Impact of Socioeconomic Status on Amygdala and Hippocampus Subdivisions in Children and Adolescents

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ABSTRACT

Socioeconomic status (SES) in childhood can impact behavioral and brain development. Past work has consistently focused on the amygdala and hippocampus, two brain areas critical for emotion and behavioral responding. While there are SES differences in amygdala and hippocampal volumes, there are many unanswered questions in this domain connected to neurobiological specificity, and for whom these effects may be more pronounced. We may be able to investigate some anatomical subdivisions of these brain areas, as well as if relations with SES vary by participant age and sex. No work to date has however completed these types of analyses. To overcome these limitations, here, we combined multiple, large neuroimaging datasets of children and adolescents with information about neurobiology and SES (N=2,765). We examined subdivisions of the amygdala and hippocampus and found multiple amygdala subdivisions, as well as the head of the hippocampus, were related to SES. Greater volumes in these areas were seen for higher-SES youth participants. Looking at age- and sex-specific subgroups, we tended to see stronger effects in older participants, for both boys and girls. Paralleling effects for the full sample, we see significant positive associations between SES and volumes for the accessory basal amygdala and head of the hippocampus. We more consistently found associations between SES and volumes of the hippocampus and amygdala in boys (compared to girls). We discuss these results in relation to conceptions of “sex-as-a-biological variable” and broad patterns of neurodevelopment across childhood and adolescence. These results fill in important gaps on the impact of SES on neurobiology critical for emotion, memory, and learning.

INTRODUCTION

Socioeconomic status (SES) in childhood has been associated with multiple negative physical and mental health outcomes, with several meta-analyses noting these links [1,2]. The mechanisms underlying these relations, however, are poorly understood. An emerging approach leverages precise quantitation of neurobiology to understand SES-gradients of health [3,4]. Neuroscientific investigations may allow a more elemental focus, as the brain determines behavioral and physiological responses [5]. This focus may be particularly valuable given the protracted nature of brain development and that the brain is shaped by experiences early in life [6].

A growing body of research has found neurobiological alterations in samples exposed to poverty or lower SES conditions [6,7]. Notably, childhood poverty has been implicated in structural differences across multiple brain regions, with differences in hippocampal and amygdala structure being commonly reported. The link between childhood poverty and smaller hippocampal volumes has been replicated by at least seven research groups [8–14]. Studies examining the directional impact of childhood poverty on amygdala structure have produced a landscape of heterogeneous results, with reports of larger and smaller amygdalae [15]. Given these areas' connections to important socioemotional functions and learning, understanding how poverty may shape these regions could shed light onto the mechanisms of SES-related disparities [16]. The amygdala is a central neural hub for vigilance and processing negative emotions [16,17]. The hippocampus plays a critical role in memory representations and using previously acquired information in service of goal-directed behavior [18,19]. As such, these brain areas are critical for emotion and behavioral responding.

While there are SES differences in amygdala and hippocampal volumes, there are many unanswered questions in this domain connected to neurobiological specificity, and for whom these effects may be more pronounced. First, related to neurobiology, while research often treats the amygdala and hippocampus as unitary structures, they are complex and heterogeneous. Different amygdala nuclei have unique connectivity profiles, patterns of developmental changes, and behavioral correlates [17,20,21]. Similarly, the hippocampus is composed of functionally distinct subregions, with differential connectivity and cytoarchitectonics [22–24]. The posterior hippocampus has been linked more to cognitive functions, while more anterior regions relate to stress and affect [25,26]. Second, there may be potential sociodemographic subgroups where the effects are more pronounced; specifically, sex and age may both moderate the impact of SES on neurobiology. Motivated in part by sex disparities in many neuropsychiatric disorders [27], there has been a growing emphasis on sex as a biological variable. After stress exposure, sex differences in neuronal firing, dendritic spines, neurogenesis, and fMRI responsivity have been found in both the amygdala and the hippocampus [28–30]. These sex differences may be due to different neuroendocrine processes, responses from the environment, or sex chromosome-specific neuroprogramming [31,32]. Sex may also be related to differential responses to stress exposure, like those associated with lower SES [33]. Related to age, there are non-linear trajectories for brain development, with many structures increasing in volume in childhood, and then showing lower volumes in adolescence and adulthood [34]. The impacts of stress on neurobiology may vary with age and development [15]. Stress may increase volumes in certain regions early in development, but then relate to "excitotoxic burnout" and smaller volumes later in time. As such, it will be critical to explore connections between neurobiology, age, sex, and SES.

Connected to neurobiological specificity, there is a growing body of past work examining amygdala and hippocampal subdivisions after childhood adversity. For the hippocampus, past projects have commonly reported smaller volumes in Cornu Ammonis (CA) 1 for those exposed

to high levels of adversity [35–38]; however, results are not perfectly uniform with other studies only finding differences in CA3, and not CA1 [39,40]. With the amygdala, less work has been completed. Two studies reported smaller basolateral amygdala volumes after exposure to adversity [41,42], but these projects also reported stress was sometimes related to differences in accessory basal, central-medial, and paralaminar subdivisions. Related to SES, limited work has examined if there are potential alterations in volumes of amygdala and hippocampal subdivisions. Three past studies have found anterior hippocampal volumes (i.e., Dentate gyrus; CA1) were positively related to different operationalizations of SES, including parental education [43], family household income [44], and socioeconomic conditions in a census tract [45]. Notably, no work to date has examined amygdala subdivisions in relation to SES.

Attempting to overcome these limitations, here, we combined multiple, large neuroimaging datasets of children and adolescents with information about neurobiology and SES (N=2,765). To improve neurobiological specificity, we examined subdivisions of the amygdala and hippocampus. We aimed to richly probe the main effects of SES on these smaller areas of the amygdala and the hippocampus, as well as examine potential sex- or age-specific impacts of SES on these volumes. In keeping with past reports of smaller volumes in lower SES youth, we predicted lower SES would be related to smaller volumes in amygdala and hippocampus subdivisions. Related to past human and non-human research in stress-exposed groups [15], we also predicted that lower SES would be related to smaller volumes in the head of the hippocampus, as well as the basolateral and central amygdala. Finally, given potential neuroprotective effects of estrogen [46,47] and developmental trajectories of brain development [48], we predicted relations between SES and volumes would be stronger for older participants, especially boys.

