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Racial discrimination as a mediator of racial disparities in insomnia disorder

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ABSTRACT

Study Objectives: Racial and ethnic minorities are more likely to suffer from insomnia that is more severe; however, few studies have examined mechanisms by which racial disparities in severity of insomnia disorder may arise. One potential mechanism for disparities in insomnia severity is perceived discrimination. This study tested discrimination as a mediator in the relationship between race and insomnia.

Methods: Participants were recruited from communities in the Detroit metropolitan area and were diagnosed with insomnia disorder using the DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition). The final sample included 1,458 individuals. Insomnia symptom severity was assessed via the Insomnia Severity Index and self-reported racial discrimination was evaluated using a single item. Racial discrimination was tested as a mediator in the relationship between race and insomnia symptom severity. Individuals were categroized as either White or a racial minority (i.e., non White individuals), with sensitivity analyses examining Black individuals and non-Black racial minority groups.

Results: Consistent with our hypothesis, racial discrimination was a significant mediator accounting for 57.3% of the relationship between race and insomnia symptom severity. Sensitivity analyses indicated that the indirect effect of racial discrimination was stronger in the non-Black racial minority group compared to Black individuals.

Conclusions: These results provide support that racial discrimination is likely an important mechanism by which racial and ethnic sleep disparities exist. Implications for prevention, intervention, and treatment of insomnia in racial minorities to reduce health disparities are discussed.

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Introduction

Among several of the fundamental and longstanding societal challenges we face is the problem of racial disparities in health. An overwhelming body of evidence indicates that living as a racial minority in the United States is frequently accompanied by excessive rates of chronic disease (e.g., hypertension, diabetes, and obesity)[1] and disproportionate mortality across the most prevalent diseases.[2–4]

Unsurprisingly, racial sleep disparities also exist. A multitude of evidence indicates that racial minority groups are more likely to report sleep disturbances compared to White individuals.[5–10] For example, Black individuals report having worse sleep quality,[10] more nonrestorative sleep,[6] and more restless sleep[11] compared to White individuals.[5,8] Indeed, a study of urban primary care patients found that the odds of reporting clinically significant sleep disturbance (based on the Pittsburgh Sleep Quality Index) was 3 times higher in Black patients than White patients.[12] Findings of insufficient sleep have also been documented in Asian Americans[13] and Native Americans.[14]

With adequate severity and chronicity, these sleep disturbances can develop into insomnia disorder. While emergent evidence for racial disparities in rates of insomnia disorder is still inconclusive,[15] extant studies suggest that when insomnia disorder occurs in racial minorities it is often more severe compared to their White counterparts.[16–18] For example, relative to White individuals, Black individuals have a 67% increased risk of insomnia disorder with short sleep,[17] which is the most severe phenotype.[19] A large prospective study also found that Black individuals were 2 times more likely to exhibit

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chronic insomnia (>1 year) compared to White individuals.[20] Another study of insomnia of army soldiers predeployment found that rates of moderate to severe insomnia symptoms were most prevalent in Native Americans compare to other racial groups.[21]

While it is important to describe racial sleep disparities, it is equally important to examine potential mechanisms driving such disparities.[22] Without established mechanisms of these disparities, race could be conflated as a risk factor as opposed to a risk *marker*. The increased risk of adverse health in racial minority groups not caused by their race; instead, race serves as a proxy for the various mechanisms by which these disparities arise. For example, racial minorities face differential exposure to stressors, barriers to education and health literacy, and limited access to health care, all of which increases risk for poor health. This issue has been at the center of ongoing discourse in the field of sleep health,[23,24] particularly because the distinction has important implications for how intervention approaches might mitigate these disparities.

