Racialized Heteroscedasticity in Connectome-Based Predictions of Brain-Behavior Relationships

Christopher T. Fields, Ph.D. 1,2,*, Matthew Rosenblatt, M.S. 2,*, Joseph Aina 3,*, Jannat Thind 1, Annie Harper, Ph.D. 1, Chyrell

Bellamy, MSW, PhD¹, Raimundo Rodriguez², Xin Zhou, Ph.D.⁴, Fahmi Khalifa, Ph.D.^{3,#}, Dustin Scheinost, Ph.D.^{2,#}

¹Program for Recovery and Community Health, Department of Psychiatry, Yale School of Medicine, New Haven, CT,

USA

²Magnetic Resonance Research Center, Department of Radiology and Biomedical Imaging, Yale School of Medicine,

New Haven, CT, USA

³Department of Electrical and Computer Engineering, Morgan State University, Baltimore, MD, USA

⁴Department of Biostatistics, Yale School of Public Health, New Haven, CT, USA

* indicates co-first authorship, # indicates shared senior authorship

Corresponding author: Christopher Fields,

Associate Research Scientist, Yale School of Medicine,

Connecticut Mental Health Center, Third Floor, 34 Park St, New Haven, CT, 06519, USA,

Email: christopher.fields@yale.edu

Preprint Disclosure: This work is a preprint and has not yet been peer reviewed.

Word Count: 3472, abstract: 337

Keywords: Resting-state fMRI, racialized heteroscedasticity, connectome-based predictive modeling, neuroimaging,

predictive modeling

Conflict of Interest and Financial Disclosure Statement: The authors have nothing to disclose.

Abstract

Background: Racialized heteroscedasticity, characterized by unequal variance in neuroimaging data across racial groups, may challenge the assumptions underlying brain-behavior predictive models. These models often assume homoscedasticity, a fundamental assumption in linear clinical models like Connectome-based predictive models (CPM), particularly when applied across diverse populations. This study investigates the presence of heteroscedasticity across racial groups in functional connectivity data from the Adolescent Brain Cognitive Development (ABCD) study and examines its implications for predictive modeling using resting-state fMRI (rs-fMRI) data.

Methods: This study analyzed neuroimaging data across six modalities—Diffusion Tensor Imaging (DTI), T1-weighted structural MRI (SMR), resting-state fMRI (rs-fMRI), and task-based fMRI (Monetary Incentive Delay, Stop-Signal Task, and nBack tasks)—from 5,084 participants in the Adolescent Brain Cognitive Development (ABCD) study. Variance differences between Black and White participants were assessed using Levene's test, adjusting for demographic and socioeconomic covariates with False Discovery Rate (FDR) correction. Behavioral variance across 36 cognitive and psychological measures was also evaluated. We predicted CPM employed to predict performance on behavioral tasks, with bootstrapping used to estimate prediction intervals and assess variance in model accuracy across groups.

Results: Racial differences in variance were observed across neuroimaging modalities, behavioral measures, and CPM variance interval analyses. After covariate regression, 68.9% of DTI features and 6.6% of rs-fMRI connectivity edges showed greater variance in Black participants, while no significant differences remained for SMR. Task-based fMRI and behavioral variance analyses revealed consistently greater variability in Black participants, with 87.8% of behavioral measures showing significant differences. Increased prediction uncertainty was observed for CPM models with Black relative to White participants across 8 out of the 15 measures tested.

Conclusion: Our findings underscore the importance of accounting for racialized heteroscedasticity in neuroimaging-based predictive models. Failing to account for differences in variance may exacerbate health disparities and lead to less reliable predictions for Black participants. These findings emphasize the need for diverse sampling and adaptive modeling approaches to ensure equitable predictive performance across populations. Addressing these disparities is essential for the development of more equitable predictive models in neuroscience and psychiatry.

Background

Recent studies have highlighted the importance of considering racial and ethnic differences in neuroimaging research.^{1,2} These differences may arise from the interaction of genetic, environmental, and sociocultural factors.³⁻⁵ For instance, experiences of racial discrimination have been shown to impact brain structure and function,^{6,7} potentially contributing to observed differences in neuroimaging data between racial groups.

The concept of heteroscedasticity, or unequal variance across groups, is well-established in statistical literature but has received limited attention in the context of neuroimaging studies. ^{8,9} In predictive modeling, the assumption of homoscedasticity (equal variance) is often made, which may lead to biased or less accurate predictions when this assumption is violated. ^{10,11} Given the growing application of neuroimaging-based predictive models in clinical decision-making, it is essential to investigate whether heteroscedasticity exists between racial groups and how it might impact the reliability and equity of these models.

The Adolescent Brain Cognitive Development (ABCD) study provides an unparalleled opportunity to examine these issues, leveraging a large and diverse dataset of adolescents. ^{1,12} Its comprehensive neuroimaging, cognitive, and behavioral assessments allow for a detailed exploration of variance differences across racial groups, offering novel insights into how race-related factors influence brain-behavior relationships.

This study aims to address a critical gap in the literature by examining racialized heteroscedasticity in rs-fMRI connectivity and its implications for neuroimaging-based psychiatric prediction models. By focusing on differences between Black and White participants, we seek to shed light on potential disparities in prediction certainty. Understanding these differences is crucial for developing more equitable and accurate predictive models in neuroscience and psychiatry, ultimately contributing to improved clinical applications and personalized interventions across diverse populations.

Methods

Study Population

This study utilized baseline demographic and year 1 neuroimaging data from the Adolescent Brain Cognitive Development (ABCD) study, which includes neuroimaging, cognitive, and behavioral assessments of youth aged 9-10 years (at baseline) from 22 research sites across the United States. Participants included in the

analysis were required to have complete rs-fMRI data, demographic information (age, gender, and parental income), and the absence of severe motion artifacts. Motion artifacts were defined as a mean framewise displacement exceeding 0.2 mm. This analysis focused on Black and White participants due to the sample size and availability of racially stratified data, allowing for robust statistical comparisons.

All participants were scanned using standardized imaging protocols across sites, and site effects were adjusted using statistical methods outlined below. Head motion was accounted for by including mean framewise displacement as a covariate in all analyses.

Neuroimaging Data Processing

Resting-state fMRI data were preprocessed using the ABCD pipeline, which included motion correction, slice-timing correction, coregistration, and normalization to a standard MNI space. Participants with mean framewise displacement exceeding 0.2 mm were excluded to minimize motion-related artifacts, and scans were visually inspected to identify additional anomalies not captured by automated thresholds. Functional connectivity matrices were computed for each participant by extracting the mean time series from 268 regions of interest (ROIs) defined by the Power atlas. Pearson correlation coefficients were calculated between each pair of ROIs, yielding a 268 x 268 connectivity matrix representing the strength of functional connections. To focus on unique and biologically meaningful patterns, redundant and diagonal edges were excluded, resulting in 35,912 unique connectivity edges for analysis.

To ensure consistency across the 22 research sites within the CPM models themselves, site-specific variability was addressed through statistical harmonization. Site effects were regressed out during preprocessing, ensuring that differences in imaging protocols and equipment did not confound the results. Additionally, nuisance regressors, including head motion parameters, were incorporated into the preprocessing pipeline to further reduce noise in the connectivity matrices. Connectivity values were z-transformed to normalize the data across participants and facilitate direct comparisons. These preprocessing and modeling steps ensured that the resulting data were of high quality and observed differences in model performance are more likely reflective of true racial differences.