METHOD

Participants. Participants between 5-18 years of age were drawn from four large neuroimaging projects: The National Consortium on Alcohol and Neuro-Development in Adolescence (NCANDA; [49]), the Healthy Brain Network (HBN; [50]), the Pediatric Imaging, Neurocognition, and Genetics (PING; [51]), and the Human Connectome Project in Development (HCP-D; [52]). Richer sample descriptions are in our supplemental materials. Across these studies, the total number of participants with usable data was N=2765 (44% Female; Mean Age= 11.9, Age SD= 3.5; Age Range = 5.04-17.99). Descriptive information for the combined sample is shown below in Table 1. Information for each project is noted in our Supplement (Table S1). A histogram of participant age by study is shown in Figure 1.

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MRI Data Acquisition and Imaging Processing. High-resolution T1-weighted structural images were acquired with varying parameters across each project. The majority of scans came from 3T scanners, with the exception of a 1.5T scanner used at one site. Scans resolution varied in in-plane resolution from 0.8 to 1.2mm. Information about MRI parameters are noted in our supplemental materials. These MRI scans were processed in Freesurfer 7.1, deployed via Brainlife.io [53]. Freesurfer is a widely documented morphometric processing tool suite, http://surfer.nmr.mgh.harvard.edu/ [54,55]. Based on hand-tracing on high-definition, ex-vivo T1-weighted 7T scans, Freesurfer can output 12 subfields and 9 amygdala subnuclei (see Refs. [56] and [57], for additional details). With the hippocampus, we focused on segmentation that divided this region into the head, body, and tail. This was motivated by: 1) work finding that

hippocampal organization and connectivity varies on its longitudinal axis (i.e., head/body/tail) [22,58]; and 2) commentary suggesting that segmentation of the longitudinal axis of the hippocampus is more appropriate, than much smaller (and potential less reliably segmented subfields, i.e., dentate gyrus, and subiculum) [59,60]. With the amygdala, subnuclei volumes were grouped into 4 divisions: a "basolateral" complex (including lateral, basal, and paralaminar subnuclei); a "medial cortical" cluster (including medial, cortical, and corticoamygdaloid subnuclei); accessory basal; and the central amygdala. A basolateral grouping was motivated by human and nonhuman animal research finding these subnuclei play distinct roles in fear and safety [61,62]. Our "medial cortical" group was motivated by research finding that medial portions of the amygdala: 1) send feedforward safety signals that oppose the fear responses signaled by the lateral portion; 2) are in close proximity to the cortical and superficial nuclei;; and 3) consistent clustering with superficial amygdala nuclei in graph theoretic analyses of human fMRI data [63]. For the 3 hippocampal and the 4 amygdala subdivision groups, we calculated the total volume by summing volumes from the left and right hemispheres of each of these hippocampal or amygdala subdivisions.

— Insert Figure 2 Approximately Here—

To exclude particularly high-motion scans and limit the impact of image quality on subcortical segmentation, we generated a quantitative metric of image quality combining noise-to-contrast ratio, coefficient of joint variation, inhomogeneity-to-contrast ratio, and root-mean-squared voxel resolution [64]. This was motivated by our work finding that T1-weighted image quality is related to Freesurfer outputs [65]. Additional details about this metric, image processing in Freesurfer, and MRI Data Acquisition are noted in our supplemental materials.

Measures of SES. We used a multimethod determination of SES, using metrics of both caregiver education and household income. For caregiver education, caregivers reported on how much school they completed (e.g., obtained a high school diploma; some college; graduate degree); this was converted into numbers of years (i.e., high school diploma = 12 years; some college = 14 years) and then took the highest value by either caregiver. For household income, caregivers selected an income range (i.e., $30,000-39,999/year; $50,000-$99,999/year), or reported a continuous value. In keeping with recommendations from demographers [66], we took the midpoint for reported ranges and then log-transformed this value ($30,000-39,999=$34,999.5; log($34,999.5)=4.54406184). For continuous income reports, we also log-transformed these values. Then, we created an SES composite by taking an average of z-scored, log-transformed income and z-scored, maximum parental education.

Analytic plan. To test relations between SES and volumes of the hippocampus and amygdala, we employed linear mixed effects models (LMEMs) using the *lme4* R package [67]. In all models, we included a random effect for study site, and examined the independent (fixed effect) variables of participant age (in years), participant sex (binary-coded), estimated Total Intracranial Volume (eTIV), image quality (CAT12 rating), and our SES composite. Analyses proceeded in two steps. First, we examined regions of interest for the full sample (N=2765). This was for 9 regions of interest (total hippocampal volume; 3 hippocampal subdivisions [head, body, and tail]; total amygdala volume, 4 amygdala subdivisions [accessory basal, central amygdala, basolateral complex, and medial cortical]. Second, to investigate potential sex and age effects, we separated our full sample by these variables. We created age tertiles with three groups and then divided those groups by sex (Age ranges and numbers of participants are detailed in our supplemental materials, specifically Table S2). We corrected these different steps of analyses for multiple comparisons using False Discovery Rate approaches [68] (9 Comparison in Step 1; 54 in Step 2). In our supplement, we also completed analyses: 1)

focused on volumes and household income or education (as individual/separate variables); and 2) for smaller parcellations of the hippocampus and amygdala output by Freesurfer.

RESULTS

Robust Relations of SES and Volume (N=2,765)
SES was associated with both amygdala and hippocampus volumes. Specifically, higher SES was related to larger (whole regional) volumes in both areas (amygdala: β=0.06, p<0.001; hippocampus: β=0.06, p<0.001). For all amygdala subnuclei, SES was related to volume (accessory basal: β=0.07, p<0.001; central amygdala: β=0.06, p<0.001; basolateral complex: β=0.05, p<0.001; medial cortical cluster: β=0.05, p<0.001). Among hippocampal subfields, SES was only related to volumes in the head of the hippocampus (β=0.09, p<0.001). After correcting for multiple comparisons, all p-values remained significant (p-fdr<0.001). All analyses were controlled for age, sex, image quality, and total brain volume, and all of these relations are displayed in Table 2.

