Anger, Fear, and Sadness: Relations to Socioeconomic Status and the Amygdala

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Abstract

■ Here, we test three often proposed hypotheses about socioeconomic status (SES), affect, and the brain, for which evidence is mixed or lacking. The first hypothesis, that negative affect is more common at lower levels of SES, has ample evidence from studies of psychiatric symptoms but is tested for the first time here across multiple measures of negative emotions in healthy young adults. The second hypothesis is actually a set of hypotheses, that SES is associated with three structural and functional properties of the amygdala. Third, and most important for the affective neuroscience of SES, is the hypothesis that SES differences in the amygdala are responsible for the affective differences. Despite the intuitive appeal of this hypothesis, it has rarely been tested and has never been confirmed. Here, we review the literature for evidence on each of these hypotheses and find in a number of cases that the evidence is weak or nonexistant. We then subject each hypothesis to a new empirical test with a large sample of healthy young adults. We confirm that negative affect is more common at lower levels of SES and we find a positive relation between SES and amygdala volume. However, evidence is weak on the relation of SES to functional properties of amygdala. Finally, the tendency toward negative affect in lower SES individuals cannot be accounted for by the structural or functional characteristics of the amygdala measured here. ■

INTRODUCTION

Socioeconomic status (SES) has a well-established relation to cognition, measured by academic achievement and intelligence tests (Sirin, 2005), and cognitive neuroscientists have begun to study the brain systems that underlie cognition (e.g., Hair, Hanson, Wolfe, & Pollak, 2015; Mackey et al., 2015; Noble et al., 2015). Less is known about the relation of SES to emotion and the affective neuroscience of SES is still nascent. The relation of SES to emotion was first documented as a clinical phenomenon, with the discovery that lower SES predicts a higher incidence of depression and anxiety disorders (Ridley, Rao, Schilbach, & Patel, 2020; Hollingshead & Redlich, 1958). This finding concerns clinical syndromes rather than the basic science of normal, healthy people, outside mental health issues. Are low SES people more likely to experience high negative affect? Here, we report a study of negative emotions in healthy young adults of varying SES and test the role of amygdala structure and function in relations between SES and negative emotions. We focus on anger, fear, and sadness—three types of emotion that, though not categorical "basic" emotions in the sense of Ekman (1999), generally differ experientially and share negative valence (Russell & Barrett, 1999). We also consider the relation of SES to negative emotion more generally by analyzing a composite of the emotions studied here.

In their comprehensive reviews of SES and negative affect, Matthews and Gallo (2011; Gallo & Matthews, 2003) discuss these negative emotions, but most of the available evidence is drawn from clinical syndromes and personality traits. For example, the studies they review use the number of subthreshold depression and anxiety symptoms. Although these could be considered proxies for the emotions of sadness and fear, the symptoms of these clinical syndromes include cognitive, behavioral, and vegetative symptoms, in addition to measures of characteristic emotional state per se. Similarly, neuroticism, the personality trait most associated with negative affect, involves lability of emotional states and attentional biases as well as a tendency to experience the states themselves (McCrae & Costa, 1987). Gallo and Matthews (2003) cite one negative emotion, anger, which has been measured and found to correlate negatively with income and education measures of SES (see, e.g., Cohen, Salonen, & Kaplan, 1999). More recent research, motivated by the question of whether money and happiness are related, has demonstrated that the daily experience of sadness is reliably higher among those of lower income (with no effect in either direction for happiness; Hudson, Lucas, Donnellan, & Kushlev, 2016; Kushlev, Dunn, & Lucas, 2015). However, a survey of a wider range of emotional states, using the Positive and Negative Affect Schedule, showed no

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association of their SES measures with any of the emotions (Crawford & Henry, 2004). In summary, evidence concerning SES and emotions in healthy individuals is both limited and mixed. Therefore, the first goal of the present study is to determine the relations of SES to anger, fear, and sadness in a large sample of healthy individuals.

Assuming that SES is found to correlate with some or all of these negative emotions, the next goal is to understand why this is so. Of course, the realities of low SES give people more to feel angry about, afraid of, and sad about, but other explanations are possible as well and may not be mutually exclusive. Here, we test a class of explanations about the neural substrates of the relation between SES and negative affect, which could reflect the physiological embodiment of different levels of social and economic hardship. Specifically, we test a set of different hypotheses that have been proposed in the literature regarding the role of amygdala structure and function as mediators of the SES-negative affect relation.

