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Explaining Racial Disparity in Bipolar Disorder Treatment:
How Do Providers Contribute?

By

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Abstract

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How Do Providers Contribute?

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Bipolar disorder is a serious mental illness that goes untreated more often among blacks than whites in the U.S. In the current study, I identified and tested a proposed mechanism underlying differential treatment in bipolar disorder, namely how treatment providers' racial biases contribute to the inadequate care of black patients with bipolar disorder. Findings from research in general and mental healthcare show that providers' clinical decisions may be related to racial biases based on stereotypes that providers hold. Moreover, general healthcare research has demonstrated the presence of implicit racial biases (defined as unconscious preferences for one race over another) among providers that are linked to the differential treatment of blacks and whites. Unfortunately, much less work has been done to show the presence and effects of implicit racial biases among mental healthcare providers. The hypothesized model in the current study proposes that implicit racial biases held by mental healthcare providers bias diagnostic and prognostic assessments as well as treatment recommendations in bipolar disorder and result in the observed treatment disparities between black and white patients. The Implicit Association Test was used to measure general racial biases in a sample of 82 mental healthcare providers. Clinical decision-making was simulated using clinical vignettes about theoretical black and white patients with psychotic and non-psychotic bipolar disorder. Three-way ANOVAs were used to test for racial disparities in decision-making, and structural equation modeling was used to determine whether providers' implicit racial biases were associated with decision-making outcomes. Results showed that providers held moderate pro-white/anti-black racial biases and neutral explicit attitudes, also known as an "aversive racism" profile. Racial disparity was observed, but confounded by a mismatch in the severity of symptoms of the hypothetical patients. Composite scores for the vignettes were used to overcome this issue in the SEM analyses. These results revealed that there was no link between racial bias and decision-making. Suggestions for future research are given to improve modeling of the relationship between racial bias and patient care. Despite limitations, findings provide some of the first evidence establishing the presence or implicit racial biases in mental healthcare.

Explaining Racial Disparity in Bipolar Disorder Treatment: How Do Providers Contribute?

The objective for the current study was to assess a proposed mechanism underlying the black-white treatment gap in bipolar disorder, namely the contribution of mental healthcare providers' racial biases to racial disparity in diagnostic and prognostic assessment and treatment recommendations. Findings from social psychological research have revealed that racial biases occur implicitly, outside of awareness and despite well-meaning intentions, and that these biases have implications for interracial provider-patient interactions and treatment outcomes (Burgess, Fu, & van Ryn, 2004). In the current study, I measured the relationship of implicit racial bias on the decision-making of mental healthcare professionals who reviewed vignettes of black and white patients with bipolar disorder. Clarifying the presence and strength of racial bias among mental healthcare providers is important, because implicit racial biases could be immediately addressed by implicit bias-reduction interventions that are already available to non-clinical populations.

Bipolar disorder, a serious mental illness characterized by severe mood disturbances, is the ninth leading cause of medical disability worldwide (World Health Organization, 2001). The disorder goes untreated far more often among black Americans than white Americans. Recently published findings showed that no black person with bipolar disorder in a large representative U.S. community sample received minimally adequate treatment (Johnson & Johnson, 2014). Black Americans with serious mental illnesses like bipolar disorder are also overrepresented in samples not surveyed in epidemiological studies, including the incarcerated and homeless populations where the need for treatment is often unmet (Snowden, 2001). This treatment disparity severely disadvantages blacks in this clinical population. If left untreated, the risk rises for relapse, suicide, hospitalizations, physical disorders, and other psychological problems (American Psychiatric Association, 2002). Indeed, blacks with bipolar disorder show a more severe illness course compared to whites, including greater likelihood of hospitalization, psychosis, and suicide attempt (Garver et al., 2006; Gonzalez et al., 2007; Kupfer, Frank, Grochocinski, Houck, & Brown, 2005). Though the need to close race gaps in treatment is often discussed in mental health and racial disparity literature, racial disparity mechanisms and targeted interventions are under-researched areas in psychiatry and clinical psychology. The unfortunate result is stunted progress: the disparities between blacks and whites in mental healthcare utilization have worsened during the last decade, despite increased attention to race gaps (Cook, McGuire, & Miranda, 2007). Until the race gap is closed, black patients with mental illnesses like bipolar disorder disproportionately and unnecessarily suffer. Findings from the current study will help to fill this gap in research by providing some of the first evidence concerning implicit racial biases among providers who are treating black patients with bipolar disorder and testing the effects of such biases on the creation of treatment disparities between black and white patients.

In the following literature review, I present (a) an overview of the black-white racial disparities documented in mental healthcare; (b) extant research on the black-white racial disparity in the treatment of bipolar disorder; (c) an overview of traditionally proposed models in mental healthcare research and bipolar disorder research for the mechanism underlying the generation of black-white disparities in treatment; (d) an overview of an emerging model of provider implicit racial bias from general medicine that has helped to explain black-white treatment disparities in that field and how it may be applied in mental healthcare; (e) a summary of the progress in understanding provider racial bias within bipolar disorder and the benefit of

beginning the investigation of these biases in bipolar disorder. This literature will be the basis and rationale for the current application of a new model of black-white disparities in bipolar disorder treatment.

Racial Disparities in Mental Healthcare

Disparities between whites and racial minorities in mental health treatment are widely documented (Cook, McGuire, & Zuvekas, 2009; Smedley, Stith, & Nelson, 2003; Snowden & Yamada, 2005; Wang, Demler, & Kessler, 2002). The 2001 U.S. Surgeon General's report, *Mental Health: Culture, Race, and Ethnicity—A Supplement to Mental Health: A Report of the Surgeon General*, summarized the main areas where racial disparities in treatment arise: compared to non-Hispanic whites, racial minorities have less access to mental healthcare, are less likely to receive treatment for mental health problems, and are more likely to be given poorer quality treatments when they are treated (U.S. Department of Health and Human Services, 2001). The supplement alerted researchers and federal agencies to the pressing concern of minority mental health and sparked a surge in research on racial disparity in mental healthcare. These included large epidemiological studies of the mental health and service delivery in nationally representative samples of Latino and Asian Americans (Alegria et al., 2004) as well as black Americans (Jackson et al., 2004). Federal agencies, including the National Center for Minority Health and Health Disparities, National Institute of Mental Health, and Agency for Healthcare Research and Quality also increased their financial commitments to monitor and address these concerns (Snowden, 2012). The Supplement also led to Congress requesting that a formal evaluation of the prevalence and sources of racial disparities in mental healthcare be made by the Institute of Medicine (Smedley et al., 2003). Despite the renewed effort that followed the landmark Supplement, racial disparities have worsened over the decade since its publication. For example, disparities between whites and racial minorities have widened on indices of medical expenditures on mental healthcare services and number of services received (Cook et al., 2009, 2007).

Although the widening of racial disparities in U.S. mental healthcare is a pressing concern for all minority populations, racial disparities in U.S. mental healthcare are most pronounced between black and non-Hispanic white Americans (U.S. Department of Health and Human Services, 2000). Findings from the National Comorbidity Survey Replication (NCSR), a study of mental health service use using nationally representative community samples of Americans from all race groups, revealed that blacks are only half as likely as whites to receive any type of treatment for mental disorders (compared to Hispanics who were 0.6 times as likely to receive any treatment compared to whites, a rate that was not statistically significant in this study; Wang et al., 2005). When blacks with mental health disorders do receive treatment, they are less likely than whites to be seen by a mental health specialist (7.2% of blacks compared to 11.8% of whites; Alegría et al., 2002). The black-white disparity persisted after adjusting for demographic characteristics, insurance coverage, and illness severity. There are also important differences in the types of treatment received by black patients. Mental healthcare for blacks occurs disproportionately in emergency room and inpatient settings (Snowden, Catalano, & Shumway, 2009; Snowden, Hastings, & Alvidrez, 2009). One study pooled two large, nationally representative U.S. samples, the National Survey of American Life (NSAL) and the NCSR, and found that rates of hospitalization for African Americans was 8.5% and 8.9% for Caribbean blacks, compared to 3.4% of whites (Snowden, Hastings, et al., 2009). In addition, hospitalizations for blacks are more often coercive, including involuntary patient stays

(Rosenfield, 1984; Segal, Bola, & Watson, 1996) and, in one study, more frequent use of seclusion and restraint than whites during inpatient stays (Flaherty & Meagher, 1980).

Black Americans have also been found to experience some mental health disorders with greater chronicity and severity than white Americans. For example, non-Hispanic blacks in the first iteration of the National Comorbidity Survey were more than 1.5 times more likely than whites to have persistent psychiatric illness, particularly mood and anxiety disorders, after controlling for demographics and educational attainment, an indicator of socioeconomic status (Breslau, Kendler, Su, Gaxiola-Aguilar, & Kessler, 2005). Among Americans surveyed in the National Survey of American Life, more than 50% of African Americans and Caribbean Americans compared to 39% of white Americans had chronic major depressive disorder (Williams et al., 2007). Blacks in this sample also reported greater severity and dysfunction associated with their depression than their white counterparts. Given the robust evidence for the persistent gap between black and white Americans, the focus of the current study was on understanding and eliminating mental healthcare disparities between these two groups.

Racial Disparities in Bipolar Disorder Treatment

Bipolar disorder is a serious mental illness marked by distinct periods of abnormally and persistently elevated, expansive, or irritable mood called manias (American Psychiatric Association, 2013). Manic episodes are defined by symptoms including inflated self-esteem or grandiosity, decreased need for sleep, being more talkative than usual or pressured to keep talking, flight of ideas or subjective experience that thoughts are racing, distractibility, increase in goal-directed activity or psychomotor agitation, and excessive involvement in pleasurable activities that have a high potential for painful consequences. Global estimates from epidemiological surveys reveal rates as high as 4-6% in adults when broad diagnostic criteria for bipolar spectrum disorders are applied (Angst, Gamma, & Lewinsohn, 2002; Judd & Akiskal, 2003; Merikangas et al., 2007; Zimmermann et al., 2009). The National Comorbidity Survey Replication (NCSR), an epidemiological study of mental disorders in the United States, shows that the U.S. has some of the highest rates for all forms of the disorder, reporting around 2 percent of people with diagnosable bipolar I and II disorder and more than 2 percent with persistent subsyndromal symptoms (Kessler et al., 2005; Merikangas et al., 2007).

The disorder tends to have a highly recurrent course. In one study, just 28 percent of individuals with bipolar disorder remained in remission after four years (Tohen, Waternaux, & Tsuang, 1990). Rates of relapse and recurrence vary widely by study, but the highest reports from longitudinal studies suggest that as many as 69 percent of individuals with bipolar disorder experience seven or more episodes (Goodwin & Jamison, 2007).

Bipolar disorder is also characterized by high rates of physical and psychiatric comorbidity and premature mortality. The Stanley Foundation Bipolar Network estimates 65 percent of patients with the disorder have a comorbid condition (Suppes et al., 2001). The most frequently occurring conditions were anxiety disorders, substance abuse disorders, and alcohol abuse. Several studies even show that the link between alcohol abuse is more strongly associated with bipolar disorder than unipolar depression (Helzer & Pryzbeck, 1988; Regier et al., 1990). Many individuals with bipolar disorder are also managing other medical conditions such as cardiovascular disease, diabetes, thyroid disease, and obesity (Krishnan, 2005; Kupfer, 2005), and these in turn are significant sources of premature mortality in this population. Indeed, there is evidence that individuals with bipolar disorder are three times more likely than the general population to die from cardiovascular disease (Sharma & Markar, 1994). Suicide, however, has

historically been the leading cause of mortality in bipolar disorder. Nearly one third of patients admit to at least one suicide attempt, and about 10-20% of individuals complete suicide (Müller-Oerlinghausen, Berghöfer, & Bauer, 2002).

Bipolar disorder is also a leading cause of disability worldwide, and is related to high rates of social dysfunction. These functional impairments have been show to be related to chronic subsyndromal symptoms, present even during remitted periods, and neurocognitive impairments associated with the disorder (Martinez-Aran et al., 2007; Sanchez-Moreno et al., 2009). This population is especially impacted in work, family, and social domains. More than half of individuals with bipolar disorder are divorced, separating or single compared with only 38% of the general U.S. population (Nolen et al., 2004). The occurrence of mania is also high in the mentally ill homeless population. In a Los Angeles study of the mentally ill homeless population, an individual was more than 17 times more likely to have a manic episode than a person in the general population (Koegel, Burnam, & Farr, 1988), rates that were dramatically elevated compared to the 2.9 fold increase in rates of major depressive episodes among the homeless as compared to the general population. These findings indicate the significant costs in productive and quality life years to the individual who lives with bipolar disorder.

When independent raters, rather than clinicians, conduct structured diagnostic assessments, there are no differences in the prevalence of bipolar disorder among blacks and whites (Merikangas et al., 2007; Simon, Fleiss, Gurland, Stiller, & Sharpe, 1973; Strakowski, McElroy, Keck, & West, 1996). For example, national epidemiological studies have shown that bipolar disorder is equally prevalent for blacks and whites in the U.S. when careful assessment tools are implemented (Breslau et al., 2006; Kessler et al., 1994). There is also no evidence for racial differences in genetic vulnerability for bipolar disorder or schizophrenia (Calkins et al., 2010; John E. Helzer, 1975; Wiener et al., 2009), nor for racial differences in the efficacy of mood-stabilizing medications (Gonzalez, Bowden, Berman, Frank, Bauer, Kogan, Alegria, et al., 2010). Despite evidence that bipolar disorder is equally prevalent and treatment-responsive for blacks and whites, findings suggest that blacks with bipolar disorder do not fare as well as whites with bipolar disorder. Bipolar disorder is more persistent among blacks than whites. One study reported that 26 percent of blacks versus 20 percent of whites with a diagnosis had experienced symptoms of bipolar disorder in the prior year (Breslau et al., 2005). Blacks with bipolar disorder, then, seem to experience disproportionate impairments associated with bipolar disorder compared to whites.

A large body of work on the black-white disparity in bipolar disorder focuses on misdiagnosis. As background, it is important to note that misdiagnosis is common in bipolar disorder generally (Dunayevich & Keck, 2000; Ghaemi, Ko, & Goodwin, 2002; Goodwin & Jamison, 2007; Meyer & Meyer, 2009). Unfortunately, on average an individual with bipolar disorder consults with up to four different physicians and takes up to ten years after first seeking care before receiving an accurate diagnosis (Hirschfeld, Lewis, & Vornik, 2003). Accurate and timely diagnosis is crucial to the health and quality of life of a person with bipolar disorder, as appropriate medications diminish rates of hospitalization, suicide, and other poor outcomes (Baldessarini et al., 2006; Scott & Pope, 2002). Although misdiagnosis is far too common, blacks with bipolar disorder in the U.S. have historically been more likely than whites to be misdiagnosed with schizophrenia. Data from national and local psychiatric hospital admissions show that blacks are 1.5 to 1.8 times as likely as whites to be diagnosed with schizophrenia, but only .5 to .75 as likely to be diagnosed with a mood disorder, including bipolar disorder and major depressive disorder (Cheung & Snowden, 1990; Lawson, Hepler, Holladay, & Cuffel,

1994; Snowden, 2001). Retrospective chart reviews and case study analysis are helpful in demonstrating the consistency with which this diagnostic error is made among blacks who do eventually receive a correct bipolar disorder diagnosis (Bell & Mehta, 1980; Mukherjee, Shukla, Woodle, Rosen, & Olarte, 1983). Unfortunately, there are no studies examining more recent clinical samples to show that diagnostic practices have improved among clinicians.

It has also been suggested that providers treating black patients with bipolar disorder may fail to appropriately weight affective symptoms into their diagnostic assessments, particularly when patients experience a mixture of affective and psychotic symptoms (McKenzie, 1999; Neighbors, Trierweiler, Ford, & Muroff, 2003). Although bipolar disorder remains the correct diagnosis when psychotic symptoms are present during a mood episode, an early epidemiological study noted that American psychiatrists seemed to focus on psychotic symptoms rather than appropriately using mood symptoms in diagnostic assessments (Kendell et al., 1971). One goal of the current study, then, was to examine the influence of psychotic symptoms on racial disparities in diagnosis.

Although the studies above document a clear problem with misdiagnosis, particularly when psychotic symptoms are present, other influential work highlights how harmful this misdiagnosis can be (Bell & Mehta, 1981). Bell & Mehta describe three adult black inpatients with bipolar disorder who were incorrectly medicated with medications for schizophrenia (in one case for up to a decade), and spent several years severely disabled despite receiving frequent professional treatment. They were often financially dependent on others and experienced unstable interpersonal relationships. They were repeatedly hospitalized (in one case up to four times in a year). Even during outpatient periods, the tranquilizing effects of anti-psychotic medications led to a constant "drugged" feeling that interfered with daily life activities. Alcoholism and oppositional behaviors such as shoplifting and combativeness worsened with the poor care, leading to arrest. Only upon receiving the correct bipolar disorder diagnosis, and thus the correct medication, did these patients achieve a sustained remission of bipolar disorder symptoms and relief from related problems. Given the frequent failure to detect bipolar disorder among blacks, diagnostic errors likely prolong impairment and suffering for black individuals at an even greater rate than the reported average.

Although the focus of black-white disparity in bipolar disorder has been on misdiagnosis and its harmful effects, there are notable racial disparities in prescription practices in bipolar disorder as well. For example, two studies of insurance claims cohorts showed that, compared to whites, blacks were less likely to receive prescriptions for mood stabilizers (49% versus 63% of whites), considered standard treatment for manic episodes of bipolar disorder (Busch et al., 2009), and more likely to be prescribed multiple medications (41% versus 34% of whites), a practice that was related to more frequent hospitalizations and emergency room visits at follow-up (Garver et al., 2006). Analyses of data from the University of Cincinnati First-Episode Mania Study showed that in a sample of demographically and clinically similar blacks and whites, blacks were more likely to be prescribed older antipsychotic medications with side effects known to be more severe than newer antipsychotic treatments even in the absence of psychotic symptoms (Fleck, Hendricks, DelBello, & Strakowski, 2002). An analysis of NCSR data showed that proper mood-stabilizing medication was non-existent among blacks with bipolar disorder (Johnson & Johnson, 2014). These prescription disparities are documented consistently despite evidence showing that there are no racial or ethnic differences in the responsiveness to psychiatric medications (Gonzalez et al., 2010). Even when adequate care is received, other types of treatment disparities persist. In a National Veterans Affair hospital sample, where

healthcare is generally more equitable and innovative than in the general population, blacks with bipolar disorder were less likely to have a follow-up outpatient visit after hospitalization or to receive a call from their doctor within 30 to 90 days of discharge, considered a standard for follow-up practice (Kilbourne et al., 2005). Altogether findings illustrate that blacks with bipolar disorder are significantly more likely to be inadequately treated than their white counterparts.

There are several negative consequences of inadequate treatment among blacks with bipolar disorder. Long-term antipsychotic use, like that described previously for blacks with bipolar disorder, is related to neurological side effects, most notably of which is tardive dyskinesia, or involuntary repetitive movements of the body that are often incurable and disruptive to quality of life (Caroff, Mann, Campbell, & Sullivan, 2002; Glazer, Morgenstern, & Doucette, 1994). Blacks with bipolar disorder in a Pennsylvania patient registry of over 2,000 patients were more likely to be improperly treated and to attempt suicide than whites with bipolar disorder (64% versus 49% attempted suicide; Kupfer et al., 2005). Blacks with inadequately treated bipolar disorder may also be at particularly high risk for incarceration, given that both black people and bipolar disorder are overrepresented in prison populations and that a black person's mental illness is often not recognized by authorities upon arresting the individual with the illness (Foulks, 2004; Hicks, 2004; Lawson & Lawson, 2013). Given the black-white gap in diagnosis and treatment of bipolar disorder, and the evidence that this poor care leads to high rates of incurable neurological side effects, suicide, hospitalization and possibly incarceration, bipolar disorder is an ideal target for research on racial disparities. Elucidating the mechanisms driving the black-white disparities in this population is an important step in improving function and quality of life for black patients with bipolar disorder.