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Relations For Sex- and Age-Specific Subgroups
Relations between SES and whole regional volumes were non-significant for pre- and early adolescent girls (all p's>.05). During pre- and early adolescence, statistical testing suggested relations between SES and hippocampus volumes for boys, but the effects did not survive correction for multiple comparisons (preadolescence: β=0.08, p=0.04, p-fdr=0.13; early adolescence: β=0.08, p=0.03, p-fdr=0.10). For the whole amygdala, the association between SES and volume was significant for boys and girls in late adolescence (boys: β=0.10, p=0.01, p-fdr=0.04; girls: β=0.12, p<0.01,p-fdr=0.02). The relation between SES and whole hippocampal volumes was also significant for both sexes in late adolescence (boys: β=0.11, p<0.01, p-fdr=0.03; girls: β=.10, p=0.01, p-fdr=0.047). In both sexes, higher SES was related to larger amygdala and hippocampus detectable by late adolescence, and the trend toward enlargement at all ages makes delayed maturation unlikely [69].

Similarly, amygdala subnuclei were most related to SES in late adolescent participants, compared to younger in either sex. Among pre-adolescent boys, the association between SES and central amygdala (β=0.11, p=0.01, p-fdr=0.03) volumes was found to be significant, whereas among pre-adolescent girls, there were no significant associations between SES and amygdala subnuclei. In early adolescence, there were no significant relations between SES and any amygdala subnuclei for either boys or girls. However, in late adolescent boys, SES was found to be significantly associated with accessory basal volumes (β=0.11, p=0.01, p-fdr=0.04). Statistical analyses initially indicated that SES was also significantly associated with volumes in the basolateral complex (β=0.10, p=0.01, p-fdr=0.06) and the medial cortical cluster (β=0.09, p=0.02, p-fdr=0.08) in late adolescent boys, but these associations did not survive correction for multiple comparisons. SES was not found to be significantly related to central amygdala volumes in late adolescent boys. In late adolescent girls, SES was significantly associated with accessory basal (β=0.13, p<0.01, p-fdr=0.02), central amygdala (β=0.11, p<0.01, p-fdr=0.02), and basolateral complex (β=0.12, p<0.01, p-fdr=0.02) volumes, and these more robust findings in girls survive correction for multiple comparisons. Only the association between SES and medial cortical cluster volumes (β=0.08, p=0.04, p-fdr=0.13) did not survive correction for multiple comparisons in late-adolescent girls. In summary, the most common manifestation of higher SES among amygdala subnuclei was greater volumes in the accessory basal nucleus, known to play a key role in social safety learning, by late adolescence.

Among hippocampal subfields, only hippocampal head volumes were found to be significantly related to SES in either sex, in any age cohort. In pre-adolescent boys, SES had a significant effect on hippocampal head volumes, but this effect did not survive correction for multiple comparisons (β=0.08, p=0.03, p-fdr=0.09). In pre-adolescent girls, the relation between SES and hippocampal head volumes was found to be nonsignificant. Among early adolescents, the association between SES and hippocampal head volume was found to be significant for early adolescent boys (β=0.12, p<0.01, p-fdr=0.02), but not for early adolescent girls. In late adolescence, SES was found to be significantly associated with hippocampal head volumes for boys (β=0.17, p<0.01, p-fdr<0.01) and also for girls (β=0.14, p<0.01, p-fdr=0.01). Relations between SES and all subdivisions for each sex- and age-specific subgroups are displayed in Table 3.

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DISCUSSION

This study investigated associations between SES and volumetric variations in the hippocampus and amygdala. Notably, we parcellated the hippocampus and amygdala into smaller subdivisions, aiming to increase neurobiological specificity. With the amygdala, we saw that SES was related to differences in all of the subnuclei investigated. For the hippocampus, effects were localized to the head of the hippocampus, with higher SES being associated with larger volumes in that subdivision. Looking at age- and sex-specific subgroups, we tended to see stronger effects in older participants, for both boys and girls. Paralleling effects for the full sample, we see significant positive associations between SES and volumes for the accessory basal amygdala and head of the hippocampus. Interestingly, some suggestive associations emerged for younger boys, in both pre- and early-adolescence. In both of these subgroups, there were relations between SES and volumes for the head of the hippocampus. Our findings are partially in line with our a priori predictions. We predicted that associations between SES and volumes of the hippocampus and amygdala would be stronger for boys compared to girls; connected to this, we did see consistent relations between SES and volumes of the head of the hippocampus.

With the hippocampus, our results are similar to previous projects that have found relations between SES and volumetric differences in the whole hippocampus [3,10–14]. With subfields and specific subdivisions, our results are mostly in line with the small number of publications focused on this question. However, each of these papers varies in the parcellation of the hippocampus and the SES variable examined. Specifically, Merz and colleagues found lower parental education was related to smaller volumes in the dentate gyrus and CA1 subfields of the hippocampus [43]. The CA1 subfield is squarely in the anterior (head) portion of the hippocampus, while their dentate gyrus subfield can span the head and body of the hippocampus. Botdorf and coworkers found smaller anterior and posterior volumes in the hippocampus with greater area deprivation index, a census-based index of SES [45]. Decker et al. found that lower household income was related to smaller anterior hippocampal volumes [44]. Clearly, the preponderance of the evidence favors smaller volumes in the head of the hippocampus.

Regarding the amygdala, there is a raft of inconsistencies in past work focused on amygdala volumes, SES, and stress exposure (see [15]. Multiple groups have reported smaller (whole) amygdala volumes in lower SES youth [11–13], but results have not been perfectly uniform (for review, see [15]). There has been no published work to date focused on SES and amygdala subnuclei. In adult samples exposed to childhood adversity, smaller volumes of basal and

accessory basal portions of the amygdala have been reported [41,42]. Oshri and colleagues also found high levels of adversity were related to smaller volumes in central-medial portions of the amygdala. Our findings extend these relations to multiple amygdala subdivisions and use the statistical power of a large sample to identify late adolescence as the period these changes are most evident.