One important potential mechanism for disparities in insomnia is racial discrimination.[22] Racial minorities are disproportionately impacted by discrimination based on actual or perceived membership of a particular racial group.[7,25] Although discrimination does occur in acute and distinct incidents, it also commonly exists in more subtle and "everyday" forms (e.g., microaggressions),[26,27] and thus can function as a chronic stressor. Like other stressors, perceived discrimination can trigger and exacerbate insomnia,[28] potentially by increasing vigilance against threat and triggering ruminative cognitions that result in difficulties falling and staying asleep.[29] Indeed, studies find that exposure to racial or ethnic discrimination is associated with self-reported insufficient sleep[30] and poorer sleep quality,[31] as well as complaints of insomnia.[32,33] However, few studies have tested discrimination as a mechanism of racial disparities in insomnia disorder. To date, only one study conducted in a sample of 133 first and second year college students demonstrated that perceived discrimination was a mediator of sleep difficulties measured using a subset of the subscales of the Pittsburgh Sleep Quality Index modeled as a latent variable.[34]

Though research in racial sleep disparities has typically focused on sleep quantity, it is important that this work extends into clinical contexts such as insomnia disorder. First, insomnia disorder rises to a level of severity that not only impairs functioning, but also significantly increases risk for other health complications. For example, insomnia has been implicated as a risk factor for obesity,[35] cardiovascular disease,[36] diabetes,[37] and depression[38,39] (see[35] for review), all of which also show disparities by race.[41] Indeed, as a robust risk factor, disparities in insomnia is likely a fundamental component in the etiology of health disparities.[36] Second, while differences in sleep characteristics (e.g., sleep duration) have been described by race, their association with adverse health consequences has been underexplored with mixed evidence[42–44]; it may be that characteristics of poor sleep is most predictive of poor health in a clinical context.[17,19,45] Finally, the chronicity and severity of insomnia disorder are stronger motivators for treatment seeking behaviors, which present opportunities for interventions to treat insomnia and prevent the cascade of morbidities leading to health disparities.

This present study aimed to examine racial discrimination as a candidate mechanism in the relationship between race and insomnia in a large sample of individuals with DSM-5 insomnia recruited from communities in the greater Detroit metropolitan area. Perceived discrimination was tested as a mediator of the racial differences of insomnia symptoms severity as measured by the Insomnia Severity Index. We hypothesized that differences in insomnia severity between racial minority groups and White individuals would be significantly mediated by perceived discrimination.

Participants and methods

Participants

Data for this study was obtained from communities in Detroit metropolitan area and the surrounding communities in southeastern Michigan. Recruitment included primary care patients from 6 hospitals, 38 medical centers, and subscribers of a major health insurance company in Michigan. Recruitment materials targeted individuals with sleep difficulties who would be interested in completing a behavioral insomnia intervention delivered via the internet, and data for this study were drawn from the screening questionnaires. All study materials were delivered in English. Eligible participants met DSM-5[46] diagnostic criteria for chronic insomnia disorder and were at least 18 years old. Exclusion criteria included diagnosed sleep disorders other than insomnia (e.g., restless legs, narcolepsy) or untreated obstructive sleep apnea, severe depression, bipolar or seizure disorders. A total of 1,458 individuals were included in the final analysis, after removing 28 individuals who declined to report their race or reported their race as unknown (see Fig. 1 for enrollment chart). Experimental protocols were approved by the local Institutional Review Board, and all participants provided informed consent for study participation.

Variables of interest

The dependent variable was insomnia severity measured using the Insomnia Severity Index (ISI).[47,48] The ISI comprises of seven components, with the first 3 items quantifying disturbance by sleep onset, maintenance, and early morning awakenings. The latter 4 queries sleep satisfaction and impact of sleep disorders on quality of life. Higher scores on the ISI indicate increased insomnia severity (range 0–28). The independent variable was race per self-report in