Traditionally Proposed Mechanisms for Black-White Treatment Disparities

The Surgeon General's report summarized various explanations of black-white disparities in mental healthcare (U.S. Department of Health and Human Services, 2001). Among these, the socioeconomic hypothesis focuses on racial differences in insurance coverage and ability to pay, which affect access to care and quality of care (Williams & Collins, 1995). As mentioned previously, however, many studies show that treatment disparities persist even after controlling for socioeconomic factors.

It is commonly proposed that stigmatization of mental health and attitudes about mental health treatment lead to low treatment-seeking among blacks. Studies have documented that blacks report high levels of concern about being perceived negatively by others if they make their symptoms known (Conner, Koeske, & Brown, 2009; Roeloffs et al., 2003). Blacks also explicitly report stigma as a key barrier to treatment-seeking in the their community (Alvidrez, Snowden, & Kaiser, 2008; Cruz, Pincus, Harman, Reynolds, & Post, 2008). In contrast, in a probability sample of 3,004 St. Louis residents, Sussman and colleagues found that low treatment-seeking among blacks with depression was related to greater distrust of treatment and fears of hospitalization more than fears of stigmatization (Sussman, Robins, & Earls, 1987). Similarly, in a focus group of 42 black participants, fear of encountering racism in their healthcare provider and being stereotyped as dangerous were main barriers to seeking professional help (Mishra, Lucksted, Gioia, Barnet, & Baquet, 2009).

Despite the presence of negative and avoidant attitudes about mental healthcare among black people, evidence for actual differences in treatment-seeking is inconsistent. Blacks have been found to be less likely than whites to seek help from a primary care physician for mental health problems in one study (Lasser, Himmelstein, Woolhandler, McCormick, & Bor, 2002),

but more likely to do so in another nationally representative study (Snowden & Pingitore, 2002). A recent analysis of the NCSR data showed that blacks in the U.S. were more willing to seek mental health treatment and have more positive attitudes about treatment than whites (Shim, Compton, Rust, Druss, & Kaslow, 2009). While negative attitudes about help-seeking among blacks are likely to be barriers to treatment, the evidence from representative samples seems to suggest that this is not the main mechanism underlying the occurrence of racial disparity in mental healthcare nor would stigmatization explain the persistent disparity among patients who have overcome this barrier and are seeking treatment.

There is evidence that these traditionally proposed mechanisms do not fully explain black-white treatment disparities in bipolar disorder in particular. One study to date conjointly investigated several traditionally proposed mechanisms as potential explanations for the black-white disparity in bipolar disorder. Using the nationally representative NCSR sample of blacks and whites with bipolar disorder, Johnson and Johnson (2014) found that both groups had comparable insurance coverage, socioeconomic status, and symptom profiles. Both groups also sought help equally often and from the same types of treatment providers. Despite these similarities in patient characteristics, no black person received minimally adequate treatment for bipolar disorder. It is likely that with more serious mental illnesses like bipolar disorder, for which professional help is often required to facilitate basic functioning for the individual, differences in patient attitudes about seeking professional help play a smaller role in the generation of racial disparities.

Another commonly proposed explanation is that black-white disparities in mental healthcare occur because symptom descriptions vary by race, which would cause racial differences in treatment. For example, blacks are thought to express psychological distress as somatic complaints more than whites, and thus they are more likely to seek help from a general healthcare provider rather than a mental healthcare specialist (Alegría & McGuire, 2003; Snowden & Yamada, 2005). In analyses of the NCS data, blacks were significantly less likely to report cardinal symptoms of depression and anxiety, and thus were less likely to be diagnosed with depression, generalized anxiety disorder, or alcohol abuse (Alegría & McGuire, 2003). Other findings, however, suggest that blacks and whites do not differ in symptom reports. For example, in one study using the Behavioral and Symptom Identification Scale (BASIS-32), a widely used clinically administered measure for symptom assessment, there were no racial differences in symptom expression between whites, blacks, and other racial minorities (Chow, Snowden, & McConnell, 2001). Other studies have identified no racial differences in affective symptomatology (Strakowski et al., 1996), including one analysis of those with bipolar disorder in the National Epidemiological Survey on Alcohol and Related Conditions (Perron, Fries, Kilbourne, Vaughn, & Bauer, 2010). Hence evidence does not provide consistent support for the idea that affective symptoms are reported differently by blacks and whites.

Above, however, I noted that misdiagnosis of bipolar disorder as schizophrenia is a particular concern among blacks. Some research is available about racial disparities in descriptions of psychosis. In the NCSR sample analyzed by Johnson and Johnson (2014), a predominately untreated sample, very few people with bipolar disorder, regardless of race, endorsed psychotic symptoms or had been misdiagnosed with schizophrenia, and so the influence of psychotic symptom presentation could not be analyzed in this study. Rates of psychosis may be higher in treated samples; several studies have shown that blacks in treated samples are more likely to experience psychosis in bipolar disorder (Patel, Delbello, & Strakowski, 2006; Strakowski et al., 2003, 1996). In an analysis of psychiatric hospital

admissions data, however, the presence of psychotic symptoms accounted for just 6.5% of variance in misdiagnosis, and race continued to predict schizophrenia misdiagnosis after controlling for the presence of psychotic symptoms (Mukherjee et al., 1983). Thus, even though blacks tend to exhibit psychosis more in bipolar disorder, this difference does not sufficiently explain the black-white disparities in diagnosis and prescription practices.

In sum, researchers have traditionally focused on patient characteristics and behaviors to explain the black-white disparity, but these do not tell the whole story of black-white treatment disparity in mental healthcare or bipolar disorder treatment. Notably, none of the traditional hypotheses account for half of the patient-provider clinical interaction – that is they do not consider the role of providers in contributing to black-white disparities. Rather, they place much of the responsibility for racial disparities on black patients and their treatment-seeking behaviors, styles of communicating symptoms, and beliefs about mental health problems and treatment. It is possible that providers also contribute to black-white disparities. A key conclusion from the Institute of Medicine report produced for Congress was that the health care system was also responsible for the generation and maintenance of racial disparities (Smedley et al., 2003). The report made a specific recommendation for research to clarify the contribution of healthcare providers to disparities, particularly by examining the role of provider biases, stereotyping, prejudices, and clinical uncertainty. The literature just reviewed suggests that this direction would be fruitful to explore in bipolar disorder given the insufficiency of traditionally proposed mechanism in explaining black-white disparities in bipolar disorder.

An Emerging Model of Provider Implicit Racial Bias

Racial biases are especially relevant to the investigation of black-white treatment disparities given the unique and long history in the U.S. of slavery, racism, and discrimination. Although overt racism is becoming less normative in contemporary U.S. society, a large body of work from social psychology has documented the presence of more subtle forms of racism, including unconscious or implicit racial bias. Implicit racial biases are the prejudices and stereotypes about a particular race group held outside of conscious awareness or control (Greenwald & Banaji, 1995). They are defined as being caused by automatically activated evaluations of the race group that are unfavorable or favorable and are related to a basic information processing strategy called automatic stereotyping (Banaji & Hardin, 1996). Stereotyping is a type of cognitive heuristic strategy that allows an individual to categorize others quickly and reduce massive amounts of information from the external world into manageable sizes for cognitive processing (Macrae & Bodenhausen, 2001). A stimulus in the environment (e.g., a black face) will trigger all learned associations related to that stimulus and initiate the process of categorization (i.e., stereotyping). On the one hand, this strategy increases cognitive efficiency and frees up cognitive resources to be used to meet other demands on the individual. The activated set of associations for black persons, however, are often negative, and appear more likely to trigger emotions like fear and disgust and expectations for hostility and aggression (Devine, 1989; Fazio, Jackson, Dunton, & Williams, 1995; Kawakami, Dion, & Dovidio, 1998). It is thought that humans universally apply stereotypes in interactions with others, especially those that are dissimilar to the individual (Brewer & Brown, 1998; Hamilton, 1981; Stangor, 2001). An extensive body of research on the IAT shows that implicit racial biases are found in the majority of samples (Nosek, Banaji, & Greenwald, 2002; Nosek et al., 2007), seemingly verifying the universality of implicit racial bias. Given the universality of implicit racial biases, healthcare providers would not be expected to be immune to holding them.

Implicit racial biases are commonly measured using Greenwald's Implicit Association Task (IAT; Greenwald, Mcghee, & Schwartz, 1998), which uses response latencies to measure the automaticity of associations between concepts. When measuring implicit racial biases in particular, test-takers are asked to quickly pair the concept "black" or "African American" (either by using the words, black names, or images of black faces) and "white" or "Caucasian American" with words that are positively and negatively valenced (e.g., "good" and "flower" versus "bad" and "insect"). The IAT has demonstrated sound reliability and validity (described later in the methodology section for the current study), and scores on the IAT are consistently better predictors of discriminatory behavior during interracial interactions than self-report measures (McConnell & Leibold, 2001; Nosek, Greenwald, & Banaji, 2005; Sabin, Nosek, Greenwald, & Rivara, 2009).

Researchers in general medicine have already used the IAT paradigm to measure the presence of racial bias (e.g., black is bad, white is good) in healthcare providers. Anti-black implicit racial bias has been documented in samples of medical students, physicians (Green et al., 2007; Sabin et al., 2009; Sabin, Rivara, & Greenwald, 2008), nurses, pharmacists, and emergency medical care providers (White-Means, Zhiyong Dong, Hufstader, & Brown, 2009). The magnitude of general racial bias effects when providers take the IAT is typically moderate to large, Cohen's d of .4 to .9 (Green et al., 2007; Sabin et al., 2008), and largest among white samples of clinicians (Sabin et al., 2009), suggesting that implicit racial levels are substantial.

Implicit racial bias theory helps to explain the paradox that healthcare providers can consider themselves to be egalitarian and unprejudiced, yet behave in ways that contribute to racial disparities in treatment. In the domain of racial bias, IAT scores and explicit measures are weakly correlated (Fazio & Olson, 2003; Gaertner & Dovidio, 1986). Several studies show that consciously egalitarian people often hold unconscious negative beliefs and attitudes (Dovidio, Kawakami, & Gaertner, 2002; Wilson, Lindsey, & Schooler, 2000). Whereas providers are expected by the public and themselves to be culturally sensitive to patients and to conduct clinical assessments and consultation in a way that is unaffected by the patient's race, they may still hold negative and stereotypical beliefs about blacks unconsciously. Indeed, in a study that administered the IAT and explicit attitude measures to 2,535 medical doctors, effect sizes for explicit measures were half as large as effect sizes for the IAT (for white MDs, Cohen's d were 1.05 and .69 for implicit and explicit effects, respectively; Sabin et al., 2009).

Not only do implicit racial biases appear to exist for medical providers, there is evidence that these biases negatively influence clinical decision-making and treatment quality. Two studies from general healthcare fields illustrate this. First, a study by Green and colleagues (2007) examined whether the magnitude of physician implicit racial bias was related to recommendations for thrombolysis treatment for theoretical black and white patients with acute coronary syndromes. Clinical vignettes about the patients were identical and varied only in racial identification. They found that physicians held strong implicit biases against black patients (Cohen's d between .5 and .9), including an association between black patients and target concepts "less cooperative generally" and "less cooperative with medical procedures." Stronger pro-white implicit bias was related to a greater likelihood of treating white patients (standardized beta = .17) and not black patients (standardized beta = -.19) with thrombolysis (betas from regression analyses were only significant for the general race IAT and not for the specific stereotype IATs). A second study by Cooper and colleagues (2012) added an assessment of quality of care based on objective ratings of real provider-patient interactions and patient post-visit satisfaction ratings to their analysis of implicit racial bias in a sample of providers treating

hypertension. Scores on the IAT suggested a moderate pro-white implicit bias (Cohen's $d = .54$). Cooper et al. administered the IAT and used generalized estimating equations and logistic regression analyses to predict the probability that a black patient would be treated differently than a white patient as the presence of pro-white bias held by the provider increased. They found that moderate pro-white bias scores on the IAT were related to a 9% greater chance that a provider would be more verbally dominant with a black patient compared than if the provider had a neutral, unbiased attitude. Moderate pro-white bias on the IAT was also related to a significant decrease in positive affect among black patients and lower probabilities among black patients of feeling respected by the clinician (15% lower than whites), liking the clinician (14% lower than whites), having confidence in the clinician (9% lower than whites), and recommending the clinician to others (13% lower than whites). These examples suggest that the IAT is a useful tool in clarifying the role of provider bias in generating black-white disparities in treatment and quality of care.

Implicit Racial Biases in Mental Healthcare

Notably, mental healthcare provider samples are largely omitted in the research examining implicit racial bias. No studies to date have used the IAT to assess the presence and effects of implicit racial bias in a mental healthcare provider sample. It could be that researchers have felt that mental healthcare providers would be relatively less likely to hold racial stereotypes given their familiarity with psychological processes and potentially their knowledge of implicit biases. Sadly, however, considerable theory and research suggests that racism may be operating even among mental healthcare providers.

Many researchers have theorized that racial bias may drive the overdiagnosis of schizophrenia and underdiagnosis of affective disorder among blacks (Adebimpe, 1981; Bhugra & Bhui, 1999; DeCoux Hampton, 2007; Jones & Gray, 1986; Lawson et al., 1994; Lewis, Croft-Jeffreys, & David, 1990; Littlewood, 1992; López, 1989). In one paper on this issue, Bell and Mehta argue that "a pervasive, covert form of racism … has been institutionalized in psychiatry to the point that low prevalence and incidence of [bipolar disorder] in blacks is given" (1980). Evidence in support of racism in the mental health field is apparent in psychiatric publications from the Jim Crowe era in the U.S., in which the (inaccurate) low prevalence of mood disorder among black people was attributed to their "primitive neurology" and lack of self-esteem and material possessions (Green, 1914; Prange & Vitols, 1962). They suggested that with little to lose, blacks could not experience a loss that would trigger an episode of depression. It is quite possible that racism such as was published in the aforementioned references has permeated psychiatric training and care and influences treatment today. Although overt racial bias has become less pronounced, it is intriguing that the profile of underdiagnosing affective disorder and overdiagnosing schizophrenia in blacks has persisted into the 20th century.

Beyond theory that racial biases continue to guide misdiagnosis in blacks with bipolar disorder, evidence from experimental vignette studies consistently indicates that patient race unduly influences clinical judgments of mental health providers. In the most frequently cited study of this kind by Loring and Powell (1988), 290 psychiatrists read two patient vignettes based on actual case studies of persons who had been originally diagnosed by their psychiatrists with DSM-III "undifferentiated schizophrenic disorder," a diagnosis perceived as less serious and dangerous than paranoid schizophrenia. All psychiatrists read the same two vignettes, though the race of each was either described as black, white, or no race was identified. When no race

information was provided about the patient, 56% of the psychiatrists diagnosed the patient with undifferentiated schizophrenic disorder. When race information was presented, white psychiatrists tended to assign the less severe disorders to white patients, and more severe disorders to black patients. Black male patients were most likely to receive the diagnosis of paranoid schizophrenia and were rarely assigned a diagnosis of depressive disorder. Several other vignette studies have replicated these effects (Lewis et al., 1990; Littlewood, 1992). Taken together, these experimental studies demonstrate that providers make more severe diagnostic evaluations of black patients. This suggests that providers hold racial biases that could potentially explain the link between patient race and clinical decision-making. Unfortunately, these studies do not inform us of the mechanism driving the effect of patient race on decision-making. Of most import, these studies have not measured racial biases.

Only one study has demonstrated the presence of unconscious racial bias in a mental health professional sample using a priming paradigm. Abreu (1999) demonstrated that therapists shown briefly flashed words related to African American stereotypes (e.g., Blacks, lazy, basketball, unemployed) rated a hypothetical patient as more hostile than therapists primed with neutral words. This suggests that racial stereotypes may affect how mental healthcare providers process information about their patients. Findings of this study were consistent with the idea that implicit and explicit measures of bias may yield different results; when therapists were told that the race of the hypothetical patient they rated was black, most changed their ratings to indicate less hostility and pathology. Thus, providers' conscious intentions to deliver equitable treatment may not match their implicit anti-black biases. Somewhat surprisingly, Abreu (1999) did not find that diagnostic decisions were affected by the presentation of African American stereotype words. That is, the therapists who saw African American stereotype words (and thought the patient was black) did not differ from the therapists who saw neutral words on the likelihood of assigning a particular diagnosis. This study was limited, though, by the absence of a measure to capture individual differences in implicit racial bias, the small number of therapists, and the narrow range of clinical decisions assessed. No studies to date have directly assessed the magnitude of implicit racial biases within mental healthcare providers, nor have they examined how individual differences in these biases relate to clinical judgments in mental healthcare or mental healthcare delivery.

The Present Study

Although there is a paucity of research on implicit racial bias in mental health, there are several reasons to suspect that mental healthcare providers will be influenced by implicit racial biases. These include the moderate to large effect sizes of implicit racial bias among general healthcare providers, the universality of implicit racial bias among humans, even those expressing overtly egalitarian attitudes, and theory and evidence for racially guided decision-making among mental healthcare providers. Furthermore, the research on provider racial bias in bipolar disorder is relatively stronger than that of other mental illnesses, in that several studies with experimental designs have shown that treatment providers do make differential evaluations of black and white patients on the basis of patient race in the treatment of bipolar disorder. These findings provide an ideal foundation for studies of implicit racial biases in bipolar disorder.

In the current study, I make the first test of the presence and strength of implicit racial biases in a mental healthcare provider sample. I also conduct the first test of the link between implicit racial biases and clinical decision-making in bipolar disorder treatment. This study improved upon previous research with the use of a validated measure of implicit racial bias. It

also improved upon previous disparity research, which has focused primarily on misdiagnosis and hospitalization, with the addition of an assessment of unexamined decision-making domains, namely overpathologizing behavior and prognostic predictions. Previous research has indicated that overpathologizing, or perceiving that more disturbed or a greater number of psychiatric disorders are present, may be more likely among minority patients than white patients, which could lead to inaccurate assessment (López, 1989). Previous findings also show physicians' and mental healthcare professionals' prognoses can be upwardly or downwardly biased by previous positive or negative experiences and expectations of their patients (Alexander & Christakis, 2008; Peris, Teachman, & Nosek, 2008). Given this, overpathologizing and prognostic assessments may be highly influenced by racial biases and are relevant in the current examination.

My objective was to show the degree of implicit racial bias present in a mental healthcare provider sample and to characterize the role of bias in creating racial disparity in clinical decision-making between black and white patients who have bipolar disorder. As such, I proposed the following three specific aims.

Aim 1: Demonstrating racially disparate decision-making. The objective of Aim 1 was to examine the relationship between patient race and provider decision-making in the treatment of bipolar disorder. As described earlier, several studies have documented that black race is consistently related to less favorable clinical decisions in bipolar disorder (e.g., misdiagnosis, inappropriate medication prescription). The goal for this step was to demonstrate the presence of black-white disparities in clinical decisions that would be potentially explained by implicit racial bias in my later aim. Patient race was randomly assigned to clinical vignettes. Decision-making was assessed in the following domains: (1) diagnostic practices, 2) prognostic predictions, and (3) treatment recommendations. Within diagnostic practices, I examined two behaviors: misdiagnoses (failure to assign a bipolar I disorder diagnosis) and overdiagnoses (number of diagnoses assigned to the patient). For prognostic predictions I examined the relative positive or negative prognosis assigned to patients. For treatment recommendations, I examined four types of decisions: referral to a psychiatrist, medication prescription, psychotherapy recommendation, and hospitalization. I hypothesized that providers would display the several patterns of racial bias corresponding with evidence from the disparity research reviewed above across the decision-making behaviors I examined. That is, black patients as compared to white patents would be (1) more likely to be misdiagnosed, (2) more likely to be overdiagnosed with multiple forms of psychopathology, (3) assigned more negative prognoses, (4) less likely to be referred to a psychiatrist, (5) less likely to be prescribed medication, (6) less likely to receive therapy, and (7) more likely to be hospitalized.