Related to age-specific effects, we see more consistent associations in older participants, and this is broadly in accord with past work. For example, Merz et al. found lower SES was significantly associated with smaller amygdala volumes in adolescent participants, but there were no significant associations at younger ages [70]. However, results are not perfectly uniform [8], and findings should be interpreted with caution given the cross-sectional nature of our work. Regarding sex-specific effects, limited work has specifically considered relations between SES and these volumes in males versus females. There are inconsistencies in past studies examining SES effects in males compared to females (with Ref. [71] noting larger structural effects for boys, but Refs. [72,73] noting the opposite for functional brain activity). Looking at preclinical work and considering “sex as a biological variable”, stress exposure often causes more significant neurobiological alterations in males compared to females [74]. Chronic stress exposure causes dendritic atrophy and spine elimination in male rodents [75]. However, in female rats, only slight changes in dendritic branch number were found [76]. This would fit with potential neuroprotective effects of estrogen [77]. It is also possible that relations may depend on both the age and sex of a participant. For example, adversity exposure before 8 years of age was more likely to impact hippocampal volumes in males, while adversity after 9 years of age impacted females more significantly [78]. Additional work, especially longitudinal studies will be critical to providing clarity about the neurobiological impacts of SES and also exposure to stress in different age- and sex-subpopulations.

Given our results, it will be crucial to think about how these neurobiological alterations may influence future behavior. We did not explore relations between volume and behavior, as specific behavioral measures greatly varied by project. In thinking about relations between SES and the head of the hippocampus, a growing literature suggests that this hippocampal subdivision may be more related to emotion and stress responding [24,79,80]. Lesions to this region in rodents [81], as well as variations in human functional connectivity of this area [82–84], have been linked to alterations in stress responding and emotional behavior. Thinking about the amygdala, we find multiple subnuclei are related to SES and these different divisions have been implicated in different aspects of fear responsivity and reward valuation [85]. More basal portions of the amygdala may serve in sensory gating for the amygdala, while the central nucleus relays emotional information to the cortex. Collectively, these alterations could create attentional biases toward negative valenced stimuli, potentially contributing to challenges in emotion regulation [42,86,87]. Preclinical work supports these broad presumptions, as mice with smaller basolateral volumes show significantly greater levels of conditioned freezing compared to those larger volumes [88]. These different behavioral processes will be important to interrogate in future work connecting SES and medial temporal lobe neurobiology.

Considering the strengths of our work, the current study benefited from a large sample size, well-used methods, and an SES composite considering both parental education and income. However, we must consider potential issues with the work. First, our work was cross-sectional in nature, based on a single MRI scan. Volumetric differences could “equalize” over time; this may be particularly true of the hippocampus, where research has demonstrated reversibility in volumetric differences if given a “stress-free” period [89]. In future work, we hope to assess other structural and functional properties of the amygdala and hippocampus through the use of longitudinal functional MRI and magnetic resonance spectroscopy [90]. Second, automated

methods, like Freesurfer, may be less accurate than hand-delineation of these subdivisions [91]. Quantification of these areas by hand is not feasible in such a large sample, but there may be novel automated approaches that more accurately quantify small amygdala and hippocampal subdivisions [92,93]. Finally, it will be important to think mechanistically about how lower SES may be impacting neurobiology. Lower SES encompass a host of challenges likely to impact development, including higher levels of stress, food insecurity, residential instability, community violence, and structural disadvantage [94,95]. This fits with past work finding relations between hippocampal volumes and rich measures of stress exposure [11]. Similarly, environmental stimulation is another proximal factor through which SES may impact these volumes, especially the hippocampus. Economically marginalized families may have lower levels of cognitive stimulation in the home, including fewer toys and educational resources [95,96]. Environmental enrichment and stimulation can impact hippocampal structure, including dendritic branching, neurogenesis, synaptic density (for review, see [97,98]). Probing different dimensions of experience, common to poverty (e.g., stress exposure; environmental stimulation) to understand the patterns reported here will be critical moving forward [99,100]. In our supplement, we examined income and parental education independently and these are important sets of results to also consider. Thoroughly isolating major drivers of neurobiological and behavioral differences could be particularly powerful, especially if this information can be translated into effective interventions to lessen different SES-related disparities.

These limitations notwithstanding, our results provide data about neurobiological alterations seen in relation to SES. Few investigations have examined subdivisions of the amygdala and hippocampus in relation to SES; and no projects to our knowledge have examined sex- and age-specific subgroups for these associations. Such neurobiological differences may connect to SES-gradients in health and well-being. Additional research is needed to clarify the complex relations among early poverty exposure and long-term mental health difficulties; our data are, however, a needed step in the ability to understand the impact of SES on neurobiology critical for emotion, memory, and learning.

Author contributions: Jamie Hanson conceived of the project, outlined approaches to statistical analyses, and wrote the majority of the manuscript. Dorthea Adkins wrote portions of the manuscript and constructed statistical models. Brendon Nacewicz guided the team on conceptual and methodological elements of amygdala neurobiology and provided feedback on draft writing. Kelly Barry aided with data cleaning, coding, and organization.

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REFERENCES

1 Peverill M, Dirks MA, Narvaja T, Herts KL, Comer JS, McLaughlin KA. Socioeconomic status and child psychopathology in the United States: A meta-analysis of population-based studies. *Clin Psychol Rev* 2021; **83**: 101933.

2 Piotrowska PJ, Stride CB, Croft SE, Rowe R. Socioeconomic status and antisocial behaviour among children and adolescents: A systematic review and meta-analysis. *Clin Psychol Rev* 2015; **35**: 47–55.

3 Hair NL, Hanson JL, Wolfe BL, Pollak SD. Association of child poverty, brain development, and academic achievement. *JAMA Pediatr* 2015; **169**. doi:10.1001/jamapediatrics.2015.1475.

4 Hair NL, Hanson JL, Wolfe BL, Pollak SD. Low household income and neurodevelopment from infancy through adolescence. *PloS One* 2022; **17**: e0262607.

5 McEwen BS. Physiology and neurobiology of stress and adaptation: central role of the brain. *Physiol Rev* 2007; **87**: 873–904.

6 Noble KG, Giebler MA. The neuroscience of socioeconomic inequality. *Curr Opin Behav Sci* 2020; **36**: 23–28.

7 Johnson SB, Riis JL, Noble KG. State of the art review: poverty and the developing brain. *Pediatrics* 2016; **137**.

8 Yu Q, Daugherty AM, Anderson DM, Nishimura M, Brush D, Hardwick A *et al.* Socioeconomic status and hippocampal volume in children and young adults. *Dev Sci* 2018; **21**: e12561.

9 Ellwood-Lowe ME, Humphreys KL, Ordaz SJ, Camacho MC, Sacchet MD, Gotlib IH. Time-varying effects of income on hippocampal volume trajectories in adolescent girls. *Dev Cogn Neurosci* 2018; **30**: 41–50.

