

Article

Subjective Socioeconomic Status and Children's Amygdala Volume: Minorities' Diminish Returns

Shervin Assari ^{1,2,*} , Shanika Boyce ³ and Mohsen Bazargan ^{1,4}

¹ Department of Family Medicine, Charles R Drew University of Medicine and Science, Los Angeles, CA 90059, USA; Mohsenbazargan@cdrewu.edu

² Department of Urban Public Health, Charles R Drew University of Medicine and Science, Los Angeles, CA 90059, USA

³ Department of Pediatrics, Charles R Drew University of Medicine and Science, Los Angeles, CA 90059, USA; ShanikaBoyce@cdrewu.edu

⁴ Department of Family Medicine, University of California, Los Angeles (UCLA), Los Angeles, CA 90095, USA

* Correspondence: assari@umich.edu; Tel.: +1-734-232-0445; Fax: +1-734-615-8739

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Abstract: Considerable research has suggested that low socioeconomic status (SES) negatively influences brain structure, including but not limited to decreased amygdala volume. Considering race and ethnicity as sociological rather than biological constructs, this study was built on minorities' diminished returns (MDRs) to test if the effects of family SES on the total amygdala volume is weaker for black and Latino children than white and non-Latino children. We borrowed data from the Adolescent Brain Cognitive Development (ABCD) study, a national multi-center brain imaging investigation of childhood brain development in the US. The total sample was 9380 9–10-year-old children. The independent variables were subjective family SES and parental education. The primary outcome was total amygdala volume. High subjective SES and parental education were independently associated with larger total amygdala size. The association between high subjective SES and larger total amygdala volume was less pronounced for black and Latino children than white and non-Latino children. For American children, family SES has unequal effects on amygdala size and function, a pattern that is consistent with MDRs. This result suggests that SES loses some of its expected effects for racial and ethnic minority families.

Keywords: socioeconomic status; socioeconomic position; amygdala; limbic system; negative emotion; emotion regulation; brain development; structural MRI

1. Introduction

High socioeconomic status (SES) is associated with positive childhood emotions and behaviors [1]. For example, children from higher SES families are protected against school drop-out [2], depression [3], suicide [4,5], antisocial behaviors [6], aggression [7], and use of tobacco [8,9], alcohol [10,11], and drugs [12]. These behavioral effects of SES are mainly attributed to the protective effects of high SES on childhood development.

The scarcity hypothesis provides an explanation for why low SES deteriorates healthy children's brain development. According to this hypothesis, low SES is a proxy of early adversity, stress, economic insecurity, and lack of resources, all operating as risk factors for poor child development. In this view, stress, adversity, and scarce resources explain the SES–brain development link [13]. Low family SES is a proxy of living in stressful environments, food insecurity, environmental toxins, and parental risk behaviors that can jeopardize healthy brain development in children [14–16]. As a result of

unhealthy brain development, children from low SES are at an increased risk of various types of psychopathologies [17–19]. In contrast, children from high SES families experience lower levels of stress, have access to stimulating environments, receive better parenting, and have access to buffers when stress occurs [20–22].

According to the minorities' diminished returns (MDRs) [23,24], however, racial groups may differ in the protective effect of high SES on brain development and function. Compared to white children, black American children show lower effects of family SES on developmental outcomes [25] such as school performance [25], mental health [26], emotion regulation [27,28], aggression [29], and substance use [26,30]. In a study, high-income black boys were more, not less, likely to be depressed [31]. Similar patterns are shown for black men [32]. In one study in blacks, the effect of discrimination on depression was larger at higher SES, suggesting that high SES may increase vulnerability to discrimination [33]. In many other studies among blacks, high SES was associated with increased proximity to whites [34,35], which increases their exposure to discrimination. Other studies have also shown a positive rather than inverse association between SES and discrimination, suggesting that high SES blacks experience more, not less, discrimination [32,34–38]. Similarly, the effects of neighborhood quality [39] and social contacts [40] are weaker for blacks than whites.

