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ORIGINAL PAPER

Testing a somatization hypothesis to explain the Black–White depression paradox

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Abstract

Purpose Epidemiologic studies document a lower prevalence of major depression in Blacks than Whites in the United States. This is paradoxical from the perspective of social stress theory. A long-standing claim in the (clinical) literature is that Blacks express depression more somatically than Whites. If true, the diagnostic algorithm may undercount depression in Blacks, since the screening symptoms privilege the psychological rather than somatic dimensions of depression. We test hypotheses that (1) Blacks express depression more somatically than Whites which (2) reduces their likelihood of endorsing screening symptoms, thereby undercounting Blacks' depression and explaining the Black–White depression paradox.

Methods We use cross-sectional data collected in 1991–92 from the National Longitudinal Alcohol Epidemiologic Survey ($n=42{,}862$) among Blacks and Whites endorsing at least one past-12-month depression symptom. We compare groups on depression somatization and test whether greater somatization in Blacks leads to lower endorsement of psychological screening symptoms, and therefore under-diagnosis.

Results Blacks have higher mean depression somatization scores than Whites (0.28, SE 0.04 vs. 0.15, SE 0.02), $t(122)=-2.15$, $p=0.03$. This difference is small and driven by Blacks' higher endorsement of 1 somatic symptom (weight/appetite change) and Whites' greater propensity to endorse psychological symptoms. However, Blacks have the same odds as Whites of endorsing screening symptoms, before and after adjusting for somatization.

Conclusions We find minimal evidence that Blacks express depression more somatically than Whites. Furthermore, this small difference does not appear to inhibit endorsement of diagnostic depression screening symptoms among Blacks, and therefore does not resolve the Black–White depression paradox.

Keywords Major depressive disorder · Race · Somatization · United States

Introduction

Psychiatric epidemiology studies in the US consistently document a paradox of a lower prevalence of depression in non-Hispanic Blacks compared with non-Hispanic Whites (hereafter Blacks and Whites) [1–6]. These findings are paradoxical in view of the social stress paradigm, the dominant framework for understanding relationships between social position and both mental and physical health [7–11]. The paradigm posits worse health in disadvantaged social groups due to greater stressor exposure and less access to coping resources [3, 12–16]. Black–White comparisons are a strong test [17] of this prediction in the US context, given Blacks' persistent disadvantaged social, political, and economic status vis-à-vis Whites [18–21]. Notably, Black–White comparisons on physical health outcomes are consistent with these predictions [3, 22, 23].

A potential explanation for this paradox is a purported Black–White difference in the expression of depressive symptomology and resulting diagnostic bias. Depression comprises both psychological and somatic dimensions [24–29]. Of the Diagnostic and Statistical Manual for Mental Disorder's (DSM) nine diagnostic symptoms, five can be considered psychological or affective (depressed mood, anhedonia, guilt or feelings of worthlessness, poor

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concentration, and suicidality) and four somatic (weight or appetite change, sleep problems, psychomotor agitation or retardation, and fatique or loss of energy) [29, 30]. Because the DSM requires endorsing either sad mood or anhedonia for a depression diagnosis, the DSM's diagnostic algorithm for major depression privileges psychological expressions of depression over more somatic expressions. Those with relatively more somatic expressions of depression are less likely to screen into the full diagnostic interview, potentially leading to under-diagnosis in this group. Clinicians and others have long suggested that some cultural groups express depression more somatically than others [26, 31–36], including Blacks compared with Whites in the US [37–40]. For example, according to one academic psychiatrist's account dating back to 1974, "The denial of natural impulses and feeling, forced on blacks by racism, has created in them those symptoms that may not be representative of the typical white depressed patient. Instead, neurotic depressions are frequently manifested through somatic complaints" (p. 99) [38]. More recently, a systematic review of barriers to depression treatment among African Americans [40] concluded: "These studies indicate that African Americans face a number of barriers in the recognition and treatment of major depression including clinical presentation with somatization…" (p. 30). Although such claims may risk overgeneralizing, positing a Black–White difference in how depression is expressed is consistent with the notion that cultural group differences may arise as a function of different structural positions across generations, in this case involving sharply divergent historical treatment and access to power in the U.S. [41, 42].