10 Hanson JL, Chandra A, Wolfe BL, Pollak SD. Association between Income and the Hippocampus. *PloS One* 2011; **6**: e18712.

11 Hanson JL, Nacewicz BM, Sutterer MJ, Cayo AA, Schaefer SM, Rudolph KD *et al.* Behavioral problems after early life stress: contributions of the hippocampus and amygdala. *Biol Psychiatry* 2015; **77**: 314–323.

12 Luby J, Belden A, Botteron K, Marrus N, Harms MP, Babb C *et al.* The effects of poverty on childhood brain development: the mediating effect of caregiving and stressful life events. *JAMA Pediatr* 2013; **167**: 1135–1142.

13 Noble KG, Houston SM, Kan E, Sowell ER. Neural correlates of socioeconomic status in the developing human brain. *Dev Sci* 2012; **15**: 516–527.

14 Noble KG, Houston SM, Brito NH, Bartsch H, Kan E, Kuperman JM *et al.* Family income, parental education and brain structure in children and adolescents. *Nat Neurosci* 2015; **18**: 773–778.

15 Hanson JL, Nacewicz BM. Amygdala allostasis and early life adversity: considering excitotoxicity and inescapability in the sequelae of stress. *Front Hum Neurosci* 2021.

16 Adolphs R. What does the amygdala contribute to social cognition? *Ann N Y Acad Sci* 2010; **1191**: 42–61.

17 LeDoux J. The amygdala. *Curr Biol* 2007; **17**: R868–R874.

18 Shohamy D, Turk-Browne NB. Mechanisms for widespread hippocampal involvement in cognition. *J Exp Psychol Gen* 2013; **142**: 1159.

19 Murty VP, Calabro F, Luna B. The role of experience in adolescent cognitive development: Integration of executive, memory, and mesolimbic systems. *Neurosci Biobehav Rev* 2016; **70**: 46–58.

20 Klein-Flügge MC, Jensen DE, Takagi Y, Priestley L, Verhagen L, Smith SM *et al.* Relationship between nuclei-specific amygdala connectivity and mental health dimensions in humans. *Nat Hum Behav* 2022; : 1–18.

21 Swanson LW, Petrovich GD. What is the amygdala? *Trends Neurosci* 1998; **21**: 323–331.
22 Vogel JW, La Joie R, Grothe MJ, Diaz-Papkovich A, Doyle A, Vachon-Presseau E *et al.* A molecular gradient along the longitudinal axis of the human hippocampus informs large-scale behavioral systems. *Nat Commun* 2020; **11**: 1–17.
23 Gruber MJ, Ranganath C. How curiosity enhances hippocampus-dependent memory: the prediction, appraisal, curiosity, and exploration (PACE) framework. *Trends Cogn Sci* 2019; **23**: 1014–1025.
24 Strange BA, Witter MP, Lein ES, Moser EI. Functional organization of the hippocampal longitudinal axis. *Nat Rev Neurosci* 2014; **15**: 655–669.
25 Poppenk J, Moscovitch M. A hippocampal marker of recollection memory ability among healthy young adults: contributions of posterior and anterior segments. *Neuron* 2011; **72**: 931–937.
26 Fanselow MS, Dong H-W. Are the dorsal and ventral hippocampus functionally distinct structures? *Neuron* 2010; **65**: 7–19.
27 Hartung CM, Lefler EK. Sex and gender in psychopathology: DSM–5 and beyond. *Psychol Bull* 2019; **145**: 390.
28 Stevens JS, Hamann S. Sex differences in brain activation to emotional stimuli: a meta-analysis of neuroimaging studies. *Neuropsychologia* 2012; **50**: 1578–1593.
29 van Eijk L, Hansell NK, Strike LT, Couvy-Duchesne B, de Zubicaray GI, Thompson PM *et al.* Region-specific sex differences in the hippocampus. *Neuroimage* 2020; **215**: 116781.
30 Yagi S, Galea LA. Sex differences in hippocampal cognition and neurogenesis. *Neuropsychopharmacology* 2019; **44**: 200–213.
31 Shansky RM, Woolley CS. Considering sex as a biological variable will be valuable for neuroscience research. *J Neurosci* 2016; **36**: 11817–11822.
32 Bale TL, Epperson CN. Sex as a biological variable: who, what, when, why, and how. *Neuropsychopharmacology* 2017; **42**: 386–396.
33 Bath KG. Synthesizing views to understand sex differences in response to early life adversity. *Trends Neurosci* 2020; **43**: 300–310.
34 Lenroot RK, Giedd JN. Brain development in children and adolescents: insights from anatomical magnetic resonance imaging. *Neurosci Behav Rev* 2006; **30**: 718–729.
35 Teicher MH, Anderson CM, Polcari A. Childhood maltreatment is associated with reduced volume in the hippocampal subfields CA3, dentate gyrus, and subiculum. *Proc Natl Acad Sci* 2012; **109**: E563–E572.
36 Yuan M, Rubin-Falcone H, Lin X, Rizk MM, Miller JM, Sublette ME *et al.* Smaller left hippocampal subfield CA1 volume is associated with reported childhood physical and/or sexual abuse in major depression: a pilot study. *J Affect Disord* 2020; **272**: 348–354.
37 Lee SW, Yoo JH, Kim KW, Kim D, Park H, Choi J *et al.* Hippocampal subfields volume reduction in high schoolers with previous verbal abuse experiences. *Clin Psychopharmacol Neurosci* 2018.
38 Margolis AE, Cohen JW, Ramphal B, Thomas L, Rauh V, Herbstman J *et al.* Prenatal Exposure to Air Pollution and Early Life Stress Effects on Hippocampal Subregional Volumes and Associations with Visual-Spatial Reasoning. *Biol Psychiatry Glob Open Sci* 2022.
39 Brody GH, Gray JC, Yu T, Barton AW, Beach SR, Galván A *et al.* Protective prevention effects on the association of poverty with brain development. *JAMA Pediatr* 2017; **171**: 46–52.
40 Malhi GS, Das P, Outhred T, Irwin L, Gessler D, Bwabi Z *et al.* The effects of childhood trauma on adolescent hippocampal subfields. *Aust N Z J Psychiatry* 2019; **53**: 447–457.
41 Nogovitsyn N, Addington J, Souza R, Placsko TJ, Stowkowy J, Wang J *et al.* Childhood trauma and amygdala nuclei volumes in youth at risk for mental illness. *Psychol Med* 2022; **52**: 1192–1199.