In light of findings that suggest that black-white disparities in bipolar disorder tend to widen when psychosis is present, I also examined the effect of psychotic symptoms in interaction with race. Clinical vignettes differed in whether psychotic symptoms were present or absent. This allowed me to determine whether providers were more likely to make particularly unfavorable decisions about black patients as compared to white patients when the bipolar disorder was accompanied by psychosis versus mood symptoms alone.

Aim 2: Measuring implicit racial biases. The objective of Aim 2 was to measure the presence of implicit racial bias in mental healthcare providers. This was the first study to measure implicit racial bias using a well-validated measure, the Implicit Association Test (IAT), in a mental healthcare provider sample. The goal for this step was to establish my proposed mechanism variable to test in the final aim. I hypothesized that mental healthcare providers

would show moderate to strong pro-white (anti-black) bias on the IAT given findings from the general medical healthcare research (reviewed above) showing this level of implicit bias among physicians. Moreover, I hypothesized that implicit biases would be uncorrelated with explicit beliefs about blacks and whites, which would be egalitarian and absent of racial prejudice, as has been demonstrated in previous studies of bias (Peris et al., 2008; Sabin et al., 2009). I also conducted analyses of demographic subgroups to examine differences in the magnitude and direction of bias in particular groups (i.e., white participants, males). I hypothesized that participants who were older, male, Non-Latino, white, less educated, and exposed less to minorities and serious mental illnesses in their practices would show stronger pro-white biases. Hypotheses were based on previous findings from similar demographic subgroup analyses in previous physician implicit bias studies (Green et al., 2007, Sabin et al., 2009).

Aim 3: Testing implicit racial bias as a mechanism for racial disparity. The objective of Aim 3 was to demonstrate a link between implicit racial bias and racially disparate clinical decisions about black and white patients. The goal for this step was to test empirically my theory that implicit racial bias underlies racial disparities in mental healthcare. I hypothesized that implicit racial bias as measured on the IAT would be associated with racial disparities in clinical decision-making. I constructed a structural equation model to test the effects of provider implicit racial bias (IAT scores) on provider decision-making for black patients while statistically controlling for decision making with white patients, explicit beliefs and potential confounding factors (e.g., provider characteristics).

Methods

Study measures and procedures were approved by an Institutional Review Board at the University of California, Berkeley.

Participants

Participants were 83 mental healthcare providers recruited through email listservs and treatment sites in California and particularly the San Francisco Bay Area. Participants were excluded if they were not licensed or practicing at the time of enrollment. Participants were paid with a $100 gift card for the online Amazon Marketplace. Descriptive characteristics of the sample demographics are summarized in Table 1. One provider who completed the survey did not reconsent after being debriefed about study procedures and their data was excluded from the current study. Thus, the total sample size for analyses was 82 providers.

Materials and Measures

Participants anonymously completed questionnaires about demographic information, information about the providers' clinical experiences, and assessments of racial bias and clinical decision-making. Measures are discussed in the order of their administration.

Demographic questionnaire. A demographic questionnaire assessed participant age, sex, Hispanic ethnicity, race, and education. Clinical experience was also assessed and included questions regarding licensure, current practice, field and setting of primary practice, prescription privilege, and psychotherapy practice. Additional questions were asked at the end of the study to avoid revealing the true study intent, including types of serious mental illnesses treated in practice, years of experience treating serious mental illness, percentage of patients with serious mental illness, and percentage of racial/ethnic minority patients.

Clinical vignettes. Five vignettes were created about hypothetical male patients (text of vignettes is presented in Appendix A): two with non-psychotic mania symptoms (i.e., mood symptoms only; Patients A and B), two with psychotic mania (i.e., mania with psychotic features; Patients C and D), and one distractor vignette describing a patient with schizophrenia (Patient E). In the psychotic mania condition, psychotic symptoms were made more salient than mood symptoms to test the hypothesis that the presence of psychotic symptoms with limited information about mood was a source of diagnostic error for providers. The distractor vignette (Patient E; Asian patient with schizophrenia) was used to prevent providers from guessing the intent to measure the role of patient race in clinical decisions about bipolar disorder. Vignettes were adapted from DSM-IV-TR and DSM-5 clinical casebooks, which were written to make the hypothetical scenario reflect likely clinical encounters (Barnhill, 2013; Spitzer, Gibbon, Skodol, Williams, & First, 2002). Thus vignettes contained some ambiguity in clinical presentation to increase how realistic each scenario was, as well as to allow clinical biases to emerge.

The patients described in the bipolar disorder vignettes were presented as black or white such that each provider saw two patients with non-psychotic mania, one black and one white, and two patients with psychotic mania, one black and one white. The patient with schizophrenia in the distractor vignette was presented as Asian to all participants. Names were used to indicate the race of the patients and, again, to make the scenario seem realistic. Vignette prose was pretested with research assistants and graduate students to ensure that the vignettes within each condition were closely matched on symptom severity and severity of dysfunction. Names were selected from a list of pre-tested names rated as "more black" (Tyrone and Lamar) or "more white" (Ian and Todd; Greenwald et al., 1998). A Korean name (Hwang) was selected from a list of common Korean names for the Asian distractor. This list was created on the basis of ratings made by Korean and Japanese raters on the typicality and the distinctness of the name as compared to common Japanese names (Greenwald et al., 1998).

Clinical decision-making questionnaire. A questionnaire was developed to assess providers' diagnostic and prognostic impressions of the case presented in the vignette and their recommendations for treatment. The questionnaire was an adapted version of the decision-making questionnaire by Peris et al. (2008), which was validated in a published study of implicit bias (mental illness stigma) in a sample of mental healthcare professionals. This method demonstrated a good ability to differentiate vignettes that were more likely to be overpathologized or assigned worse prognoses from those in which patients were managing non-clinical levels of distress.

Providers were asked to select a probable diagnosis from a broad list of mental illnesses. The seven variables of interest were overpathologizing, defined as the total number of diagnoses assigned to one patient, and misdiagnosis, defined dichotomously as the correct or incorrect identification of the true diagnosis (bipolar disorder). A prognosis score was summed from ratings on 7-point Likert scales of the likelihood of treatment adherence, benefit from treatment, treatment dropout, and the degree to which the patient was assessed to be a danger to self and others. To assess treatment recommendations, providers were asked whether they would recommend psychiatric consultation, medication, psychotherapy, and hospitalization of the patients described in the vignettes.

Race Attitudes Implicit Association Test (Race IAT; Greenwald, Mcghee, and Schwartz, 1998). The Race IAT was used to measure the strength of implicit racial biases in providers. The IAT is a computer-administered categorization task that uses response latencies to assess the strength of associations between pairs of concepts (Greenwald et al., 1998). On the

Race IAT these concepts are *race* (black vs. white depicted in images of faces) and *valence* (pleasant vs. unpleasant words). The IAT is the most widely accepted measure of implicit social cognition due to its sensitivity to subtle, often undesired and unconscious racial biases. The measure has demonstrated good reliability (Cunningham, Preacher, & Banaji, 2001; Nosek, Greenwald, & Banaji, 2013). The IAT also demonstrates robust validity, including correlations with other implicit attitude measures, and better prediction of discriminatory behavior during interracial interactions than self-report measures (McConnell & Leibold, 2001; Nosek et al., 2005; Sabin et al., 2009). Faster associations between concepts are interpreted as the index of strength of bias. For example, a profile of faster responses to pairing "white" and "good" or "black" and "bad" compared to responses pairing the reverse is interpreted as a pro-white racial bias. On the task, participants are asked to work as quickly as possible, and to categorize images of black and white faces with value-laden words using two computer keys. The task is comprised of seven blocks of trials presented in random order. Before test blocks, participants complete practice blocks, in which they are asked to respond as quickly as possible to a single construct. In two test blocks, participants categorize *race* and *valence* in "stereotype compatible" ways (white faces + pleasant words; black faces + unpleasant words) and "stereotype incompatible" ways (black faces + pleasant words; white faces + unpleasant words).

The presentation of instructions for the IAT measure in this study required some sensitivity. Findings from a previous study indicated that instructions that describe the IAT as sensitive to actual racial attitudes can threaten test-takers and paradoxically cause participants to perform worse on the IAT than they would have otherwise (Frantz, Cuddy, & Burnett, 2004). The same study showed that affirming instructions that do not focus on racism do not lead to the same level of threat or score inflation, and provide valid IAT indices of racial biases. Therefore, administration of the Race IAT in the current study modeled these "non-threatening" instructions (for full description see Frantz et al., 2004). These instructions described the IAT as a measurement of "knowledge about cultural stereotypes" and that it was unrelated to personal beliefs or behaviors about other races.

IAT D score, the main measure of bias, was calculated using the updated scoring algorithm, which yields a standardized difference score (Greenwald, Nosek, & Banaji, 2003). The IAT D score was derived by taking the difference between mean response times on the two test blocks and dividing that difference by the standard deviation of all response times in those two blocks. An IAT D score of zero indicated no bias for white or black race. Positive scores indicated pro-white/anti-black racial bias and negative scores indicated pro-black/anti-white racial bias. The D score has a range of -2 to +2 with break points for "slight bias" (.15), "moderate bias" (.35) and "strong bias" (.65), which are based on conventional break points for effect size. Standard data cleaning procedures for the IAT were applied, including removing trials with long response latencies (potential inattention) and trials with very fast responses (failure to process stimuli).

Feelings thermometer (Greenwald, Mcghee, and Schwartz, 1998). Explicit racial biases were assessed using a traditional feelings thermometer validated in several IAT and racial attitudes studies as a measure of conscious attitudes about members of other races (Dasgupta, Greenwald, Mcghee, Mellott, & Nosek, 2001; Greenwald et al., 1998; Sabin et al., 2008). Participants rated their feelings towards black and white people on two items with an 11-point Likert scale (0 = cold, 10 = warm; *"My feelings towards Black Americans are…"* and *"My feelings towards White Americans are…"*). A single score for explicit bias was calculated as the difference between responses on these two items.

Procedures

Participating providers were emailed a link to a website for the study survey. All study measures were completed via the Internet on a survey hosted by Qualtrics at the participants' convenience. The study was described as a project to assess provider decision-making with the intent of providing evidence to help the reduction of clinical decision-making errors and improve care for patients with serious mental illnesses. No reference to bipolar disorder, race, or bias was made so as not to tip off the providers of the specific study intent.

Providers completed informed consent procedures online followed by the demographic questionnaire. They were then randomly assigned to one of two vignette conditions such that half of the sample was in Vignette Condition 1 and half was in Vignette Condition 2 (see Figure 1). Providers in each condition saw the same five vignettes, however, the patient race displayed in each vignette differed by assignment. Providers in Vignette Condition 1 saw a white Patient A (non-psychotic mania), black Patient B (non-psychotic mania), white Patient C (psychotic mania), black patient D (psychotic mania), and the distractor Patient E (schizophrenia). Providers in Vignette Condition 2 saw a black Patient A (non-psychotic mania), white Patient B (non-psychotic mania), black Patient C (psychotic mania), white patient D (psychotic mania), and the distractor Patient E (schizophrenia). This design allowed for tests of between- and within-subject differences in decision-making that were related to patient race and psychotic symptom presence.

Vignette Condition 1				
Non-Psychotic Mania		Psychotic Mania		Distractor
IAN	**TYRONE**	**TODD**	**LAMAR**	**HWANG**
(white)	(black)	(white)	(black)	(Asian)
Patient A	Patient B	Patient C	Patient D	Patient E
Vignette Condition 2				
Non-Psychotic Mania		Psychotic Mania		Distractor
TYRONE	**IAN**	**LAMAR**	**TODD**	**HWANG**
(black)	(white)	(black)	(white)	(Asian)
Patient A	Patient B	Patient C	Patient D	Patient E

Figure 1. Randomly assigned vignette conditions. Vignettes were presented in random order.

In both vignette conditions, the five clinical vignettes were presented in random order, and providers completed the clinical decision-making questionnaire for each case immediately after each individual vignette was presented. Next, providers completed implicit and explicit measures of racial bias (Race IAT and the Feelings Thermometer, respectively). Finally, providers answered additional demographic questions and a question about their awareness of the study intent to measure racial bias. After completing the survey, providers read a debriefing form explaining the true study purpose and were asked to provide reconsent for use of their data. All providers were then directed to a separate survey to provide their email addresses if they wished to receive the $100 compensation for their participation.

Analysis

Analyses were conducted using version 24 of SPSS statistical software (SPSS Inc., Chicago, Ill, USA). Mplus version 7 was used for SEM analyses (Muthen and Muthen, 2007). Alpha was set to .05. Descriptive statistics were used to characterize the provider sample. T-tests, chi-squares, and ANOVAs were conducted to assess whether providers in the randomly assigned vignette conditions were matched on key background variables.

Before conducting tests of hypotheses, statistical assumptions were tested, including review of the univariate distributions of variables. To test Hypothesis 1, three-way mixed ANOVAs were conducted to examine the main effects of and interactions between patient race, psychosis, and vignette condition on each of the decision-making outcomes. The distractor vignette was not included in analyses. To test Hypothesis 2, a one-sample t-test was used to assess whether the mean IAT d-score and explicit bias score differed significantly from zero (i.e., neutral or unbiased on the IAT). To further characterize racial bias in the provider sample, the absolute and relative strengths of explicit and implicit biases in specific demographic subgroups were examined using correlations, t-tests and one-way ANOVAs. These follow-up analyses included comparisons of group scores against zero as well as independent samples to each other. To test Hypothesis 3, structural equation modeling (SEM) was used to test the fit of a hypothesized model of implicit and explicit bias scores predicting decision-making outcomes among black patients controlling for decision-making in white patients (see Figure 11).

Results

Table 1 describes demographic and clinical characteristics of the participants, as well as mean IAT scores for subgroups. Several demographic subgroups had low endorsement, so variables were collapsed into broader categories. The provider sample was primarily female, non-Hispanic, and white. Most had a Ph.D. or Psy.D., were practicing in clinical psychology, and in private practice. Most participants were therapists, and no one prescribed medication. The majority had experience treating serious mental illnesses, with about half having experience with bipolar disorder. On average, providers reported that 38.5% of their patients were racial or ethnic minorities and 10.7% had bipolar disorder.

Potential Confounding Variables

Providers in Vignette Condition 1 and Vignette Condition 2 were matched on thirteen of fourteen clinical background and demographic characteristics, but differed in their field of practice, $\chi^2 = 12.8$, df = 3, $p < .01$. There were more clinical psychologists in Vignette Condition 2 ($n = 42$) than in Vignette Condition 1 ($n = 40$), $\chi^2 = 6.0$, df = 1, $p < .05$, and all the counseling psychologists were randomized to Vignette Condition 1, $\chi^2 = 12.0$, df = 1, $p < .01$. Clinical psychology background was also correlated with IAT scores, Pearson $r = ..24$, $N = 82$, $p < .05$.

Given the associations between field and other variables of interest, provider field was included as a covariate in the multivariate model for the SEM analyses in Hypothesis 3 tests. Two binary variables for clinical and for counseling psychology (i.e., 0 = not a clinical psychologist, 1 = clinical psychologist) were created for this purpose.

Analysis of Vignette Matching on Symptom Severity Ratings

Despite pre-testing to match symptom severity within the non-psychotic mania vignettes and the psychotic mania vignettes, paired sample t-tests revealed that vignettes were not matched. Of the two non-psychotic mania vignettes, providers rated Patient A's symptoms as more severe than Patient B's symptoms. Of the two psychotic mania vignettes, they rated Patient D's symptoms as more severe than Patient C's symptoms. This is important to consider in interpreting following results, as providers in Vignette Condition 1, who saw Patient A as white and Patient B as black, would be expected to rate the non-psychotic white patient as more severe and in need of more care than the non-psychotic black patient. These vignette discrepancies between the nonpsychotic vignettes work against hypotheses. There is a similar concern for providers in Vignette Condition 2, who saw Patient D as white and Patient C as black. Given the vignette severity mismatch, providers in Vignette Condition 2 would be expected to rate the psychotic black patient as more severe and in need of care than the psychotic white patient. Thus, racial disparities observed between black and white patients, especially that follow this pattern of discrepancy, could be the result of vignette severity mismatch rather than bias. Thus, effects of Vignette Condition below may be indicators of confounding effects of the mismatched vignettes. The results of analyses for Hypothesis 1 tests were examined for patterns of racial disparities that mirrored the vignette severity mismatch, and the effect of this methodological concern is discussed in the conclusions for this paper.

Descriptive Analyses

Means and variances for each outcome variable are presented in Table 3. Inter-correlations of outcome variables are presented in Table 4. As shown, variability was extremely limited for referrals to psychiatrists and for medications, limiting ability to study these outcomes; it is also the case that these two outcomes were highly correlated. Referrals to a psychiatrist are typically made with the intent to help the client access medication, which may explain the correlation. Combining the two variables was considered, however, alpha reliability of the combined variable did not reach an acceptable level (α = .65). Thus, the medication variable was dropped from main analyses[1]. The remaining outcome variables appeared highly independent from each other.

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[1] There were no main effects of race or race by psychosis interactions for medication prescriptions. There was a significant Race x Vignette Condition interaction, $F(1, 78) = 5.12$, $p < .05$, partial eta^2 = .06, suggesting that providers in the two vignette conditions differed in the rates of medication prescription they made to black and white patients. This effect of race, however, was not present among Vignette Condition 1 providers, $F(1, 37) = 1.63$, $p = .21$, partial eta^2 = .09, and only marginally significant among Vignette Condition 2 providers, $F(1, 41) = 3.84$, $p = .06$, partial eta^2 = .04. Pairwise comparisons showed that there was a slight tendency in Vignette Condition 2 providers to recommend medication prescription more often to black patients (mean = .95, 95% CI [.91, 1.00]) than white patients (mean = .88, 95% CI [.81, .96]), $p = .06$.

Effects of Patient Race and Psychosis on Diagnosis, Prognosis, and Treatment Decisions

　　　　Main effects, two-way interactions, and three-way mixed ANOVAs of race, psychosis, and vignette condition were conducted with the decision-making variables as outcomes, to determine whether providers in the whole sample and within each vignette condition treated patients differently because of their race and if the presence of psychosis exacerbated any biases in decision-making. Assumptions of normality and homogeneity of variances were not met for several cells within variables as assessed using, respectively, Shapiro-Wilk's test and Levene's test for equality of variances. Transformations were not performed in order to maintain the interpretability of results and because the ANOVA is robust to deviations in normality, as well as variance in heterogeneity when sample sizes of subgroups are relatively equal. These deviations appeared to be caused by outliers, as assessed by inspection of boxplots. There were six outliers in misdiagnosis and psychiatrist referral; eight outliers in prognosis; and ten outliers in overpathologizing, therapy, and hospitalization. One participant was identified as an outlier on three of six variables. Rather than performing transformations, a comparison was made of results with and without outliers. Ultimately, outliers were kept in the analysis, because they did not materially affect the results. Mauchly's test of sphericity was met for the three-way interaction effects and the simple two-way interaction effects. Sidak corrections for multiple comparisons were used to adjust p-values in all pairwise comparisons; these adjusted p-values are reported below. Figures are provided for statistically significant three-way and two-way interactions.