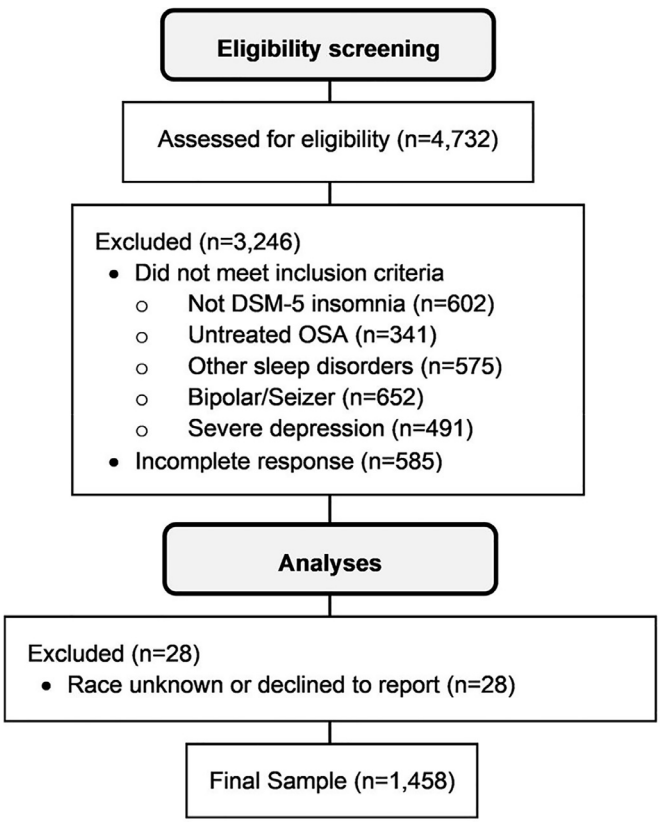


Fig. 1. Study enrollment flowchart.

the following categories: White, Black or African American, Asian, American Indian/Alaska Native, More than one race, Unknown (or do not wish to report). Analyses compared non-White to White indviiduals, with sensitivity analyses further comparing White to Black individuals and White individuals to non-Black racial minority groups (see Analytical Approach below). Though racial discrimination is commonly experienced across races, discrimination may manifest differently between racial groups. As such, analyses powered to disaggregate racial groups may reduce masking or distortion of these associations. In all models, race was included as a categorical variable with White as the reference group.

The mediator was racial discrimination as assessed via a single-item measure from the Commonwealth Fund 2001 Health Care Quality Survey[49] and the 2003 California Health Interview Survey.[50] These are validated and population-based surveys that are widely cited in academic research,[51–53] and used in briefings for Congress and other governmental agencies to increase equity in health care policies. The prompt was "*Thinking about your race or ethnicity, how often have you felt treated badly or unfairly because of your race or ethnicity.*", and responses included "Never" (0), "Rarely" (1), "Sometimes" (2), "A lot" (3), and "All the time" (4). The construct validity of this measure has been demonstrated using health outcomes known to be associated with other measures of racism.[51,53,54]

All final models covaried for age, sex, and socioeconomic status (annual household income and education) due to their established relationship with insomnia and racial discrimination as variables of interest. Medical comorbidities and body mass index were tested as covariates during model building but were removed in the final analyses due to nonsignificance across all models. Annual household income was operationalized as a categorical variable with four ordered levels: very low, low, middle, and high. The lowest category of household income was operationalized as an annual household income less than 15k, which is consistent with the poverty threshold for a 2-person household in 2016.[55] The cutoffs for low, middle, and high income were <35k, <75k, and ≥75k, respectively. Education was similarly operationalized with four ordered levels: high school or less, some college, college, and graduate school. These categories correspond to the International Standard Classification of Education[56] levels 3 or below, 4 and 5, 6, and 7 or higher.