42 Oshri A, Gray JC, Owens MM, Liu S, Duprey EB, Sweet LH *et al.* Adverse childhood experiences and amygdalar reduction: high-resolution segmentation reveals associations with subnuclei and psychiatric outcomes. *Child Maltreat* 2019; **24**: 400–410.
43 Merz EC, Desai PM, Maskus EA, Melvin SA, Rehman R, Torres SD *et al.* Socioeconomic disparities in chronic physiologic stress are associated with brain structure in children. *Biol Psychiatry* 2019; **86**: 921–929.
44 Decker AL, Duncan K, Finn AS, Mabbott DJ. Children's family income is associated with cognitive function and volume of anterior not posterior hippocampus. *Nat Commun* 2020; **11**: 1–11.
45 Botdorf M, Dunstan J, Sorcher L, Dougherty LR, Riggins T. Socioeconomic disadvantage and episodic memory ability in the ABCD sample: Contributions of hippocampal subregion and subfield volumes. *Dev Cogn Neurosci* 2022; **57**: 101138.
46 McEwen BS. Sex, stress and the hippocampus: allostasis, allostatic load and the aging process. *Neurobiol Aging* 2002; **23**: 921–939.
47 McEwen BS. Hormones and behavior and the integration of brain-body science. *Horm Behav* 2020; **119**: 104619.
48 Aoki C, Romeo RD, Smith SS. Adolescence as a critical period for developmental plasticity. *Brain Res* 2017.
49 Brown SA, Brumback T, Tomlinson K, Cummins K, Thompson WK, Nagel BJ *et al.* The national consortium on alcohol and neuro-development in adolescence (NCANDA): A multisite study of adolescent development and substance use. *J Stud Alcohol Drugs* 2015; **76**: 895–908.
50 Alexander LM, Escalera J, Ai L, Andreotti C, Febre K, Mangone A *et al.* An open resource for transdiagnostic research in pediatric mental health and learning disorders. *Sci Data* 2017; **4**: 1–26.
51 Jernigan TL, Brown TT, Hagler Jr DJ, Akshoomoff N, Bartsch H, Newman E *et al.* The pediatric imaging, neurocognition, and genetics (PING) data repository. *Neuroimage* 2016; **124**: 1149–1154.
52 Somerville LH, Bookheimer SY, Buckner RL, Burgess GC, Curtiss SW, Dapretto M *et al.* The Lifespan Human Connectome Project in Development: A large-scale study of brain connectivity development in 5–21 year olds. *Neuroimage* 2018; **183**: 456–468.
53 Avesani P, McPherson B, Hayashi S, Caiafa CF, Henschel R, Garyfallidis E *et al.* The open diffusion data derivatives, brain data upcycling via integrated publishing of derivatives and reproducible open cloud services. *Sci Data* 2019; **6**. doi:10.1038/s41597-019-0073-y.
54 Dale AM, Fischl B, Sereno MI. Cortical surface-based analysis: I. Segmentation and surface reconstruction. *NeuroImage* 1999. doi:10.1006/nimg.1998.0395.
55 Fischl B, Dale AM. Measuring the thickness of the human cerebral cortex from magnetic resonance images. *Proc Natl Acad Sci U S A* 2000; **97**: 11050–11055.
56 Iglesias JE, Augustinack JC, Nguyen K, Player CM, Player A, Wright M *et al.* A computational atlas of the hippocampal formation using ex vivo, ultra-high resolution MRI: Application to adaptive segmentation of in vivo MRI. *Neuroimage* 2015; **115**: 117-137.
57 Saygin ZM, Kliemann D, Iglesias JE, van der Kouwe AJ, Boyd E, Reuter M *et al.* High-resolution magnetic resonance imaging reveals nuclei of the human amygdala: manual segmentation to automatic atlas. *Neuroimage* 2017; **155**: 370–382.
58 Genon S, Bernhardt BC, La Joie R, Amunts K, Eickhoff SB. The many dimensions of human hippocampal organization and (dys)function. *Trends Neurosci* 2021; **44**: 977–989.
59 Kahhale I, Buser NJ, Madan CR, Hanson JL. Quantifying Numerical and Spatial Reliability of Amygdala and Hippocampal Subdivisions in FreeSurfer. *bioRxiv* 2021. doi:10.1101/2020.06.12.149203.
60 Wisse LE, Chételat G, Daugherty AM, de Flores R, la Joie R, Mueller SG *et al.* Hippocampal subfield volumetry from structural isotropic 1 mm3 MRI scans: a note of

