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Evidence for Differential Predictive Performance of the Prime Screen between Black and White Help-Seeking Youths

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Abstract

Objective: Self-report screening instruments for emerging psychosis have the potential to improve early detection efforts by increasing the number of true-positives among those deemed to be at “clinical high-risk” for the disorder, but their practical utility depends on their validity across race. This study sought to examine whether a commonly-used self-report screening tool for psychosis-risk performs equally among black relative to white youths in its ability to predict risk status.

Method: Black ($n = 58$) and white ($n = 50$) help-seeking individuals ages 12–25 (61% female) were assessed with the Prime Screen and the Structured Interview for Psychosis-risk Syndromes (SIPS). A logistic regression model estimated race differences in the strength of relations between Prime Screen scores and SIPS-defined risk status.

Results: Higher Prime Screen scores significantly predicted risk status among white ($p < .01$) but not black ($p = .23$) participants. Self-reported prime screen scores among black youths at low risk more closely resembled scores of participants at high risk than scores of white peers who were also at low risk.

Conclusions: Results suggest that consideration of race or ethnicity and associated cultural factors is important when screening for clinical high-risk status. Findings support the need to develop culturally valid early psychosis screening tools to promote appropriately tailored early intervention efforts.

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Keywords

clinical high-risk; psychosis; screening instrument; racial disparities

Introduction

Individuals at “clinical high-risk” for psychosis are those experiencing recent attenuated psychotic syndromes or other indicators of susceptibility during adolescence or young adulthood, a key period of risk for first episode psychosis (1). As only 25% of these individuals develop a formal psychotic illness in the years after identification (2), false positive identification of psychosis-risk syndromes is a limitation of psychosis prevention efforts (3). Evidence suggests that recent trends toward recruiting participants from the general, non-help-seeking population contributes to these limitations (4). In conjunction with the developing consensus that high-risk syndromes warrant clinical attention regardless of eventual psychosis (due to frequently high levels of distress and impairment; [5]), these findings raise questions about the most appropriate ways to identify individuals on a path toward worsening prognosis. The use of brief, self-report screening instruments prior to clinical assessment referral may contribute to an efficient and cost-effective solution to this problem (6). Self-report screens can create a pretest signal indicating one’s probability of meeting high-risk criteria (7), and have strong validity in the prediction of subsequent psychosis (8).

Given that normative experiences and item interpretation can vary as a function of factors related to race, ethnicity, and culture, validation of instruments designed to capture psychological and behavioral abnormalities requires close examination of instruments’ performance across different racial and cultural backgrounds (9). Historically, many psychometric instruments lack sensitivity to important cultural factors (9–14), suggesting that the validity of psychosis-risk instruments may differ between members of ethnic majority and ethnic minority populations (10, 15). This can contribute to sociodemographic health disparities by limiting the benefits of screening, including early intervention, for racial minorities (16, 17).

Decades of research demonstrate that black individuals are more likely than white individuals to be misdiagnosed with schizophrenia (18–20), further compounding what may be an actual underlying disparity in prevalence rates (21, 22) and quality of treatment (23). Given the importance of early detection and intervention in curbing the burden of serious psychopathology (24, 25), these findings highlight the need to develop screening tools that can signal emerging psychosis among black, help-seeking youths, who may be both at risk of an eventual misdiagnosis of schizophrenia and, paradoxically, at risk of the onset of an actual (not misdiagnosed) psychotic disorder.

This study aimed to determine whether a commonly-used self-report pre-screen for clinical high-risk criteria, the Prime Screen, performs equivalently across black and white help-seeking youths. Building from literature demonstrating limited cultural sensitivity of many psychometric instruments (9, 10, 14), and extending previous work suggesting a strong predictive relation between Prime Screen scores and clinical high-risk status, we aimed to

determine whether the magnitude of these Prime-risk relations was weaker among black participants relative to white participants. To address the possibility that differences in self-rated symptoms could be due to group differences in levels of clinician-rated psychopathology, clinician bias, or disparities in socioeconomic status (26), we examined rates of high-risk diagnoses across race groups, the magnitude of relations between Prime Screen scores and clinician-rated positive symptoms, and whether family income accounted for any differential relation between Prime scores and risk status. In exploratory analyses, we examined the specific Prime Screen items that may contribute to any observed racial differences.