If Blacks do express depression more somatically than Whites, then, all else being equal, depression may be disproportionately undercounted among Blacks in the psychiatric epidemiology studies documenting the Black–White depression paradox. Previous examinations [27, 43, 44] of group differences in endorsement patterns across depression symptoms, using data from the studies documenting the Black–White paradox, have not found meaningful Black–White differences. However, in these studies, information on all depression symptoms was only available for those who had previously endorsed in the diagnostic interview at least one psychological screening symptom. Group differences in the tendency to express depression either more psychologically or somatically may, therefore, be muted in these samples. However, one large epidemiologic study in the U.S.—the National Longitudinal Alcohol Epidemiologic Survey (NLAES)—inquired about all nine depression symptoms in the diagnostic interview, regardless of screener endorsement. Because all study participants provided information on all nine diagnostic symptoms for depression in the NLAES, we are able to test if, controlling for somatic health, Blacks are more likely than Whites to endorse somatic symptoms in a sample not conditioned on endorsing a psychological screening symptom. We can further test if a more somatic expression of depression suppresses Blacks' relative likelihood of endorsing a screening symptom. Accordingly, the hypotheses we test in the NLAES sample are: (1) Blacks express depression more somatically than Whites; (2) Blacks have lower odds than Whites of endorsing a depression screening symptom; and (3) greater depression somatization in Blacks than Whites explains Blacks' lower likelihood of endorsing a screening symptom. To the degree our results support these hypotheses, we will have specified a mechanism to explain at least a portion of the Black–White depression paradox.

Methods

Study samples and designs

NLAES was conducted cross-sectionally in 1991–1992 using a nationally representative household sample ($n = 42{,}862$) of English-speaking adults 18 and older in the 48 contiguous states and the District of Columbia. Blacks and persons aged 18–29 were oversampled. Professional non-clinicians conducted face-to-face interviews with participants in their homes, using the Alcohol Use Disorders and Associated Disabilities Interview Schedule, a fully structured diagnostic interview based on the diagnostic criteria of the fourth edition of the DSM (AUDASIS-IV). The household response rate was 91.9 percent and the person response rate was 97.4 percent [45].

Measures

Dependent variable

Major depression screening symptom endorsement This is a dichotomous variable defined by endorsing in the AUDADIS-IV interview for depression, either sad mood or anhedonia occurring over a two-week or longer period in the last 12 months. Test–retest reliability of the AUDADIS-IV major depression interview has ranged from $\kappa = 0.65$ to $\kappa = 0.73$ [46–48]. Clinical reappraisal studies of major depressive disorder using the AUDADIS-IV had agreement in the $\kappa = 0.64$–0.68 range [49] and evidence from convergent validity studies has also been good [48, 50–52].

Independent variables

Race Those who self-reported being non-Hispanic and Black (unweighted $n = 5955$; weighted percent = 12.84%) or non-Hispanic and White (unweighted $n = 31{,}938$; weighted percent = 87.16%) comprise the study sample.

Somatization We operationalized depression somatization as a difference score between the number of somatic symptoms endorsed (possible range 0–4) and the number of psychological symptoms endorsed (possible range 0–3) in the past-12-month major depression interview. Somatic symptoms pertain to weight/appetite change, sleep deprivation, psychomotor function, and low energy. Psychological symptoms, excluding the two screening items, refer to feelings of worthlessness/guilt, poor concentration, and suicidality. We excluded the screening symptoms because they comprise the dependent variable in the second and third hypotheses. The difference scores range from + 4 to − 3, with higher scores indicating greater somatization.

Underlying depression To account for underlying depression, we use a summary score of the seven non-screening symptoms endorsed in the major depressive episode interview. Scores range from 0 to 7. Because four of the seven symptoms of underlying depression are somatic, high levels of underlying depression may artifactually inflate somatization scores; therefore, we adjust for underlying depression when testing somatization.

Somatic illness NLAES asked participants about 23 conditions "causing problems in the last 12 months," including stomach ulcer, cirrhosis of the liver, "high blood cholesterol, high blood fat, or high lipid content," hardening of the arteries or arteriosclerosis, high blood pressure, chest pain or angina, heart attack or myocardial infarction, stroke or cerebrovascular disease, emphysema, "arthritis, osteoporosis or other joint or bone diseases," cancer, and "any other physical health problem." We generated summary scores of conditions endorsed with a possible range from 0 to 23. If significantly different between Blacks and Whites, we will adjust for somatic illness whenever testing somatization, since endorsing somatic symptoms as part of the depression interview could be due to somatic illness rather than depression.