Research has established racial/ethnic differences in each SES indicator's role in children's brain development [20,41–43]. In several studies, the magnitude of the effects of family SES on a wide range of developmental and health outcomes is weaker for blacks and Latinos than whites and non-Latinos [32,36,44–49]. As a result of MDRs, middle-class ethnic minority children remain at risk for poor developmental and health outcomes [50–54]. For example, high SES black and Latino children remain at risk of anxiety [55], depression [31], poor health [45], poor school performance [56,57], as well as high-risk behaviors [54] such as aggression [54] and tobacco use [58,59]. Differential effects of SES across racial and ethnic groups of children are robust [44–46,60,61]. Data from the fragile families and child wellbeing study (FFCWS) shows that high parental education and family income is associated with better outcomes in impulsivity, school performance, school bonding, attention-deficit/hyperactivity disorder (ADHD), obesity, aggression, depression, and self-rated health for non-Latino and white children than Latino and black American children [52,61–63]. Subjective SES and parental education each impact brain imaging findings in a certain way [20,41–43]. Various SES indicators may also be the underlying mechanisms by which racial and ethnic disparities emerge in children's development [44,45,64].

At least some of the effects of high SES on emotional and behavioral outcomes [1] can be attributed to the role of family SES on structure and function of the amygdala [20]. Across various brain structures, the amygdala has shown the most consistent pattern of association with SES [20,42,65]. While amygdala reactivity has a central role in emotion regulation, altered size and function of the amygdala is shown to predict high-risk behaviors such as aggression and substance use [66–70].

For at least three reasons, the intersections of race, SES, and amygdala structure was analyzed in this study. First, most of the literature is conducted mainly in white samples. For example, all studies by Javanbakht et al. were conducted exclusively or mainly in white individuals [20,42,65]. There is a need for brain imaging data from large, racially, and ethnically diverse samples to compare racial and ethnic groups. Most previous studies have used sample sizes of less than 100 or at most a couple of hundred participants [20,42,65]. Studies with large sample sizes are rare. As such, they are needed to compare diverse subsamples. The adolescents brain cognitive development (ABCD) study has a large sample size [71–75], which enhances the statistical power and generalizability of the results for comparison of black, white, Latino, and non-Latino groups for the effects of SES on children brain imaging. Thus, using large datasets, there is still a need for research on whether race and ethnicity change the functional and structural brain correlates of SES resources.

Second, there is a need to advance the current literature, where more information exists on the effects of family SES during childhood on brain structure in adulthood and less is known about the effects of childhood SES on childhood brain structure [20,41–43]. While we know that childhood poverty is a predictor of undesired brain changes during adulthood [20,41–43,76], less is known about

the brain imaging finding of children while living under poverty. For example, all studies by Javanbakht investigated the effects of childhood family SES on adulthood brain function and structure [20,42,65]. Thus, we need to extend the research from the well demonstrated role of childhood SES in predicting adults' neural circuits to the connection of childhood poverty to childhood brain development.

The third reason is a need for additional studies exploring the salience of family SES indicators other than income and poverty. There is particularly a need to study the effects of subjective SES and parental education on American children's brain function [20,41–43,76]. Thus, we need more studies that compare the effects of subjective family SES on neural regulatory structures and functions across diverse groups [77].

Aims

To extend the existing knowledge on the complexities of social determinants of children's brain development in the US, we explored racial and ethnic group variations in the effects of two family SES indicators, namely subjective family SES and parental education, on total amygdala volume among 9–10-year-old children. We expected to find racial and ethnic differences in the magnitude of the association between our family SES indicators and total amygdala volume, in line with the observed MDRs [23,24,54]. More specifically, we expected to find weaker SES effects on total amygdala volume for black and Latino than white and non-Latino children. This expectation is in line with the other research on a wide range of phenotypes and behaviors [23,24,54].

2. Materials and Methods

2.1. Design and Settings

We conducted a secondary analysis of the Adolescent Brain Cognitive Development (ABCD) study data [71–75]. With a cross-sectional design, we applied data from the baseline of the ABCD study. ABCD is a national, state-of-the-art brain imaging study of childhood brain development [71,78]. The advantages of the ABCD study include a national sample, a large sample size, a large sample of blacks and Latinos, available data, robust measures of brain development, and considerable socioeconomic factors [71–75].

2.2. Participants and Sampling

Participants of the ABCD study were selected across multiple cities across states. This ABCD sample was primarily recruited through school systems with sampling (school selection) informed by race, ethnicity, sex, SES, and urbanicity. More details of ABCD sampling are published elsewhere [79]. Inclusion criteria were being an either non-Latino, Latino, black, or white child between ages 9 and 10, and having valid data on total amygdala volume. We excluded any child from any other race/ethnicity (Asian, mixed, other).