Method

Procedures

The study took place within the context of an ongoing longitudinal study of psychosis-risk, beginning in 2010. Participants and/or parents (if the child was < 18 years old) spoke by phone with a trained researcher who described study procedures and determined eligibility (see below). Visits took place in a private room within university clinics. Following informed consent, youths completed self-report measures alone while parents (when present) were interviewed regarding the youth's psychiatric history. Subsequently, participants completed psychiatric interviews with the researcher. The study was approved by the university institutional review boards.

Participants

Individuals ages 12–25 were recruited from community clinics, hospitals, schools, and private practitioners in Baltimore, MD. Additional inclusion criteria required only that participants were receiving mental health services at the time of enrollment. Participants were typically referred for mental health assessment and diagnosis due to suspected emerging psychosis or other psychiatric concerns (e.g., affective disorders). The participants could be divided into three categories: individuals at clinical high-risk; help-seeking controls; and individuals with diagnosable psychosis (e.g., schizophrenia). Given interest in the performance of the Prime Screen in predicting psychosis-risk among black relative to white youths, participants with psychosis ($N = 26$) or who were not black or white ($N = 27$) were excluded from analyses.

Measures

Race.—Race was reported by the participant or their parent using a demographics questionnaire derived from the National Institute of Health's definitions for racial and ethnic categories. The response item corresponding to black race was worded "Black or African American. A person having origins in any of the black racial groups of Africa. Terms such as 'Haitian' or 'Negro' can be used in addition to 'black or African American.'" The item corresponding to white race was worded "White. A person having origins in any of the original peoples of Europe, the Middle East, or North Africa." Racial subgroups were not identified.

Structured Interview for Psychosis-Risk Syndromes (SIPS).—The SIPS is a gold-standard semi-structured interview designed to identify and rate the severity of clinical high-risk syndromes (27). Although no study to our knowledge has directly evaluated the cross-cultural performance of the SIPS, a recent comprehensive review of its reliability and validity across the 31 countries in which it has been used found no evidence of differential performance by culture (28). To meet criteria for a risk syndrome, participants must have experienced either (1) 1 attenuated positive psychotic symptom occurring at least weekly, (2) an illness episode of psychotic-level intensity that is too brief to meet criteria for formal psychosis, and/or (3) a recent, 30% decline in functioning in the context of schizotypal personality disorder (SPD) or a family history of psychosis. Given evidence that risk of transition to psychosis among adolescents with SPD is comparable to the degree of risk among those meeting other SIPS criteria (e.g., 21% [29]), we included individuals with SPD but no family history or significant functional decline in the high-risk group. The SIPS items for unusual thought content, suspiciousness, grandiosity, perceptual abnormalities, and disorganized communication are rated on a 0–6 (absent–severe) scale and summed to create a measure of positive symptom severity. All SIPS raters (9 white, 1 Asian) were certified following an official two-day workshop and achieved excellent inter-rater agreement (ICC = .82 for positive symptoms; κ = 1 for diagnosis). Raters were blind to participants' Prime Screen scores.

Kiddie Schedule for Affective Disorders and Schizophrenia (KSADS).—We used the KSADS to characterize the sample clinically. The KSADS is a well-validated semi-structured diagnostic interview used to identify DSM diagnoses among youths (30). KSADS diagnoses are made based on separate interviews with the child and their parents. Training included expert instruction, rating of audio recordings, *in vivo* interview observation and co-rating, and supervised administration until diagnostic agreement with experienced raters was achieved for 3 participants and approval was given by the principal investigators.

The Prime Screen, Revised.—The Prime Screen is a 12-item self-report questionnaire developed by the SIPS authors as a brief way to estimate the probability of meeting clinical high-risk criteria (31). Items are rated on a 7-point scale, ranging from 0 to 6 (“definitely disagree; somewhat disagree; slightly disagree; not sure; slightly agree; somewhat agree; definitely agree”). Participants who endorse 2 items at a 5 or 6 are considered to screen positive. The sum of Prime-rated positive symptoms is strongly correlated with the sum of SIPS-rated positive symptoms (7) and has been shown to predict subsequent transition to psychosis in those at clinical high-risk (8). The average administration time of the instrument is 1 minute 40 seconds, and the Flesh-Kincaid reading-level estimate is 6:8. Cronbach's alpha in the present sample is .89.

Statistical Analyses

Preliminary analyses.—Prime Screen scores were computed by using each participant's frequency of items endorsed at a level of 5 or 6 (hereafter referred to as Prime Screen “cutoff” scores), consistent with author recommendations and with previous validity studies (7). Primary analyses were also conducted using the sum of raw Prime Screen scores; as the results were the same regardless of the scoring method, for simplicity they are not reported

here but are available upon request. Black and white participants were compared on the demographic and clinical variables displayed in Table 1 using chi square or *t* tests.