The idea that SES differences in the amygdala are implicated in the relation between SES and negative affect has been articulated by many authors in many forms (Smith & Pollak, 2020; Hanson et al., 2019; Palacios-Barrios & Hanson, 2019; Kim, Evans, Chen, Miller, & Seeman, 2018; Merz, Tottenham, & Noble, 2018; Barch et al., 2016; McEwen & Gianaros, 2010; Gianaros et al., 2008). For example, Merz et al. (2018) propose the hypothesis that "associations between SES and amygdala volume may be relevant for understanding socioeconomic differences" in internalizing traits (anxiety, fear, self-consciousness, and sadness). Amygdala reactivity has been discussed by Gianaros et al. (2008) as a reflection of "internalized distress" arising from low social status. Decreased amygdala–mPFC functional connectivity (FC) after childhood poverty has been offered by Javanbakht et al. (2015) as "a candidate neural mechanism for negative social-emotional bias."

Although the foregoing mediation hypotheses are intuitively plausible, to date they have received little support. Testing them requires analyzing measures of SES, negative affect, and amygdala structure and function in the same group of participants. Therefore, once we have tested the relation of SES to negative emotion, we will then test the relations between SES and amygdala structure and function, and the mediating role of the amygdala in the SES–negative emotion relation.

Regarding SES–amygdala relations, extensive evidence has generally shown a positive relation between amygdala volume and SES (e.g., Lotze et al., 2020; Gur et al., 2019; McDermott et al., 2019; Hanson et al., 2015; Luby et al., 2013; Butterworth, Cherbuin, Sachdev, & Anstey, 2012). Nevertheless, some studies find no relation (e.g., Lawson et al., 2017; Noble et al., 2015; Hanson, Chandra, Wolfe, & Pollak, 2011), and a negative relation has been reported (Noble, Houston, Kan, & Sowell, 2012). As a first step toward testing the mediation hypothesis, we will test the relation between SES and amygdala volume in a large sample of healthy young adults.

Amygdala function has also been observed to vary with SES, although, here, the evidence base is smaller and less consistent than for volume. Three relatively early studies found higher amygdala reactivity to negative facial expressions in people of lower SES (Kim, Capistrano, Erhart, Gray-Schiff, & Xu, 2017; Muscatell et al., 2012; Gianaros et al., 2008). However, this pattern is sometimes only found in subgroups of participants (Assari, 2020; White et al., 2019; Javanbakht et al., 2016) or for faces of one race but not another (Muscatell, McCormick, & Telzer, 2018). One finding of higher reactivity in lower SES was general to all faces rather than specific to emotional faces (Demenescu et al., 2014). Finally, one study failed to find the effect (Demidenko et al., 2021), and another found it to be reversed, with blunted rather than enhanced reactivity to negative facial expressions in low SES (Gard et al., 2017). The evidence suggests that lower SES may well, on average, show higher amygdala responses to negative facial expressions, but additional data are needed before this can be concluded with confidence.

FC between the amygdala and prefrontal regions associated with emotion regulation has only occasionally been studied in relation to SES. Of five previous analyses, two found an SES effect on resting FC with the ventromedial PFC (vmPFC, an area associated with implicit emotion regulation; Etkin, Büchel, & Gross, 2015), consisting of higher FC in higher SES (Hanson et al., 2019; Javanbakht et al., 2015). A third found a similar positive relation between connectivity of basolateral amygdala to vmPFC and SES in children only, with no difference by the early 20s (Ramphal et al., 2020). A fourth showed a positive SES effect on connectivity between amygdala and ventrolateral PFC (vlPFC), an area associated with explicit emotion regulation (Etkin et al., 2015) during an explicit emotion regulation task (Kim et al., 2013). The fifth study did not find any SES effect on amygdala connectivity with either the vmPFC or vlPFC but did find one between the amygdala and the superior frontal gyrus (SFG). In summary, the evidence base on SES and amygdala–PFC FC is quite limited and not entirely consistent.