2.3. Study Process

Structural magnetic resonance imaging (sMRI) data were used to measure total amygdala volume. As described in detail by Casey et al. (2018), participants completed a high-resolution T1-weighted structural MRI scan (1-mm isotropic voxels) using scanners from Philips Healthcare (Philips, Andover, Massachusetts, USA), GE Healthcare (General Electrics, Waukesha, WI, USA), or Siemens Healthcare (Siemens, Erlangen, Germany) [72]. All the structural MRI data were processed using FreeSurfer version 5.3.0, available at <http://surfer.nmr.mgh.harvard.edu/> [80,81], according to standard processing pipelines [72]. Processing included removal of nonbrain tissue, segmentation of gray and white matter structures [82], and cortical parcellation [83]. All scan sessions underwent radiological review whereby scans with incidental findings were identified. Quality control for the structural images comprised visual inspection of T1 images and FreeSurfer outputs for quality [84]. Quality review was conducted by the ABCD team. Subjects whose scans failed inspection (due to severe artifacts or irregularities)

were excluded. The Desikan–Killiany atlas was used for cortical parcellation [84]. Regions of interest included caudal middle frontal, lateral orbitofrontal, medial orbitofrontal, rostral middle frontal, superior frontal, and frontal pole. In this analysis, we used the volumetric data provided by the ABCD data.

2.4. Study Variables

The study variables included demographic factors, family SES indicators, and total amygdala volume. A detailed explanation of the procedures and harmonization of the structural MRI in the ABCD study is available here [72].

2.4.1. Primary Outcome

The primary outcome was total amygdala volume, measured by structural MRI. Amygdala volume is shown to be under the influence of exposure to poverty, trauma, and adversity [65,85,86].

2.4.2. Moderator

Race. Race, a self-identified and a dichotomous variable, was coded as 1 for black and 0 for white (reference).

Ethnicity. Parents were asked if they are of Latino ethnic background. This variable was coded as Latino = 1 and non-Latino = 0.

Independent Variable

Subjective Family SES. Subjective family SES in this study was financial difficulties measured by the following seven items: “In the past 12 months, has there been a time when you and your immediate family experienced any of the following:” (1) “Needed food but could not afford to buy it or could not afford to go out to get it?”, (2) “Were without telephone service because you could not afford it?”, (3) “Did not pay the full amount of the rent or mortgage because you could not afford it?”, (4) “Were evicted from your home for not paying the rent or mortgage?”, (5) “Had services turned off by the gas or electric company, or the oil company would not deliver oil because payments were not made?”, (6) “Had someone who needed to see a doctor or go to the hospital but did not go because you could not afford it?”, and (7) “Had someone who needed a dentist but could not go because you could not afford it?” Responses to each item were either 0 or 1. We calculated a mean score with a potential range between 0 and 1—a higher score indicating higher subjective family SES. Our variable was a continuous measure [33,87–92].

Parental Educational Attainment. Participants reported their years of schooling. This variable was operationalized as a continuous (interval) variable ranging from 0 for no formal education to 21 doctoral degrees.

2.4.3. Confounders

Age. Age was a dichotomous variable coded 1 or 0 for 10 years and 9 years of age. Parents reported the age of the children.

Sex. Sex was 1 for males and 0 for females.

Parental marital status. Parental marital status was 1 for married and 0 for any other condition (reference).

Parental employment status. Parental employment status was 1 for employed and 0 for unemployed (reference).

2.5. Data Analysis

We used SPSS 22.00 (IBM, NY, USA) for data analysis. Frequencies (n and %) and mean (standard deviations (SDs)) were reported for descriptive purposes. To estimate bivariate analyses between the study variables, we used the Pearson correlation test in the pooled sample. To perform our

multivariable analyses, we performed multiple linear regressions. The independent variable was the family SES. The outcome was the total amygdala volume. All these models controlled for all confounders. All models were performed in the pooled sample. Model 1 did not have interaction effects. Model 2 was performed with two interaction terms. For sensitivity analysis, we also ran models that included total intracranial volume as a covariate. Adding this variable did not alter our results of interaction. Thus, we reported the most parsimonious model (models without total intracranial volume as a covariate). Unstandardized regression coefficient (b), SE, and *p*-values were reported for each model. A *p*-value of equal or less than 0.05 was significant.

2.6. Ethical Aspect

Our analysis was exempt from a full review. The ABCD study protocol, however, was approved by the University of California, San Diego (UCSD) Institutional Review Board (IRB) [78].

3. Results

3.1. Descriptives

The sample included 9380 9–10-year-old children. Table 1 presents the descriptive statistics of the pooled sample.