Primary analyses.—To examine whether a weaker relation between Prime Screen scores and risk status was present among black relative to white participants, a moderated logistic regression was performed in which dichotomous risk status was regressed on race, Prime Screen cutoff scores, and their crossproduct. In a second linear regression, SIPS positive symptoms were regressed on these same predictors. In the case of moderation, simple effects were computed to examine the effect of Prime Screen cutoff scores on the outcome variable at each level of race (32). Regression analyses were then repeated controlling for family income and demographic/clinical variables that differed significantly by race.

Exploratory analyses.—To explore which specific Prime Screen items were differentially related to risk status among black versus white participants, a 2×2 (race \times risk status) analysis of covariance (ANCOVA) was conducted for each of the 12 raw Prime Screen items. ANCOVAs examined mean item differences across risk groups within each race. Due to the exploratory nature of these analyses, no correction for multiple comparisons was applied. Finally, we computed sensitivity and specificity values for the entire sample and for the black and white groups separately.

Results

A total of 108 participants ($N = 43$ clinical high-risk, $N = 65$ help-seeking control) were included in the analyses, similar in size to several other psychosis-risk screening studies (6). Of these, $N = 58$ were black and $N = 50$ were white (Table 1). Prime Screen scores were missing for 8, the sum of SIPS positive symptoms scores was missing for 2, and family income data were missing for 11 individuals due to incomplete research procedures. Data were excluded pairwise per analysis. The continuous variables of interest displayed acceptable skew and kurtosis (i.e., < 2 [33]; Table 2). Black participants were on average younger than white participants ($t = 2.68$, $df = 106$, $p = .009$) and less likely to have a mood disorder (46.6% versus 68%; $\chi^2 = 5.03$, $df = 1$, $p = .025$). Age ($r = -.26$, $p = .008$) and mood disorder ($t = -2.18$, $df = 105$, $p = .032$) were related to Prime Screen cutoff scores and were considered covariates. The race groups did not differ on any of the other demographic or clinical variables.

As demonstrated in Table 1, the race groups did not significantly differ on rates of high-risk diagnoses or on the severity of Prime Screen cutoff scores or SIPS positive symptoms. Results from a moderated logistic regression, however, revealed a significant interaction between race and Prime Screen cutoff scores in the predicted probability of meeting high-risk criteria (Table 3). Simple effects analyses suggested that higher Prime Screen cutoff scores significantly increased the probability of meeting these criteria for white but not black participants. The effect remained significant when controlling for household income, age, and mood disorder (interaction: $b = -.51$, Wald $\chi^2 = 4.66$, $df = 1$, $p = .031$, $\text{Exp}[B] = .60$, 95% CI [.38, .96]; see also Table S1). When we included participants who met criteria for a formal psychotic disorder in the high-risk group (see Methods), the pattern of findings remained the same (Table S2).

Table 4 displays means and standard errors of individual Prime Screen items, plus results of 2×2 and within-race ANCOVAs comparing scores on each item across groups (with covariates). These analyses sought to determine which Prime Screen items account for the differential response pattern described above. Statistically significant race by risk status interactions were observed for six items (items 1, 2, 5, 6, 8, and 9). For black youths, mean differences between high-risk and help-seeking control groups were substantially smaller (items 1 and 5) or in the opposite direction (items 2, 6, 9, and 12) as was seen in white participants. For these latter items, black help-seeking controls reported numerically *higher* scores than black youths at risk.

Within-race contrasts explored risk-group differences on Prime Screen items separately among black and white participants (Table 4). White controls consistently scored lower than white youths at high-risk, whereas a mixed pattern of results was observed among black youths, with black controls frequently endorsing items at a level comparable to or even numerically greater than those at risk. In the combined sample, sensitivity and specificity of the Prime Screen were .43 and .90, respectively. Splitting by race, these values were .27 and .90 for the black group, and .61 and .90 for the white group.

A linear regression predicting the sum of positive symptoms from race, Prime Screen cutoff scores, and their crossproduct revealed no significant interaction, suggesting that the relation between participant-rated Prime Screen scores and clinician-rated positive symptom severity (irrespective of risk status; Table 2) was roughly equal across black and white participants.

Discussion

We found that the Prime Screen, a frequently used self-report assessment of clinical high-risk criteria, did not reliably distinguish black help-seeking youths who were at risk for psychosis from those who were not, even though it did distinguish these groups among white participants. The findings were not explained by differences in income, age, mood disorder, rates of clinical high-risk diagnosis, or clinician-rated symptom severity. Item-level analyses suggested that that most items displayed differential performance across race, suggesting a relatively widespread versus item-specific effect.