With these varied findings as context, the current project aims to address three open questions: First, are negative emotions more pronounced among healthy individuals of lower SES? Do anger, fear, and sadness figure more prominently in the emotional experience of lower SES adults? Second, do the structure and function of the amygdala, measured in terms of volume, reactivity, and FC, vary by SES? Third, does amygdala structure or function account for SES differences in negative emotional states? For each of these questions, SES is the primary variable of interest. However, the possibility of moderation by gender, age, or race (see, e.g., Steptoe & Zaninotto, 2020; Assari, Preiser, & Kelly, 2018; Whittle, Yücel, Yap, & Allen, 2011) also informs the research plan. We test these

hypotheses in young adults from the Human Connectome Project.

METHODS

Participants

Participants were healthy young adults, 22–35 years old at recruitment, with no history of major psychopathology, from the publicly available repository of the Human Connectome Project S1200 release (www.humanconnectome.org/). Informed consent was obtained for all participants (see Van Essen et al., 2012, 2013, for further information on inclusion/exclusion criteria and consent). These participants broadly reflected the racial composition of the U.S. population.

We removed individuals who did not report their SES (6) or who were studying toward a degree (238), because their income and educational attainment would likely misrepresent (underestimate) their SES, resulting in a sample of 962. One participant of the 962 was missing affect measures, resulting in $n = 961$, as shown in Table 1. Because of different amounts of missing data and different numbers of excluded participants for different dependent measures (specified later in Methods, with the descriptions of the measures), sample sizes differed. Table 1 shows the sizes of the different samples and their highly similar demographic characteristics.

Measures

SES

The SES measure was a composite of income and education (correlation = .437), calculated by *z*-transforming each to place them on a common scale and then averaging them. Education was measured by the year of education completed (<11 = 11, 12, 13, 14, 15, 16, 17+ = 17); for example, a person who completed undergraduate study had 16 years of education. Income was measured as total household income (<$10,000 = 1; $10,000–$19,999 = 2; $20,000–$29,999 = 3; $30,000–$39,999 = 4; $40,000–$49,999 = 5; $50,000–$74,999 = 6; $75,000–$99,999 = 7; ≥$100,000 = 8).

Negative Affect

Affect was assessed with the Negative Affect subscale of the NIH Toolbox for Assessment of Neurological and Behavioral Function. This is a brief yet comprehensive instrument developed for use in epidemiologic and clinical research (Gershon et al., 2010). The Emotion Battery, including the Negative Affect scales, has good psychometric properties, including test–retest reliability and factor structure (Salsman et al., 2013). The Negative Affect measures, defined within the Toolbox as anger, fear, and sadness, assessed anger, fear, and sadness with statements to which participants respond 1–5, which corresponds to

Table 1. Demographic Information for the Samples Used for Analyses of the Variables Listed

	Sample with Behavior	*Sample with Amygdala Volume and Behavior*	*Sample with Amygdala Reactivity and Behavior*	*Sample with Amygdala FC and Behavior*
Sample size	961	885	832	631
Gender	Women 539 or 56%	Women 497 or 56%	Women 458 or 55%	Women 351 or 56%
Racial component	White 74.7%	White 75.8%	White 77.0%	White 76.6%
	Black 16.0%	Black 15.2%	Black 14.1%	Black 13.3%
	Asian 4.7%	Asian 4.7%	Asian 4.9%	Asian 6.0%
	Other 4.6%	Other 4.3%	Other 4.0%	Other 4.1%
Education, years	Range 11–17	Range 11–17	Range 11–17	Range 11–17
	Mean 14.90	Mean 14.97	Mean 15	Mean 15
	Median 16	Median 16	Median 16	Median 16
	SD 1.88	*SD* 1.85	*SD* 1.84	*SD* 1.84
Income	Range 1–8	Range 1–8	Range 1–8	Range 1–8
	Mean 5.21	Mean 5.27	Mean 5.31	Mean 5.31
	Median 6	Median 6	Median 6	Median 6
	SD 2.13	*SD* 2.09	*SD* 2.07	*SD* 2.07

Racial component "other" refers to unknown, more than one, or American Indian/Alaskan Native. Numerical values for income and education are as defined by the Human Connectome Project and described in the section on SES measures, below.

"extremely untrue of me" to "extremely true of me," "not at all" to "extremely," or "never" to "always," depending on the wording of the item. Anger was assessed with three subscales, anger affect, anger aggression, and anger hostility, combined for an overall anger score with subscales weighted by the number of items in each. Fear was assessed with two subscales, fear-affect and fear-somatic arousal, similarly combined. Sadness was assessed with a single scale. Examples from each subscale are "In the last 7 days I was irritated more than people knew" (anger-affect), "I wonder why I sometimes feel so bitter about things" (anger-hostility), "I get into fights a little more than the average person" (anger-aggression), "In the past 7 days I felt frightened" (fear-affect), "In the past 7 days my heart was racing or pounding" (fear-somatic arousal), and "I felt that I had nothing to look forward to" (sadness).