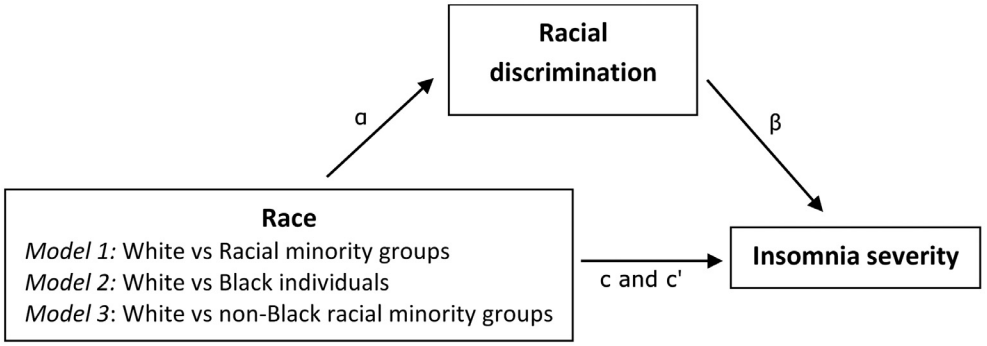
Analytical approach

The hypothesis was tested using mediation analyses conducted in accordance with procedures outlined by Fairchild and MacKinnon,[57] with significance testing of the indirect effect using the distribution of the product approach (i.e., the PRODCLIN method) implemented in R.[58] This method is less vulnerable to Type I errors compared to traditional significance tests,[59] and does not assume a normal distribution, which allows for asymmetric confidence intervals,[60,61] Statistical significance was determined if the 95% CI for the indirect effect did not include zero. The parameters required for the PRODCLIN approach (pathways α, β, c, and c') were determined via three ordinary least squares regression models with covariates (see Fig. 2). The direct effect (pathway c) was obtained by regressing race on insomnia severity controlling for covariates. Pathway α was obtained by regressing race on racial discrimination controlling for covariates. The remaining parameters were obtained in a third regression that tested the effect of the mediator (racial discrimination) on the outcome variable (insomnia severity) controlling for the predictor (race; pathway β) and covariates, and the effect of the predictor (race) after controlling for the mediator (racial discrimination; pathway c') and covariates. The indirect (i.e., mediated) effect of the predictor on the outcome variable was tested using the product of the α and β parameter estimates. Sensitivity analyses were also conducted to examine the specific effects within racial groups. The proportion of the mediated effect was calculated using a ratio of the indirect effect to the total effect.

In order to account for potential differences by racial groups, the analytical approach included sensitivity analyses parsed by racial minority groups. A power analysis indicated that a minimum sample size of 78 in each racial minority group was needed to achieve 0.8 power to detect statistical significance. As such, we were able to include one sensitivity analysis that compared the specific effect of Black (n = 271) compared to White individuals, and an option of a second analysis that compared the aggregated effect of non-Black racial minority groups to White individuals. In building the latter model, exploratory analyses were run by each non-Black racial group (Asian American, American Indian/Alaska Native, More than one race), and groups that demonstrated similar results were aggregated to achieve adequate statistical power. This approach optimized statistical power while minimized the chance of masking intergroup differences.

Results

The final sample included 1,458 individuals with insomnia disorder as defined by the DSM-5. Of the total sample, 74.2% were White (n = 1082) and 25.8% were a racial minority (Black: n = 271, Asian-American: n = 33, American Indian/Alaska Native: n = 14, more than



$$c: \quad Y_{Insomnia} = \beta_0 + \underline{\beta_1 Race} + \beta_2 Covariate_1 + \ldots \beta_n Covariate_n + \varepsilon$$

$$\alpha: \quad Y_{Discrimination} = \beta_0 + \underline{\beta_1 Race} + \beta_2 Covariate_1 + \ldots \beta_n Covariate_n + \varepsilon$$

$$\beta \text{ and } c': \quad Y_{Insomnia} = \beta_0 + \underline{\beta_1 Race} + \underline{\beta_2 Discrimination} + \beta_3 Covariate_1 + \ldots \beta_n Covariate_n + \varepsilon$$

Fig. 2. Schematic of the mediation approach.

Table 1
Sample characteristics by race

	White	Black	Asian	AI/AN	Multiracial
N	1082	271	33	14	58
Sex (F)	77.6%	84.1%	69.7%	71.4%	79.3%
Age	42.2 ± 16.3	43.5 ± 14.4	38.3 ± 13.8	37.8 ± 13.5	32.2 ± 16.2
Hispanic/Latinx	5.9%	1.1%	3.0%	28.6%	37.9%
Income					
Very low	17.3%	19.9%	12.1%	35.7%	31.0%
Low	35.6%	30.6%	18.8%	28.6%	34.5%
Middle	22.8%	33.6%	36.4%	28.6%	25.9%
High	24.3%	15.9%	33.3%	7.1%	8.6%
Education					
≤ High school	24.7%	17.7%	12.1%	42.9%	36.2%
Some college	43.4%	46.1%	18.2%	42.9%	44.8%
College	16.9%	18.8%	30.3%	7.14%	10.3%
Graduate school	15.0%	17.3%	39.4%	7.14%	8.6%
ISI	17.3 ± 4.2	18.1 ± 4.3	17.5 ± 4.7	17.1 ± 5.8	17.9 ± 4.2
Discrimination	0.4 ± 0.7	1.5 ± 1.1	1.6 ± 1.1	1.2 ± 1.0	1.3 ± 1.2