A long history suggests many psychometric instruments do not perform equivalently across cultures (9–14). Instruments may not measure the same constructs across racial/ethnic groups, may use language that conveys different meaning across these groups, and/or may concern constructs that are more familiar to some groups than others (34). Questionnaires may be inherently subject to certain of these limitations. The Prime Screen, for example, was designed to convey risk-level experiences by adding contingency words (e.g., “I *think* that I have *felt*...” [italics added]), a convention that may have differentially influenced responses across race. Questionnaires also may restrict the opportunity to provide important contextual information associated with endorsements, such as the degree of associated distress or impairment. By contrast, diagnostic interviews allow clinicians to use age- and culturally-appropriate language and to clarify the circumstances surrounding endorsements. Addition of a “distress scale” to the Prime Screen, as is included in a similar measure (the Prodromal Questionnaire – Brief; PQ-B), may partly address this issue.

We found that Prime Screen cutoff scores among black help-seeking controls more closely resembled scores of participants at clinical high-risk than scores of white help-seeking controls. Notably, the frequency of high-risk diagnoses and the severity of clinician-rated positive symptoms did not differ between racial groups. These findings are important as they suggest that black youths in our sample appear highly symptomatic when considering only their self-reported Prime Screen scores. Following a structured interview administered by a trained diagnostician, however, it appears that black and white participants in this sample do not differ on their clinical level of psychosis-risk. Given the history of misdiagnosis of schizophrenia in black individuals (18, 19), reduced access to health screening and quality treatment (16, 17), and generally high levels of discrimination and risk factors for psychosis to which people of color are often exposed (22, 35, 36), these findings highlight the need to carefully consider the most appropriate referral and treatment options for black youths who, based on these and other findings, are at increased risk for inappropriate referral, diagnosis, and intervention.

An alternative explanation for our results is that Prime Screen ratings are the more accurate measure of psychosis-risk among the present black controls, but the SIPS clinicians did not accurately rate these symptoms, potentially due to limitations of the SIPS or cultural differences between participants and majority white clinicians. This is unlikely, however, as (1) all clinician-measured indices of psychopathology were either equal or lower among black relative to white participants, including SIPS-rated positive symptoms, rates of high-risk diagnosis, and DSM diagnoses; (2) formal psychotic disorders are frequently over-diagnosed in black individuals, in contrast with the roughly equal rates of high-risk diagnoses we observed; and (3) clinicians were blind to participants' Prime Screen scores during assessment. Therefore, our results point to the screen as the primary source of inaccuracy in assessment.

Two general population studies recently found evidence of measurement invariance across multiple racial/ethnic groups for the PQ-B, another tool designed for psychosis-risk screening (37, 38). Although these results may appear to contrast with ours, a critical distinction between these studies and ours is that only our study assessed participants with both a screening instrument and the gold-standard SIPS. Notably, black and white participants in our pooled sample did not differ on their Prime Screen cutoff scores; only when the clinician-rated risk status was considered did a differential response pattern emerge. Given that we observed such a pattern for nearly all Prime Screen items, our results suggest that this instrument may not capture the same constructs across racial or cultural populations. The field would benefit from studies incorporating measurement invariance analysis of multiple psychosis-risk screening instruments with direct comparisons against gold-standard assessments.

A strength of our study is its use of a clinical control group to assess the performance of the Prime Screen, a screening tool used in real-world clinical settings. Help-seeking controls are optimal comparators in studies like ours because these individuals are more clinically representative than healthy controls of the population for which the instrument was designed (39, 40). Nonetheless, high-risk participants in our sample tended to have more DSM diagnoses than help-seeking controls, suggesting greater overall illness severity. Although

specificity estimates of the Prime Screen were excellent and our main findings held when adjusting for racial differences in mood disorder, because black participants on average presented with fewer DSM diagnoses than white participants, it remains possible that general illness severity contributed to the differential performance of the Prime Screen.

Limitations and Future Directions

With federal funding for clinical high-risk intervention programs, large-scale dissemination of screening tools is underway. Findings from the present study may inform these efforts, but our relatively small sample may not generalize to larger programs with more inclusive recruitment strategies or broader sociodemographic ranges. Our requirement that participants had already contacted a mental healthcare provider, for example, likely distinguishes our sample from individuals whose initial psychosis-risk assessment may be their first lifetime contact with services; is also possible that referral patterns were differentially distributed across clinical or racial groups in our study. As screening thresholds may vary by help-seeking status (41) and referral source (42), identifying interactions between idiographic factors such as these may advance early identification efforts.