In recognition of the importance of negative valence as a general construct (Russell & Barrett, 1999) and the possibility that valence per se is more predictive than states of anger, fear, and sadness measured individually, we created an overall composite of negative affect to increase analytic power. The composite was computed by weighting the three affect scales by their number of items and simply averaging them.

Structural MRI

T1-weighted images were obtained, per HCP protocol, on a 3-T Siemens Skyra scanner (Siemens AG) with a 32-channel head coil, using a 7 min 40 sec 3-D MPRAGE scan. Resolution was 0.7 mm isotropic, with field of view = 224 mm, matrix = 320, 256 sagittal slices, repetition time = 2400 msec, and echo time = 2.14 msec. Left and right amygdala volume were calculated by the HCP using Freesurfer pipeline. Of the 961 participants with appropriate SES information and affect data, six were missing amygdala volume data in the HCP data base, resulting in n = 885, as shown in Table 1.

fMRI

Resting fMRI was acquired, using a gradient-echo EPI sequence, in a session that lasted 14 min 33 sec. Resolution was 2.0 mm isotropic, with field of view = 208 × 180 mm (RO × PE), matrix = 104 × 90 (RO × PE), 72 sagittal slices with 2.0 mm slice thickness, repetition time = 720 msec, and echo time = 33.1 msec. Acquisition of tfMRI data was similar to resting state, except that run durations were determined by task and the seven tasks (14 scan runs) for a total of 1 hr of scan time.

Brains were normalized to fslr32k via the MSM-AII registration. CompCor and five principal components from the ventricles a white matter masks were used to regress out nuisance signals from the time series. In addition, the 12 detrended motion estimates provided by the Human Connectome Project were regressed out from the regional time series. The mean global signal was removed, and then time series were band-pass filtered from 0.009 to 0.08 Hz (Baker et al., 2019; Smith et al., 2015). All preprocessing was executed with Connectome Workbench and custom software available (https://github.com/ThomasYeoLab/CBIG/tree/master/stable_projects/preprocessing). All data are publically available from the Human Connectome Project.

Reactivity. Amygdala reactivity was acquired when participants performed an emotional face matching task with a shape-matching control condition, adapted from Hariri, Tessitore, Mattay, Fera, and Weinberger (2002). The amygdala reactivity measure was extracted from the beta coefficients of contrast face–shape, which was processed with FLS's FEAT MSM-AII with 2 mm minimal smoothing. Participants were presented with blocks of trials that either asked them to decide which of two faces presented on the bottom of the screen matched the face at the top of the screen, or which of two shapes presented at the bottom of the screen matched the shape at the top of the screen. The faces had either an angry or fearful expression. One hundred twenty-eight of the 961 participants were missing amygdala reactivity data, and one additional participant was removed owing to a right amygdala activation value more than 7 *SD*s away from the mean (all other values were within 4.5 *SD*s).

Although this task involves the perception of a different person's emotional state, not one's own, it has been found to be a sensitive marker of a participants' emotional state (e.g., Pichon, Rieger, & Vuilleumier, 2012) and of vulnerability to mood disorder (Mattson, Hyde, Shaw, Forbes, & Monk, 2016). We did not anticipate emotion-specific amygdala responses given the lack of differentiation found in most studies (see Fitzgerald, Angstadt, Jelsone, Nathan, & Phan, 2006), as well as lesion evidence of a general role for the amygdala in negative emotions (Adolphs et al., 1999).

FC. Consistent with commonly used parameters (Schaefer et al., 2018; Power et al., 2011), the following criteria were adopted to mitigate effects of motion: Frames with greater than 0.2 mm framewise displacement or a derivative root mean square above 75 were removed as outliers, and segments of less than five uncensored time points were also removed. In addition, sessions composed of greater than 50% outlier frames were not further analyzed. After removal of these sessions, 631 subjects had FC data for analysis.