Table 1. Descriptive data overall (*n* = 9380).

Characteristics	<i>n</i>	%
Race		
White	7181	76.6
Black	2199	23.4
Ethnicity		
Non-Latino	7779	82.9
Latino	1601	17.1
Sex		
Male	4443	47.4
Female	4937	52.6
Age (year)		
9	5052	53.9
10	4328	46.1
Parental employment status		
Employed	2906	31.0
Unemployed	6474	69.0
Parental marital status		
Not married	3041	32.4
Married	6339	67.6
	Mean	SD
Parental education (years)	16.71	2.63
Subjective family SES	0.93	0.16
Amygdala volume	1563.26	231.67

3.2. Unadjusted Bivariate Correlations

Table 2 presents the results of the unadjusted bivariate correlations based on the Spearman test. Family SES is positively correlated with the total amygdala volume.

Table 2. Bivariate associations ($n = 9380$).

	1	2	3	4	5	6	7	8	9
1 Race (black)	1.00	-0.12 **	-0.02	0.00	-0.41 **	-0.05 **	-0.30 **	-0.27 **	-0.18 **
2 Ethnicity (Latino)		1.00	-0.01	-0.02	-0.08 **	-0.04 **	-0.19 **	-0.07 **	-0.05 **
3 Sex (male)			1.00	0.02	0.01	-0.01	-0.01	-0.01	0.32 **
4 Age (10)				1.00	0.00	0.01	0.00	0.02	0.07 **
5 Parents married					1.00	0.03 **	0.36 **	0.30 **	0.12 **
6 Parents employed						1.00	0.25 **	0.13 **	0.04 **
7 Parental education (years)							1.00	0.34 **	0.11 **
8 Subjective family SES								1.00	0.10 **
9 Total amygdala volume									1.00

** $p < 0.01$.

3.3. Pooled-Sample Associations

Table 3 reports the results of two pooled sample regression models. Model 1, which only included the main effects, showed that high subjective family SES and parental education were associated with total amygdala volume. Model 2 showed that subjective family socioeconomic status (SES) and race and ethnicity interact, meaning that the effects were less pronounced for black and Latino than white and non-Latino children. These interactions were statistically significant. Parental education did not show a statistically significant interaction between race and ethnicity.

3.4. Stratified Associations

Table 4 reports the results of four regression models by race. Model 3, which was conducted in whites, showed that high subjective family SES and parental education were associated with total amygdala volume. Model 4, which was conducted in blacks, did not show an effect of subjective family SES or parental education on total amygdala volume.

3.5. Stratified Associations

Table 5 reports the results of four regression models by ethnicity. Model 5, which was conducted in non-Latinos, showed that high subjective family SES and parental education were associated with total amygdala volume. Model 6, which was conducted in Latinos, showed an effect of parental education, but not subjective family SES on total amygdala volume.

Table 3. Linear regressions in the pooled sample ($n = 9380$).

	Model 1						Model 2					
	b	SE	95% CI		t	p	b	SE	95% CI		t	p
Race (black)	-78.52	5.99	-90.27	-66.78	-13.11	<0.001	35.49	40.78	-44.46	115.43	0.87	0.384
Ethnicity (Hispanic)	-27.13	6.19	-39.26	-14.99	-4.38	<0.001	76.07	43.12	-8.46	160.60	1.76	0.078
Sex (male)	145.28	4.44	136.58	153.97	32.75	<0.001	145.40	4.43	136.71	154.10	32.79	<0.001
Age (10)	28.59	4.44	19.88	37.30	6.44	<0.001	28.62	4.44	19.92	37.33	6.45	<0.001
Married household	16.92	5.46	6.22	27.63	3.10	0.002	16.43	5.48	5.68	27.17	3.00	0.003
Parents employed	10.51	4.97	0.77	20.25	2.11	0.034	11.05	4.97	1.30	20.80	2.22	0.026
Parental education (years)	3.99	0.98	2.06	5.91	4.05	0.000	4.75	1.44	1.93	7.58	3.30	0.001
Subjective family SES	44.19	14.66	15.44	72.94	3.01	0.003	95.68	23.14	50.32	141.04	4.14	<0.001
Parental education (years) x race							-3.47	2.20	-7.78	0.84	-1.58	0.114
Parental education (years) x ethnicity							0.09	2.09	-4.02	4.19	0.04	0.968
Subjective family SES x race							-63.86	30.46	-123.56	-4.16	-2.10	0.036
Subjective family SES x ethnicity							-110.82	37.28	-183.89	-37.76	-2.97	0.003

Table 4. Linear regressions by race ($n = 9380$).