Age We adjust for age in all analyses because of its appreciably different distribution in Blacks and Whites (18–24: Blacks = 17.3%, Whites = 12.4%; 25–44: Blacks = 48.4%, Whites = 42.9%; 45–64: Blacks = 17.1%, Whites = 26.6%; ≥ 65: Blacks = 11.7%, Whites = 18.1%; $\chi^2 = 320.14$, $df = 3$, $p < 0.0001$) and its statistically significant association with screener endorsement ($\chi^2 = 719.67$, $df = 3$, $p < 0.0001$; NCS-R: $\chi^2 = 59.01$, $df = 3$, $p < 0.0001$).

Sex We also adjust for sex in all analyses because of different distributions in Blacks and Whites in this sample (Black female = 55.3%; White female = 51.9%; $\chi^2 = 30.77$, $df = 1$, $p < 0.0001$) and its statistically significant association with screener endorsement ($\chi^2 = 279.68$, $df = 1$, $p < 0.0001$).

Analytic strategy

All analyses were conducted using SAS software's (version 9.3, SAS Institute, Inc., Cary, NC) survey procedures to account for NLAES' survey weights and complex sampling design. Results are weighted to the US population and standard errors account for the multi-strata sampling plan. Our analyses were conducted among those ($n = 12{,}574$) endorsing at least one past-12-month depression symptom, and who therefore provided information germane to our hypotheses.

All analyses adjust for age and sex, through standardization when means or probabilities are compared, or in multivariable logistic regression analyses. The second and third hypotheses were tested using the SURVEYLOGISTIC procedure. Mean differences between Blacks and Whites in underlying depression, somatization, somatic health, and the underlying dimensions of somatization were tested using SURVEYREG procedures which allowed for multivariable adjustments and age- and sex-standardizing (to the White distribution of these variables) using the *estimate* procedure.

Results

We first report that in the NLAES sample, Blacks had a lower prevalence than Whites of past-year major depression (Black = 2.03%, White = 3.63%; $\chi^2 = 39.22$, $p < 0.0001$; OR = 0.60, 95% confidence interval (CI), 0.53–0.67), adjusting for age and sex. Sociodemographic characteristics of the sample are displayed in Table 1.

Table 2 shows mean somatization scores in the first row. Consistent with our first hypothesis, Blacks have a statistically significant higher mean somatization score than Whites. There were no group differences on somatic health and therefore, we did not adjust for it in our analyses.

Table 2 also shows results of post hoc Black–White comparisons on the underlying symptom structure of the somatization measure, unadjusted for underlying depression and somatic health. Among the four somatic symptoms, Blacks are more likely than Whites to endorse weight/appetite change, and Whites are more likely than Blacks to endorse low energy. Among the five psychological symptoms, Whites are slightly more likely than Blacks to endorse each. In a composite measure summing probabilities across the four somatic symptoms, Blacks and Whites are not different. In the composite measure of the five psychological symptoms, Whites have a slightly higher mean score than Blacks, both when excluding and including the screening symptoms. Our results indicate that Blacks' higher mean somatization score is driven by the weight/appetite symptom and Whites' greater tendency to endorse psychological symptoms; it does

Table 1 Sociodemographic characterstics of non-Hispanic Blacks and non-Hispanic Whites in the National Longitudinal Alcohol Epidemiologic Survey (1991–1992)