We employed parcellation generated from gradients and similarity of intrinsic FC patterns (Schaefer et al., 2018). Specifically, we extracted time-varying mean BOLD signal from n = 100 cortical ROIs in this alternative parcellation. The processing pipeline used here has previously been suggested to be ideal for removing false relations between connectivity and behavior (Rodriguez, Izquierdo, & Ahn, 2019). The prefrontal regions we selected were based on Schaefer parcellation 17 networks:

LH_DefaultB_PFC_2 and RH_DefaultB_PFCv_2 for vlPFC, LH_DefaultA_PFCm_1 and RH_DefaultA_PFCm_1 for vmPFC, and LH_DefaultB_PFCd_1 and RH_DefaultB_PFCd_1 for SFG.

Following Kraus et al. (2021) and Elliott et al. (2019), activity during resting state with the HCP's seven task activation scans were combined. We were not attempting to contrast patterns of connectivity during affective and nonaffective processes; rather, we were seeking characteristic patterns of connectivity across individuals that could correlate with characteristic affective states. Individual differences in FC are similar between task and resting states, and this choice allows us to sample the brain's FC in a diverse range of cognitive states as well as increasing measurement accuracy given the increase in the length of the time series. In fact, functional networks are dominated by common organizational principles and stable individual features, with substantially more modest contributions from task state and day-to-day variability (Gratton et al., 2018). In summary, to maximize the accuracy of our FC calculations, we measured FC across a range of states; no task-based activation maps were generated. Pearson r correlation values were Fisher z-transformed and then averaged across all states.

SES-related Analyses

All analyses involved multiple regression, with different dependent measures, according to the hypotheses being tested. SES was the independent variable of interest, and covariates were gender, age, and race, along with the within-subject factor of hemisphere for the imaging dependent measures. Also included in the regressions were interactions of the between-subject covariates with hemisphere and SES (justified by Steptoe & Zaninotto, 2020; Assari et al., 2018; Whittle et al., 2011), to be dropped from the model when nonsignificant. A subsidiary analysis of the SES main effects will also be carried out without covariates to determine whether the SES effects are retained or abolished by the addition or subtraction of covariates. These results will be reported and interpreted only if eliminating covariates changes the conclusion regarding SES and the outcomes.

To test the SES–negative affect relations, four separate regression analyses were conducted with the four dependent measures (composite negative affect score and anger, fear, and sadness scores). False discovery rate (FDR) correction was applied to the results of these four analyses of SES main effects. To assess possible moderation of SES effects by covariates and given the number of interaction effects involving SES and covariates, we also computed FDR q-values for the interactions. All measures of negative affect were predicted to be lower at higher levels of SES.

Next, we analyzed the relation of SES to the five specific measures of amygdala structure and function. These were mixed-effect models with subject as the random effect, so that the amygdala in each hemisphere was nested within individuals. Five regressions were run, with analogous use of covariates, interactions, and correction as for affect measures. Significant interactions would be probed by post hoc analysis to reveal relations among the SES effects observed in the cells of the research design. The estimated marginal means (emmeans) package in R (Russell et al., 2021) was used to compute marginal means of linear relations and then pairwise compare them, which corrects for multiple comparisons with the Tukey method. FC analyses also included head motion (mean relative root-mean-square displacement over the whole time series) as a covariate. We analyzed amygdala FC to three prefrontal regions whose connectivity varies by SES, vmPFC, vlPFC, and SFG, the first two of which play important roles in implicit and explicit emotion regulation, respectively (Etkin et al., 2015). On the basis of prior literature, summarized earlier, we expected amygdala volume to be larger, amygdala reactivity to be lower, and amygdala–PFC connectivity to be higher at higher levels of SES.

For any significant relations found between SES and emotional, structural, or functional outcomes, we repeated the analysis of the outcome measure in question with the samples used for the other outcome measures. This enabled us to gauge the robustness of any specific significant findings obtained with one sample to the smaller samples.

Finally, to test if amygdala structure or function mediates the association between SES and negative affect, the PROCESS macro in SPSS (Hayes, 2022) was used to estimate indirect pathways with 95% bias-corrected confidence intervals based on bootstrapping with 50,000 samples. This approach is robust to the distribution of the indirect effect. To reduce the number of tests, thereby minimizing Type I error, testing of pathways was restricted to affect measures that showed a significant link to SES and to those amygdala measures that showed significant associations with SES in the form of main effects or interactions. Results were reported as standardized beta coefficients throughout to show effect sizes.