	Model 3						Model 4					
	b	SE	95% CI		t	p	b	SE	95% CI		t	p
Ethnicity (Hispanic)	-32.20	6.94	-45.81	-18.60	-4.64	<0.001	14.89	15.43	-15.36	45.15	0.97	0.335
Sex (male)	150.34	5.13	140.28	160.41	29.28	<0.001	130.11	8.75	112.95	147.27	14.87	<0.001
Age (10)	31.34	5.14	21.26	41.42	6.10	<0.001	19.13	8.77	1.94	36.33	2.18	0.029
Married household	11.85	6.55	-0.99	24.69	1.81	0.070	28.10	9.88	8.73	47.47	2.85	0.004
Parents employed	7.87	5.78	-3.47	19.21	1.36	0.174	19.73	9.66	0.78	38.67	2.04	0.041
Parental education (years)	4.73	1.17	2.44	7.03	4.05	<0.001	0.22	1.86	-3.42	3.86	0.12	0.907
Subjective family SES	63.40	20.20	23.81	103.00	3.14	0.002	17.14	20.69	-23.44	57.71	0.83	0.408

Table 5. Linear regressions by ethnicity ($n = 9380$).

	Model 5					Model 6						
	b	SE	95% CI		t	p	b	SE	95% CI		t	p
Race (black)	-82.21	6.67	-95.28	-69.14	-12.33	<0.001	-41.32	15.89	-72.48	-10.16	-2.60	0.009
Sex (male)	143.35	4.92	133.71	152.99	29.15	<0.001	155.26	10.21	135.24	175.28	15.21	<0.001
Age (10)	28.06	4.92	18.42	37.71	5.70	<0.001	30.30	10.26	10.17	50.43	2.95	0.003
Married household	20.13	6.32	7.75	32.51	3.19	0.001	1.91	10.79	-19.26	23.09	0.18	0.859
Parents employed	11.86	5.54	0.99	22.72	2.14	0.032	6.96	11.18	-14.98	28.89	0.62	0.534
Parental education (years)	3.14	1.23	0.73	5.54	2.56	0.011	4.98	1.63	1.79	8.18	3.06	0.002
Subjective family SES	58.06	16.64	25.43	90.68	3.49	<0.001	-14.37	30.71	-74.60	45.86	-0.47	0.640

4. Discussion

This study had two primary findings. Although higher subjective family socioeconomic status (SES) and parental education predicted a larger volume of the amygdala (first finding), subjective family SES showed a stronger effect for white and non-Latino than black and Latino children (second finding).

Our first results can be compared with the literature on the effects of SES and adversity on amygdala volumes. These effects may be because stress, adversities, trauma, maternal depression, and SES impact children's amygdala volume [65,85,86,93–100]. There are multiple studies that have documented enlarged amygdala volumes in children who have been raised in an orphanage [98], have had maternal depression [101]), or have had infant attachment insecurity [102]. However, in our study, high SES was associated with larger amygdala size. Our study finding is more in line with the results of studies showing smaller amygdala size in low SES children. For example, Merz et al. studied 1196 children and adolescents who were between ages of 3 and 21. These individuals were selected from the pediatric imaging, neurocognition, and genetics study and showed that lower family income and parental education are significantly associated with smaller amygdala volume in adolescents (13–21 years) but not in younger children (3–12 years) [86].

While our first finding documented a link between SES and total amygdala volume, most of the literature on the SES–amygdala relationship has focused on amygdala function rather than structure. For example, three studies by Javanbakht have linked high family SES to amygdala response to negative facial expressions. While social adversities show cumulative (additive) effects on amygdala structure and function (including amygdala response to threatening faces) [65], these effects may be more pronounced for females than males [42]. Childhood poverty is also linked to a reduced functional connectivity between the amygdala and the medial prefrontal cortex [20]. Brody et al. also documented a link between poverty and a reduced connectivity in neural networks involved in emotion regulation (e.g., amygdala) [103]. Barch et al. documented a link between childhood poverty and reduced connectivity between the amygdala and hippocampus with brain regions including the lingual gyrus, superior frontal cortex, posterior cingulate, as well as putamen [104]. The literature has also shown the effects of family SES indicators such as poverty and household income on the brain [77] and behavior [20,41–43,76].