	Unweighted *n*, weighted % (SE)			
	Non-Hispanic Black		Non-Hispanic White	
Sex*				
Male	2100	44.70 (0.63)	13,600	48.13 (0.23)
Female	3855	55.30 (0.63)	18,338	51.88 (0.23)
Age*				
18–24	857	17.27 (0.43)	3361	12.42 (0.33)
25–44	2686	48.40 (0.55)	13,604	42.91 (0.27)
45–64	1401	22.66 (0.27)	7925	26.59 (0.29)
65+	1011	11.67 (0.42)	7048	18.08 (0.25)
Education*				
< HS	1908	28.44 (0.65)	5427	16.13 (0.29)
HS or GED	1838	31.92 (0.39)	10,133	32.73 (0.29)
Some college or AA	1400	26.40 (0.56)	8348	26.96 (0.29)
BA or higher	730	13.25 (0.33)	7749	24.19 (0.26)
Unknown	79		281	
Relationship status*				
Married or cohabitating	1926	43.07 (0.61)	17,934	65.59 (0.33)
Divorced, separated, widowed	2013	23.62 (0.38)	8107	16.73 (0.17)
Never married	1962	33.31 (0.59)	5618	17.69 (0.36)
Unknown	54		279	
Region*				
Northeast	1057	17.30 (0.50)	7072	21.95 (0.22)
Midwest	1221	19.08 (0.59)	8880	26.96 (0.35)
South	3151	55.53 (1.24)	9883	32.45 (0.44)
West	526	8.10 (0.28)	6103	18.64 (0.33)

*Statistically significant difference between non-Hispanic Blacks and non-Hispanic Whites using χ^2 test at the $p < 0.05$ level

not indicate a greater general inclination of Blacks compared with Whites to endorse somatic symptoms.

Results from testing our second hypothesis, that Blacks are less likely than Whites to endorse depression screening symptoms, are not supported (Table 3, model 1). Blacks have essentially the same odds as Whites of endorsing a screening symptom. Since there were no group differences on underlying depression or somatic health, we did not adjust for either variable when testing this hypothesis.

Our original hypothesis 3 was that adjusting for Blacks' greater depression somatization compared with Whites would explain Blacks' lower odds of endorsing the screening symptoms (hypothesis 2). Consequent to the results not supporting our second hypothesis, we speculated that Blacks' higher depression somatization scores compared with Whites might still suppress their relative odds of endorsing screening symptoms, such that adjusting for this variable might reveal *greater* odds of endorsing screening symptoms than Whites. Our results do not support this supposition. Table 3, model 2 shows that the Black–White odds ratio of endorsing a screening symptom remains essentially unchanged after adding somatization to the model.

Discussion

This study tested a methodological explanation for the paradox in psychiatric epidemiology of Blacks in the US having a lower prevalence than Whites of major depression. The explanation derived from a suggestion in the primarily clinical literature that Blacks express depression more somatically than Whites. If true, we reasoned, this could suppress Blacks' endorsement of the psychological screening symptoms for major depression, leading to a disproportionate undercount of major depression in Blacks compared with Whites.

Our evidence does not support this explanation. Although Blacks have slightly higher somatization scores than Whites in the NLAES sample, that difference is driven by Blacks' higher score on one somatic item and Whites' higher scores across the psychological symptoms. Therefore, evidence based on this nationally representative sample is not consistent with the notion that Blacks broadly express depression more somatically than Whites. Further, the small somatization difference we find has no impact on Blacks' relative propensity to endorse the depression screening symptoms. In the NLAES sample, Blacks' odds of endorsing screening symptoms are not statistically significantly different from Whites' odds, and, controlling for depression somatization does not affect the Black–White screener endorsement odds ratio.

Our conclusion of no consequential Black–White difference on somatization is based on data from a nationally representative psychiatric epidemiology study in which the Black–White depression paradox is present. Moreover, the AUDADIS-IV diagnostic interview for depression comprises the common somatic and psychological expressions of depression. Therefore, our test of the somatization hypothesis makes us inclined to rule out this explanation in the future. The three other nationally representative psychiatric epidemiology studies that have been conducted in the US (the National Comorbidity Survey, the National Comorbidity Survey-Replication, and the National Epidemiologic Survey on Alcohol and Related Conditions) do not lend themselves to test the somatization hypothesis because they collected full symptom information only from those endorsing screening symptoms. As we have noted, these sub-samples potentially exclude those with more somatic expressions of depression.