　　　　Misdiagnosis. Misdiagnosis of bipolar disorder was coded as binary (i.e., 0 = not diagnosed with bipolar disorder, 1 = diagnosed with bipolar disorder). There were no main effects of race or vignette condition on misdiagnosis. There was a main effect of psychosis, such that patients were more likely to be correctly diagnosed with bipolar disorder when they were psychotic than when they were non-psychotic, $F(1, 80) = 4.86$, $p < .05$, partial eta^2 = .06. For the two-way interactions, there was no significant race by psychosis interaction for this outcome, indicating that the presence of psychosis did not exacerbate racial disparities in bipolar disorder diagnosis as hypothesized, $F(1, 80) = .18$, $p = .67$, partial eta^2 = .02. There was also no psychosis by vignette condition interaction, indicating that providers in each assigned condition diagnosed bipolar disorder similarly in both psychotic and non-psychotic patient vignettes, $F(1, 80) = 4.86$, $p = .44$, partial eta^2 = .01. There was a significant two-way interaction between patient race and vignette condition, $F(1, 80) = 30.14$, $p < .001$, partial eta^2 = .27, indicating that the effects of patient race on bipolar disorder diagnosis differed in the two vignette conditions. As shown in Figure 2, in Vignette Condition 1, providers gave bipolar disorder diagnoses less often to black patients (mean = .51, 95% CI [.40, .63]) than white patients (mean = .75, 95% CI [.65, .85]), $F(1, 39) = 25.19$, $p < .01$, partial eta^2 = .39, as hypothesized. In contrast with hypotheses, providers in Vignette Condition 2 gave bipolar disorder diagnoses more often to black patients (mean = .70, 95% CI [.59, .81]) than white patients (mean = .50, 95% CI [.38, .62]), $F(1, 41) = 10.03$, $p < .01$, partial eta^2 = .20. Finally, there was no significant three-way interaction between race, psychosis, and vignette condition for misdiagnosis, $F(1, 80) = 1.25$, $p = .28$, partial eta^2 = .02, showing that the race by psychosis interactions aforementioned did not differ by assigned vignette condition.
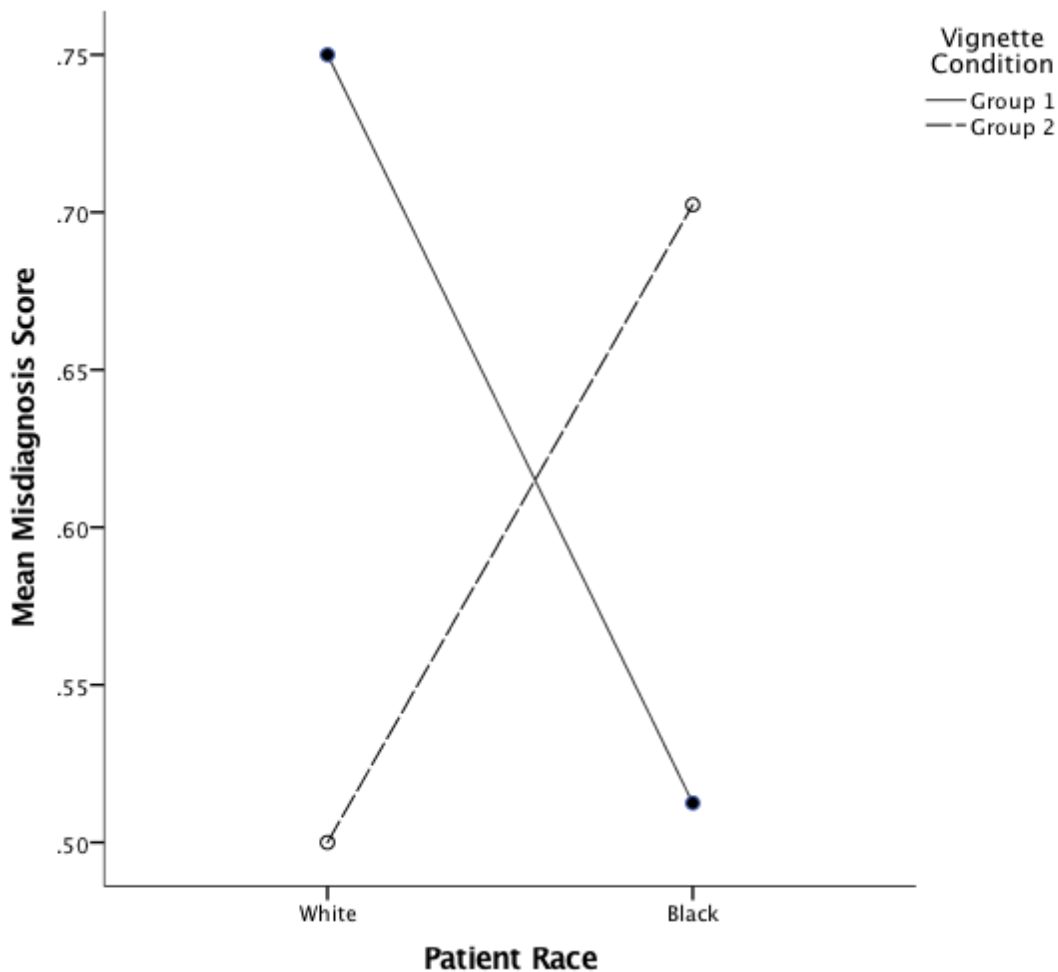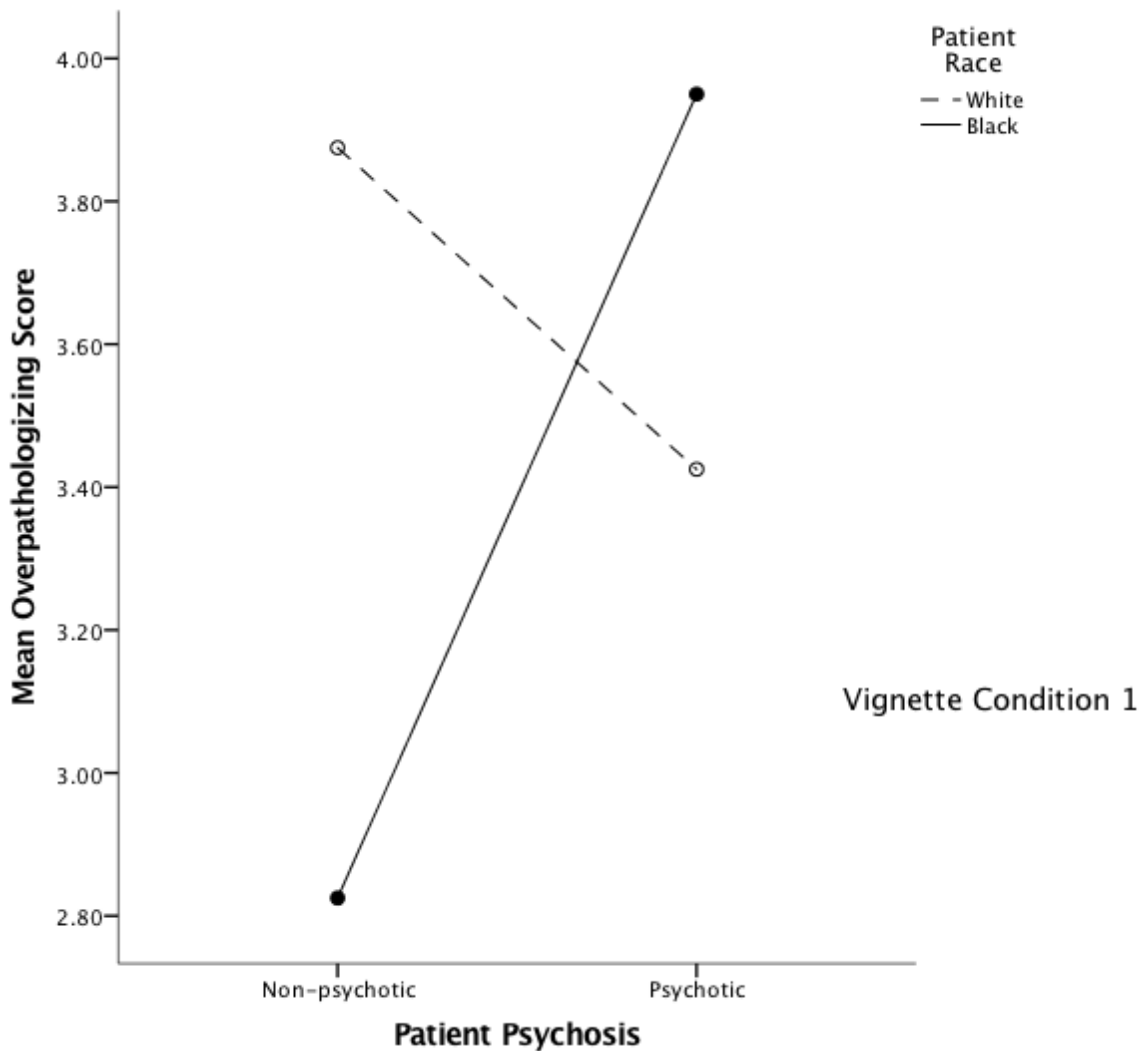
Figure 2. Race x Vignette Condition interaction for Misdiagnosis. Range was 0 to 1 with higher scores indicating a correct diagnosis of bipolar disorder and lower scores indicating that bipolar disorder was not diagnosed.

Overpathologizing. There were no main effects of race, psychosis, or vignette condition for overpathologizing, nor two-way interactions of these variables. There was a significant three-way interaction between race, psychosis, and vignette condition on overpathologizing, $F(1, 80) = 14.25$, $p < .001$, partial eta$^2 = .15$, indicating that the race by psychosis interaction differed in the two assigned vignette conditions (see Figure 3). Specifically, the directions of the two two-way effects were opposite and driven by the responses to non-psychotic vignettes. Providers in Vignette Condition 1 assigned, on average, 1.05 more diagnoses (overpathologized) to the non-psychotic white patient (mean = 3.86, 95% CI [2.84, 4.91]) than to the non-psychotic black patient, (mean = 2.83, 95% CI [2.05, 3.61]), $F(1, 39) = 4.94$, $p < .05$, partial eta$^2 = .11$. Providers in Vignette Condition 2 assigned, on average, 0.71 more diagnoses to the non-psychotic black patient (mean = 3.05, 95% CI [2.16, 3.94]) than the non-psychotic white patient (mean = 2.33, 95% CI [1.74, 2.93]), $F(1, 41) = 4.34$, $p < .05$, partial eta$^2 = .10$. There was no effect of race on overpathologizing among psychotic patients in either vignette condition,

There was a main effect of psychosis in Vignette Condition 1 among black patients, $F(1, 39) = 10.48$, $p < .01$, partial eta$^2 = .21$. As hypothesized, providers in Vignette Condition 1

assigned 1.13 more diagnoses to black patients when they were psychotic (mean = 3.95, 95% CI [3.08, 4.81]) than when they were not, (mean = 2.83, 95% CI [2.05, 3.61]). This effect was not significant among white patients treated by Vignette Condition 1 providers. There was no main effect of psychosis in Vignette Condition 2 among black or white patients.
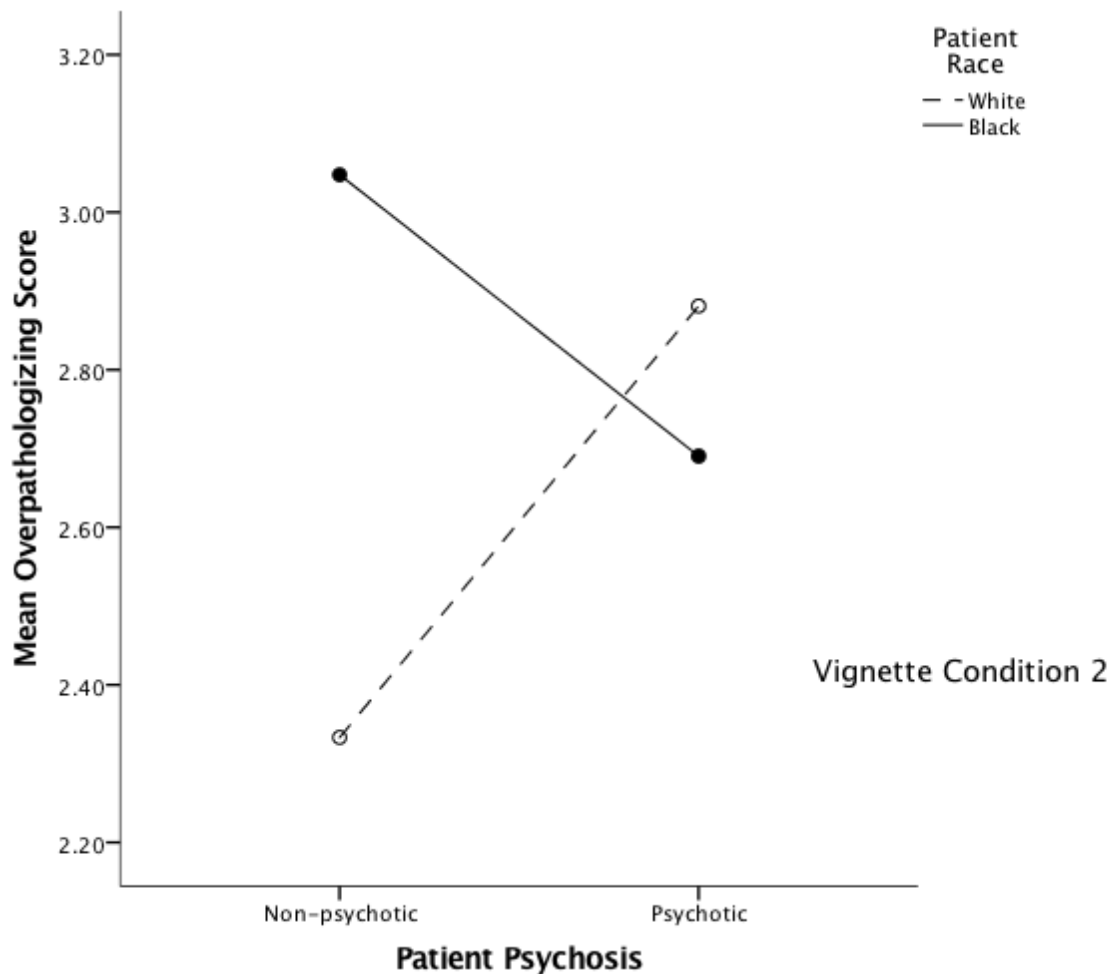
Figure 3. Race x Psychosis x Vignette Condition interaction for Overpathologizing. Y-axis is number of diagnoses assigned to a patient. Thus, higher scores indicate greater overpathologizing.

Prognosis. There were no main effects of race or vignette condition on prognosis. There was a main effect of psychosis in the expected direction, namely that the psychotic patients (mean = 23.63, 95% CI [22.99, 24.26]) received worse prognoses than the non-psychotic patients (mean = 24.47, 95% CI [23.70, 25.24]), $F(1, 79) = 5.01$, $p < .05$, partial eta^2 = .06. There was no significant Race x Psychosis interaction in the total sample. There was a significant Race x Vignette Condition interaction $F(1, 79) = 4.70$, $p < .05$, partial eta^2 = .06 (Figure 4) and a significant Psychosis x Vignette Condition interaction $F(1, 79) = 4.57$, $p < .05$, partial eta^2 = .06 (Figure 5), indicating that providers in the assigned conditions treated patients differently on the basis of race as well as psychosis presence. Pairwise comparisons, however, showed that the difference between black and white patients was only marginally significant in Vignette Condition 2, $p = .06$, and not significant in Vignette Condition 1. The Psychosis x Vignette Condition interaction was driven by a difference in prognoses given to patients seen by the Vignette Condition 2 providers as compared to Vignette Condition 1 providers, specifically the assignment of worse prognoses to psychotic patients (mean = 23.58, 95% CI [22.61, 24.56]) than to non-psychotic patients in Vignette Condition 2 (mean = 25.24, 95% CI [24.11, 26.37]), $F(1,$

41) = 10.03, $p < .01$. In Vignette Condition 1, prognoses were comparable for nonpsychotic patients (mean = 23.71, 95% CI [22.63, .24.78]) and psychotic patients, (mean = 23.67, 95% CI [22.84, 24.49]), $F(1, 38) = .01$, $p = .95$. The three-way interaction of Race x Psychosis x Vignette Condition on prognosis was significant, suggesting that the Race x Psychosis interaction differed in the two vignette conditions, $F(1, 79) = 44.76$, $p < .001$, partial eta$^2 = .36$ (see Figure 6).
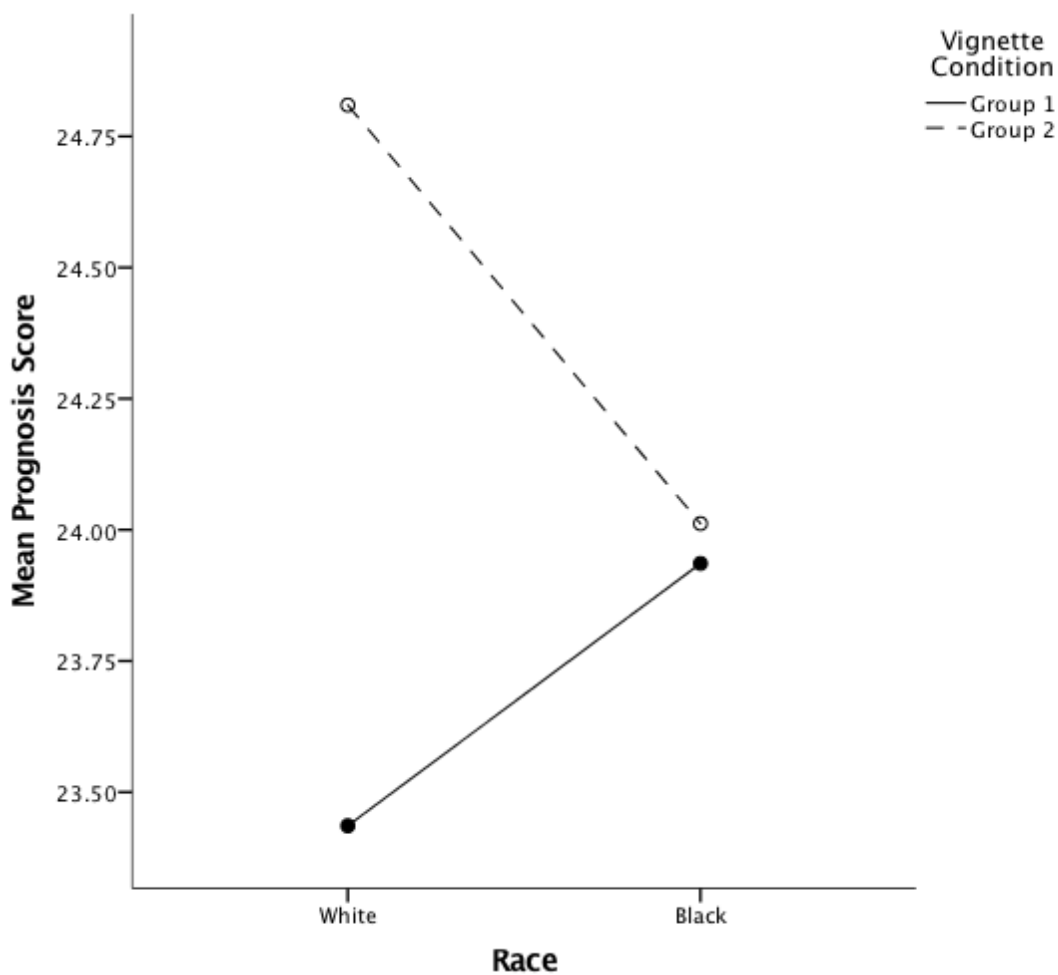