AI/AN = American Indian or Alaska Native.

one race: n = 58). The sample was predominantly female (racial minority: 81.6%, White: 77.6%), with a mean age of 41.9 ± 15.9 SD (racial minority: 41.1 ± 14.6 SD, White: 42.2 ± 16.3 SD). The mean ISI score of the sample was 17.4 ± 4.27 SD, indicating moderate insomnia severity. As expected, racial minorities reported significantly more frequent discrimination compared to White individuals (see Table 1). As a group, insomnia symptoms were more severe in racial minorities (t [1456] = 2.62, P< .01), though the effect (Cohen's d = 0.16) was small, likely because the sample comprised those meeting criteria for a diagnosis of insomnia. Individuals who reported higher levels of racial discrimination also reported more severe insomnia (t [1456] = 3.29, P = .001); however, the effect size of racial discrimination was over twice the effect size of race (Cohen's d = 0.37) (see Fig. 3). An exploratory post-hoc multivariate regression analysis with the components of the ISI found that racial discrimination was associated with higher severity across all components except for difficulty staying asleep and sleep satisfaction.

Mediation analyses

The first regression revealed a significant direct effect where beloning to a racial minority group was associated with higher ISI scores (pathway c: B = 0.14 ± 0.06 SE, P < .05). Educational attainment was the only covariate that reached statistical significance, with higher education associated with lower insomnia severity (B = −0.15 ± 0.07 SE, P < .05). As expected, beloning to a racial minority group was also associated with higher reported discrimination (pathway α: B = 1.09 ± 0.05 SE, P < .001). Finally, while racial discrimination remained a significant predictor of insomnia severity after accounting for race (pathway β: B = 0.07 ± 0.03 SE, P < .05), racial minority status was no longer significantly associated with