We note here an ostensible, and related, paradox in the psychiatric epidemiology literature. Comparisons of US

Table 2 Non-Hispanic Blacks' and non-Hispanic Whites' age- and sex-standardized means and prevalences among those endorsing at least one past-year depression symptom in the National Longitudinal Alcohol Epidemiologic Survey (1991–1992)

	Non-Hispanic Black 2122 (13.64%)[a]	Non-Hispanic White 10,452 (86.36%)	*t* value	*p* value
Somatization mean (SE), range −3 to +4[b]	0.28 (0.04)	0.15 (0.03)	−2.15	0.03
Somatic health conditions mean (SE), range 0–22	1.06 (0.04)	1.02 (0.02)	−0.81	0.42
Underlying depression mean (SE), range 1–7	2.52 (0.05)	2.57 (0.03)	0.69	0.49
Underlying dimensions of somatization and depression				
Somatic symptom endorsement probabilities, range 0–1 (SE)				
Weight/appetite change	0.55 (0.01)	0.45 (0.01)	−4.41	< 0.0001
Sleep problems	0.41 (0.01)	0.43 (0.01)	0.88	0.38
Psychomotor changes	0.25 (0.01)	0.24 (0.01)	−0.44	0.66
Low energy	0.37 (0.01)	0.42 (0.01)	2.21	0.03
Psychological symptom endorsement probabilities, range 0–1 (SE)				
Sad mood	0.34 (0.01)	0.36 (0.01)	0.88	0.38
Anhedonia	0.26 (0.01)	0.29 (0.01)	1.32	0.19
Poor concentration	0.31 (0.01)	0.32 (0.01)	0.44	0.66
Guilt/worthlessness	0.23 (0.01)	0.27 (0.01)	1.77	0.08
Suicidality	0.39 (0.02)	0.43 (0.01)	1.77	0.08
Somatic symptom probability sums, range 0–4 (SE)	1.59 (0.04)	1.55 (0.02)	−0.84	0.40
Psychological symptom probability sums, range 0–3 (SE)[c]	0.93 (0.03)	1.02 (0.01)	3.46	< 0.001
Psychological symptom probability sums, range 0–5 (SE)[d]	1.53 (0.05)	1.66 (0.02)	2.61	0.01

All comparisons are based on weighted sample

[a]Unweighted *n* (weighted %)

[b]Adjusted for underlying depression

[c]Excluding screener symptoms

[d]Including screener symptoms

Table 3 Logistic regression of race predicting screener endorsement, adjusting for underlying depression, age, sex, and somatization, among those endorsing at least one past-year depression symptom in the National Longitudinal Alcohol Epidemiologic Survey (1991–1992)

	Model 1		Model 2	
	OR	95% CI	OR	95% CI
Race (ref = non-Hispanic White)	0.96	0.90–1.02	1.01	0.93–1.11
Age	0.77	0.74–0.80	0.83	0.80–0.87
Sex (ref = male)	1.35	1.26–1.44	1.11	1.03–1.21
Somatization			0.78	0.76–0.80
Underlying depression			2.23	2.17–2.29

Both analyses are based on weighted sample

Blacks and Whites on major depression and psychological distress consistently yield divergent results [6] in studies using nationally representative samples. In these studies, Blacks reliably score equal to or higher than Whites on psychological distress, despite consistently having a lower prevalence of major depression. Though major depression and psychological distress are different constructs, there is sufficient conceptual [53, 54] and measurement [30, 55] overlap (i.e., most distress measures borrow heavily from depression and anxiety diagnostic categories) between them that one would expect consistent results across group comparisons on these outcomes. We speculated that if Blacks were to express depression more somatically than Whites, they would also express distress more somatically. Yet, because distress measures do not have screening symptoms but are typically reported as simple sums of endorsed symptoms, they are not biased against more somatic expressions of distress. This, in turn, could explain why Blacks score equal to or higher than Whites on these measures, as social stress theory would predict. To the extent Blacks did express depression and distress more somatically than Whites, this could further explain the second paradox of divergent results when comparing Blacks and Whites on depression and distress. In ancillary analyses (results not shown), we examined whether Blacks expressed distress more somatically in the National Comorbidity Survey-Replication's measure of non-specific psychological distress (the K10). Our results were remarkably consistent with those from our Black–White comparisons of depression somatization in the NLAES sample.

Specifically, the NCS-R data also did not support a broad somatization hypothesis among Blacks.

What of other explanations for the Black–White depression paradox? Substantive explanations include positing greater levels in Blacks than Whites of religiosity and racial socialization [56], self-esteem [57], and social networks [58], factors thought to have protective dimensions for mental health [59–66]. To our knowledge, only the social networks hypothesis has been tested, and the findings failed to support it [58, 67]. A more recent hypothesis [3, 68] proposes an interaction between race, stress, and poor health behaviors (e.g., overeating) such that at higher stressor levels, these behaviors are more protective against depression in Blacks than in Whites, while simultaneously leading to worse somatic health outcomes in Blacks. Tests of this hypothesis have yielded mixed results [3, 69, 70].