Our second results can be explained through two mechanisms. First, there is high exposure to racial and ethnic discrimination in high SES black and Latino families. Racial and ethnic discrimination affect the amygdala's structure and function [105–110]. In a study, discrimination was associated with increased connectivity with multiple brain regions. In the presence of racial discrimination, the amygdala shows a more robust connection with the thalamus (place reference here). Similarly, chronic discrimination increases connections between the amygdala and the putamen, anterior insula, the caudate, medial frontal gyrus, and anterior cingulate [105]. As high SES, particularly high subjective SES, is a proxy of high not low discrimination [32–38,111,112], high SES black American children still report lower than expected amygdala sizes, because of the effect of discrimination.

Our second finding can be also seen as a reflection of the MDRs. Many studies have shown more significant effects of SES on outcomes for white than black American children [44,45,113].

For example, family SES has shown larger effects on ADHD [63], anxiety [55], aggression [54], tobacco dependence [54], school bonding [114], school performance [56,57], obesity [52], and health [51] for white than black American children. Family SES has also shown a more salient role in shaping the impulsivity of white than black American children [50]. As a result of this pattern, higher than expected risk of poor self-rated health, obesity, poor mental health, chronic disease, impulsivity, aggression, smoking, and low school performance are observed in high SES black American children [52,61,63]. The higher than expected risks for high SES black American children seem to be robust as they hold across SES indicators, outcomes, population groups, birth cohorts, age groups, and settings [23,24].

As shown by this study and previous work [23], family SES may differently influence the outcomes of black and white children [60,115], adolescents [116], adults [117], and older adults [118,119]. In an unequal society, not only parental education [54] but also their own educational attainment [58,62,120], employment [111], marital status [46], and even coping style [121,122] generates unequal outcomes for blacks and whites. Regardless of their types, SES resources seem to always generate unequal effects for blacks and whites, a pattern which may be indicative of social stratification, segregation, and deeply rooted societal inequalities [123–132].

Differential effects of family SES indicators for black and white families contribute to the transgenerational transmission of inequalities [50–54]. Differential effects of SES mean that the same level of SES may generate unequal outcomes for the next generation, which results in the reproduction of inequalities across generations. However, most of the previous studies on MDRs have relied on self-reported outcomes. Thus, the evidence lacked biological studies that test the differential effects of SES on children's brain imaging. This paper extends the existing literature by testing such patterns on brain development.

The observed MDRs suggest that Latino and black American children are at two jeopardies. The first risk is that they live in low SES families. The second risk is that their SES shows a weaker impact on their brain development. The weakened effect of high SES for black and Latino children may be due to other unique stressors in the lives of racial and ethnic minorities across all SES levels.

There is a need for an enhanced understanding of the role of the amygdala in explaining why high SES shows a more significant impact on children's behavioral and emotional outcomes of white and non-Latino than black and Latino families. Without such knowledge, our policymakers may fall into a false thinking trap that suggests equalizing SES may be enough to equalize the brain function of racial and ethnic groups. However, past research has shown that high SES may still be associated with some residual adversities for non-white families. For example, high SES black families remain in poor neighborhoods, experience high levels of stress, have a poor diet, are more obese, have higher risk social networks, and are sent to high-risk schools (add references here). As a result, despite family-level SES, black and Latino children remain at risk of poor developmental outcomes across multiple emotional and behavioral domains.

It should be emphasized again that we see race and ethnicity as social factors (as proxy of social status, treatment by society, access to opportunity structure, interpersonal discrimination, environmental injustices, societal obstacles, and historical injustice) on how the individual is treated by society. Across various brain mechanisms, we focused on the amygdala, which is highly involved in emotion regulation, emotion expression, social relations, aggression, and impulsivity. An alteration of the amygdala volume and function is expected to be involved in many outcomes across various domains. As our results suggested, race and ethnicity alter the implications of family SES for amygdala structure and function, which are at the core of and salient to brain functions across domains.

5. Conclusions

In summary, in a large national sample of American children, high subjective family SES correlates with larger amygdala volume. However, this effect is unequal across racial and ethnic groups with the marginal return of SES being smaller for racial and ethnic minority families than non-Latino and white people. Policy solutions that wish to achieve equality should go beyond SES inequalities and be aware

that SES generates unequal outcomes across racial and ethnic groups. More research is needed on implications of the observations that black, white, Latino, and non-Latino children may show different associations between SES and brain development. The influence of social determinants on children's brain development is complex and multiplicative rather than simple and additive.

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