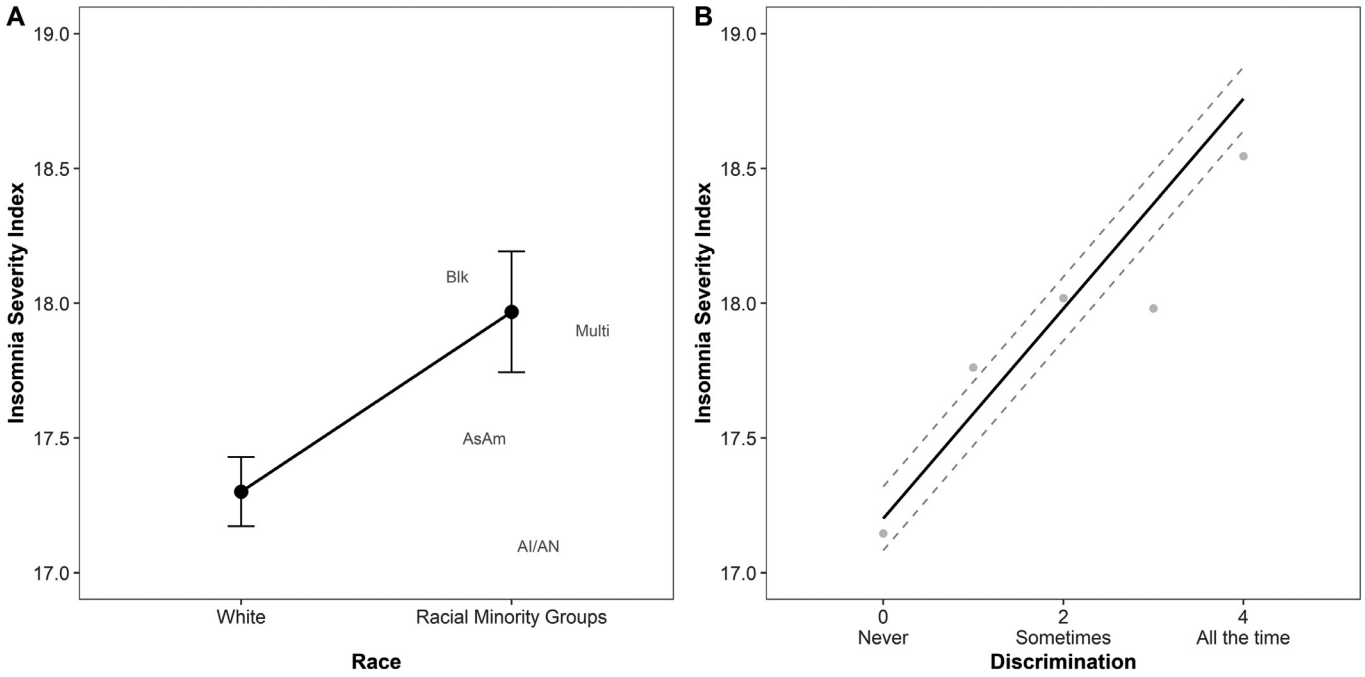


Fig. 3. Panel A) Comparison of insomnia severity by White versus non-White individuals. Insomnia values for specific racial groups in the non-White category indicated with the following labels: Blk = Black ; Multi = More than one race; AsAm = Asian American; AI/AN = American Indian/Alaska Native. Panel B) Insomnia severity by discrimination. Error bars indicate one standard error.

higher ISI scores (pathway c': B = 0.06 ± 0.07 SE, P = .439). The 95% confidence interval (CI) of the indirect effect did not overlap with zero ($\alpha \times \beta$: 0.08, 95% CI [0.01, 0.15]), and the indirect effect accounted for 57.3% of the relationship between race and insomnia severity.

Black individuals versus White individuals

A significant direct effect indicated that Black individuals reported more severe insomnia symptoms compared to White individuals (pathway c: B = 0.17 ± 0.07 SE, $P < .05$). Educational attainment was the only covariate that reached statistical significance, with higher education associated with less insomnia severity (B = −0.15 ± 0.07 SE, $P < .05$). Being Black was also associated with higher reported discrimination (pathway α: B = 1.18 ± 0.06 SE, $P < .001$). Finally, racial discrimination remained a significant predictor of insomnia severity even after adjusting for race (pathway β: B = 0.09 ± 0.03 SE, $P < .01$), but being Black was no longer a statistically significant predictor of insomnia severity after adjusting for racial discrimination (pathway c': B = 0.07 ± 0.08 SE, P = .39). The 95% CI of the indirect effect did not overlap with zero ($\alpha \times \beta$: 0.10, 95% CI [0.03, 0.18]), and the indirect effect accounted for 60.8% of the relationship between race and insomnia severity in Black and White participants.

Non-Black racial minority groups versus White individuals

Exploratory analyses indicated that individuals who identified as Asian American and multiracial exhibited similar patterns of results, and thus were aggregated in the final analysis. Results did not show a significant direct effect of race on insomnia severity for Asian American or multiracial individuals compared to White individuals (pathway c: B = 0.11 ± 0.11 SE, P = .31); however, being Asian American or multiracial was significantly associated with more reported discrimination compared to White individuals (pathway α: B = 1.18 ± 0.10 SE, $P < .001$). Educational attainment was the only covariate that reached statistical significance, with higher education associated with less insomnia severity (B = −0.24 ± 0.07 SE, $P < .001$). As testing the significance of an indirect effect does not require a direct effect,[62–65] we continued to test for a significant indirect pathway from race through discrimination. Racial discrimination remained a significant predictor of insomnia severity even after adjusting for race (pathway β: B = 0.08 ± 0.03 SE, $P < .05$), but the relationship between race and insomnia severity was close to zero after accounting for racial discrimination (pathway c': B = 0.02 ± 0.12 SE, P = .98). The 95% CI of the indirect effect did not overlap with zero ($\alpha \times \beta$: 0.09, 95% CI [0.02, 0.17]), and the indirect effect accounted for 84.0% of the relationship between race and insomnia.