Fifteen behavioral and cognitive tasks from the ABCD dataset were included in this analysis to capture a broad range of neurodevelopmental and mental health outcomes. These tasks assessed domains such as working memory (lmt_scr_perc_correct), processing speed (pcps_speed), cognitive flexibility (nihtbx_cardsort_uncorrected), inhibitory control (nihtbx_flanker_uncorrected), and verbal intelligence (nihtbx_picturevocab_uncorrected). Additional tasks evaluated reading ability (nihtbx_reading_uncorrected), impulsivity (uppps_y_ss_positive_urgency, uppps_y_ss_negative_urgency), delay discounting and decision-making (cash_choice_perc_higher, ddl_total_cum_dscore, ddl_indifference_point), and motivational influences (nacc_reinforce_bias). Pre-morbid intellectual functioning was assessed using the Wechsler Test of Adult Reading (wtar_uncorrected).

These measures were chosen based on their relevance to brain-behavior relationships and their utility in predicting neurodevelopmental trajectories. Tasks were selected to ensure a representative sampling of cognitive, emotional, and behavioral domains, allowing for robust analysis of variability across racial groups. Behavioral scores were standardized across participants to facilitate direct comparisons, and any missing data were excluded pairwise to maximize the sample size for each task. These measures were selected to capture a range of cognitive and behavioral domains relevant to neurodevelopment and mental health outcomes.

Statistical Analysis

To test for differences in variance between Black and White participants, Levene's test was conducted for each of the 35,912 unique resting-state fMRI connectivity edges, using race as a grouping factor. This approach was chosen for its sensitivity to heteroscedasticity, enabling the detection of group differences in variance. Demographic covariates, including age, gender, parental income, and head motion, were included to account for potential confounding effects. Multiple comparisons were corrected using the False Discovery Rate (FDR) method, with significant results identified at p < 0.05.

For the Connectome-Based Predictive Modeling (CPM) analysis, whole-brain rs-fMRI connectivity was used as predictors to model performance across 15 cognitive and behavioral tasks. The dataset was split into training and testing sets, employing a "leave-3-sites-out" cross-validation approach to mitigate site-specific variability and improve generalizability. Prediction intervals for Black and White participants were estimated using bootstrapping procedures, generating 95% confidence intervals based on 1,000 iterations. Differences in

prediction intervals between the two groups were assessed using permutation tests, with FDR correction applied to ensure robust comparisons across multiple tasks.

Software

All analyses were conducted using Python and MATLAB for data preprocessing, statistical analysis, and Connectome-Based Predictive Modeling (CPM). The CPM analysis specifically used the code package provided by the Yale Magnetic Resonance Research Center (Yale MRRC), which is publicly available at https://github.com/YaleMRRC/CPM. Python packages such as NumPy and SciPy were used for statistical tests, and pandas was employed for data management. Bootstrapping procedures and model evaluations were implemented using custom Python code, and results were visualized using Matplotlib.

Ethical Approval

The ABCD study operates under a centralized Institutional Review Board (IRB) at the University of California, San Diego, as well as local IRBs at each of the 22 participating recruitment sites. All participants and their parents/guardians provided written informed consent and assent for data collection. The present analysis was conducted on de-identified data obtained from the ABCD study's publicly available repository, in accordance with all relevant ethical guidelines and regulations.

Results

Racial Differences in Variance in Structural, Resting-State, and Task-Based Connectivity

We examined differences in variance across six key neuroimaging modalities—Diffusion Tensor Imaging (DTI), T1-weighted structural MRI (SMR), resting-state fMRI (rsfMRI), and task-based fMRI during the Monetary Incentive Delay (MID), Stop-Signal Task (SST), and nBack tasks—between Black and White participants in the ABCD study. For Diffusion Tensor Imaging (DTI, FA), 151 features corresponding to white matter tracts were examined. For T1-weighted structural MRI (SMR), 151 features representing regional gray matter volumes and cortical thickness were included. For fMRI, we constructed a connectivity matrix comprising 71,854 edges (from a 268 x 268 matrix). After excluding redundant connections, 35,912 unique edges were tested

for differences in variance using Levene's test. We focused on these unique edges to ensure that analyses captured non-redundant, meaningful patterns in functional and structural connectivity.

Across all modalities, significant differences in variance were observed between Black and White participants, with the majority of features showing greater variance in Black participants. When covariates were not included in the models (**Figure 1A**), significant findings emerged across all six imaging modalities.

For DTI fractional anisotropy (FA), 29.1% (44/151) of the features showed significant variance differences, with 75% (33/44) exhibiting greater variance in Black participants (p<0.001). Similarly, for T1-weighted structural MRI (SMR-T1), 20% (30/151) of the features displayed significant variance differences, with 63% (19/30) showing increased variance in Black participants (p=0.199).

In the resting-state fMRI analysis, 2.5% (875/35,912) of the unique edges exhibited significant differences in functional connectivity variance between groups, with 70% (573/875) showing greater variance in Black participants (p<.001).

For the task-based fMRI modalities, a similar pattern of increased variance in Black participants was observed. During the Monetary Incentive Delay (MID) task, 6.7% (2409/35,912) of edges showed significant variance differences, with 95.7% (2330/2409) demonstrating greater variance in Black participants (p<.001). For the Stop-Signal Task (SST), 1.9% (678/35,912) of edges exhibited significant variance differences, with 95.1% (672/678) showing increased variance in Black participants (p<.001). Finally, for the nBack task, 0.87% (311/35,912) of edges displayed significant differences in variance, with 84.9% (152/311) showing greater variance in Black participants (p<.001).

When including covariates (age, sex, head motion) and site correction in the Levene's test, significant racial differences in variance persisted across modalities (**Figure 1B**). The exception here was T1-weighted structural MRI (SMR), which did not have any features that survived the addition of covariates to Levene's test. For DTI fractional anisotropy (FA), 68.9% (104/151) of features showed significant variance differences, with 100% (104/104) demonstrating greater variance in Black participants (p<.001). In the resting-state fMRI analysis, 6.6% (2373/35,912) of edges exhibited significant differences in functional connectivity variance, with 66% (1204/2373) showing greater variance in Black participants (p<.001). Task-based fMRI modalities similarly demonstrated persistent differences. During the MID task, 16.3% (5398/35,912) of edges were significant, with 92% (4953/5398) exhibiting greater variance in Black participants (p<.001). For the Stop-Signal Task (SST),

11.0% (3968/35,912) of edges showed significant variance differences, with 87% (3451/3968) demonstrating greater variance in Black participants (p<.001). Finally, for the nBack task, 5.9% (2126/35,912) of edges showed significant variance differences, with 83% (1762/2126) exhibiting greater variance in Black participants (p<.001). These findings indicate that racialized variance differences are robust even when controlling for site and demographic factors.

This racial skew in Levene's test results was further validated using chi-square tests, as detailed in Supplementary Table 1. We analyzed the number of features showing significantly greater variance in one group compared to the other across neuroimaging modalities. Supplementary Table 1 provides a breakdown of features with significantly greater variance in Black participants versus White participants, as determined by Levene's test. The chi-square test assessed whether the distribution of features with greater variance differed significantly between the two groups. The results demonstrated a pronounced skew toward greater variance in Black participants across most modalities, even after accounting for demographic covariates and site effects.

While controlling for multiple comparisons is a rigorous approach for identifying significant differences, edge weight selection for CPM models is typically not subjected to multiple comparison correction, as it prioritizes predictive utility rather than strict statistical significance. Nonetheless, even after controlling for multiple comparisons, robust increases in variance across neuroimaging measures were observed for Black compared to White participants. Supplementary Figure 1 presents the variance histograms for all modalities under the less strict FDR threshold (pN), while Supplementary Figure 2 presents the variance histograms under the more strict FDR threshold (pID). Additionally, Supplementary Figure 3 and Supplementary Figure 4 provide pN and pID variance histograms for covariate adjusted Levene's analyses.