Aside from the lack of evidence, a central problem with substantive explanations for the Black–White depression paradox is that they fail to account for the Black–White distress findings. Proponents of substantive hypotheses must explain why a given factor would protect Blacks from depression but not distress, or would protect Blacks from depression so much more effectively than from distress. If anything, one might think that protective factors would work in the short term against distress, but have diminishing benefit over time, or limited benefits in the face of larger life stressors, and ultimately fail to protect against depression.

Artifactual explanations for the Black–White depression paradox include a greater tendency to misdiagnose depression as a psychotic spectrum disorder in Blacks than Whites [71–73], and differential item functioning between Blacks and Whites of the items used to diagnose major depression [43]. Differential item functioning occurs when groups have different probabilities of endorsing an item or symptom, controlling for underlying levels of the construct being measured [43]. To date, results from tests of these hypotheses explain, at best, only a trivial portion of the Black–White depression paradox. The prevalence of psychotic spectrum disorders [74] is too low to account for the large prevalence gap in depression, and mood disorders more broadly, found between Blacks and Whites. Tests of differential item functioning have detected only minor differences in how the items and symptoms of the diagnostic interview for depression function between Blacks and Whites [43, 44].

Finally, we note that in the NLAES sample, Blacks and Whites had similar probabilities of endorsing a screening symptom and similar levels of underlying depression as measured with the remaining seven diagnostic symptoms for depression. However, Blacks had 40 percent lower odds of a depression diagnosis in the sample. This suggests points other than symptom endorsements in the diagnostic algorithm at which Blacks diverge from Whites. The diagnostic interview for major depression inquires about the nine symptoms discussed here, but also entails numerous exclusion criteria, including ostensible cases following death or loss and cases induced by medication or non-medical substance use. Importantly, distress measures do not have exclusion criteria. Future investigations of the Black–White depression paradox should compare Blacks' and Whites' responses across these exclusion criteria to determine the extent to which they account for the lower prevalence of major depression in Blacks. These comparisons may provide more specific guidance in developing substantive or artifactual explanations for the Black–White depression paradox.

Strengths and limitations

With regard to testing our hypothesized explanation for the Black–White depression paradox, NLAES is the only large psychiatric epidemiology study using a nationally representative US sample in which all participants were asked about all nine depression symptoms, regardless of screener endorsement. This made NLAES uniquely suited for assessing depression somatization in a sample unbiased by conditioning on endorsement of a psychological screening symptom.

Several limitations of this study are noted as well. First, depression symptomatology may have been misreported, leading to misclassification at the symptom level. To the extent this misclassification was differential by race, estimates of somatization and underlying depression may be under- or over-estimated in each race group. Second, the measure of somatic health relied on participants identifying conditions causing problems in the past year that in many cases require diagnosis by a medical professional. Because Blacks in the US have less access to health care and receive poorer care [21, 75–79] than Whites, somatic health may have been disproportionately underestimated in Blacks. In turn, when comparing Blacks and Whites on depression somatization, somatic health may have been under-adjusted for among Blacks, thereby disproportionately inflating their somatization score. This would mean, if anything, that somatization in Blacks was *over*-estimated in our sample.

Conclusion

We find no evidence in our study of a meaningful difference between Blacks and Whites on the somatization of depression. Blacks' slightly higher somatization score is driven by one somatic symptom and a greater tendency in Whites to endorse psychological symptoms. Adjusting for Blacks' slightly higher level than Whites of depression somatization has no impact on Blacks' relative likelihood of endorsing a screening symptom. Therefore, the data do not support a somatization explanation for the paradox of lower

depression in Blacks than Whites. Future attempts to resolve the Black–White depression paradox need to simultaneously address the second paradox of discordant Black–White distress findings. Moreover, examinations of group differences in the responses to the exclusion criteria of the major depression diagnostic algorithm are warranted.

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Compliance with ethical standards

Conflict of interest On behalf of all authors, the corresponding author states that there is no conflict of interest.

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