In contrast, exploratory analyses in American Indian/Alaska Native individuals (n = 14) revealed a small and non-significant decrease in insomnia symptom severity compared to White individuals (B = −0.08 ± 0.28 SE, P = .75), when controlling for covariates. As was the case with the other models, educational attainment was the only covariate that reached statistical significance, with higher education associated with less insomnia severity (B = −0.25 ± 0.08 SE, $P < .01$). After accounting for racial discrimination, the relationship between race and insomnia severity remained nonsignificant, though the coefficient increased in strength (B = −0.17 ± 0.27 SE, P = .52).

Discussion

This study examined the role of racial discrimination as a potential mechanism for racial sleep disparities in a large clinical sample comprising White and racial minority groups with DSM-5 insomnia. Results supported our hypothesis that racial discrimination was indeed a significant mediator in the relationship between race and insomnia severity. In general, we found that racial discrimination explained almost 60% of the differences in insomnia severity between White individuals and racial minority gorups after accounting for covariates, including socioeconomic status (i.e. income and education). Indeed, the effect size of the difference in insomnia severity by racial discrimination was twice that of the difference by race. These results are consistent with prior research implicating racial discrimination as a contributor to sleep disturbances in the general population,[32,33,66] and extends these findings to racial disparities in insomnia disorder.

Insomnia is likely an important part of the landscape of health disparities because it may serve as a fundamental pathway to health disparities.[36] Insomnia exacerbates stress and potentiates risk for multiple morbidities, including but not limited to depression, anxiety, suicide, substance use disorders, impaired immune functioning, cardiovascular diseases, and chronic pain (see[40,67] for review). Though racial minorities experience an enhanced baseline risk for these morbidities, insomnia may facilitate their progression,[36] particularly as insomnia may manifest more intensely or as more severe phenotypes in racial minorities.[16–18] Furthermore, the consequences of insomnia are often higher for those already disenfranchised.[17,18] For example, whereas absenteeism and diminished work productivity are commonly associated with insomnia,[68] those with the least access to social capital and economic resources are most vulnerable to the cascading consequences such as lost wages or employment termination.[69]

Insomnia is also important in the landscape of health disparities because it represents an opportunity for both intervention and prevention.[16,70] In contrast to many other risk factors for health disparities (e.g., socioeconomic status, residential segregation, and food insecurity) insomnia is highly modifiable via behavioral intervention.[71,72] In fact, our research using Cognitive Behavioral Therapy (delivered digitally for increased accessibility) demonstrated comparable efficacy between Black and White individuals[16] despite historically established differences in treatment engagement and adherence in other forms of psychotherapy.[73,74] Furthermore, we have also demonstrated that early intervention of insomnia can prevent incident depression.[70] Together, these data suggest that interventions that target and/or mitigate the impact of racial discrimination on insomnia may play an important role in reducing health disparities.

Though racial discrimination has been well-documented as a predictor of poor health, mental health, and coping,[31] few studies have examined the role of racial discrimination in the development of insomnia. In the 3-P etiological model of insomnia,[28] stress is the most common precipitating factor of sleep disturbances. Indeed, evidence from large epidemiological studies have found that psychosocial stress—including racial discrimination—was associated with less sleep, worse sleep quality, and incidence of insomnia symptoms.[33,75] Another study in older Black women also found that racial discrimination was associated with worse insomnia symptoms.[32] However, the evolution from acute sleep perturbations to insomnia disorder is understood to be perpetuated by common compensatory behaviors that inadvertently exacerbate and maintain insomnia symptoms (e.g., daytime napping, extending time in bed, etc.), such that the insomnia disorder persists even if the precipitating stressor dissipates. Accordingly, precipitating stressors (e.g., racial discrimination) may not be a robust predictor of symptom severity in the context of a clinical disorder. Consistent with this model, the direct association between the frequency of racial discrimination and insomnia severity in this clinical sample of DSM-5 insomnia was small, albeit stronger than the association between race and insomnia severity. Importantly, racial discrimination is also a chronic or repetitive stressor, and thus may also contribute to the maintenance of insomnia symptoms. These results add additional evidence for racial discrimination as a mechanism driving insomnia in racial minorities and lend support that racial disparities in insomnia are likely a consequence of social inequities (e.g., discrimination) and other determinants of health.