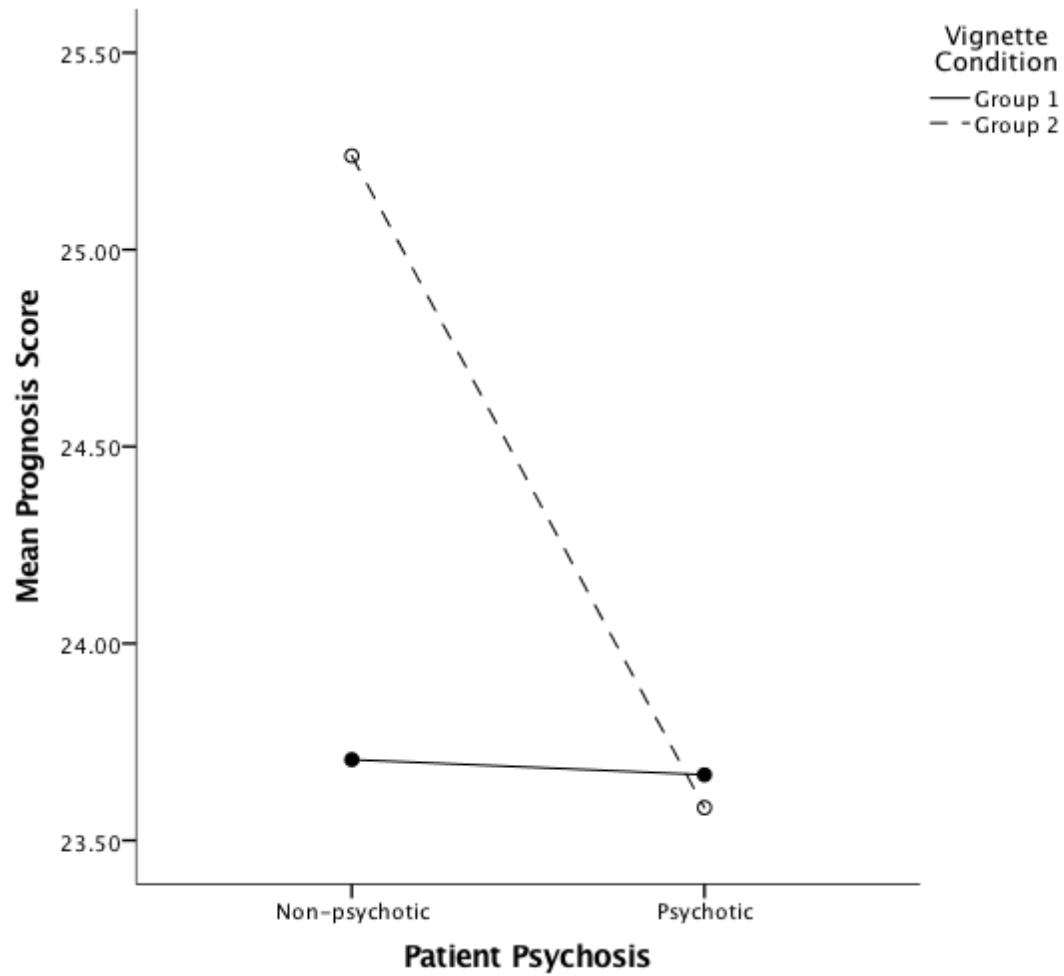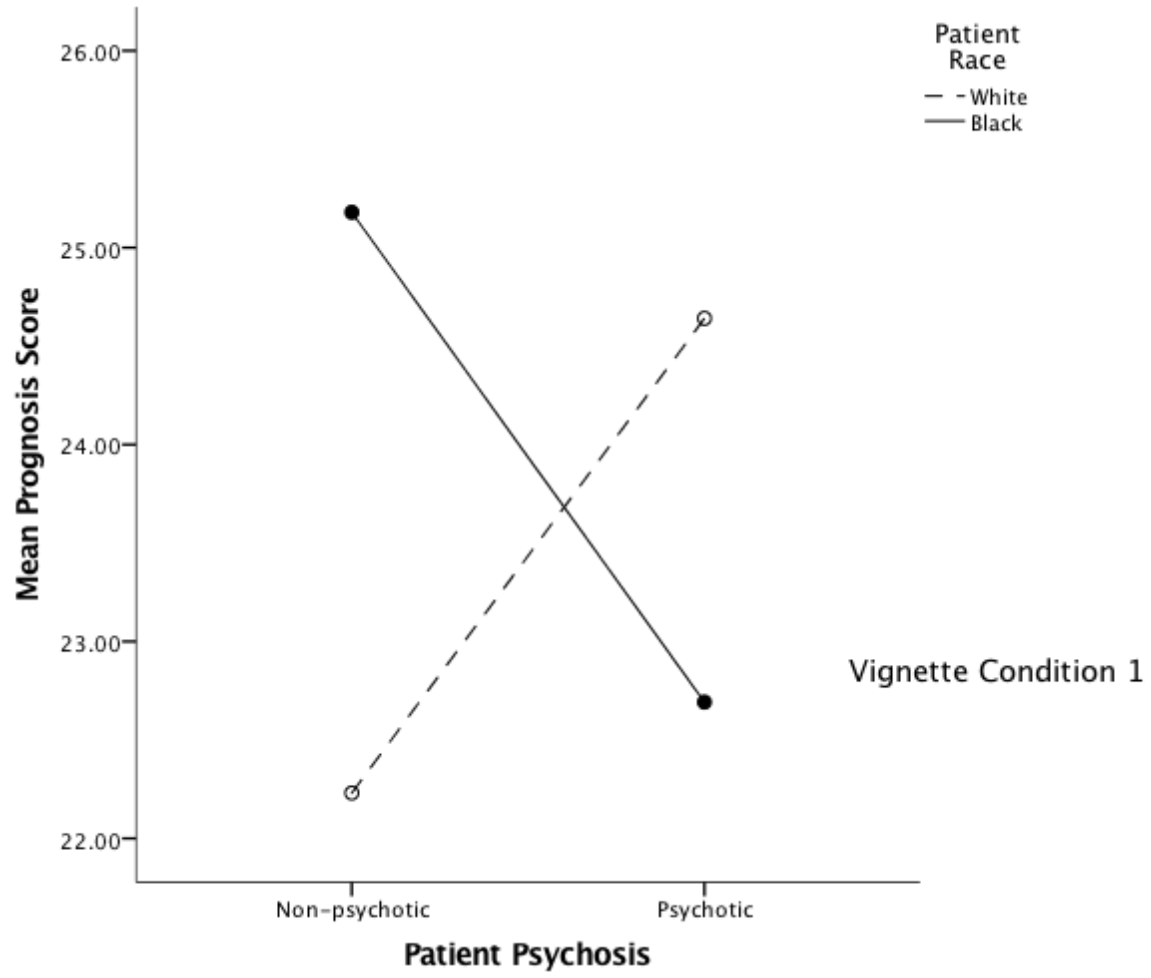*Figure 4*. Race x Vignette Condition interaction for Prognosis. Higher scores indicate better prognoses.

Figure 5. Psychosis x Vignette Condition interaction for Prognosis. Higher scores indicate better prognoses.
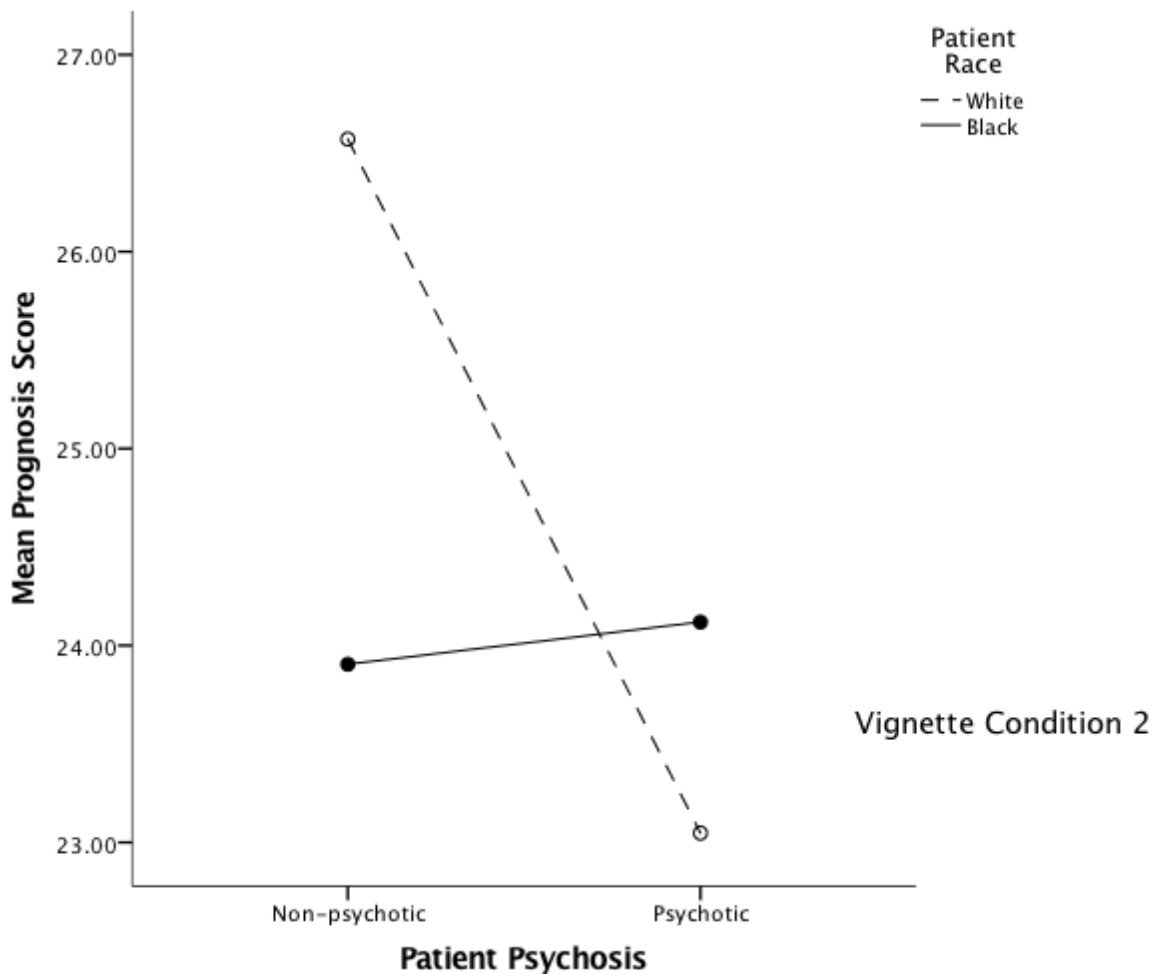
Vignette Condition 1

Figure 6. Race x Psychosis x Vignette Condition interaction for Prognosis. Higher scores indicate better prognoses.

Follow-up analyses of the three-way interaction revealed that the two-way interaction effects (Race x Psychosis) in the vignette conditions were similar in magnitude but opposite in direction, Vignette Condition 1, $F(1, 38) = 26.22$, $p < .001$, partial eta$^2 = .41$, and Vignette Condition 2, $F(1, 41) = 18.42$, $p < .001$, partial eta$^2 = .31$ (see Figure 6). Pairwise comparisons revealed that providers in Vignette Condition 1 gave non-psychotic black patients a prognosis score that was, on average, 2.95 points higher (better prognosis) than what they gave non-psychotic white patients, 95% CI of the difference [1.70, 4.20], $F(1, 38) = 22.69$, $p < .001$; whereas providers in Vignette Condition 2 gave non-psychotic black patients a score that was 2.67 points lower (worse prognosis) than what they gave non-psychotic white patients, 95% CI of the difference [-3.89, -1.45], $F(1, 41) = 19.46$ $p < .001$. For psychotic trials, providers in Vignette Condition 1 gave black patients a prognosis score that was 1.90 points lower than what they gave white patients, 95% CI of the difference [-3.21, -.59], $F(1, 39) = 8.59$, $p < .01$. This effect was not significant for psychotic black patients treated by Vignette Condition 2 providers.

There was a main effect of psychosis in Vignette Condition 1 among black patients, $F(1, 39) = 10.50$, $p < .01$. Providers in Vignette Condition 1 gave black patients who were psychotic prognoses that were 2.48 points lower than non-psychotic black patients, 95% CI [.93, 4.02], $p <$

.01. This effect of psychosis was also present for white patients seen by Vignette Condition 1 providers, $F(1, 38) = 12.90$, $p < .01$; this group gave white patients who were psychotic prognoses that were 2.41 points higher than white patients who were not psychotic. There was no effect of psychosis in Vignette Condition 2 among black patients, but there was an effect among white patients, $F(1, 41) = 21.33$, $p < .001$. Vignette Condition 2 providers gave white patients who were non-psychotic prognoses that were 3.52 points higher than when they were psychotic, 95% CI [1.98, 5.07].

Psychiatrist referral. There were no main effects of race or vignette condition for psychiatrist referrals. There was a main effect of psychosis in the expected direction, namely that the psychotic patients (mean = 1.00, 95% CI [1.00, 1.00]) received more referrals to psychiatrists than non-psychotic patients (mean = .96, 95% CI [.93, .99]), $F(1, 79) = 7.59$, $p < .01$, partial eta^2 = .09. The Psychosis x Vignette Condition and Race x Psychosis interactions were not significant. There was a nonsignificant trend toward a two-way Race x Vignette Condition interaction, $F(1, 79) = 3.60$, $p = .06$, partial eta^2 = .04. There was also a nonsignificant trend toward three-way interaction between race, psychosis, and vignette condition for psychiatrist referral, $F(1, 79) = 3.60$, $p = .06$, partial eta^2 = .04.

Psychotherapy recommendation. There were no main effects of race or vignette condition on psychotherapy recommendations. There was a main effect of psychosis in the expected direction, namely that the psychotic patients (mean = .81, 95% CI [.74, .88]) received fewer psychotherapy recommendations than the non-psychotic patients, (mean = .93, 95% CI [.89, .98]), $F(1, 80) = 15.49$, $p < .01$, partial eta^2 = .16. There were no significant Psychosis x Group or Race x Psychosis interactions for this outcome. There was a significant Race x Vignette Condition interaction $F(1, 80) = 4.70$, $p < .05$, partial eta^2 = .06, suggesting that providers in the assigned conditions treated patients differently on the basis of race (see Figure 7). Pairwise comparisons, however, were not significant. Providers in Vignette Condition 1 showed a tendency to recommend therapy to white patients (mean = .91, 95% CI [.81, .93]) more than black patients (mean = .85, 95% CI [.77, .94]), $p = .13$. In Vignette Condition 2, providers showed a tendency of recommending therapy more often to black patients (mean = .89, 95% CI [.81, .98]) than to white patients (mean = .83, 95% CI [.75, .92]), $p = .13$.
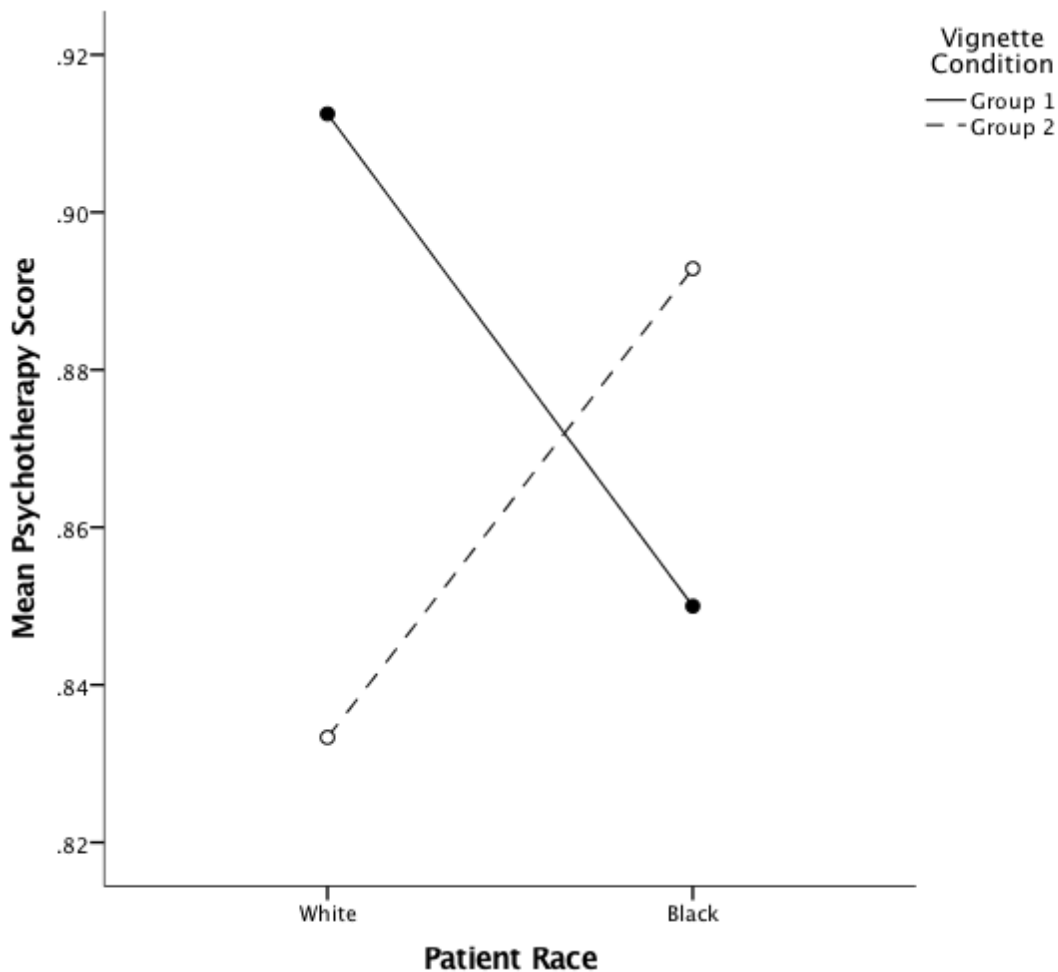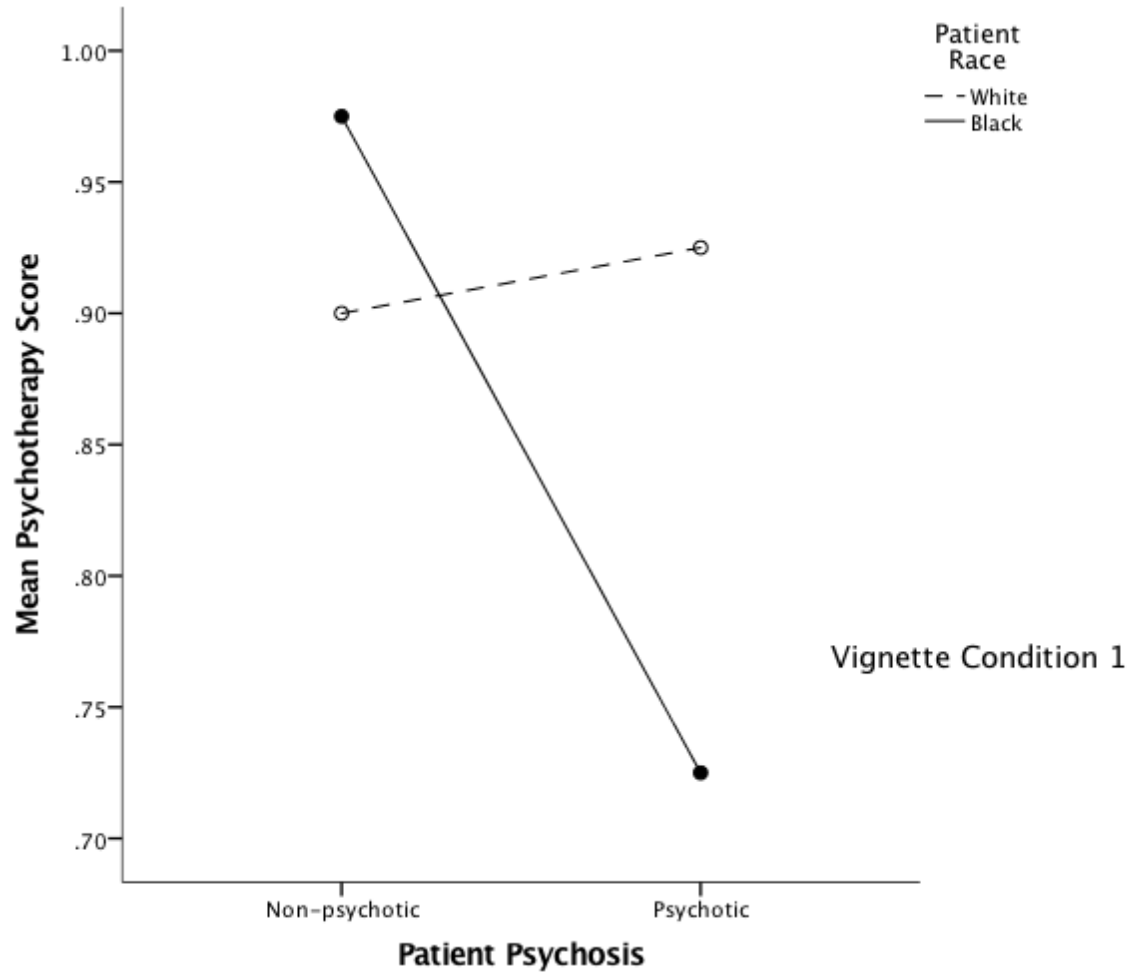
Figure 7. Race x Vignette Condition interaction for Psychotherapy. Higher scores indicate higher frequency of psychotherapy recommendations.