Behavioral Variance Differences Across Racial Groups

In addition to neural measures, we analyzed variance differences across 36 behavioral measures from the ABCD dataset, using Levene's test to assess differences in variance between Black and White participants (Table 1). These measures spanned several domains, including cognition, personality traits, emotional regulation, and behavioral health. Both non-covaried and covaried models were used, with the covaried models controlling for age, gender, parental income, study site, and head motion. To account for multiple comparisons, False Discovery Rate (FDR) correction was applied to the results.

As shown in Table 1, of the 36 behavioral measures analyzed, 33 measures (87.8%) exhibited significant differences in racial variance as measured by Levene's test. Of these, 29 showed significantly greater variance in Black participants compared to White participants. Among these measures, several from the Child Behavior Checklist (e.g., rule-breaking, aggression, and attention problems) and personality domains (e.g., sensation-seeking and impulsivity) exhibited the most pronounced differences. These findings suggest that race-related factors contribute to greater behavioral heterogeneity across multiple psychological and cognitive domains, even after accounting for demographic and site-related covariates. This pattern parallels the increased variability observed in neural measures, further indicating that racialized experiences may influence a broad range of behavioral outcomes.

Conversely, a smaller subset of measures exhibited greater variance in White participants. In the non-covaried models, 4 measures showed significantly greater variance in White participants. These measures primarily involved domains related to cognitive abilities, such as reading performance and crystallized intelligence. While the proportion of measures with greater variance in White participants was smaller, these results highlight the importance of considering variability patterns across racialized groups for specific behavioral constructs.

Connectome-Based Predictive Modeling (CPM) Variance in Prediction Intervals

We conducted Levene's tests to assess racial differences in variance for composite CPM scores correlated with each of the 36 behavioral measures (Table 2). CPM scores were calculated as is typically done in Connectome-Based Predictive Modeling, with each score optimized to correlate most strongly with the corresponding behavioral measure, selecting the top 10% of edges. The results, presented in Table X, revealed significant racial differences in variance for 13 out of the 36 behavioral measures. These findings suggest that variance differences between Black and White participants extend beyond neuroimaging features and into the predictive performance of CPM models.

Building on this, we evaluated how these variance differences manifest in the prediction intervals of CPM models. Using whole-brain rs-fMRI connectivity as predictors, we modeled performance across 15 cognitive and psychological test outcomes from the ABCD dataset. The CPM approach links brain connectivity patterns with behavioral and cognitive outcomes, allowing us to assess the variability in prediction intervals for Black and

White participants. The variance estimates for the prediction intervals were calculated by fitting CPM models across the 15 cognitive/psychological tests. Ninety-five percent confidence intervals (CIs) were generated using a "leave 3-sites out" bootstrapping method, which involved leaving out data from three of the 21 ABCD research sites in each iteration. This method was repeated 1,000 times to ensure robust cross-validation and reliable variance estimates for model predictions in both racial groups. Significantly wider variance intervals were observed for Black participants compared to White participants across several cognitive and behavioral domains. For example, Black participants had significantly greater variance in the prediction intervals for the NIH Toolbox Card Sorting Task, a measure of executive functioning, as well as for the Fluid Cognition Composite, which evaluates cognitive flexibility and problem-solving abilities. Similarly, the NIH Toolbox List Sorting Task, assessing working memory, and the Pattern Comparison Task, a measure of processing speed, both showed wider variance intervals for Black participants. In total, 8 out of the 15 tested models demonstrated significantly greater variance intervals for Black participants after FDR correction (p < 0.05).

Discussion

Our study reveals significant differences in the variance of resting-state functional connectivity (rs-fMRI) between Black and White participants in the Adolescent Brain Cognitive Development (ABCD) study, as well as disparities in the prediction intervals of connectome-based predictive models across various cognitive and behavioral tasks. These findings have important implications for neuroimaging research, particularly in the context of diverse populations and the development of predictive models for clinical applications. ¹³⁻¹⁵

The observation that 10.5% of the examined rs-fMRI connectivity edges showed significant differences in variance between Black and White participants, with 98.5% of these edges demonstrating greater variance in Black participants, is a striking finding. This pervasive heteroscedasticity suggests that the assumption of homogeneous variance across racial groups in neuroimaging studies may be flawed. Such an assumption could lead to biased or less accurate predictions when applying models developed primarily on one racial group to another. The greater variability in connectivity patterns among Black participants persisted even after controlling for socioeconomic and demographic factors. This suggests that race-related factors beyond commonly considered variables like socioeconomic status may influence neural connectivity patterns. These factors could

include experiences of racial discrimination, differences in environmental exposures, or other sociocultural influences that may impact brain development and function. 4,18-21

The wider prediction intervals observed for Black participants across several cognitive and behavioral domains in our connectome-based predictive modeling (CPM) analysis further underscore the importance of considering racial differences in neuroimaging-based predictive models. The significantly greater variance in prediction intervals for 8 out of 15 tested models, including measures of executive functioning, cognitive flexibility, working memory, and processing speed, indicates a higher degree of uncertainty in model predictions for Black participants. This increased uncertainty has critical implications for the application of these models in clinical settings. If not properly accounted for, it could lead to less reliable predictions or misinterpretations of brain-behavior relationships in Black individuals. This finding aligns with growing concerns about the generalizability of neuroimaging findings across diverse populations and the potential for exacerbating health disparities through the uncritical application of predictive models.^{22,23}

Several factors may contribute to the observed differences in variance and prediction uncertainty between Black and White participants. Genetic diversity within African populations could contribute to increased variability in brain structure and function. However, it's crucial to note that genetic explanations alone are insufficient and can perpetuate harmful racial stereotypes if not contextualized within broader socio-environmental factors. ²⁴ Differences in environmental exposures, including pollution, nutrition, and access to healthcare, may contribute to greater variability in brain development and function among Black participants.

These factors are often linked to systemic racial inequalities and may have cumulative effects on brain health. ^{25,26}

Chronic stress related to experiences of racial discrimination and systemic racism can impact brain structure and function. 6,27 The variability in these experiences and individual coping mechanisms could contribute to greater heterogeneity in brain connectivity patterns among Black participants. It's also possible that current neuroimaging techniques and analysis methods may be less sensitive or more variable when applied to Black participants due to factors such as differences in brain morphology or hemodynamic responses. This highlights the need for developing and validating neuroimaging methods across diverse populations. 28

Our findings emphasize the need for more nuanced approaches to neuroimaging research and predictive modeling that account for potential heteroscedasticity across racial groups. Neuroimaging studies should prioritize diverse and representative sampling to capture the full spectrum of variability across populations. This includes

not only racial diversity but also considerations of socioeconomic status, geographic location, and other relevant demographic factors.²

The observed differences in variance and prediction uncertainty between racial groups raise important ethical considerations for the development and application of neuroimaging-based predictive models. Researchers must be cautious in interpreting and communicating these findings to avoid reinforcing harmful stereotypes or biological determinism.²⁹ It is crucial to emphasize that these differences likely reflect a complex interplay of social, environmental, and biological factors rather than inherent racial characteristics. Responsible reporting of these results should always contextualize them within the broader societal and historical factors that contribute to health disparities.³⁰

We use the term "racialized heteroscedasticity" to acknowledge that the observed differences in variance are not inherent to race itself, but rather are a result of societal processes of racialization. This terminology emphasizes that race is a social construct, and the disparities we observe are likely due to the lived experiences, environmental factors, and systemic inequalities associated with racial categorization in society. By framing it as "racialized" heteroscedasticity, we aim to shift the focus from biological determinism to the social and structural factors that contribute to these disparities in neuroimaging data.

While our study provides important insights into racialized heteroscedasticity in neuroimaging data, several limitations should be considered. First, the binary categorization of race (Black vs. White) does not capture the full spectrum of racial and ethnic diversity. Future studies should examine these patterns across more diverse racial and ethnic groups. Second, while we controlled for several demographic and socioeconomic factors, there may be other unmeasured confounding variables that contribute to the observed differences in variance.