RESULTS

SES and Affect

As expected, lower SES is generally associated with higher negative affect across the various sample sizes corresponding to the availability of different brain measures. These results are shown in Table 1. The negative affect composite shows a small but significant negative relation to SES for all samples, such that lower SES individuals reported higher general negative affect. Of the specific emotions, anger shows the strongest relation to SES across samples, followed by sadness (significant in all but the smallest sample) and fear (smaller effect and significant in only two of the samples). The effect of SES on negative affect does not differ by gender, race, or age. In addition, men report

Table 2. SES and Affect: Standardized Coefficient (*SE*)

	Sample with Behavior n = 961	*Sample with Amygdala Volume and Behavior n = 885*	*Sample with Amygdala Reactivity and Behavior n = 832*	*Sample with Amygdala FC and Behavior n = 631*
Negative affect composite	**−0.123 (0.030)***** $q < 0.001$	**−0.134 (0.031)***** $q < 0.001$	**−0.133 (0.032)***** $q < 0.001$	−0.081 (0.037)$^{+}$ $q = 0.056$
Anger	**−0.178 (0.035)***** $q < 0.001$	**−0.180 (0.036)***** $q < 0.001$	**−0.185 (0.037)***** $q < 0.001$	**−0.128 (0.044)*** $q = 0.016$
Fear	−0.061 (0.036)$^{+}$ $q = 0.085$	**−0.080 (0.037)*** $q = 0.031$	**−0.079 (0.039)*** $q = 0.041$	−0.045 (0.044) $q = 0.301$
Sadness	**−0.130 (0.035)***** $q < 0.001$	**−0.141 (0.037)***** $q < 0.001$	**−0.135 (0.039)***** $q < 0.001$	−0.071 (0.045) $q = 0.152$

Significant relations in **bold** font; levels of FDR corrected significance: $^{+}(q < 0.1)$, $*(q < 0.05)$, $**(q < 0.005)$, $***(q < 0.001)$.

significantly more anger and less fear than women. One interaction is found to be significant for anger, with the race, specifically Black participants showing a stronger SES effect than Asian ($q = 0.022$) and other race participants ($q = 0.022$; Table 2).

SES and Amygdala Volume

SES has a small but highly significant effect on amygdala volume, as shown in Table 3. This effect is not moderated by any covariates, as indicated by no significant interactions. To determine whether the difference in SES effects observed for volume is robust across the smaller sample sizes available for the other imaging measures, we repeated the analysis with these samples. The main effect of SES on amygdala volume remains significant with the smaller reactivity sample ($b = 0.114$, $SE = 0.029$, $q < 0.001$) and the smallest FC sample ($b = 0.126$, $SE = 0.033$, $q < 0.001$).

SES and Amygdala Reactivity

Contrary to expectations concerning amygdala reactivity in the face matching task, SES is not related to amygdala reactivity, and the nonsignificant trend shows a positive association, such that higher SES is associated with greater reactivity. A borderline significant interaction between SES, hemisphere, and gender is also observed, with women showing higher reactivity with higher SES in the right amygdala. There is no obvious interpretation for this pattern, and at $q = 0.056$, we refrain from seeking one.

SES and Amygdala FC

We begin with FC between amygdala and vmPFC, which is associated with implicit emotion regulation (Etkin et al., 2015) and has previously shown a positive relation to SES (Hanson et al., 2019). In the present sample, there is no relation between SES and amygdala–vmPFC, nor is there moderation of this relation by any covariates. Turning to vlPFC, an area implicated in explicit emotion regulation (Etkin et al., 2015), we again observe no main effect of SES on FC and no moderation by covariates. Finally, FC between amygdala and SFG (Barch et al., 2016; Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008) is unrelated to SES and shows no moderation.

Of note, analyses of SES effects without covariates find that the amygdala–vmPFC relation becomes significant when race is eliminated as a covariate ($q = 0.030$). This is plausibly interpreted as the result of the well-known relation of race and accompanying racism to SES in our society. It suggests that studies of SES, whether behavioral or neural, should covary for race or otherwise take it into account.