There was a three-way interaction of Race x Psychosis x Vignette condition for psychotherapy recommendation, $F (1, 80) = 23.02$, $p < .001$, partial eta$^2 = .22$. The Race x Psychosis interactions in each Vignette Condition were similar in magnitude but opposite in direction (Vignette Condition 1) $F(1, 39) = 11.83$, $p < .01$, partial eta$^2 = .23$, (Vignette Condition 2) $F(1, 41) = 11.18$, $p < .01$, partial eta$^2 = .21$ (see Figure 8). Further analyses of the three-way interaction showed that there was no main effect of race for the non-psychotic patient vignettes, however there was an effect for the psychotic vignettes in both vignette conditions, (Vignette Condition 1) $F(1, 39) = 9.75$, $p < .01$, (Vignette Condition 2) $F(1, 41) = 8.20$, $p < .01$. Pairwise comparisons for the psychotic trials showed that providers in Vignette Condition 1 recommended psychotherapy less often to black patients than to white patients who were psychotic, mean difference $= .20$, 95% CI of the difference $[-.33, -.07]$, $p < .01$, whereas in contrast, providers in Vignette Condition 2 recommended psychotherapy more often to black patients than to white patients who were psychotic, mean difference $= .17$, 95% CI $[.05, .28]$, $p < .01$.
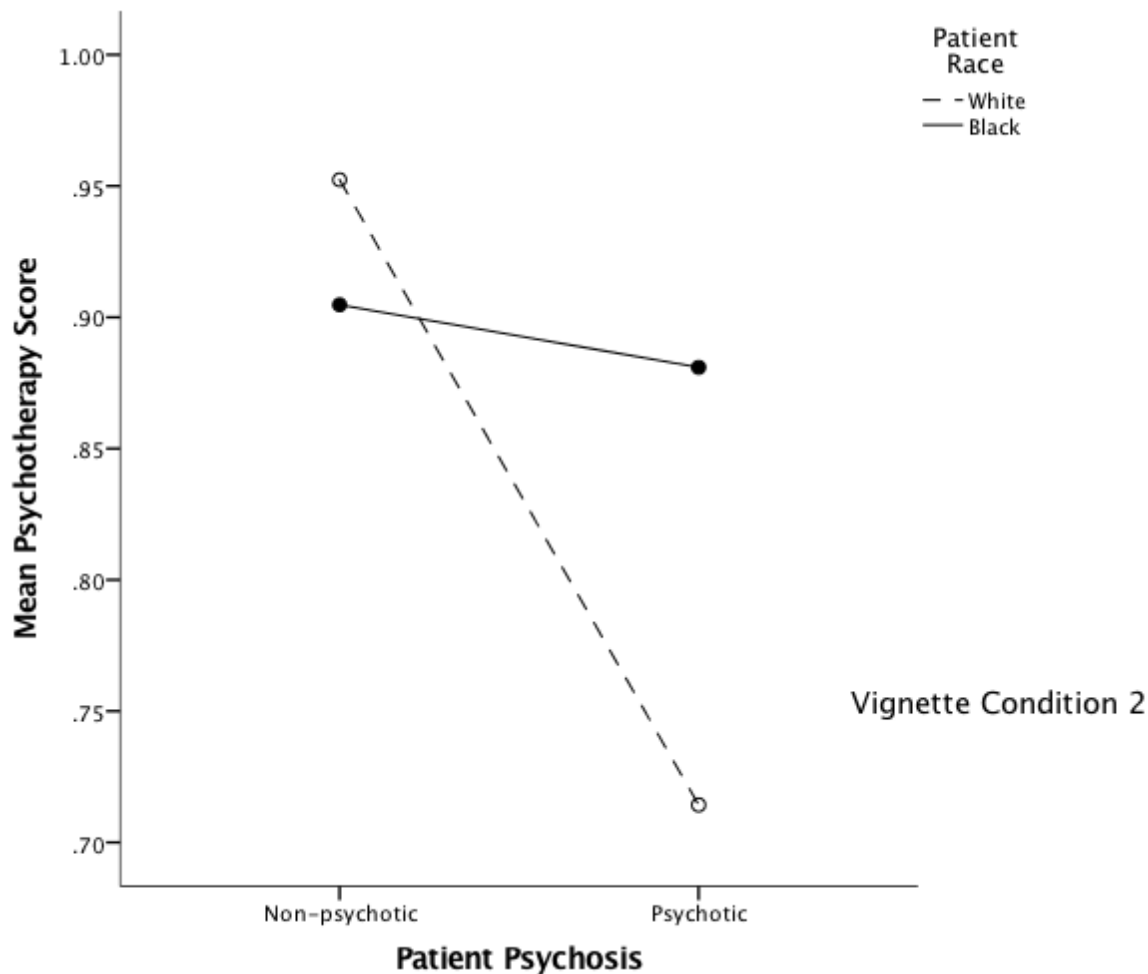
Figure 8. Race x Psychosis x Vignette Condition interaction for Psychotherapy. Higher scores indicate higher frequency of psychotherapy recommendations.

There was a main effect of psychosis in Vignette Condition 1 among black patients, F(1, 39) = 10.26, p < .01. Providers in Vignette Condition 1 recommended psychotherapy more often to black patients who were non-psychotic than black patients who were psychotic, mean difference = .25, 95% CI [.09, .41], p < .01. There was no difference in the treatment given to psychotic and non-psychotic black patients by Vignette Condition 2 providers. In contrast, the effect of psychosis was present for white patients seen by Vignette Condition 2, F(1, 41) = 12.81, p < .01, but not for Vignette Condition 1. Vignette Condition 2 providers recommended psychotherapy more often to white patients who were non-psychotic than white patients who were psychotic, mean difference = .24, 95% CI [.10, .37], p < .01, and Vignette Condition 1 providers treated non-psychotic and psychotic white patients equally.

Hospitalization. There were no main effects or race or vignette condition for hospitalization decisions. There was a main effect of psychosis in the expected direction, namely that the psychotic patients (mean = .76, 95% CI [.69, .83]) received fewer psychotherapy recommendations than the non-psychotic patients, (mean = .24, 95% CI [.17, .31]), F(1, 79) = 150.40, p < .001, partial eta^2 = .66. There were no significant Race x Vignette Condition or Race x Psychosis interactions. There was a significant Psychosis x Vignette Condition interaction F(1,

79) = 10.16, $p < .05$, partial eta^2 = .11, suggesting that providers in the assigned conditions treated patients differently on the basis of psychosis presence (see Figure 9). Pairwise comparisons showed that, although both vignette condition provider groups recommended hospitalization more to psychotic patients than non-psychotic patients, the discrepancy in hospitalization rates was greater among providers in Vignette Condition 2, mean difference = .66, 95% CI of the difference [.54, .77], $p < .001$, than providers in Vignette Condition 1, mean difference = .39, 95% CI of the difference [.26, .51], $p < .001$.

Figure 9. Psychosis x Vignette Condition interaction for Hospitalization. Higher scores indicate higher frequency of hospitalization recommendations.

There was a three-way interaction of Race x Psychosis x Vignette Condition for hospitalization recommendation, $F(1, 79) = 49.85$, $p < .001$, partial eta^2 = .39. The simple two-way interactions (Race x Psychosis) in both vignette conditions were similar in magnitude but opposite in direction, (Vignette Condition 1) $F(1, 38) = 24.70$, $p < .001$, partial eta^2 = .39, (Vignette Condition 2) $F(1, 41) = 27.88$, $p < .001$, partial eta^2 = .41 (see Figure 10). Pairwise comparisons showed that when patients were non-psychotic, providers in Vignette Condition 1 recommended hospitalization less often to black patients than to white patients, mean difference

= .31, 95% CI [.12, .49], *p* < .01, while providers in Vignette Condition 2 recommended hospitalization more often to black patients than to white patients, mean difference = .19 95% CI [.07, .31], *p* < .01. When patients were psychotic, providers in Vignette Condition 1 recommended hospitalization more often to black patients than to white patients, mean difference = .35, 95% CI [.18, .52], *p* < .001, while providers in Vignette Condition 2 recommended hospitalization less often to black patients than to white patients, mean difference = .21, 95% CI [.09, .34], *p* < .01.
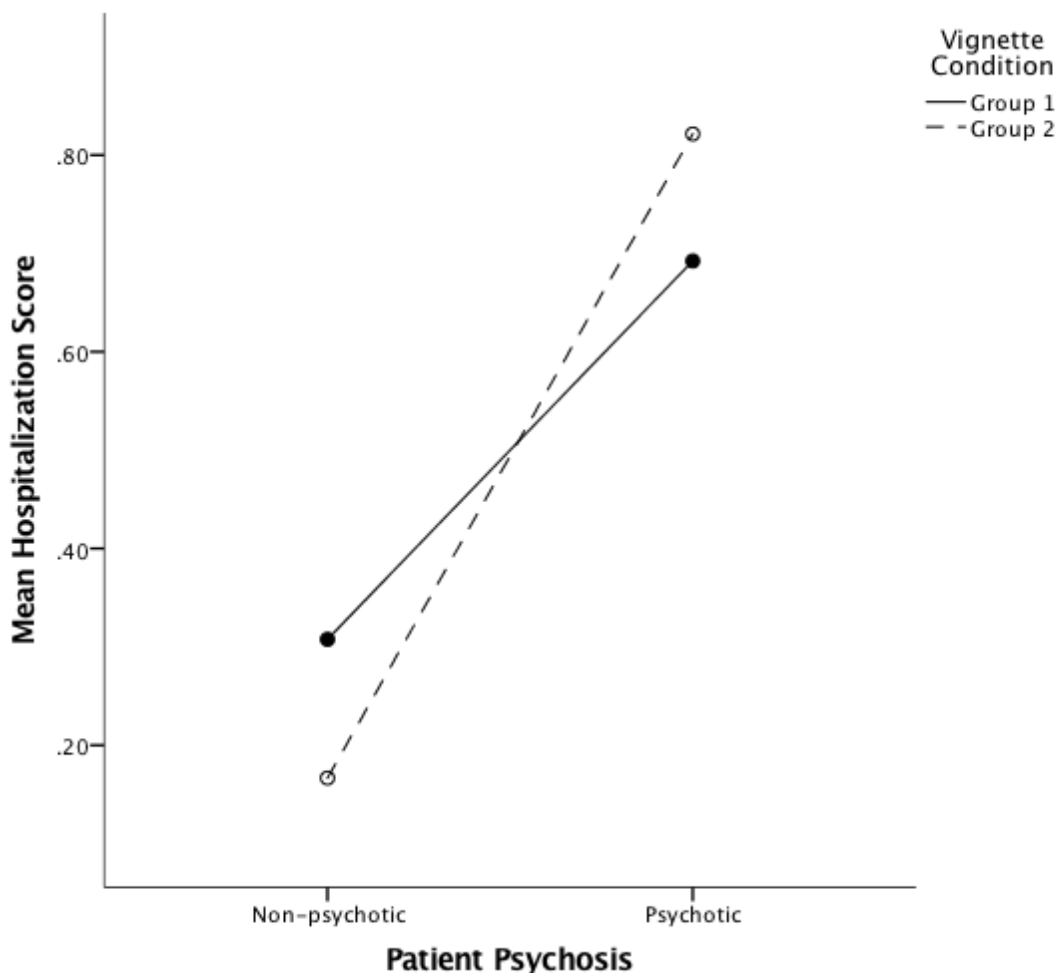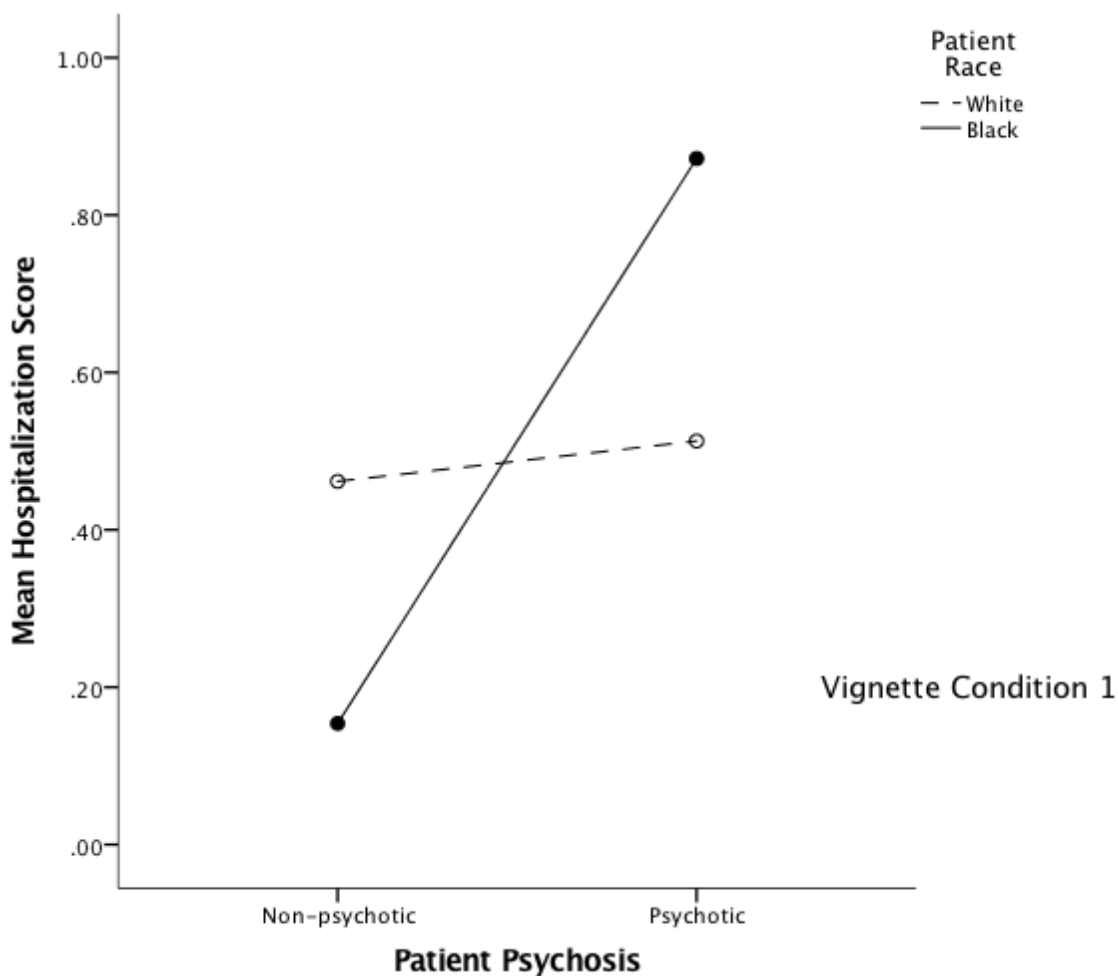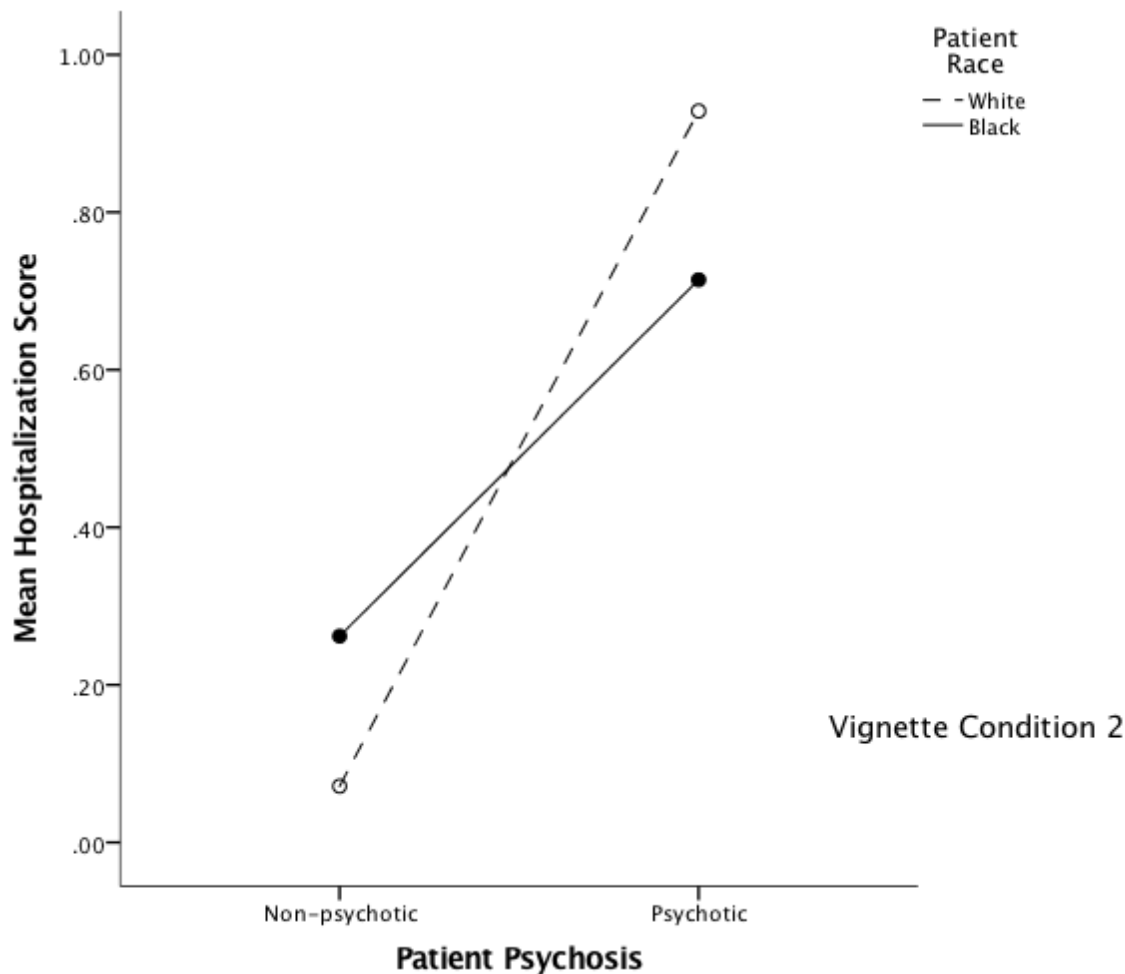
Figure 10. Race x Psychosis x Vignette Condition interaction for Hospitalization. Higher scores indicate higher frequency of hospitalization recommendations.

There were also main effects of psychosis in both vignette conditions among black patients, (Vignette Condition 1) $F(1, 39) = 102.82$, $p < .001$, (Vignette Condition 2) $F(1, 41) = 28.41$, $p < .001$. Providers in both vignette conditions were more likely to recommend hospitalization to black patients when they were psychotic than when they were non-psychotic, (Vignette Condition 1) mean difference = .73, 95% CI [.58, .87], $p < .001$, (Vignette Condition 2) mean difference = .45, 95% CI [.28, .62]. There was no main effect of psychosis among white patients seen by Vignette Condition 1, but the effect was present among white patients seen by Vignette Condition 2 providers. Providers in Vignette Condition 2 recommended hospitalization more often to white patients when they were psychotic than when they were non-psychotic, mean difference = .86, 95% CI [.75, .97].