More comprehensive assessments of environmental exposures, experiences of discrimination, and cultural factors, particularly those gathered through community-engaged research, could provide additional insights. 32,33

In conclusion, our study highlights the presence of racialized heteroscedasticity in rs-fMRI connectivity and its impact on connectome-based predictions of brain-behavior relationships. These findings underscore the importance of considering diversity and potential sources of variability in neuroimaging research and predictive modeling. By acknowledging and addressing these differences, we can work towards developing more equitable and accurate predictive models that serve diverse populations effectively. Moving forward, it is essential for the neuroimaging community to prioritize diverse sampling, develop adaptive modeling techniques, and engage in

279	responsible interpretation and communication of results. By doing so, we can advance our understanding of brain
280	behavior relationships across diverse populations and contribute to more equitable and effective applications of
281	neuroimaging in clinical and research settings.
282	

Funding: The Adolescent Brain Cognitive Development (ABCD) Study was supported by the National Institutes of Health (NIH) and additional federal partners under the following awards: U01DA041022, U01DA041025, U01DA041028, U01DA041048, U01DA041089, U01DA041093, U01DA041106, U01DA041117, U01DA041120, U01DA041134, U01DA041148, U01DA041156, U01DA041174, U24DA041123, and U24DA041147. The ABCD federal partners include the National Institute on Drug Abuse (NIDA), the National Institute on Alcohol Abuse and Alcoholism (NIAAA), the National Cancer Institute (NCI), the National Institute of Mental Health (NIMH), the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), the National Heart, Lung, and Blood Institute (NHLBI), the National Institute of Neurological Disorders and Stroke (NINDS), the National Institute on Minority Health and Health Disparities (NIMHD), the NIH Office of Behavioral and Social Sciences Research (OBSSR), the NIH Office of Research on Women's Health (ORWH), the Centers for Disease Control and Prevention (CDC) - Division of Violence Prevention, the National Institute of Justice (NIJ), the CDC – Division of Adolescent and School Health, the National Science Foundation (NSF), and the National Endowment for the Arts (NEA). This work was supported by grant support to the authors: National Institute of Mental Health (NIMH) grant F32MH129052 to CTF; and NIMH grant 5R01MH121095-05 and Yale ASCEND grant to DS. The funding organizations had no role in the analysis and interpretation of the data, preparation, review, or approval of the manuscript, or the decision to submit the manuscript for publication. This manuscript reflects the views of the authors and may not represent the opinions or views of the National Institutes of Health (NIH) or the ABCD consortium investigators.

302 Author Contributions:

CTF led the conceptualization and performed data analysis, data interpretation, and the initial draft of the manuscript. MR and JA conducted statistical analysis, contributed to data interpretation, and revised the manuscript. JKT, AH, CB, and RR contributed to data interpretation and manuscript revision. XZ and FK supervised the project, assisted with data interpretation, and revised the manuscript. DS provided project supervision and contributed to manuscript revision.

308

309

310

283

284

285

286

287

288

289

290

291

292

293

294

295

296

297

298

299

300

301

303

304

305

306

307

Data Availability: The data supporting the findings of this study were obtained from the Adolescent Brain Cognitive Development (ABCD) Study. Due to restrictions, the data are available upon reasonable request from

the corresponding author or from the ABCD Study Data Access portal. The source data used in this study were openly available prior to its initiation. The ABCD data used in this report came from the fast track data release 5.1, which is accessible to qualified researchers through the NIMH Data Archive (NDA). The raw data are available at https://nda.nih.gov/study.html?id=2313, and the data dictionary for ABCD can be found at https://data-dict.abcdstudy.org/. Additional details about the measures assessed for the ABCD study are provided at https://wiki.abcdstudy.org/release-notes/start-page.html. Instructions for obtaining NDA data use certification are available at https://nda.nih.gov/nda/access-data-info.

Acknowledgments: The authors would like to thank the ABCD Study Consortium for providing access to the data, as well as the participants and families who contributed to this research. Special thanks to the Yale School of Medicine for its institutional support through the ASCEND grant program.

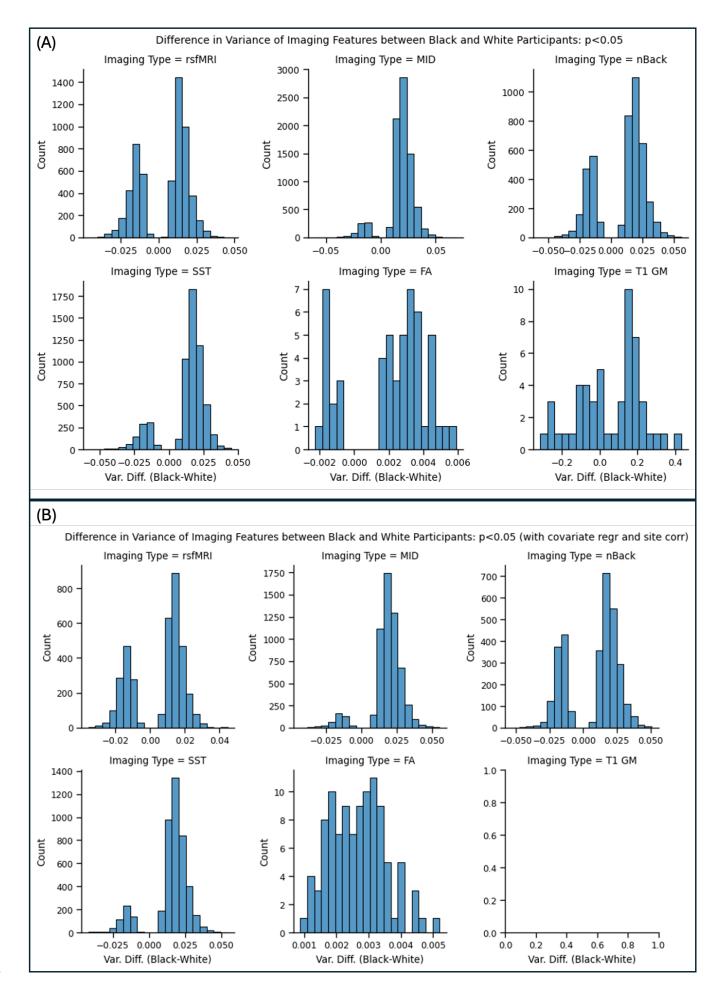


Figure 1. Histograms depict the distribution of variance differences (Black-White) across six key neuroimaging modalities. Figure 1A presents results without covariates, and Figure 1B includes covariate regression and site correction (age, sex, head motion, and site). Neuroimaging modalities include (A) resting-state fMRI (rsfMRI), (B) Monetary Incentive Delay task (MID), (C) nBack task, (D) Stop-Signal Task (SST), (E) Diffusion Tensor Imaging fractional anisotropy (FA), and (F) T1-weighted gray matter structural MRI (T1 GM). The x-axis represents the difference in variance (positive values indicate greater variance in Black participants, while negative values indicate greater variance in White participants), and the y-axis represents the count of features with corresponding variance differences. Across all modalities, features demonstrating significant variance differences (p < 0.05) are included.