Table 3. SES and Amygdala Volume, Reactivity, and FC: Standardized Coefficient (*SE*)

	Amygdala Volume	*Amygdala Reactivity*	*Amygdala-vmPFC FC*	*Amygdala-vlPFC FC*	*Amygdala-SFG FC*
SES effect	**0.106 (0.028)***** $q < 0.001$	0.033 (0.034) *ns*	0.056 (0.034) *ns*	0.015 (0.031) *ns*	−0.026 (0.033) *ns*

Significant relations in **bold** font; levels of FDR corrected significance: $^{+}(q < 0.1)$, $*(q < 0.05)$, $**(q < 0.005)$, $***(q < 0.001)$.

Table 4. Indirect Effect of Amygdala in the Association of SES and Negative Affect: Standardized 95% Confidence Interval of Indirect Path

	Amygdala Volume
Negative affect composite	**(0.002, 0.024)**
Anger	**(0.001, 0.022)**
Fear	**(0.001, 0.022)**
Sadness	**(0.001, 0.022)**

Bold font denotes significant effect.

Mediation of SES–Affect Relations by Amygdala

The foregoing analyses indicate that only amygdala volume is related to SES and could therefore be considered a possible mediator for the SES-negative affect relation. To test this, the indirect pathway from SES to negative affect was assessed for the negative affect composite and anger, fear, and sadness separately. Contrary to the prediction that amygdala volume mediates these relations and thus reduces the strength of the SES–negative affect relations when taken into account, the results show that SES–affect relations are stronger when amygdala volume is accounted for, as shown in Table 4. That is, people of low SES do not experience more negative affect because of their amygdala volume (or unmeasured factors related to amygdala volume); they experience more negative affect despite their amygdala volume. Regarding the often-proposed hypothesis that the amygdala is responsible for higher negative affect in lower SES people, these results are disconfirming. Whereas a nonsignificant mediation of the expected form would not disconfirm that hypothesis so much as fail to confirm it, leaving open the possibility that the study was simply not sufficiently sensitive to reject the null hypothesis, the present results are decisive evidence that amygdala volume does not mediate the relation between SES and negative affect.

DISCUSSION

Here, we assessed three widely cited ideas about SES and emotion: First, that negative affect is more common at lower levels of SES; second, that SES is associated with structural and functional properties of the amygdala; and third, that these SES differences in the amygdala are responsible for the affective differences. We reviewed existing evidence for these ideas, found it on the whole to be quite limited, and subjected the ideas to empirical test. Conclusions ranged from strong support to strong disconfirmation.

Previous evidence linking SES and negative affect is substantial but confined mostly to clinical disorders of affect. Such disorders are not simply stronger versions of the emotions of anger, fear, and sadness; they include many nonaffective symptoms that typically cluster in a syndrome with the affective symptoms. Fewer studies have examined the relation between SES and negative affect in healthy people, as done here, and previous results have been mixed. The present study is the first to seek and find a relation of SES to nonclinical measures of three distinct negative emotions. The results provided strong confirmation of the hypothesis that normal healthy individuals of low SES typically experience more negative affect than their higher SES counterparts. Although the effect sizes are not large, with standardized betas for the SES–negative affect relations in Table 2 ranging from −0.061 to −0.178, neither are they negligible, particularly when the effects impact tens of millions of young adults in the United States alone.

Turning to the relations between SES and the amygdala, there are varying degrees of evidence in the literature on SES differences in amygdala structure and function. The present study assessed amygdala volume, reactivity, and FC, which have all been posited to vary by SES. The most thoroughly investigated measure in this literature is amygdala volume, examined in relation to SES primarily in child and adolescent samples. Previous findings were a mix of positive relations (higher SES and larger amygdala volume) and null results, along with one report of a negative relation. Few if any studies have involved healthy young adults (but see Lawson et al., 2017, for a small sample of 25- to 35-year-olds, for whom neither childhood nor concurrent SES predicted amygdala volume). The present findings therefore added strong confirmation of a positive relation between SES and amygdala volume and did so uniquely in a large sample of healthy young adults.

Functional properties of the amygdala, specifically reactivity and FC, have also been reported to vary with SES. However, as reviewed earlier, the evidence base here is smaller and less consistent than for volume. Although SES may be related to amygdala responses to negative facial expressions, simple generalizations do not appear possible. The present study further reduced hope for a simple generalization.

Amygdala–PFC FC has obvious relevance to negative affect, as interactions between these parts of the brain are implicated in emotion regulation, and the most common goal of emotion regulation is the reduction of negative emotion. Unfortunately, as reviewed earlier, relatively little is known about differences in amygdala–PFC FC across levels of SES. The present analysis substantially extended the sparse evidence base that exists, albeit with null findings. In summary, for the relation of SES to amygdala reactivity and FC finds some support in the existing literature but is far from firmly established, and the present findings only add reason for caution.