caution. *Hum Brain Mapp* 2021; **42**: 539–550.
61 Adhikari A, Lerner TN, Finkelstein J, Pak S, Jennings JH, Davidson TJ *et al.* Basomedial amygdala mediates top-down control of anxiety and fear. *Nature* 2015; **527**: 179–185.
62 Marowsky A, Yanagawa Y, Obata K, Vogt KE. A specialized subclass of interneurons mediates dopaminergic facilitation of amygdala function. *Neuron* 2005; **48**: 1025–1037.
63 Caparelli EC, Ross TJ, Gu H, Liang X, Stein EA, Yang Y. Graph theory reveals amygdala modules consistent with its anatomical subdivisions. *Sci Rep* 2017; **7**: 1–14.
64 Gaser C, Dahnke R, Thompson PM, Kurth F, Luders E. CAT-a computational anatomy toolbox for the analysis of structural MRI data. *BioRxiv* 2022.
65 Gilmore AD, Buser NJ, Hanson JL. Variations in structural MRI quality significantly impact commonly used measures of brain anatomy. *Brain Inform* 2021; **8**: 1–15.
66 Parker RN, Fenwick R. The Pareto curve and its utility for open-ended income distributions in survey research. *Soc Forces* 1983; **61**: 872–885.
67 Bates D, Mächler M, Bolker B, Walker S. Fitting Linear Mixed-Effects Models Using lme4. *J Stat Softw* 2015; **67**: 1–48.
68 Benjamini Y, Hochberg Y. Controlling the false discovery rate: a practical and powerful approach to multiple testing. *J R Stat Soc Ser B Methodol* 1995; **57**: 289–300.
69 Shaw P, Eckstrand K, Sharp W, Blumenthal J, Lerch J, Greenstein D *et al.* Attention-deficit/hyperactivity disorder is characterized by a delay in cortical maturation. *Proc Natl Acad Sci* 2007; **104**: 19649–19654.
70 Merz EC, Tottenham N, Noble KG. Socioeconomic Status, Amygdala Volume, and Internalizing Symptoms in Children and Adolescents. *J Clin Child Adolesc Psychol* 2018; **47**: 312–323.
71 King LS, Dennis EL, Humphreys KL, Thompson PM, Gotlib IH. Cross-sectional and longitudinal associations of family income-to-needs ratio with cortical and subcortical brain volume in adolescent boys and girls. *Dev Cogn Neurosci* 2020; **44**: 100796.
72 Kim D-J, Davis EP, Sandman CA, Glynn L, Sporns O, O'Donnell BF *et al.* Childhood poverty and the organization of structural brain connectome. *NeuroImage* 2019; **184**: 409–416.
73 Rakesh D, Seguin C, Zalesky A, Cropley V, Whittle S. Associations between neighborhood disadvantage, resting-state functional connectivity, and behavior in the adolescent brain cognitive development study: the moderating role of positive family and school environments. *Biol Psychiatry Cogn Neurosci Neuroimaging* 2021; **6**: 877–886.
74 Shansky RM, Murphy AZ. Considering sex as a biological variable will require a global shift in science culture. *Nat Neurosci* 2021; **24**: 457–464.
75 McEwen BS. Plasticity of the hippocampus: adaptation to chronic stress and allostatic load. *Ann N Y Acad Sci* 2001; **933**: 265–277.
76 Galea L, McEwen B, Tanapat P, Deak T, Spencer R, Dhabhar F. Sex differences in dendritic atrophy of CA3 pyramidal neurons in response to chronic restraint stress. *Neuroscience* 1997; **81**: 689–697.
77 McEwen BS. Invited review: Estrogens effects on the brain: multiple sites and molecular mechanisms. *J Appl Physiol* 2001; **91**: 2785–2801.
78 Teicher MH, Anderson CM, Ohashi K, Khan A, McGreenery CE, Bolger EA *et al.* Differential effects of childhood neglect and abuse during sensitive exposure periods on male and female hippocampus. *Neuroimage* 2018; **169**: 443–452.
79 Herman J, Dolgas C, Carlson S. Ventral subiculum regulates hypothalamo–pituitary–adrenocortical and behavioural responses to cognitive stressors. *Neuroscience* 1998; **86**: 449–459.
80 Kheirbek MA, Drew LJ, Burghardt NS, Costantini DO, Tannenholz L, Ahmari SE *et al.* Differential control of learning and anxiety along the dorsoventral axis of the dentate gyrus. *Neuron* 2013; **77**: 955–968.

81 Henke PG. Hippocampal pathway to the amygdala and stress ulcer development. *Brain Res Bull* 1990; **25**: 691–695.
82 Gryglewski G, Baldinger-Melich P, Seiger R, Godbersen GM, Michenthaler P, Klöbl M *et al.* Structural changes in amygdala nuclei, hippocampal subfields and cortical thickness following electroconvulsive therapy in treatment-resistant depression: longitudinal analysis. *Br J Psychiatry* 2019; **214**: 159–167.
83 Hanson JL, Gillmore AD, Yu T, Holmes CJ, Hallowell ES, Barton AW *et al.* A family focused intervention influences hippocampal-prefrontal connectivity through gains in self-regulation. *Child Dev* 2019; **90**: 1389–1401.
84 Hubachek S, Botdorf M, Riggins T, Leong H-C, Klein DN, Dougherty LR. Hippocampal subregion volume in high-risk offspring is associated with increases in depressive symptoms across the transition to adolescence. *J Affect Disord* 2021; **281**: 358–366.
85 Phelps EA, LeDoux JE. Contributions of the amygdala to emotion processing: from animal models to human behavior. *Neuron* 2005; **48**: 175–187.
86 Barry KR, Hanson JL, Calma-Birling D, Lansford JE, Bates JE, Dodge KA. Developmental connections between socioeconomic status, self-regulation, and adult externalizing problems. *Dev Sci* 2022; : e13260.
87 Hanson JL, Albert WD, Skinner AT, Shen SH, Dodge KA, Lansford JE. Resting state coupling between the amygdala and ventromedial prefrontal cortex is related to household income in childhood and indexes future psychological vulnerability to stress. *Dev Psychopathol* 2019; **31**: 1053–1066.
88 Yang RJ, Mozhui K, Karlsson R-M, Cameron HA, Williams RW, Holmes A. Variation in mouse basolateral amygdala volume is associated with differences in stress reactivity and fear learning. *Neuropsychopharmacology* 2008; **33**: 2595–2604.
89 Vyas A, Mitra R, Rao BS, Chattarji S. Chronic stress induces contrasting patterns of dendritic remodeling in hippocampal and amygdaloid neurons. *J Neurosci* 2002; **22**: 6810–6818.
90 Nacewicz BM, Angelos L, Dalton KM, Fischer R, Anderle MJ, Alexander AL *et al.* Reliable non-invasive measurement of human neurochemistry using proton spectroscopy with an anatomically defined amygdala-specific voxel. *Neuroimage* 2012; **59**: 2548–2559.
91 Hanson JL, Suh JW, Nacewicz BM, Sutterer MJ, Cayo AA, Stodola DE *et al.* Robust automated amygdala segmentation via multi-atlas diffeomorphic registration. *Front Neurosci* 2012; **6**: 166.
92 DeKraker J, Haast RA, Yousif MD, Karat B, Lau JC, Köhler S *et al.* Automated hippocampal unfolding for morphometry and subfield segmentation with HippUnfold. *Elife* 2022; **11**: e77945.
93 Liu Y, Nacewicz BM, Zhao G, Adluru N, Kirk GR, Ferrazzano PA *et al.* A 3D fully convolutional neural network with top-down attention-guided refinement for accurate and robust automatic segmentation of amygdala and its subnuclei. *Front Neurosci* 2020; **14**: 260.
94 Masarik AS, Conger RD. Stress and child development: A review of the Family Stress Model. *Curr Opin Psychol* 2017; **13**: 85–90.
95 Evans GW. The environment of childhood poverty. *Am Psychol* 2004; **59**: 77.
96 Merz EC, He X, Myers B, Noble KG. Socioeconomic disadvantage, chronic stress, and hippocampal subfield development in children. *Neurosci Insights* 2020; **15**: 2633105520931098.
97 Ohline S, Abraham W. Environmental enrichment effects on synaptic and cellular physiology of hippocampal neurons. *Neuropharmacology* 2019; **145**: 3–12.
98 Van Praag H, Kempermann G, Gage FH. Neural consequences of enviromental enrichment. *Nat Rev Neurosci* 2000; **1**: 191–198.
99 Palacios-Barrios EE, Hanson JL, Barry KR, Albert WD, White SF, Skinner AT *et al.* Lower