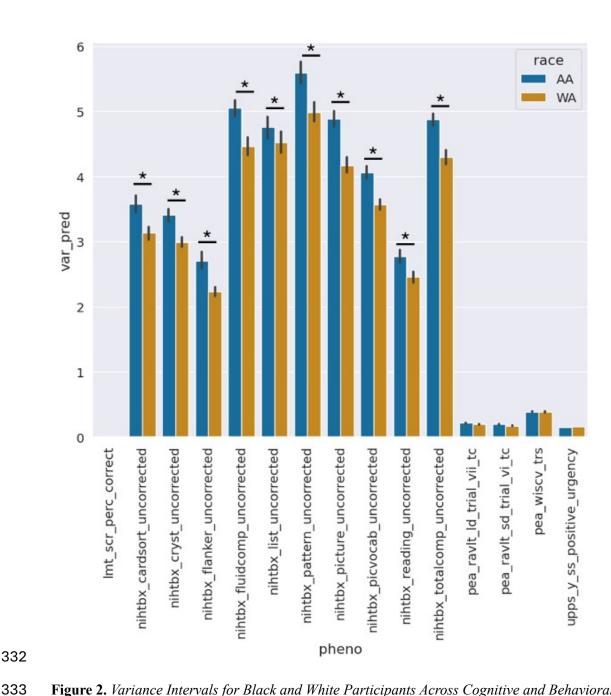


Figure 2. Variance Intervals for Black and White Participants Across Cognitive and Behavioral Tasks.

This figure illustrates the 95% variance intervals for Black and White participants in Connectome-Based Predictive Modeling (CPM), using resting-state fMRI connectivity to predict performance across 15 behavioral and cognitive tasks. The wider variance intervals for Black participants indicate increased variability in model predictions. Confidence intervals (CIs) were generated using a leave-3-sites-out bootstrapping approach over 1,000 iterations. In 10 of the 15 tasks, the variance for Black participants was significantly greater than for White participants (p < 0.05, FDR-corrected), demonstrating higher prediction uncertainty for Black participants. The tasks evaluated in this analysis include lmt_scr_perc_correct, which assessed working memory, and pcps_speed, which measured processing speed. Cognitive flexibility was measured by nihtbx_cardsort_uncorrected, while

nihtbx_flanker_uncorrected evaluated inhibitory control and attention. nihtbx_picturevocab_uncorrected assessed verbal intelligence, and nihtbx_reading_uncorrected evaluated reading ability. Impulsivity measures included uppps_y_ss_positive_urgency for positive urgency and uppps_y_ss_negative_urgency for negative urgency. The Monetary Choice Task was represented by cash_choice_perc_higher for delayed gratification and cash_choice_perc_smaller for preference for smaller immediate rewards. Additional tasks included cpt_dprime, which measured sustained attention, and ddl_total_cum_dscore and ddl_indifference_point, both assessing decision-making in delay discounting. Motivational influences on decision-making were evaluated by nacc_reinforce_bias, and wtar_uncorrected was used to assess pre-morbid intellectual functioning. In these tasks, the variance intervals were consistently wider for Black participants, particularly in measures related to working memory, inhibitory control, and impulsivity, underscoring the importance of accounting for variance differences when developing predictive models to ensure more equitable outcomes.

b_sd

w_sd

b_sd/w_sd

N

variable_names	P_Deliavior	I_DCHGAIOI	D_3u	W_3u	D_3U/W_3U	
cbcl_scr_syn_rulebreak_r	6.04646858816722e-65	294.3341208	2.342404174	1.649217262	1.42031267	9007
pgbi_p_ss_score	4.46604314945146e-59	266.5666511	3.519408775	2.438690549	1.443155129	9005
nihtbx_flanker_uncorrected	3.00250397369565e-54	243.8325318	10.81015897	8.196994281	1.318795476	8899
pps_y_ss_severity_score	2.78744420222196e-35	154.958436	12.18591678	9.60795525	1.268315314	9007
nihtbx_cardsort_uncorrected	1.68339864429146e-33	146.6879704	11.04408035	8.645392807	1.277452696	8900
lmt_scr_rt_correct	2.04433799311084e-26	113.846689	527.6762681	449.2832114	1.174484723	8765
pps_y_ss_number	3.14632839361133e-22	94.50330961	3.914569525	3.363105885	1.16397451	9006
upps_y_ss_positive_urgency	1.64459520568086e-19	82.00161389	3.198911434	2.830895446	1.129999851	9001
bis_y_ss_bas_drive	4.79564802116352e-19	79.86768186	3.290705699	2.932191959	1.122268168	9001
cbcl_scr_syn_aggressive_r	9.07797002462527e-19	78.59559795	5.007591376	4.155597116	1.205023306	9007
lmt_scr_perc_correct	1.3253772783297e-17	73.26515413	0.154085622	0.168816703	0.912739195	8768
cbcl_scr_syn_attention_r	1.25945509316862e-16	68.7796803	3.824114497	3.373571391	1.133550785	9007
cbcl_scr_syn_social_r	9.5129085602602e-15	60.19801799	2.52022957	2.188398518	1.151631912	9007
upps_y_ss_lack_of_planning	1.45008688258898e-13	54.8058633	2.57150919	2.322017522	1.107446075	9001
nihtbx_fluidcomp_uncorrected	1.31145517039e-12	50.4582502	11.14030346	9.822922332	1.134112954	8845
nihtbx_list_uncorrected	1.74222309122435e-12	49.89725501	12.63076482	11.27016869	1.120725445	8875
upps_y_ss_negative_urgency	4.37199668389686e-12	48.08078035	2.852918994	2.576014654	1.107493309	9001
cbcl_scr_syn_withdep_r	4.56389806147375e-10	38.94151411	1.888536221	1.646749665	1.14682654	9007
nihtbx_totalcomp_uncorrected	1.03934338516559e-09	37.33031551	9.231211867	8.221664893	1.122791063	8845
pea_wiscv_trs	4.38277541783795e-08	30.02482606	3.930705124	3.599203541	1.09210415	8838
bis_y_ss_bis_sum	4.75579033573064e-08	29.86496656	3.996035027	3.646203975	1.095943906	9001
pea_ravlt_sd_trial_vi_tc	1.451382480854e-07	27.69787812	3.159366394	2.915074164	1.083803092	8862
nihtbx_reading_uncorrected	3.09279704140809e-07	26.23096229	7.181428376	6.44683388	1.113946553	8895
cbcl_scr_syn_thought_r	3.09675609756959e-07	26.22798558	2.400566126	2.143449644	1.119954524	9007
upps_y_ss_sensation_seeking	5.64589789996424e-05	16.23339651	2.816170612	2.642571341	1.065693315	9001
nihtbx_pattern_uncorrected	0.000131616	14.63136155	15.07024641	14.21199047	1.060389566	8889
nihtbx_picture_uncorrected	0.000270671	13.27381405	11.43263821	12.0066545	0.952191821	8897
bis_y_ss_bas_fs	0.000442791	12.35152911	2.785740433	2.580763881	1.07942476	9001
nihtbx_cryst_uncorrected	0.002838287	8.913584971	6.835698782	6.522514194	1.048015931	8883
cbcl_scr_syn_anxdep_r	0.003991468	8.291967872	3.021992896	3.103539863	0.97372453	9007
cbcl_scr_syn_somatic_r	0.006887617	7.30541226	2.077063395	1.923682957	1.079732701	9007
lmt_scr_efficiency	0.023384802	5.141492584	0.067103029	0.069337076	0.967779905	8765