Providers' Implicit and Explicit Racial Biases

The distribution of IAT D-scores was approximately normal as assessed by inspection of the Q-Q plot and skewness and kurtosis statistics. The average IAT D-score for the entire sample was .37, $SD = .60$, one-sample $t(81) = 5.64$, $p < .01$, considered to be a moderate pro-white bias. The average explicit bias difference score was .13, $SD = 1.62$, which was not different from zero

or "neutral," one-sample $t(81) = .75$, $p = .45$. IAT d-scores and explicit bias difference scores were positively correlated such that the stronger the explicit pro-white or pro-black bias, the stronger the implicit pro-white or pro-black bias, Pearson $r = .39$, $N = 82$, $p < .01$.

There were no differences in IAT or explicit bias scores based on gender, Hispanic ethnicity, type of serious mental illness experience, degree type, field, and setting of practice (see Table 1). Age, years of serious mental illness experience, and proportion of racial or ethnic minority patients, patients with serious mental illness or bipolar disorder specifically were not significantly correlated with implicit and explicit bias (see Table 2). There was a difference in explicit bias scores depending on provider race, $F(3) = 4.65$, $p < .01$. Given that there was only one black provider, post-hoc comparisons could not be examined between providers of black and other races. The effect of provider race on explicit bias, however, was no longer significant after running the one-way ANOVA without the black provider, $F(2) = 1.10$, $p = .34$.

T-tests of implicit and explicit bias scores against the value zero ("neutral") within each vignette condition were conducted. Providers in Vignette Condition 2 had moderate pro-white implicit bias, mean = .52, $SD = .50$, $t(41) = 6.67$, $p < .001$, and moderate pro-white explicit bias, mean = .55, $SD = 1.17$, $t(41) = 3.03$ $p < .01$, scores (consonant attitudes about blacks and whites). Providers in Vignette Condition 1 had slight pro-white implicit bias, mean = .22, $SD = .66$, $t(39) = 2.14$, $p < .05$, but neutral explicit bias, mean = -.30, $SD = 1.90$, $t(39) = -1.00$, $p = .32$, scores (dissonant attitudes about blacks and whites). Although Vignette Condition 1's mean explicit bias raw score was in the pro-black range, this did not reach statistical significance. Independent samples t-tests showed that the vignette conditions differed in the strength of their implicit and explicit biases. Vignette Condition 2 had a stronger pro-white implicit bias than Vignette Condition 1, $p < .05$, as well as stronger pro-white explicit bias, $p < .05$.

Linking Racial Disparities in Clinical Decision-Making to Racial Bias

Only the four decision-making variables in which racial disparities were present (in interaction with other variables) at analyses for Hypothesis 1 were retained in the SEM analyses: overpathologizing, prognosis, psychotherapy recommendation, and hospitalization. The decision-making variables were the dependent variables in this model, which was computed as the average of the decision-making variable score for the psychotic and non-psychotic black patient vignettes. Similar averages were computed for the non-psychotic and psychotic white patient vignettes to create four parallel variables that were entered as control variables. Thus, the model (see Figure 11) was constructed to test for the effects of measured implicit and explicit racial biases on decision-making among black patients beyond the effects observed among the treatment of white patients (i.e., racial disparity). Given that the field of practice, specifically clinical or counseling psychology background, differed by assigned vignette condition, these variables were added to the model as additional covariates. Correlations of the model variables are presented in Table 5. The fit of the hypothesized model and the significance of the paths connecting the bias variables to the decision-making variables were examined. The maximum likelihood estimation procedure was used for data estimation. The identified model was a non-saturated model. A backward elimination method was used to improve model fit, which included removing non-significant paths (i.e., setting a path to zero) in the diagram and the adjusted model was compared to the initial model. Given that the distributions of the decision-making variables were non-normal, bootstrapping (500 iterations) was conducted to obtain bias-corrected standard errors for coefficients in the path model. Error terms were left uncorrelated.
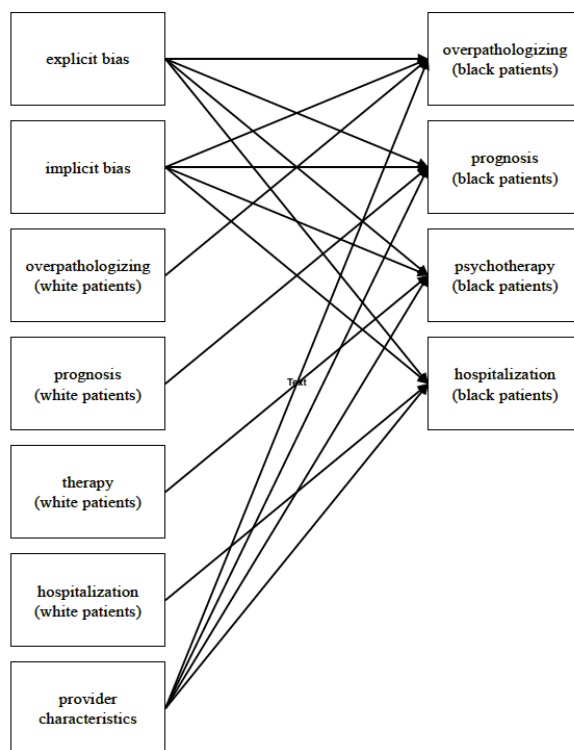
Figure 11. Hypothesized SEM model of implicit and explicit bias and decision-making outcomes in black patients.

Model fit was assessed using the chi-square statistic, root-mean-square error of approximation index (RMSEA), the comparative fit index (CFI), and the Tucker-Lewis index (TLI). The chi-square and RMSEA are indices of absolute fit, which are fundamental measures of how well the identified model fits the data using no comparison to a baseline model. The CFI and the TLI are indices of relative fit in which the proposed model is compared to a baseline model for which the null hypothesis is that all variables in the model are uncorrelated. Non-significant chi-square values are considered indicators of good model fit. RMSEA values of less than .08 are considered to be an acceptable fit, indicative of parsimony (Browne and Cudeck, 1993). CFI and TLI values above .90 indicate acceptable fit (Hu & Bentler, 1999).

All four goodness-of-fit indices indicated poor fit to the data, chi-square = 69.30 (df = 24, $p < .001$), RMSEA = .15, 95% CI [.11, .19], CFI = .64, TLI = .43. Removing non-significant paths did not improve the model enough to reach acceptable fit on any of the indices. Table 6 presents the standardized beta coefficients for all paths in the model, controlling for decision-making among white patients and field of practice. Significant paths are shown in Figure 12. Implicit and explicit bias scores were not significantly associated with any of the decision-making outcomes in blacks. There was a nonsignificant trend in the hypothesized direction of greater IAT scores (e.g., stronger pro-white bias) predicting worse prognoses given to black patients, standardized beta = -1.10, SE = .60, $p = .07$.
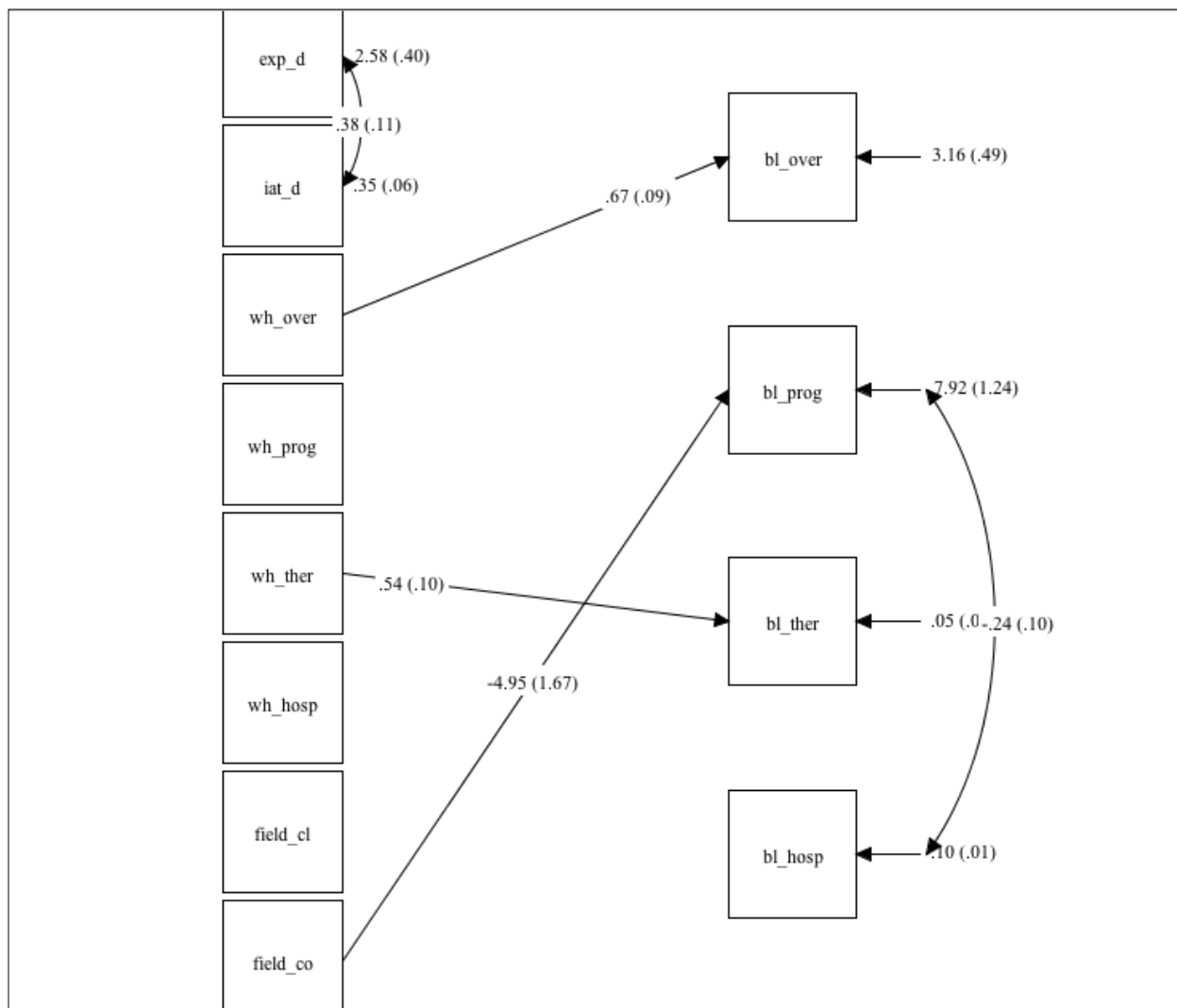
Figure 12. Structural equation model for provider sample with significant standardized coefficients between bias and decision-making outcomes in black patients, controlling for decision-making among white patients and field of practice. "Exp_d" = explicit racial bias difference score; "iat_d" = implicit racial bias d-score; "over" = overpathologizing; "prog" = prognosis; "ther" = therapy; "hosp" = hospitalization; "bl" = black patient vignettes; "wh" = white patient vignettes; "field_cl" = clinical psychologist; "field_co" = counseling psychologist.

Discussion

The objective of the current study was to determine whether racial bias indices explain racial disparities in decision-making among black and white patients with bipolar disorder (bipolar disorder). This was the first investigation in psychiatric research of the presence and effects of measured racial bias in a mental healthcare provider sample. Although previous work has documented profound racial disparities in the treatment and outcomes of bipolar disorder, mechanisms driving disparity in bipolar disorder had not been examined before now. The current study drew from well-validated and effective methods in social psychology for understanding unconscious prejudices, like the IAT, to empirically measure racial bias among mental healthcare providers. It also extended the literature on implicit bias from a focus on diagnostic inaccuracy to

two other key aspects of clinical decision-making: prognostic assessments and treatment decisions.

Only one of three hypotheses was supported. The aim 1 hypothesis, that providers would make more favorable decisions among white patients than black patients with comparable symptom presentations, and that this disparity would be exaggerated when psychosis was present, was not supported. Contrary to hypotheses, the main effects of race and the interactions of race and psychosis were not significant. Any of the significant effects of race or Race x Psychosis were qualified by the effect of assigned vignette condition. Evidence showed that providers in the two vignette conditions were not matched on implicit bias scores, despite random assignment, which could have produced vignette condition effects where one group treated patients differently than another because of racial bias. The effects observed in overpathologizing, prognosis, psychotherapy recommendation, and hospitalization, however, followed the pattern described in preliminary analyses as potentially confounded with the vignette severity mismatch. That is, providers overpathologized, diagnosed bipolar disorder, gave worse prognoses to, and made fewer psychotherapy recommendations and more hospitalizations with the more severe vignette out of the two non-psychotic vignettes and the psychotic vignettes, which may have been unrelated to the patients' race. Given that vignettes were not matched in severity, and that providers assigned to the two conditions were not matched in their level of implicit bias, it is not possible to tease apart the effects due to true racial biases and those due to severity of the vignettes. It cannot be concluded on the basis of the current findings that racial disparities were present in mental healthcare decision-making. Moreover, contrary to the aim 3 hypothesis, implicit and explicit racial biases were unrelated to decision-making among black patients across diagnosis, prognosis, and treatment domains examined.

The hypotheses of aim 2, that mental healthcare providers would show moderate to strong pro-white (anti-black) bias on the IAT and that implicit biases would be uncorrelated with explicit beliefs, was supported. Providers in the current sample showed moderate pro-white bias and neutral explicit beliefs on measures of racial bias. My hypothesis that participants who were older, male, Non-Latino, white, less educated, and exposed less to minorities and serious mental illnesses in their practices would show stronger pro-white biases was not supported. Slight to moderate pro-white implicit bias appeared universally present across a variety of demographic and background experience variables in this sample.

At the same time that implicit biases were observable and significant, providers expressed consciously egalitarian beliefs about black and white people. This dissonance between implicit and explicit racial attitudes has been characterized previously in racial bias studies as "aversive racism" (Abreu, 2001; Gaertner & Dovidio, 1986; Penner, Dovidio, West, Gaertner, & Albrecht, 2010). People with an aversive racism profile consider themselves to be non-prejudiced and consciously affirm racial equality, yet have unconscious negative attitudes about a particular race or ethnic group. The term "aversive" was chosen to capture the aversive reaction people with this profile have at the suggestion that they are racially biased. It is considered to be a "contemporary form of prejudice found among liberal and well-educated Whites" that still produces discrimination and disparity, but in subtle, indirect ways that are not threatening to the individual's belief that they are unbiased (Dovidio, Penner, et al., 2008). It has been theorized that aversive racism's is linked to discrimination and disparity by providers' racial anxieties. Researchers have suggested that white individuals with this profile experience anxiety and discomfort out of fear of inadvertently appearing racist, leading to avoidance of face-to-face interactions with a black person (Dovidio et al., 2008; West, Shelton, & Trail, 2009). One study

of racial anxiety among college students has supported the idea that anti-black implicit racial biases of white individuals are positively correlated with anxiety before interacting with a black person (Amodio & Hamilton, 2012). Several studies have shown that white individuals' performance on a response inhibition task (i.e., Stroop test) is impaired after interracial interactions, because they engage in self-regulation during the interaction (trying not to appear racist) that leads to the depletion of their executive attentional capacity (Engle, Conway, Tuholski, & Shisler, 1995; Muraven & Baumeister, 2000). This effect is stronger among individuals with high levels of racial bias (Richeson & Shelton, 2003; Richeson et al., 2003) and in interracial interactions with increased self-regulation demands (Richeson & Trawalter, 2005). Continued investigation of racial anxiety and increased self-regulation may, therefore, help clarify the underlying mechanisms for racial disparities in mental healthcare, particularly among serious mental illness populations where provider perceptions of patient unpredictability and potential for violence may fuel racial anxieties and exacerbate these dynamics.

As mentioned above, the aim 3 hypothesis that racial bias, particularly implicit racial biases, would explain racial disparities in decision-making was not supported by findings. Implicit and explicit racial biases were unrelated to decision-making among black patients across all outcomes examined in the SEM analyses. This may have been related to the heterogeneity of the sample, which mainly included clinical and counseling psychologists. Findings indicated, however, that racial bias profiles differed in these two fields; counseling psychologists in the current sample had neutral implicit and explicit profiles, and clinical psychologists had moderate pro-white implicit and neutral explicit profiles. Counseling psychology programs may provide more systematic training in cultural competency and bias reduction than clinical psychology programs. Due to a limited sample size of counseling psychologists in the current sample, however, differential analysis of the two subgroups could not be conducted. Further examination of mental healthcare providers by field of practice in future research could provide some insight into racial bias in treatment.

Limitations

There were several limitations of the current study. Patient vignettes were not matched in symptom severity despite pre-testing to control for this. Findings from analyses of the vignettes suggested that this mismatching was the underlying causes of apparent racial differences between black and white patient vignettes rather than true racial disparities. Vignette matching was important in the current study because of the interest in the interaction of race and psychosis, however, other vignette studies have limited their examination to the effects of race alone. The SEM analyses in the current study addressed this limitation to some degree by collapsing across psychotic and non-psychotic vignettes. Future research should focus on improving on the vignette paradigm and developing new paradigms that facilitate analyses of the intersection of race and other common factors in clinical presentations of serious mental illness.

It should also be noted that an absence of variability precluded gaining a true understanding of some outcome variables. That is, almost all providers indicated that they would make psychiatrist referrals and recommend medication. Perhaps as a result of the low base rate, no racial disparities were observed in referrals to psychiatrists and only marginal nonsignificant differences were present in medication recommendations for black versus white hypothetical patients. Five of seven decision-making variables were binary, which tend to have lower statistical power to detect effects.

Aside from the limited range on decision-making variables, other, more nuanced decision-making outcomes may be more sensitive to effects of racial biases. For instance, among physicians, large effects of implicit racial bias as measured using the IAT were found on verbal dominance, patient affect, patient ratings of interpersonal care, patient centeredness, speed and duration of talk-time (Cooper et al., 2012). Racial anxiety related to aversive racism in a provider may lead to poorer interpersonal interactions with racially dissimilar patients, like less patient-centeredness and less time spent with these patients. These outcome variables were continuous and included subjective and objective outcomes, which allowed for greater variability and increased sensitivity to racial bias. Future research should incorporate such outcomes into their investigations of the effects of racial bias in mental healthcare.

The current study only simulated mental healthcare decision-making, and thus could not approximate the complexity of interracial interactions between providers and patients. For example, time constraints and patient caseload might exasperate the effects of racial biases on decision-making. The length of time working with an individual patient might modulate the effects of racial bias on mental healthcare outcomes. One possibility for future research would be to ask providers to rate vignettes under rapid decision-making simulations to approximate time and workload constraints in the actual treatment setting. The current study does not address such factors, though they should be considered in future research of racial bias in mental healthcare.

Another limitation was that the provider sample was drawn from California. Given that California is relatively liberal compared to states in the southern and mid-western regions of the U.S., it is important to note that significant moderate pro-white (anti-black) racial bias were still observed; racial bias and its effects on mental healthcare outcomes may be stronger in states with longer and more pervasive histories of discrimination and racism against black people. It is also possible that, although bias may be stronger, providers in the latter states might hold consonant, rather than dissonant implicit and explicit pro-white (anti-black) attitudes about black people, as observed in the current sample. One study has suggested that nonverbal cues of discomfort and anxiety from a provider with an aversive racism profile cause more dissatisfaction among black patients than they experience with a provider who is racially biased, but holds non-anxious consonant attitudes about black and white people (Penner et al., 2010). Future research should examine racial bias in other regions of the U.S. and the world to characterize racial bias profiles and their links to clinical care.