It is important to consider that self-reported race is only a crude proxy for numerous cultural, historical, geographic, and socioeconomic factors (among many others [43]) that may influence a person's mental health status or response to questionnaires. Community studies designed to carefully measure these factors would allow researchers to tease apart their relative influences on psychosis-risk screening in ways that our study could not; they may also have enhanced ability to detect influences on racial/ethnic cultural subgroups (e.g., of specific Caribbean, African descent or European descent). A valuable approach may be to develop a maximally and cross-culturally effective screening tool based on combinations of items from previously validated psychosis-risk questionnaires. Qualitative interviews with respondents of varying backgrounds may help to promote development of novel screening items.

Conclusions

Mental health screening is a critical juncture in pathways to care. The potentially inadequate performance of psychosis-risk screens among black youths may represent a rupture at this junction, further compounding racial disparities in access to accurate diagnosis and treatment. Greater attention to cultural and contextual influences on clinical assessment may foster more accurate diagnosis and early, targeted intervention.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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References

1. McGorry PD, Yung AR, Phillips LJ: The “close-in” or ultra high-risk model: a safe and effective strategy for research and clinical intervention in prepsychotic mental disorder. *Schizophrenia bulletin* 29:771–90, 2003 [PubMed: 14989414]
2. Fusar-Poli P, Cappucciati M, Rutigliano G, et al.: At risk or not at risk? A meta- analysis of the prognostic accuracy of psychometric interviews for psychosis prediction. *World Psychiatry* 14:322–32, 2015 [PubMed: 26407788]
3. Wiltink S, Velthorst E, Nelson B, et al.: Declining transition rates to psychosis: the contribution of potential changes in referral pathways to an ultra-high-risk service. *Early intervention in psychiatry* 9:200–6, 2015 [PubMed: 24224963]
4. Fusar-Poli P, Schultze-Lutter F, Cappucciati M, et al.: The dark side of the moon: meta-analytical impact of recruitment strategies on risk enrichment in the clinical high risk state for psychosis. *Schizophrenia bulletin* 42:732–43, 2016 [PubMed: 26591006]
5. Carpenter WT, Schiffman J: Diagnostic concepts in the context of clinical high risk/attenuated psychosis syndrome. *Schizophr Bull* 41:1001–2, 2015 [PubMed: 26163478]
6. Kline E, Schiffman J: Psychosis risk screening: a systematic review. *Schizophr Res* 158:11–8, 2014 [PubMed: 25034762]
7. Kline E, Wilson C, Ereshefsky S, et al.: Psychosis risk screening in youth: a validation study of three self-report measures of attenuated psychosis symptoms. *Schizophr Res* 141:72–7, 2012 [PubMed: 22921375]
8. Kline E, Thompson E, Demro C, et al.: Longitudinal validation of psychosis risk screening tools. *Schizophr Res* 165:116–22, 2015 [PubMed: 25975827]
9. Sternberg RJ: Cultural explorations of human intelligence around the world. *Online Readings in Psychology and Culture* 4, 2002
10. Helms JE: Why is there no study of cultural equivalence in standardized cognitive ability testing? *Am Psychol* 47:1083, 1992
11. Neighbors HW, Trierweiler SJ, Munday C, et al.: Psychiatric diagnosis of african americans: diagnostic divergence in clinician-structured and semistructured interviewing conditions. *J Natl Med Assoc* 91:601–12, 1999 [PubMed: 10641496]
12. Sternberg RJ: Culture and intelligence. *Am Psychol* 59:325, 2004 [PubMed: 15511120]
13. Strakowski S, McElroy S, Keck P, et al.: Racial influence on diagnosis in psychotic mania. *Biol Psychiatry* 39:157–62, 1995
14. Suzuki LA, Valencia RR: Race–ethnicity and measured intelligence: educational implications. *Am Psychol* 52:1103, 1997
15. Groth-Marnat G: *Handbook of Psychological Assessment* Oxford, England
16. Kenik J, Jean-Jacques M, Feinglass J: Explaining racial and ethnic disparities in cholesterol screening. *Preventive medicine* 65:65–9, 2014 [PubMed: 24806331]
17. Wallace DAC, Baltrus PT, Wallace TC, et al.: Black white disparities in receiving a physician recommendation for colorectal cancer screening and reasons for not undergoing screening. *J Health Care Poor Underserved* 24:1115, 2013 [PubMed: 23974385]
18. Olbert CM, Nagendra A, Buck B: Meta-analysis of black vs. white racial disparity in schizophrenia diagnosis in the united states: do structured assessments attenuate racial disparities? *J Abnorm Psychol* 127:104, 2018 [PubMed: 29094963]
19. Schwartz RC, Blankenship DM: Racial disparities in psychotic disorder diagnosis: a review of empirical literature. *World J Psychiatry* 4:133, 2014 [PubMed: 25540728]
20. Strakowski SM, Keck PE, Arnold LM, et al.: Ethnicity and diagnosis in patients with affective disorders. *J Clin Psychiatry* 64:747–57, 2003 [PubMed: 12934973]
21. Bresnahan M, Begg MD, Brown A, et al.: Race and risk of schizophrenia in a US birth cohort: another example of health disparity? *Int J Epidemiol* 36:751–8, 2007 [PubMed: 17440031]
22. van Os J, Kenis G, Rutten BP: The environment and schizophrenia. *Nature* 468:203, 2010 [PubMed: 21068828]