4.4079529

1.225755175

1.19460239

0.279455107

2.331296994

3.198346881

2.972596672

7.602234514

2.219419238

3.085944315

2.884979706

7.568627266

1.050408573

1.036424042

1.030370046

1.004440336

9001

8824

9001

8906

0.035799521

0.268264333

0.274432419

0.597071758

f_behavior

upps_y_ss_lack_of_perseverance

nihtbx picvocab uncorrected

pea_ravlt_ld_trial_vii_tc

bis_y_ss_bas_rr

342

343

344

345

346

347

348

349

350

351

352

353

variable_names

p_behavior

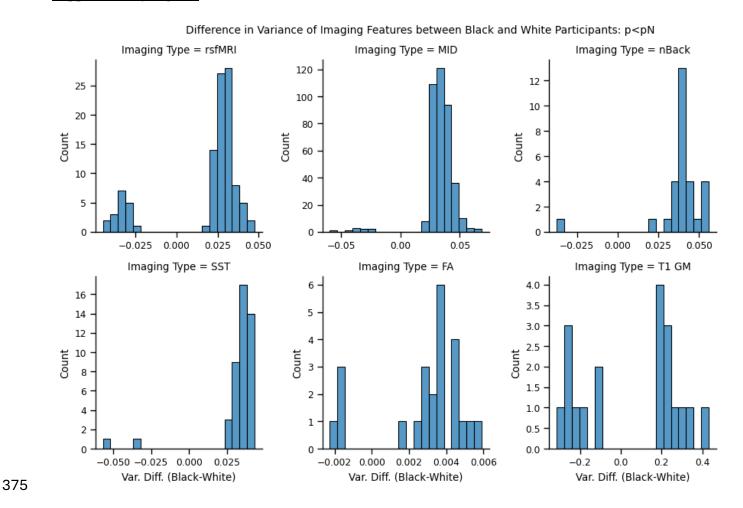
Table 1. Variance differences in behavioral measures between Black and White participants. The table presents results of Levene's test for variance differences across 36 behavioral measures, including the p-value (p_behavior), F-statistic (f_behavior), and standard deviations for Black (b_sd) and White (w_sd) participants. The ratio of standard deviations (b_sd/w_sd) quantifies the relative variability between the two groups, with values >1 indicating greater variability among Black participants and values <1 indicating greater variability among White participants. Measures with statistically significant variance differences (p < 0.05) are highlighted. Sample sizes (N) vary across measures due to data availability. These results highlight racialized patterns of variability in cognitive, emotional, and behavioral domains, with most measures showing greater variability among Black participants.

behavior	lev_stat	p_value
cbcl_scr_syn_anxdep_r_cpm	27.68885878	1.48E-07
cbcl_scr_syn_rulebreak_r_cpm	22.16599835	2.57E-06
pgbi_p_ss_score_cpm	21.39088771	3.84E-06
cbcl_scr_syn_somatic_r_cpm	20.98736643	4.73E-06
cbcl_scr_syn_thought_r_cpm	17.32967971	3.19E-05
upps_y_ss_lack_of_planning_cpm	15.13352067	0.000101452
upps_y_ss_positive_urgency_cpm	12.72602278	0.000363935
upps_y_ss_lack_of_perseverance_cpm	10.52367626	0.001186191
bis_y_ss_bas_drive_cpm	9.725175043	0.001827801
nihtbx_list_uncorrected_cpm	6.734315519	0.009484807
lmt_scr_perc_correct_cpm	5.217080963	0.022407227
pea_wiscv_trs_cpm	5.040174444	0.024808937
upps_y_ss_negative_urgency_cpm	4.187921048	0.040764471
lmt_scr_efficiency_cpm	3.826817067	0.050493469
pea_ravlt_ld_trial_vii_tc_cpm	3.643809445	0.056334441
bis_y_ss_bis_sum_cpm	3.510974745	0.061021064
bis_y_ss_bas_fs_cpm	2.128954403	0.144601284
pea_ravlt_sd_trial_vi_tc_cpm	2.047220328	0.152546011
nihtbx_picvocab_uncorrected_cpm	2.040252392	0.153245838
nihtbx_flanker_uncorrected_cpm	1.771998759	0.183194776
cbcl_scr_syn_attention_r_cpm	1.434651131	0.231062872
cbcl_scr_syn_aggressive_r_cpm	1.23238888	0.266995619
lmt_scr_rt_correct_cpm	1.168477882	0.27976564
cbcl_scr_syn_withdep_r_cpm	1.049970178	0.305562851
upps_y_ss_sensation_seeking_cpm	0.989842434	0.319828377
nihtbx_cryst_uncorrected_cpm	0.97817608	0.322696724
bis_y_ss_bas_rr_cpm	0.295175043	0.586947058
pps_y_ss_severity_score_cpm	0.132177542	0.716200048
pps_y_ss_number_cpm	0.069115695	0.792639389
nihtbx_totalcomp_uncorrected_cpm	0.046576312	0.829140282
nihtbx_fluidcomp_uncorrected_cpm	0.039905301	0.841673864
nihtbx_reading_uncorrected_cpm	0.012822364	0.909848037
cbcl_scr_syn_social_r_cpm	0.011149385	0.915911321
nihtbx_picture_uncorrected_cpm	0.001777261	0.966374742
nihtbx_cardsort_uncorrected_cpm	0.00025083	0.987364553
nihtbx_pattern_uncorrected_cpm	2.34E-06	0.998779071

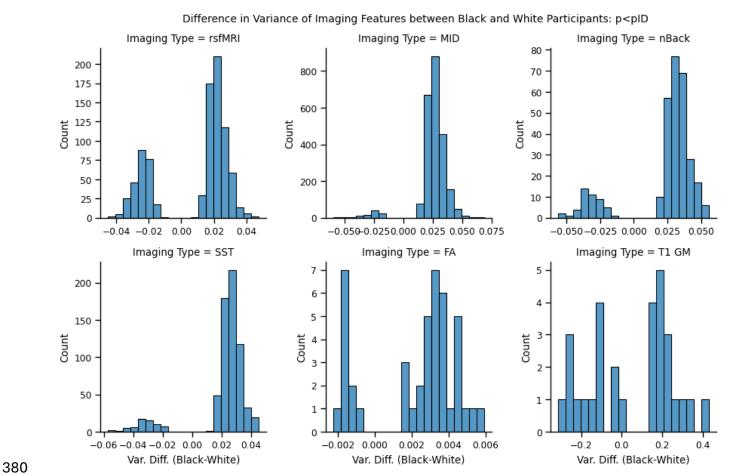
Table 2. Results of Levene's tests for racial differences in variance in composite CPM scores across behavioral measures. Each row corresponds to a behavioral measure, with Levene's test statistics (lev_stat) and associated p-values (p_value) reported. Composite CPM scores were calculated using the top 10% of edges most strongly correlated with each behavioral measure. Significant results (p < 0.05) are highlighted, indicating behavioral measures where variance differed significantly between Black and White participants. These results emphasize the

presence of racialized heteroscedasticity in CPM models and its potential impact on predictive accuracy across behavioral domains.

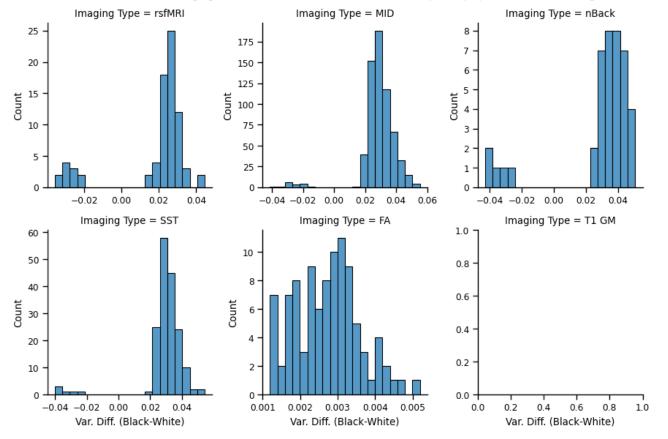
Supplementary Figures



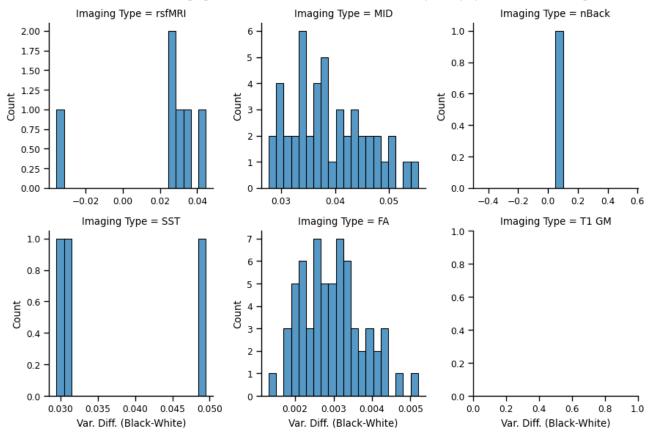
Supplementary Figure 1. Variance histograms for neuroimaging modalities (resting-state fMRI, DTI, T1-weighted structural MRI, MID, SST, and nBack tasks) under the less stringent FDR threshold (pN). The histograms illustrate the distribution of variance differences across features (Black-White), with positive values indicating greater variance for Black participants. Features with significant differences (p < 0.05) are highlighted.