Implications

Several suggestions have been proposed for future study of racial bias in mental healthcare, including the development of simulation paradigms and measures for decision-making and the incorporation of nuanced outcome measures that may be more sensitive to effects of race. In addition to this, some researchers have suggested that white providers' in-group bias, or favoring and putting in extra effort to help a patient who is racially similar to the provider, can be sufficient for creating racial disparity even if providers give standard care to black patients (Burgess, et al., 2004). Understanding whether in-group favoritism or bias directed against the out-group (racially dissimilar patients), or both, contributes to racial disparity may help to elucidate other models for mechanisms of a link between bias and racial disparity in mental healthcare.

If the link between provider racial bias and racial disparity can be demonstrated, it would pave the wave for the application of available bias-reducing, equity-promoting techniques that would counter racial biases among mental healthcare providers in real clinical settings. One

example is psychoeducation about the development and activation of implicit bias (Devine, Forscher, Austin, & Cox, 2012). The mere awareness of bias is this study was shown to lead to significant bias reductions on the IAT. Another is empathy-building, which encourages providers to take the perspective of minorities they are treating (Galinsky & Moskowitz, 2000). Physicians who participated in these interventions showed improvements in awareness about biases in clinical consultation and greater concern over discrimination experienced by their patients (Galinsky & Moskowitz, 2000).

Conclusions

In summary, findings did not support the core hypothesis about a link between racial biases and racial disparities in the mental healthcare of black and white patients with bipolar disorder. One possibility is that difficulties in the vignettes used, and with vignette approaches more generally, may have limited ability to find effects of racial bias on decision-making. Findings do, however, demonstrate the presence of implicit racial bias among mental healthcare providers despite consciously held egalitarian beliefs, a profile known as aversive racism. Aversive racism has been characterized in previous research as a contemporary form of racism that, though less obvious than the overt racism observed through American history, has damaging effects on interracial relationships and could potentially influence the trajectory of mental healthcare among interracial provider-patient dyads. Despite limitations, the current study is both novel and important as it provides the first direct measurement of racial bias in a mental healthcare provider sample. This evidence has implications for the design of mental healthcare professionals' training, particularly curricula related to issues of interracial relationships and addressing one's own biases in the treatment of racially dissimilar patients. Attention should be given to the curricula taught in counseling psychology programs in particular, as this group showed the lowest levels of bias in the current study.

Table 1

Characteristics of Provider Sample (N = 82)

Characteristic	n	% or M (SD)	IAT M (SD)[a]	Explicit Bias M (SD)[a]
Vignette Condition 1	40	48.8	.22 (.66)*	-.30 (1.90)
Vignette Condition 2	42	51.2	.52 (.50)**	.55 (1.17)**
Age (years)	82	43.3 (10.0)	--	--
Gender				
Female	55	67.1	.36 (.56)**	.07 (1.84)
Male	27	32.9	.38 (.62)**	.26 (1.02)
Ethnicity				
Hispanic/Latino	2	2.4	-.07 (.53)[d]	0 (.00)[d]
Non-Hispanic/Latino	80	97.6	.38 (.60)**	.14 (1.64)
Race				
Non-white[b]	16	19.5	.45 (.70)*	.31 (2.18)
White	66	80.5	.35 (.59)**	.09 (1.46)
Degree				
Master	14	17.1	.19 (.46)	0 (2.00)
Doctorate[c]	68	82.9	.41 (.62)**	.16 (1.54)

Field	n	%		
Clinical psychology	68	82.9	.44 (.59)**	.16 (1.53)
Counseling psychology	10	12.2	.03 (.62)	-.10 (2.42)
Other	4	4.9	.11 (.33)[d]	.25 (.50)[d]
Treatment Setting				
Private practice	51	62.2	.36 (.56)**	.06 (1.53)
Outpatient	21	25.6	.38 (.70)*	.24 (2.12)
Hospital	10	12.2	.43 (.61)[e]	.30 (.67)
Conducts psychotherapy	78	95.1	.35 (.60)**	.08 (1.63)
SMI Experience				
No experience	15	18.3	.48 (.68)*	.10 (1.68)
Depressive disorders	73	89.0	.38 (.60)**	.10 (1.68)
Bipolar disorders	42	51.2	.47 (.55)**	.07 (1.74)
Psychotic disorders	21	25.6	.52 (.49)**	.14 (.65)
Years of SMI experience	81	9.9 (9.1)	--	--
% Patients with SMI	82	31.1 (24.2)	--	--
% Patient caseload with bipolar disorder	81	10.7 (14.3)	--	--
% Patient caseload racial/ethnic minority	82	38.5 (26.3)	--	--

Note. SMI = serious mental illness.

[a]Implicit Association Test (IAT) and explicit scores: positive values represent pro-white bias and negative values represent pro-black bias.

[b]Non-white providers were Asian ($n = 13$), Black or African-American ($n = 1$), and Biracial/Multiracial ($n = 2$; Asian + Black/African-American and Asian + White).

[c]Doctorate degrees included Ph.D. ($n = 37$), M.D./Ph.D. ($n = 5$), and Psy.D. ($n = 26$).

[d]Statistical significance not reported for subsamples smaller than n = 10. IAT scores for these groups should be interpreted with caution.

[e]$p = .05$

*Values are significantly different from 0 at $p < .05$.

**Values are significantly different from 0 at $p < .01$.

Table 2

Pearson Correlations of Provider Characteristics with Implicit and Explicit Bias Scores (N = 82)

Provider Characteristics	IAT	Explicit Bias
Age (years)	-.02	.15
Years of SMI experience	-.13	-.06
% Patients with SMI	-.11	-.17
% Patient caseload with bipolar disorder	-.06	-.14
% Patient caseload racial/ethnic minority	-.01	-.18

Note. No correlations were significant at α = .05; SMI = serious mental illness.

Table 3

Means and Standard Deviations of Severity Ratings and Decision-Making Outcomes by Patient Vignette (N = 82 except where noted)

Outcome	Patient A M (SD)	Patient B M (SD)	Patient C M (SD)	Patient D M (SD)	Grand Mean M (SD)
Severity[c, d] (scale 0-7)	5.19 (.76)	4.75 (.70)	5.65 (.87)	6.13 (.75)	5.42 (.60)
Misdiagnosis[b, d] (scale 0-1)	.65 (.48)	.49 (.50)	.80 (.40)	.52 (.50)	.62 (.30)
Overpathologizing[c] (scale 1-109)	3.45 (3.06)	2.57 (2.18)	3.05 (2.74)	3.40 (2.63)	3.12 (2.28)
Prognosis[c, d] (scale 1-42)	23.10 (3.35)[a]	25.94 (4.61)	24.40 (3.40)	22.93 (3.57)	24.11 (2.72)
Psychiatrist (scale 0-1)	.99 (.11)[a]	.91 (.28)	1.00 (.00)	1.00 (.00)	.97 (.08)
Medication[b] (scale 0-1)	.94 (.24)[a]	.83 (.38)[a]	.99 (.11)	.96 (.19)	.93 (.14)
Psychotherapy[d] (scale 0-1)	.90 (.30)	.96 (.19)	.90 (.30)	.72 (.45)	.87 (.24)
Hospitalization[c, d] (scale 0-1)	.36 (.48)[a]	.11 (.32)	.62 (.49)	.90 (.30)	.50 (.26)

Note. Symptom severity scale: 1 = no symptoms; 2 = minimal; 3 = mild; 4 = moderate; 5 = moderately severe; 6 = severe; 7 = very severe.
[a]missing one observation
[b]significant difference between Patient A and Patient B vignettes at α = .05
[c]significant difference between Patient A and Patient B vignettes at α = .01
[4] significant difference between Patient C and Patient D vignettes at α = .05

Table 4

Correlations Between Decision-Making Outcome Variables (N = 82)

	Misdiagnosis	Overpathologizing	Prognosis	Psychiatrist	Medication	Psychotherapy
Overpathologizing	.178					
Prognosis	-.052	-.087				
Psychiatrist	.330**	.053	-.232*			
Medication	.405**	-.058	-.178	.573**		
Psychotherapy	-.166	.056	.154	-.039	-.127	
Hospitalization	.113	-.278*	-.288**	.195	.187	-.201

*Values are significantly different from 0 at $p < .05$.

**Values are significantly different from 0 at $p < .01$.

Table 5

Correlations Between Structural Equation Model Variables

	Overpath-ologizing (Black)	Prognosis (Black)	Therapy (Black)	Hospitali-zation (Black)	Explicit Bias	Implicit Bias	Overpath-ologizing (White)	Prognosis (White)	Therapy (White)	Hospitali-zation (White)	Clinical Psychol-ogist
Prognosis (Black)	-.043										
Therapy (Black)	.010	.142									
Hospitali-zation (Black)	-.129	-.257	-.218								
Explicit Bias	-.255	-.014	.054	.000							
Implicit Bias	-.148	-.133	-.006	.096	.393						
Overpath-ologizing (White)	.692	-.052	-.010	-.113	-.253	-.159					
Prognosis (White)	.090	-.114	.162	-.012	.014	-.162	.070				

Therapy (White)	.096	.067	.534	-.327	.068	.093	.084	-.046			
Hospitalization (White)	.093	-.146	.154	.002	.009	-.167	.072	.998	-.053		
Clinical Psychologist	-.004	.057	.025	.052	.038	.244	-.032	-.046	-.096	-.050	
Counseling Psychologist	.109	-.218	-.100	-.060	-.054	-.216	.121	.034	.108	.041	-.821

Note. "Black" = black patient vignettes; "white" = white patient vignettes.

Table 6

Standardized Coefficients With Bootstrapped Standard Errors for the Structural Equation Model

Path	Estimate	S.E.	Est./S.E.	P-Value
Overpathologizing (Black) on				
Implicit Bias	-0.065	0.370	-0.176	0.860
Explicit Bias	-0.127	0.136	-0.935	0.350
Overpathologizing (White)	0.671	0.085	7.857	0.000
Clinical Psychologist	0.860	0.930	0.924	0.356
Counseling Psychologist	0.982	1.066	0.921	0.357
Prognosis (Black) on				
Implicit Bias	-1.095	0.595	-1.838	0.066
Explicit Bias	0.106	0.212	0.501	0.616
Prognosis (White)	-0.009	0.006	-1.568	0.117
Clinical Psychologist	-2.732	1.462	-1.869	0.062
Counseling Psychologist	-4.950	1.667	-2.970	0.003
Therapy (Black) on				
Implicit Bias	-0.047	0.046	-1.023	0.306
Explicit Bias	0.008	0.017	0.481	0.631
Therapy (White)	0.543	0.097	5.571	0.000
Clinical Psychologist	-0.101	0.114	-0.884	0.377
Counseling Psychologist	-0.242	0.130	-1.854	0.064
Hospitalization (Black) on				
Implicit Bias	0.058	0.066	0.887	0.375
Explicit Bias	-0.009	0.023	-0.385	0.700
Hospitalization (White)	0.000	0.001	0.267	0.789

Clinical Psychologist	-0.011	0.161	-0.069	0.945
Counseling Psychologist	-0.048	0.184	-0.262	0.794
Implicit Bias with				
Explicit Bias	0.376	0.114	3.314	0.001
Prognosis (Black) with				
Overpathologizing (Black)	0.114	0.554	0.206	0.837
Therapy (Black) with				
Overpathologizing (Black)	0.003	0.043	0.071	0.943
Prognosis (Black)	0.053	0.071	0.750	0.453
Hospitalization (Black) with				
Overpathologizing (Black)	-0.043	0.062	-0.690	0.490
Prognosis (Black)	-0.240	0.100	-2.394	0.017
Therapy (Black)	-0.004	0.008	-0.470	0.638

Note. "Black" = black patient vignettes; "white" = white patient vignettes.

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Appendix A. Vignettes of hypothetical patients.

Non-Psychotic Mania Vignette A (Patient A)

A, a stock analyst and married father of two children in his 30s, is brought to the emergency room (ER) after 10 days of what his wife describes as "another dark period," marked by a hair-trigger temper. She notes that these dark periods have gone on as long as she has known him but that he has experienced at least a half dozen of them in the prior year. She says they typically improve within a month. She adds that she wonders whether alcohol worsens his symptoms, because he routinely ramps up its use after the dark periods begin.

A's wife says she decided to bring him to the ER after she discovered that he had recently created a blog entitled A's Best Stock Picks. Such an activity not only is out of character, but also, given his job as a stock analyst for a large investment bank, strictly against company policy and grounds for dismissal. She says that he has been working on these stock picks around the clock, forgoing his own meals, sleep, as well as his responsibilities at work and with his children. He counters that he is fine and that his blog would "make them as rich as Croesus." His wife disagrees and describes how, when his drive escalates, he becomes extremely arrogant and irritable. When she or the kids try to interrupt his work, he will yell that his family is not appreciating him or respond with biting sarcasm.

The patient had been diagnosed with depression in college, after the death of his father from suicide. His father had been a wildly erratic, alcohol-abusing businessman whom the patient loved very much. His paternal grandmother had several "nervous breakdowns."

On examination, the patient is pacing angrily in the exam room. He is dressed in jeans and an unbuttoned shirt. Upon the examiner's entrance, he sits and explains that this is all a miscommunication, that he is fine and needs to get home immediately to tend to his business. He is very difficult to interrupt. He denies hallucinations but admits, with a smile, to a unique ability to predict the stock market. He refuses cognitive testing, saying he would decline the opportunity to be a "trained seal, a guinea pig, Mr. Ed, and a barking dog, thank you very much, and may I leave now?"

Non-Psychotic Mania Vignette B (Patient B)

When B's wife finally convinces him to get a psychiatric evaluation, he is in his 30's and has been unemployed for several weeks. After this last week in which B stayed up most of each night fixing things around the house, his wife said that she would leave him if he did not see a doctor. The wife reports that they also had several fights this week in which B was verbally abusive, which is unusual for him.

B's troubles began seven months earlier when he was working as an insurance adjuster and had a few months of mild, intermittent anxiety, fatigue, insomnia, and loss of appetite. At the time, he attributed these symptoms to stress at work, and within a few months, he was back to his usual self. At that point, he worked feverishly to catch up on work he had let slide before. About four months ago, his effort seemed to pay off and he received a promotion at work. B has enjoyed the increase in energy and productivity he is having and does not think anything is wrong with him despite his wife's growing concern. He feels a surge in self-confidence and believes the promotion is proof that he is on top of things. With some pressing, he admits that, although he has been feeling great, there are times when everything seems to grate on him. That's when he would yell at his wife or, when he was working, snap at a co-worker for no apparent reason.

B lost his job about a month ago. He had used company funds to buy a high-priced stereo system and several new computers for the office, thinking this would help his coworkers be as productive as him. When his boss confronted him about the purchases, saying they were extravagant and unnecessary, B lost his temper and yelled several insults at his boss. His boss then fired him for "being belligerent and showing poor judgment."

B reports that he is not taking any medications. He is estranged from his relatives and is unaware of any family history of mental illness. Both B and his wife deny any drug use. An emergency room psychiatrist noted B to be a fast-talking man with no evidence of delusions or hallucinations. Results of B's physical examination and cognitive testing are unremarkable.

Psychotic Mania Vignette A (Patient C)

C, a man in his 30s, is brought to the emergency room (ER) by concerned friends. C is a graduate student in his second year at the English department of a top local university. He is doing well academically and enjoys a large circle of friends. In the midst of an uneventful period in the first semester, he began to feel depressed; experienced loss of appetite, with a weight loss of about 10 pounds; and had trouble falling asleep and waking up too early. He did not seek professional help at the time because he attributed his depression to the "usual graduate student experience."

Now in the second semester, these problems seem to have gone away. He reports feeling increasingly energetic and productive. He has stayed up through the night many times drafting papers that are not due for several months. About a month ago, his "best" ideas came at time when he didn't sleep for more than a week. He had "thousands of ideas" coming to him for paper topics that he felt were profound and could "shift paradigms" in his field. He stayed awake to take care to write each one down and keep notes on all of them.

In the last week, however, C has started to believe that comments on television shows are referring to his new ideas, suspecting that people on the shows have stolen them from him either by reading his thoughts or by radar. At times he also feels that thoughts from other people are intruding in his head via radar, controlling his thoughts or producing emotions of anger or euphoria that are beyond his control. He wonders if there is a hole in his head through which the messages are being sent to him. He describes once hearing voices, which spoke about him in the third person and ordered him to perform sexual acts.

The ER psychiatrist's notes state that C's friends describe him "as if he were high, talking so fast they could hardly understand him and behaving irritably for no apparent reason." C, however, denies any drug use.

C's father, when in his 40s, had had a severe episode of depression, characterized by hypersomnia, profound psychomotor retardation, and suicidal ideation.

Psychotic Mania Vignette B (Patient D)

D, who appears to be in his 30s, is brought to an emergency room (ER) by police. One officer says that D bribed them with sex while in their patrol car. D refers to himself as the "New Jesus" and declines to offer another name. He refuses to sit and instead runs through the ER. He is put into restraints and receives intramuscularly administered sedatives.

Despite being restrained, he remains giddily agitated, talking rapidly about receiving messages from God and having "an important role on Earth." When asked when he last slept, he says he no longer needs sleep, indicating that he had "been touched by Heaven." His speech is disorganized and difficult to understand. After an additional 45 minutes of agitation, he receives

another dose of a sedative. This calms him, but he still does not sleep. His restraints are removed.

D is disheveled and smells bad, though he does not smell of alcohol. He makes poor eye contact, instead looking at nearby patients, a ticking clock, the examiner, a nearby nurse—at anything or anyone that moves. His leg bounces rapidly up and down, but he does not get out of his chair. He describes his mood lately as "not bad." His affect is labile, often laughing for no particular reason but getting angry when he feels misunderstood. He denies having hallucinations. When asked the date, he responds with an extended discussion about the underlying meaning of the day's date, which he misses by a single day. He remembers the names of the two police officers who had brought him to the hospital. He refuses more cognitive testing and remains tense and jumpy.

D's sister arrives an hour later, after having been called by a neighbor who had seen her brother taken away by police. She says D had seemed strange a week earlier, uncharacteristically arguing with relatives at a holiday gathering. He had claimed not to need sleep at that time and had been talking about his "gifts," all of which seemed very unusual for him. She has tried to contact him since then, but he has not responded to phone, e-mail, or text messages. She says that D does not typically use drugs. She also says he is a middle school math teacher who had just finished a semester of teaching.

Schizophrenia Distractor Vignette (Patient E)

E, a man in his 30s, is brought to the emergency room (ER) by the campus police of the university from which he had been suspended several months earlier (E is a graduate student in physics). The police had been called by a professor who reported that E had walked into his classroom shouting, "I am the Joker, and I am looking for Batman." When E refused to leave the class, the professor contacted security.

Although E has had much academic success as a student, his behavior has become increasingly odd during the past year. He quit seeing his friends and spends most of his time lying in bed staring at the ceiling. He lives with several family members but rarely speaks to any of them. He has been suspended from the university because of lack of attendance. His sister, who is reached by phone, says that she has recurrently seen him mumbling quietly to himself and notes that he would sometimes, at night, stand on the roof of their home and wave his arms as if he were "conducting a symphony." Although his father and sister have tried to encourage him to see someone, E has never seen a psychiatrist and has no prior hospitalizations. E's sister says that she has never known him to use illicit substances or alcohol. When asked about drug use, E appears angry and does not answer.

The ER psychiatrist notes that E is a well-groomed young man who is generally uncooperative and appears "constricted, guarded, inattentive, and preoccupied." He becomes enraged when the ER staff bring him a snack. He loudly insists that all of the hospital's food is poisoned and that he will only drink a specific type of bottled water. He appears to be internally preoccupied, although he denies hallucinations. E states that his mood is "OK" and has no disturbance in his sleep or appetite. He is oriented and speaks articulately but refuses formal cognitive testing.

E's mother and grandmother, were both reportedly "crazy." E's mother had abandoned the family when E was young, and he and his sister were raised by his father.