23. Kuno E, Rothbard AB: Racial disparities in antipsychotic prescription patterns for patients with schizophrenia. *Am J Psychiatry* 159:567–72, 2002 [PubMed: 11925294]
24. McGorry PD: Early intervention in psychosis: obvious, effective, overdue. *J Nerv Ment Dis* 203:310, 2015 [PubMed: 25919380]
25. Nieman DH, McGorry PD: Detection and treatment of at-risk mental state for developing a first psychosis: making up the balance. *Lancet Psychiatry* 2:825–34, 2015 [PubMed: 26360901]
26. Wicks S, Hjern A, Gunnell D, et al.: Social adversity in childhood and the risk of developing psychosis: a national cohort study. *Am J Psychiatry* 162:1652–7, 2005 [PubMed: 16135624]
27. McGlashan T, Walsh B, Woods S: *The Psychosis-Risk Syndrome: Handbook for Diagnosis and Follow-Up* New York, NY: Oxford University Press, 2010
28. Woods SW, Walsh B, Powers AR III, et al.: Reliability, validity, epidemiology, and cultural variation of the Structured Interview for Psychosis-risk Syndromes (SIPS) and the Scale Of Psychosis-risk Symptoms (SOPS). ; in *International Handbook of Attenuated Psychosis Syndrome in Youth and Young Adults: Early Identification and Intervention Across Cultures* Edited by Li H, Shapiro D, Seidman LJ. New York: Springer, In press
29. Woods SW, Addington J, Cadenhead KS, et al.: Validity of the prodromal risk syndrome for first psychosis: findings from the north american prodrome longitudinal study. *Schizophr Bull* 35:894–908, 2009 [PubMed: 19386578]
30. Kaufman J, Birmaher B, Brent D, et al.: Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): initial reliability and validity data. *J Am Acad Child Adolesc Psychiatry* 36:980–8, 1997 [PubMed: 9204677]
31. Miller T: The SIPS-screen: a brief self-report screen to detect the schizophrenia prodrome. *Schizophr Res* 70:78, 2004
32. Aiken LS, West SG, Reno RR: *Multiple Regression: Testing and Interpreting Interactions* Thousand Oaks, CA: Sage, 1991
33. Curran PJ, West SG, Finch JF: The robustness of test statistics to nonnormality and specification error in confirmatory factor analysis. *Psychol Methods* 1:16–29, 1996
34. Lonner WJ: Psychological tests and intercultural counseling. *Counseling across cultures*:275–303, 1981
35. Smedley A, Smedley BD: Race as biology is fiction, racism as a social problem is real: Anthropological and historical perspectives on the social construction of race. *Am Psychol* 60:16, 2005 [PubMed: 15641918]
36. Williams DR, Mohammed SA, Leavell J, et al.: Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. *Ann N Y Acad Sci* 1186:69–101, 2010 [PubMed: 20201869]
37. Karcher NR, Barch DM, Avenevoli S, et al.: Assessment of the Prodromal Questionnaire–Brief Child Version for Measurement of Self-reported Psychoticlike Experiences in Childhood. *JAMA psychiatry* 75:853–61, 2018 [PubMed: 29874361]
38. Cicero DC, Krieg A, Martin EA: Measurement invariance of the Prodromal Questionnaire–Brief among white, Asian, Hispanic, and multiracial populations Assessment:1073191116687391, 2017
39. Youngstrom E, Meyers O, Youngstrom JK, et al.: Comparing the effects of sampling designs on the diagnostic accuracy of eight promising screening algorithms for pediatric bipolar disorder. *Biological Psychiatry* 60:1013–9, 2006 [PubMed: 17056395]
40. Millman ZB, Gold JM, Mittal VA, et al.: The critical need for help-seeking controls in clinical high-risk research. *Clinical Psychological Science*, In press
41. Savill M, D’ambrosio J, Cannon TD, et al.: Psychosis risk screening in different populations using the Prodromal Questionnaire: a systematic review. *Early intervention in psychiatry* 12:3–14, 2018
42. Millman ZB, Schiffman J: False positives and clinical heterogeneity among youth at clinical high-risk for psychosis: Clinical and ethical implications for assessment and treatment. *Journal of Ethics and Mental Health* 10, 2018
43. Helms JE, Jernigan M, Mascher J: The meaning of race in psychology and how to change it: A methodological perspective. *American Psychologist* 60:27, 2005 [PubMed: 15641919]