The third idea, that the amygdala is responsible for the higher levels of negative affect in lower SES individuals, appears throughout the literature (Smith & Pollak, 2020; Hanson et al., 2019; Palacios-Barrios & Hanson, 2019; Kim et al., 2018; Merz et al., 2018; Barch et al., 2016; McEwen & Gianaros, 2010; Gianaros et al., 2008). It is

typically proposed as a plausible hypothesis awaiting direct confirmation, with most studies reporting SES–amygdala relations per se rather than a test of mediation.

Two exceptions to this were the studies of Barch et al. (2016) and Hanson et al. (2019), neither of which supported the mediation hypothesis in question. The present study was the first attempt to systematically test the amygdala mediation hypothesis with amygdala volume, reactivity, and FC. Mediation analysis failed to show a mediating role for amygdala volume in the SES–negative affect relation, instead revealing a suppression effect. Thus, despite the intuitive appeal of this oft-cited hypothesis, it is strongly disconfirmed here.

Regarding SES, brain, and behavior more generally, the published findings reviewed here illustrate the challenge of replicable research in the behavioral and neural sciences, particularly regarding complex social attributes like SES. Our own findings are a further reminder that such research is often marked by inconsistencies and only partial replications. The challenge of drawing firm conclusions in the affective neuroscience of SES can be partly attributed to the field's early stage of development. The publications providing the hypotheses to be tested date back only to 2008.

In addition, studies vary in numerous ways that may be scientifically relevant but were not distinguished here. Participant age may play a role in the ways that SES is manifested in brain and affect. Much of the literature on SES and the brain concerns children and adolescents. The effects of SES may depend on developmental stage (Piccolo, Merz, He, Sowell, & Noble, 2016), and this may be particularly true for the amygdala (Tottenham & Sheridan, 2010).

Another source of variability arises from the different meanings of SES and the ways it is measured. For youth samples, one or more of the following indices of SES are typically used: parental income, educational attainment or occupational status, or the young person's subjective social status or neighborhood SES. For adults, the same measures have been used, either referring to the adult subjects' own current SES or childhood SES. Fortunately, different measures of SES tend to be somewhat correlated (Galobardes, Shaw, Lawlor, Lynch, & Smith, 2006), although they have also been shown to have distinctive relations to brain and behavioral traits. Examples of SES constructs with distinctive roles include subjective social status and neighborhood SES. These refer to one's perceived place in social hierarchies and to the socioeconomic characteristics of one's neighborhood, respectively, independent of more objective measures of personal economic and social resources. We adopted a "lumping" rather than a "splitting" strategy for our review of the background literature, not distinguishing studies by age or SES measure. Nevertheless, disagreements among past studies are not explicable just based on these differences.

In conclusion, typical daily feelings differ by SES; our data indicate that even normal healthy young adults report on average feeling more negative emotion the lower their SES. Amygdala volume varies with SES as well. However, we established that this could not be responsible SES differences in negative affect, as has often been supposed. Findings on amygdala reactivity and FC also failed to show a relation to SES. If the prevalence of negative affect at lower levels of SES is to be explained by neuroscience, new hypotheses will need to be tested.

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Data Availability Statement

We analyzed the public available dataset – the Human Connectome Project.

Diversity in Citation Practices

Retrospective analysis of the citations in every article published in this journal from 2010 to 2021 reveals a persistent pattern of gender imbalance: Although the proportions of authorship teams (categorized by estimated gender identification of first author/last author) publishing in the *Journal of Cognitive Neuroscience* (*JoCN*) during this period were M(an)/M = .407, W(oman)/M = .32, M/W = .115, and W/W = .159, the comparable proportions for the articles that these authorship teams cited were M/M = .549, W/M = .257, M/W = .109, and W/W = .085 (Postle and Fulvio, *JoCN*, 34:1, pp. 1–3). Consequently, *JoCN* encourages all authors to consider gender balance explicitly when selecting which articles to cite and gives them the opportunity to report their article's gender citation balance. The authors of this article report its proportions of citations by gender category to be as follows: M/M = .517; W/M = .155; M/W = .19; W/W = .138.

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