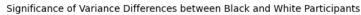
Supplementary Figure 2. Variance histograms for neuroimaging modalities (resting-state fMRI, DTI, T1-weighted structural MRI, MID, SST, and nBack tasks) under the more stringent FDR threshold (pID). The histograms show the distribution of variance differences across features (Black-White), emphasizing significant findings after strict correction for multiple comparisons.

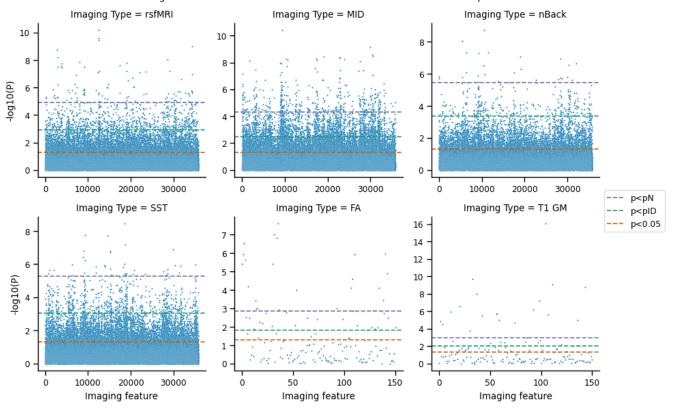


Supplementary Figure 3. Covariate-adjusted variance histograms for neuroimaging modalities (resting-state fMRI, DTI, T1-weighted structural MRI, MID, SST, and nBack tasks) under the less stringent FDR threshold (pN). Variance differences (Black-White) are adjusted for age, gender, head motion, and site effects, with significant features (p < 0.05) highlighted.

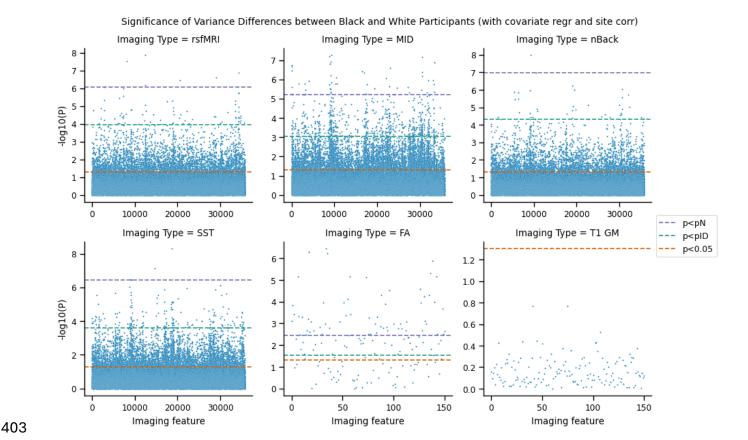


Supplementary Figure 4. Covariate-adjusted variance histograms for neuroimaging modalities (resting-state fMRI, DTI, T1-weighted structural MRI, MID, SST, and nBack tasks) under the more stringent FDR threshold (pID). These histograms reflect variance differences (Black-White) with covariate adjustments, focusing on features with significant differences (p < 0.05).





Supplementary Figure 5. Significance of variance differences between Black and White participants across six neuroimaging modalities. The -log10(p) values represent the statistical significance of Levene's test for each imaging feature. Dashed lines indicate significance thresholds: p < 0.05 (orange), less stringent FDR threshold (pN, purple), and more stringent FDR threshold (pID, green). Results are shown for resting-state fMRI (rsfMRI), task-based fMRI (Monetary Incentive Delay [MID], nBack, and Stop-Signal Task [SST]), Diffusion Tensor Imaging (FA), and T1-weighted structural MRI (T1 GM). Positive findings highlight features with greater variance in Black participants compared to White participants.



Supplementary Figure 6. Significance of variance differences between Black and White participants across six neuroimaging modalities, adjusted for covariates and site effects. The -log10(p) values represent the statistical significance of Levene's test for each imaging feature, after controlling for age, gender, head motion, and site effects. Dashed lines indicate significance thresholds: p < 0.05 (orange), less stringent FDR threshold (pN, purple), and more stringent FDR threshold (pID, green). Results are shown for resting-state fMRI (rsfMRI), task-based fMRI (Monetary Incentive Delay [MID], nBack, and Stop-Signal Task [SST]), Diffusion Tensor Imaging (FA), and T1-weighted structural MRI (T1 GM). The covariate adjustment ensures that observed variance differences are not confounded by demographic or site-specific factors.

Imaging Type	covar	p_thresh	positive_var_	negative_var	pchisq
FA		0.05	39	13	0.000311491
MID		0.05	7438	674	0
SST		0.05	4911	896	0
T1 GM		0.05	32	20	0.096092329
nBack		0.05	3076	1375	2.17193618357484e-143
rsfMRI		0.05	3572	2155	3.13599205760491e-78
FA		pID	33	11	0.000911119
MID		pID	2305	104	0
SST		pID	615	63	9.66553123960432e-100
T1 GM		pID	16	14	0.715000655
nBack		pID	264	47	8.51985534851393e-35
rsfMRI		pID	614	261	7.9098987534593e-33
FA		pN	20	4	0.001090835
MID		pN	383	9	1.3823123276378e-79
SST		pN	43	2	9.84400630995219e-10
T1 GM		pN	11	8	0.491297124
nBack		pN	28	1	5.33726360033909e-07
rsfMRI		pN	85	18	4.0645251997321e-11
FA	covar_regr_	0.05	56	1	3.21842226079615e-13
MID	covar_regr_	0.05	7444	503	0
SST	covar_regr_	0.05	5132	628	0
nBack	covar_regr_	0.05	2765		3.25620314478851e-138
rsfMRI	covar_regr_	0.05	3656		1.06028557928629e-175
T1 GM	covar_regr_	0.05	0		NA
FA	covar_regr_	pID	38		7.0744630989707e-10
MID	covar_regr_	pID	2270	60	0
SST	covar_regr_	pID	639		7.33282662642109e-121
nBack	covar_regr_	pID	136		2.17454108453107e-22
rsfMRI	covar_regr_	pID	473		9.60383335660292e-55
T1 GM		pID	0		NA
FA	covar_regr_	pN	19		1.3071845366763e-05
MID	covar_regr_		285		2.25331894650011e-61
SST	covar_regr_	pN	16		6.33424836662398e-05
nBack	covar_regr_	pN	9	1	
	covar_regr_	pN	69		0.011412036 3.61178637726065e-12
rsfMRI	covar_regr_	pN	0		NA
T1 GM	covar_regr_	pN 0.05	104		2.02316054223403e-24
FA	site_corr_covar_regr_				
MID	site_corr_covar_regr_	0.05	5398	430	0
SST	site_corr_covar_regr_	0.05	3968	545	0 1.41057371695111e-80
nBack	site_corr_covar_regr_	0.05	2126		
rsfMRI	site_corr_covar_regr_	0.05	2373		4.47286394361418e-85
T1 GM	site_corr_covar_regr_	0.05	0		NA
FA	site_corr_covar_regr_	pID	98		4.18382560777942e-23
MID	site_corr_covar_regr_	pID	616		6.79081461861592e-126
SST	site_corr_covar_regr_	pID	167		1.88625239988304e-34
nBack	site_corr_covar_regr_	pID	36		1.28936236148417e-06
rsfMRI	site_corr_covar_regr_	pID	66		3.66110147762755e-10
T1 GM	site_corr_covar_regr_	pID	0		NA
FA	site_corr_covar_regr_	pN	63		2.06706581807826e-15
MID	site_corr_covar_regr_	pN	47		7.09867044520102e-12
SST	site_corr_covar_regr_	pN	3	0	
nBack	site_corr_covar_regr_	pN	1	0	
rsfMRI	site_corr_covar_regr_	pN	5	1	0.102470435
T1 GM	site_corr_covar_regr_	pN	0	0	NA