Highlights:

- The Prime Screen self-report measure of psychosis-risk syndromes significantly predicted clinician-established risk status for participants who were white.
- The Prime Screen did not significantly predict clinician-established psychosis risk status for participants who were black.
- Consideration of individual participant characteristics is important when considering results from screening tools designed to detect psychosis risk.
- Intervention efforts for early psychosis will be augmented by the development of culturally valid psychosis-risk screening tools.

Table 1.

Demographic and Clinical Characteristics Across Race and Risk Status

	Black				White			
	Clinical High-Risk		Help-Seeking Control		Clinical High-Risk		Help-Seeking Control	
	N	%	N	%	N	%	N	%
Number of Participants	24	41.4	34	58.6	19	38	31	62
Female	16	27.6	19	32.8	14	28	17	34
Annual Family Income								
< 20,000	8	13.8	14	24.1	2	4.0	3	6.0
20,000–39,999	7	12.1	9	15.5	3	6.0	2	4.0
40,000–79,999	3	5.2	5	8.6	4	8.0	10	20.0
> 80,000	4	6.9	2	3.4	8	16.0	13	26.0
DSM Diagnoses ¹								
Mood Disorder	15	25.9	12	20.7	15	30.0	19	38
Anxiety Disorder	12	20.6	11	19	16	32	19	38
PTSD	6	10.3	7	12.1	6	12.0	6	12.0
ADHD	10	17.2	17	29.3	10	20.0	15	30.0
Substance Use	1	1.7	0	.0	2	4.0	5	10.0
No Diagnosis	0	.0	5	8.6	0	.0	1	2.0
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age	14.88	2.01	15.75	3.14	16.70	2.87	16.97	3.12
SIPS Positive	12.46	4.74	4.87	3.42	12.16	3.22	5.13	2.85
Prime Cutoff	2.95	2.77	2.09	2.39	3.50	2.81	.79	1.59
Prime Raw	29.00	17.42	25.23	14.72	33.56	17.26	12.52	14.70

Percentages reflect the proportion of individuals in that race group. Due to small cell sizes, annual family income is presented in 4 categories. For primary analyses involving family income, however, this variable was coded in 6 levels (< 20,000; 20,000 – 39,999; 40,000 – 59,999; 60,000 – 79,999; 80,000 – 99,000; > 100,000). For annual family income, N = 97, for SIPS Positive symptoms, N = 106, for Prime variables, N = 100; otherwise, N = 108. For SIPS positive symptoms, scores range from 0–30. For Prime Cutoff, scores range from 0–12. For Prime Raw, scores range from 0–72. For all three of these measures, higher scores indicate more severe positive symptoms. DSM = diagnostic and statistical manual of mental disorders, PTSD = posttraumatic stress disorder, SIPS Positive = structured clinical interview for psychosis-risk syndromes, positive symptom domain.

¹More than one diagnosis was common, percentages therefore exceed 100%.

Table 2.

Correlation Coefficients and Normality Estimates for Primary Study Variables

	1	2	3	4	Mean	SD	Skew	Kurtosis
1. Risk Status	-							
2. Race	.03	-						
3. SIPS Positive	.72**	.03	-		7.97	5.10	.51	-.32
4. Prime Cutoff	.34**	.12	.53***	-	2.16	2.54	1.20	-.62

For SIPS Positive, scores range from 0–30. For Prime Cutoff, scores range from 0–12. For both, higher scores indicate more severe symptoms. SIPS Positive = structured interview for psychosis-risk syndromes, positive symptom domain.