Interestingly, sensitivity analyses indicated that the indirect effect of racial discrimination was stronger in Asian American or multiracial individuals relative to Black individuals. While the results from this study do not speak to why racial discrimination might play a differential role between racial minority groups, it is unlikely that racial discrimination is a less potent mediator of insomnia severity in Black individuals. Black individuals have a long and complex history of social, political, and economic disenfranchisement; notably, racism—particularly anti-Black racism has expanded beyond overt forms of discrimination (e.g., accepted use of anti-Black slurs, overt racial segregation) to more insidious and covert forms of discrimination (e.g., racial gerrymandering enacted via partisan gerrymandering,[76] mortgage redlining,[77] discriminatory hiring practices[78,79]) that relate to social determinants of health (e.g., neighborhood and physical environmental conditions, education and health literacy, access to resources, food, and health care, etc.). However, these covert forms of discrimination may not be adequately captured on an instrument that elicits more proximal experiences of maltreatment due to race. On the other hand, overt forms of discrimination against non-Black minorities have persisted (e.g., yellowface in entertainment industry,[80] overt anti-Chinese rhetoric and violence during the COVID-19 pandemic,[81] Native American mascots,[82] etc.). Future research should test whether such differences between racial groups are replicated in larger samples.

Limitations

Importantly, we had limited statistical power for a more fine-grained analysis that parses non-Black racial minority groups and that further examines differences by ethnicity. However, because of the paucity of research in non-Black racial minorities, we opted to include sensitivity analyses that aggregated non-Black racial minority groups that exhibited similar trends; this allowed us to achieve adequate statistical power while minimizing the risk of masking intergroup differences. Regrettably, there was insufficient power to parse analyses by both race and ethnicity. The underrepresentation of non-Black racial minorities in research samples is likely because investigators largely have not set intentional recruitment goals for non-Black racial minorities,[83] and the minority of investigators that have made a priori minority recruitment goals often fall short of those goals.[83] These data imply that research questions (and thus study and recruitment designs) have not focused on these minority groups, perhaps in part because there is little extant research to build from. As such, we also opted to include these sensitivity analyses so they may be considered in the generation of hypotheses for additional research, which should aim to replicate these findings in larger samples and to further examine the complexities and nuances in the mechanisms by which sleep disparities arise within different racial and ethnic minority groups.

The present study used a cross-sectional design and is therefore limited in the determination of temporal precedence. However, our results are consistent with prior research indicating that racial discrimination was a significant mediator of the prospective relationship between race and sleep disturbances in a sample of college students,[34] and extend this finding to clinically significant insomnia. Additionally, interpretation and generalization of results should take into consideration racial differences in help seeking[84,85] as the sample comprised individuals interested in receiving a behavioral intervention for insomnia. A common limitation of health disparities research is that many instruments have been developed and validated in predominantly White samples. That said, there is some evidence that the ISI shows cross cultural and racial invariance.[86,87] Another limitation may be the use of a single item that broadly measured the frequency of racial discrimination. Though commonly used in large epidemiological studies, the use of more complex instruments may capture nuances and dimensions of racial discrimination.

Conclusions

Overall, this study implicates racial discrimination as an important mechanism driving racial disparities in symptom severity in insomnia disorder, and adds novel information regarding potential differences in the role of racial discrimination between racial minority groups. Future research should replicate and extend these findings with a prospective design using more comprehensive instruments measuring racial discrimination along with additional relevant predictors in a sample that better represents the diverse racial groups. The potential for treatment and prevention of insomnia in racial minorities to reduce health disparities should also be explored.

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