzon, 2017) used depressive symptomology as the outcome, again limiting the applicability of the findings to the depression paradox for reasons discussed previously. Importantly, the existence of racial patterning across a number of different elements of religiosity is suggestive of multiple mechanisms through which religiosity could impact the depression paradox; each one may be worth further investigation.

The results of this review also speak to broader understandings of relationships between social positioning and psychopathology. Social stress theory posits that due to marginalized status, members of stigmatized social groups (such as racial minorities) should evince worse mental health (relative to their dominant group counterparts) because of their increased exposure to stressors and lack of resources for salubrious coping in response to these stressors, due to socioeconomic deprivation and discrimination (Dressler et al., 2005; Horwitz, 2002; Pearlin et al., 1981; Schwartz & Meyer, 2010; Turner, 2010). Said differently, it is because of expectations derived from social stress theory that observed racial patterns in depression are deemed “paradoxical”. For this reason, stressor exposure is an integral component of the depression paradox phenomenon, even when not explicitly mentioned as part of an articulated model, as societally patterned stressor exposure is what drives expectations of a heightened burden of depression for Black people. As a result, the lack of support for artefactual explanations for the paradox represents a challenge to social stress theory, as an etiologic mechanism would imply the existence of a causal element that extends beyond those depicted in the theory. Explicit discussions of social stress theory, and the implications for it of the depression paradox, should it prove valid, have been broadly absent from the etiologic mechanism literature but are worth including moving forward.

Each of the etiologic mechanisms included in this review – social support, racial socialization, and the EA Model - similarly would account for the paradox through a mechanism of racialized stressor coping; each is posited to lessen the impact of stressors experienced by Black people, relative to white people. Importantly, each mechanism should manifest in the same way when evaluating the causal relationships between race, stressor exposure, and depression: the association between stressor exposure and depression should be of a lesser magnitude for Black individuals than for white individuals. This specific causal structure (also inconsistent with social stress theory) is implicitly (if not explicitly) implied by each of the etiologic mechanisms discussed (as well as self-esteem and religiosity), though it too, has not been formally tested empirically, to our knowledge. However, if this causal structure does not accurately represent the relationships between race, stressor exposure, and depression, then it would also suggest the need for broader reconceptualization of the ways in which a lower prevalence of depression among Black individuals could arise.

Finally, the evidence presented in this review and the lack of resolution regarding the meaning of the observed racial patterning in major depression, raise important questions about how we assess and address mental health burden in Black communities. An invalid paradox would suggest a potentially sizeable unrecognized burden of depression experienced by Black individuals. However, even if the paradox is valid (meaning a true lower prevalence of DSM diagnosed depression for Black relative to white individuals), it would raise questions as to whether DSM depression is the appropriate target of mental health focus and resources. Although the pattern of lower or similar prevalence among Black individuals has been documented for a number of psychiatric disorders (excluding lifetime post-traumatic stress disorder) (Erving et al., 2019), the majority of the literature has focused on racial patterns in unipolar depression. However, despite the lower prevalence of depressive disorder, Black individuals routinely report a higher burden of depressive symptoms and a higher prevalence of non-specific psychological distress (Kessler & Neighbors, 1986; Mehta et al., 2015; Vega & Rumbaut, 1991; Weissman et al., 2015; Wellman, 1993). For example, the Black-white prevalence ratio of meeting major depressive disorder symptom criteria in the National Health and Nutrition Examination Survey ranges from 1.35 – 2.35 (Barnes & Bates, 2017). The discordance between racial patterns in depressive symptoms and depressive disorder is a paradox in its own right that has been discussed previously and that deserves further attention (Barnes & Bates, 2017). The higher burden of distress in conjunction with the gravity, magnitude, and pervasiveness of structural racism in American society, and the documented relationship between racism/racial discrimination and mental health broadly (Paradies, 2006; Pascoe & Smart Richman, 2009; R. Williams & Williams-Morris, 2000; Williams & Mohammed, 2008; Williams et al., 2003), highlights the limited utility of focusing on a lower burden of DSM depression if at the expense of other mental health markers. Efforts to address the burden of mental health among Black populations will also require attention to issues of treatment availability and institutionalized racism in the healthcare system that undermine access to care.

The goal of this review was to interrogate the strength of the current evidence surrounding different proposed explanations for the Black-white depression paradox. In doing so, this review highlights the very limited evidence in support of any of the extant putative mechanisms, as well as the extent to which the literature is referencing mechanisms despite the lack of supportive evidence and, in some cases, the presence of contradictory evidence. By synthesizing and evaluating the state of the extant evidence regarding these mechanisms, this review hopes to guide investigators to better focus the field’s resources on mechanisms that have yet to be falsified, as well as highlight the need to identify and test new hypothesized mechanisms. Although this review does not cover a fully exhaustive list of possible mechanisms, additional hypotheses (e.g., “John Henryism”(K. M. Keyes et al., 2011)) have not to our knowledge received sufficient conceptual or empirical attention in the literature to merit inclusion.

In conclusion, this review suggests the need for a renewed investment in efforts to explain the Black-white depression paradox, but one more characterized by rigorous discernment between DSM criteria-based depression and depressive symptomology, testing of promising etiologic mechanisms that have not been sufficiently investigated empirically, and the identification and testing of alternative potential artefactual and etiologic explanations.

Empirical substantiation of an underlying etiologic explanation for the paradox, thereby rendering it a valid phenomenon, would have important implications for social stress theory, the causal model underpinning the paradox. Alternatively, formally testing whether the causal structure implied by social stress theory (and seemingly contradicted by the depression paradox) relating racial group membership, stressor exposure, and depression is accurate, might also shed light on potential new paths toward identifying explanations for the paradox. Finally, efforts to explain lower rates of major depression among Black Americans relative to their white counterparts should not come at the expense of a commitment to better understand the broader mental health consequences and manifestations of structural racism and the potentially significant unmet need for mental health resources in Black communities.

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- Reviewed artefactual explanations were not supported by empirical evidence
- Etiologic explanations were either unsupported by evidence or under tested
- Racial socialization and religiosity merit further empirical examination
- Development of new hypothesized explanatory mechanisms is needed

Table 1.

Overview of operationalizations of social support used in studies testing social support as an explanation for the Black-white Depression Paradox

Study	Data	Construct	Operationalization
Kiecolt et al. 2008	NCS	Social integration	Marital status
			Extended kin in household
			Frequency of contacts with kin outside of household
			Frequency of contacts with friends
			Church attendance
			Endorsing having someone they can open up to about private feelings
		Perceived supportiveness of spouse/partner	Six item factor scale
		Perceived supportiveness of relatives	Six item factor scale
		Perceived Supportiveness of friends	Six item factor scale
		Perceived strain from spouse/partner	Six item factor scale
		Perceived strain from relatives	Six item factor scale
		Perceived strain from friends	Six item factor scale
Shim et al. 2012	NSAL	Support from family	“How close do you feel towards your family members?”
			“How often do your family members help you out?”
		Support from friends	“How close do you feel towards your friends?”
			“How often do your friends help you out?”
Mouzon 2013	NSAL	Family-based social support	Frequency of interactions
			Frequency of instrumental support received
			Frequency of instrumental support given
			Balanced instrumental support
			Subjective family closeness
			Emotional Support
			Emotional Strain
Mouzon 2014	NSAL	Friend-based social support	Frequency of interactions
			Frequency of overall support received
			Frequency of overall support given
			Balanced overall support
			Subjective friend closeness
			Number of fictive kin
			Overall support from fictive kin