Supplementary Table 1. Counts of features showing significantly greater variance in Black vs. White participants across neuroimaging modalities. Levene's test was used to assess variance differences for each feature, and a chi-square test was conducted to determine whether the distribution of features with greater variance differed significantly between groups. Positive values indicate the number of features with greater

- variance in Black participants, while negative values indicate greater variance in White participants. Results are shown for resting-state fMRI, task-based fMRI (Monetary Incentive Delay, Stop-Signal Task, and nBack tasks),
- 420 Diffusion Tensor Imaging (DTI), and T1-weighted structural MRI.

421 References

- 422 1. Falk EB, Hyde LW, Mitchell C, et al. What is a representative brain? Neuroscience meets population
- 423 science. *Proc Natl Acad Sci U S A* 2013; **110**(44): 17615-22.
- 424 2. LeWinn KZ, Sheridan MA, Keyes KM, Hamilton A, McLaughlin KA. Sample composition alters
- associations between age and brain structure. *Nat Commun* 2017; **8**(1): 874.
- 426 3. Assari S. Health Disparities due to Diminished Return among Black Americans: Public Policy Solutions.
- 427 *Social Issues and Policy Review* 2018; **12**(1): 112-45.
- 428 4. Assari S. Unequal Gain of Equal Resources across Racial Groups. *Int J Health Policy Manag* 2018; 7(1):
- 429 1-9.
- 430 5. Fields C, Black C, Thind JK, et al. Governance for Anti-Racist AI in Healthcare: Integrating Racism-
- 431 Related Stress in Psychiatric Algorithms for Black Americans. *PsyArXiv (Preprint)* 2024.
- 432 6. Berger M, Sarnyai Z. "More than skin deep": stress neurobiology and mental health consequences of
- 433 racial discrimination. *Stress* 2015; **18**(1): 1-10.
- 434 7. Fani N, Harnett NG, Bradley B, et al. Racial Discrimination and White Matter Microstructure in Trauma-
- 435 Exposed Black Women. *Biol Psychiatry* 2022; **91**(3): 254-61.
- Baek EJ, Jung HU, Chung JY, et al. The effect of heteroscedasticity on the prediction efficiency of
- genome-wide polygenic score for body mass index. Front Genet 2022; 13: 1025568.
- 438 9. Breusch TS, Pagan AR. A Simple Test for Heteroscedasticity and Random Coefficient Variation.
- 439 *Econometrica* 1979; **47**(5): 1287-94.
- 440 10. Godfrey LG, Orme CD. The robustness, reliability and power of heteroskedasticity tests. *Econometric*
- 441 Reviews 2007; **18**(2): 169-94.
- 442 11. White H. A Heteroskedasticity-Consistent Covariance Matrix Estimator and a Direct Test for
- 443 Heteroskedasticity. *Econometrica* 1980; **48**(4).
- Volkow ND, Koob GF, Croyle RT, et al. The conception of the ABCD study: From substance use to a
- broad NIH collaboration. *Dev Cogn Neurosci* 2018; **32**: 4-7.
- 446 13. Cogburn CD, Roberts SK, Ransome Y, Addy N, Hansen H, Jordan A. The impact of racism on Black
- American mental health. *Lancet Psychiatry* 2024; **11**(1): 56-64.

- 448 14. Obermeyer Z, Powers B, Vogeli C, Mullainathan S. Dissecting racial bias in an algorithm used to manage
- 449 the health of populations. *Science* 2019; **366**(6464): 447-53.
- 450 15. Ricard JA, Parker TC, Dhamala E, Kwasa J, Allsop A, Holmes AJ. Confronting racially exclusionary
- practices in the acquisition and analyses of neuroimaging data. *Nat Neurosci* 2023; **26**(1): 4-11.
- 452 16. Greene AS, Shen X, Noble S, et al. Brain-phenotype models fail for individuals who defy sample
- 453 stereotypes. *Nature* 2022; **609**(7925): 109-18.
- Li J, Bzdok D, Chen J, et al. Cross-ethnicity/race generalization failure of behavioral prediction from
- resting-state functional connectivity. *Sci Adv* 2022; **8**(11): eabj1812.
- 456 18. Assari S, Mincy R. Racism May Interrupt Age-related Brain Growth of African American Children in the
- 457 United States. *J Pediatr Child Health Care* 2021; **6**(3).
- 458 19. Hatzenbuehler ML, Weissman DG, McKetta S, et al. Smaller Hippocampal Volume Among Black and
- Latinx Youth Living in High-Stigma Contexts. *J Am Acad Child Adolesc Psychiatry* 2022; **61**(6): 809-19.
- 460 20. Williams DR, Lawrence JA, Davis BA. Racism and Health: Evidence and Needed Research. Annu Rev
- 461 *Public Health* 2019; **40**: 105-25.
- 462 21. Williams DR, Mohammed SA. Racism and Health I: Pathways and Scientific Evidence. Am Behav Sci
- 463 2013; **57**(8).
- Basu A. Use of race in clinical algorithms. Sci Adv 2023; 9(21): eadd2704.
- 465 23. Pfohl SR, Foryciarz A, Shah NH. An empirical characterization of fair machine learning for clinical risk
- prediction. *Journal of Biomedical Informatics* 2021; **113**.
- 467 24. Tishkoff SA, Reed FA, Friedlaender FR, et al. The genetic structure and history of Africans and African
- 468 Americans. *Science* 2009; **324**(5930): 1035-44.
- 469 25. Farah MJ. The Neuroscience of Socioeconomic Status: Correlates, Causes, and Consequences. *Neuron*
- **470** 2017; **96**(1): 56-71.
- 471 26. Hackman DA, Farah MJ. Socioeconomic status and the developing brain. *Trends Cogn Sci* 2009; **13**(2):
- 472 65-73.
- 473 27. Fani N, Carter SE, Harnett NG, Ressler KJ, Bradley B. Association of Racial Discrimination With Neural
- Response to Threat in Black Women in the US Exposed to Trauma. *JAMA Psychiatry* 2021; **78**(9): 1005-12.

- Webb EK, Carter SE, Ressler KJ, Fani N, Harnett NG. The neurophysiological consequences of racism-
- 476 related stressors in Black Americans. *Neurosci Biobehav Rev* 2024; **161**: 105638.
- 477 29. Yudell M, Roberts D, DeSalle R, Tishkoff S. SCIENCE AND SOCIETY. Taking race out of human
- 478 genetics. *Science* 2016; **351**(6273): 564-5.
- 479 30. 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* 2005; **60**(1): 16-26.
- 481 31. Omi M, Winant H. Racial formation in the United States. Third edition. ed. New York: Routledge/Taylor
- 482 & Francis Group; 2015.
- 483 32. Breland-Noble A, Streets FJ, Jordan A. Community-based participatory research with Black people and
- Black scientists: the power and the promise. *Lancet Psychiatry* 2024; **11**(1): 75-80.
- 485 33. George S, Duran N, Norris K. A systematic review of barriers and facilitators to minority research
- 486 participation among African Americans, Latinos, Asian Americans, and Pacific Islanders. Am J Public Health
- 487 2014; **104**(2): e16-31.