**
p < .01

p < .001

Table 3.

Logistic Regression Analysis with Simple Effects Predicting Risk Status from Race, Prime Screen Cutoff Scores, and their Interaction

	b	s_b	Wald χ^2	p	Exp(B)	95% CI
Model predicting risk status from Prime Screen cutoff scores						
Race	-.10	.47	.05	.82	.90	[.36, 2.25]
Prime Cutoff	.34	.11	10.14	.00	1.41	[1.14, 1.74]
Race \times Prime Cutoff	-.44	.22	4.03	.05	.62	[.42, .99]
Simple effects of Prime Screen cutoff scores on predicted probability of meeting high-risk criteria, at levels of race						
Black	.13	.11	1.43	.23	1.14	[.92, 1.42]
White	.58	.19	9.16	.00	1.78	[1.23, 2.59]

df = 1. Model terms are centered at zero. CI = confidence interval.

Table 4.

Results of Exploratory 2×2 ANCOVAs Examining Mean Differences in Raw Prime Screen Scores Across Race and Risk Status, and ANCOVAs Examining Mean Differences Within Race Groups.

	Black				White				Race × Risk Interaction					
	Clinical High-Risk	Help-Seeking Control	SE	Mean	F	η^2	SE	Mean	SE	η^2	F	η^2		
1. I think that I have felt that there are odd or unusual things going on that I can't explain.	3.03	.49	2.79	.42	.13	.00	3.55	.51	1.55	.39	9.22**	.204.74*	4.74*	.06
2. I think that I might be able to predict the future.	1.05	.53	2.26	.45	2.98	.07	1.67	.47	.70	.36	2.52	.06 4.93*	4.93*	.06
3. I may have felt that there could possibly be something interrupting or controlling my thoughts, feelings, or actions.	2.13	.50	2.20	.42	.01	.00	2.27	.49	1.14	.38	3.16	.08	1.96	.02
4. I have had the experience of doing something differently because of my superstitions.	2.06	.49	2.29	.42	.13	.00	2.81	.52	1.31	.40	4.96*	.12	3.01	.04
5. I think that I may get confused at times whether something I experience or perceive may be real or may just be part of my imagination or dreams.	3.77	.48	3.36	.41	.42	.01	4.12	.53	1.54	.41	14.25**	.28	5.83*	.07
6. I have thought that it might be possible that other people can read my mind, or that I can read other's minds.	.79	.43	1.53	.37	1.64	.04	1.50	.47	.69	.36	1.79	.05	4.57*	.05
7. I wonder if people may be planning to hurt me or even may be about to hurt me.	3.23	.51	1.82	.44	4.51*	.10	2.63	.46	1.54	.36	3.33	.08	.03	.00
8. I believe that I have special natural or supernatural gifts beyond my talents and natural strengths.	1.17	.53	2.43	.45	3.10	.07	1.31	.40	.62	.31	1.77	.05	3.59	.04
9. I think I might feel like my mind is "playing tricks" on me.	2.19	.50	2.75	.43	.70	.02	3.61	.49	1.36	.38	12.32**	.25	8.64**	.10
10. I have had the experience of hearing faint or clear sounds of people or a person mumbling or talking when there is no one near me.														

	Black					White					Race × Risk Interaction			
	Clinical High-Risk		Help-Seeking Control			Clinical High-Risk		Help-Seeking Control			F		η^2	
	Mean	SE	Mean	SE	F	η^2	Mean	SE	Mean	SE	F		η^2	
11. I think that I may hear my own thoughts being said out loud.	2.83	.50	2.20	.43	.87	.02	4.18	.56	1.46	.43	14.28**	3.47	.28	.04
12. I have been concerned that I might be “going crazy.”	2.01	.50	2.31	.43	.12	.00	2.52	.48	.91	.37	6.75*	3.56	.15	.04
	2.01	.53	2.03	.46	.00	.00	3.27	.46	1.22	.36	11.54**	4.32*	.24	.05

For within-race contrasts, $df = 1, 42$ for the white group, and $df = 1, 47$ for the black group. Levene's test estimated unequal variances at the population level for items 4 and 10 for the black group, and items 2, 6, and 11 for the white group. For interactions, $df = 1, 89$, and F statistic and η^2 represent the interaction of race (black vs. white) and risk status (clinical high-risk vs. help-seeking control). Analyses control for family income, age, and mood disorder. Raw Prime Screen scores range from 0–72, with higher scores indicating more severe symptoms. ANCOVAs = analyses of covariance.

* $p < .05$

** $p < .01$