neural value signaling in the prefrontal cortex is related to childhood family income and depressive symptomatology during adolescence. *Dev Cogn Neurosci* 2021; **48**: 100920.
100 Palacios-Barrios EE, Hanson JL. Poverty and self-regulation: Connecting psychosocial processes, neurobiology, and the risk for psychopathology. *Compr Psychiatry* 2019; **90**: 52–64.

Table and Figure Legend

Table 1*.* Demographic information for the full sample, including the means and standard deviations for age (in years), income (in US dollars), highest parental education (in years), image quality, and total brain volume, as well as the distribution of sex in this sample.

Table 2. Beta (standardized) coefficients, t-statistics, p-values, and FDR-corrected p-values for all linear mixed effects models of the relations between socioeconomic status and volumes of the whole amygdala; whole hippocampus; amygdala subnuclei; and hippocampal subfields. Significant (uncorrected) p-values are shaded in yellow-green, and significant FDR-corrected p-values are shaded in green.

Table 3. Beta (standardized) coefficients for relations between SES and whole volume or subregional volume of the hippocampus or amygdala. These are divided by age and sex subsamples. Beta (standardized) coefficients are shaded yellow-green where the corresponding p-values were initially significant but did not survive corrections for multiple comparisons, and green where the corresponding p-values were significant after correction (using FDR methods).

Figure 1. Stacked histogram depicting the number of participants at each age, for each study, between the ages of 5 and 18. The top set of bins represents the numbers of participants in the HBN study at each age (colored in blue); stacked below are the bins for the HCPD (purple), NCANDA (green), and HBN (orange) studies, respectively. The dashed and dotted lines display the mean ages for each study (with each color corresponding to a different study; blue = HBN; purple = HCPD; green = NCANDA; orange = PING).

Figure 2. Depiction of our amygdala and hippocampal regions of interest. The left side of this figure shows our 4 amygdala subnuclei groups (Group 1: a basolateral complex [red]; Group 2: a medial cortical cluster [orange]; Group 3: the accessory basal subnuclei [blue]; Group 4: central amygdala subnuclei [green], overlaid on an average T1-weighted scan. The right side of this figure shows our 3 hippocampal subdivisions (Hippocampal head [yellow]; hippocampal body [turquoise]; hippocampal tail [magenta]).

Table 1. Demographic information for the full sample, including the means and standard deviations for age (in years), income (in US dollars), highest parental education (in years), image quality, and total brain volume, as well as the distribution of sex in this sample.

Table 1. Full Sample Demographics

Variable	N = 2,765[1]	Distributions
Age [Years]	11.9 (3.5)	
Sex		
F	1,228 (44%)	
M	1,537 (56%)	
Income($)	123,641 (99,112)	
Highest Parental Education [Years]	17.49 (2.95)	
Image Quality	0.856 (0.021)	
Total Brain Volume	1,501,276 (172,642)	

[1] Mean (SD); n (%)

Table 2. Beta (standardized) coefficients, t-statistics, p-values, and FDR-corrected p-values for all linear mixed effects models of the relations between socioeconomic status and volumes of the whole amygdala; whole hippocampus; amygdala subnuclei; and hippocampal subfields. Significant (uncorrected) p-values are shaded in yellow-green, and significant FDR-corrected p-values are shaded in green.

Statistics for the Effect of SES on Regional Volumes in the Full Sample

Region	β Coefficient	T Statistic	P Value	BH-corrected P-value
Whole Regions				
Whole Amygdala	0.058	4.085	**<0.001**	**<0.001**
Whole Hippocampus	0.060	4.160	**<0.001**	**<0.001**
Amygdala Subnuclei				
Accessory Basal	0.068	4.637	**<0.001**	**<0.001**
Central Amygdala	0.057	3.644	**<0.001**	**<0.001**
Basolateral Complex	0.048	3.361	**<0.001**	**0.001**
Medial Cortical Cluster	0.055	3.627	**<0.001**	**<0.001**
Hippocampal Subfields				
Hippocampal Head	0.093	6.192	**<0.001**	**<0.001**
Hippocampal Body	0.015	1.016	0.31	0.31
Hippocampal Tail	0.020	1.167	0.24	0.27

Table 3. Beta (standardized) coefficients for relations between SES and whole volume or subregional volume of the hippocampus or amygdala. These are divided by age and sex subsamples. Beta (standardized) coefficients are shaded yellow-green where the corresponding p-values were initially significant but did not survive corrections for multiple comparisons, and green where the corresponding p-values were significant after correction (using FDR methods).

Standardized Coefficients for the Effect of SES on Hippocampal and Amygdala Regions by Group									
	Whole Regions		**Amygdala Subnuclei**				**Hippocampal Subfields**		
Group	Whole Amygdala	Whole Hippocampus	Accessory Basal	Central Amygdala	Basolateral Complex	Medial Cortical Cluster	Hippocampal Head	Hippocampal Body	Hippocampal Tail
Pre Adolescence									
Boys	0.04160	0.07518	0.06625	0.11224	0.02249	0.05993	0.08225	0.05470	0.06753
Girls	0.07774	0.02440	0.06546	0.04217	0.07285	0.07297	0.06463	-0.00939	-0.04838
Early Adolescence									
Boys	0.05003	0.08166	0.05069	0.04539	0.04268	0.02965	0.11866	0.01500	0.04380
Girls	0.01019	0.02194	0.04801	0.02369	-0.00792	0.02532	0.04951	-0.00797	-0.00159
Late Adolescence									
Boys	0.10297	0.11444	0.10695	0.05349	0.09653	0.09476	0.17001	0.03593	0.03054
Girls	0.12221	0.10252	0.12839	0.11220	0.11701	0.08195	0.14373	0.01691	0.07665

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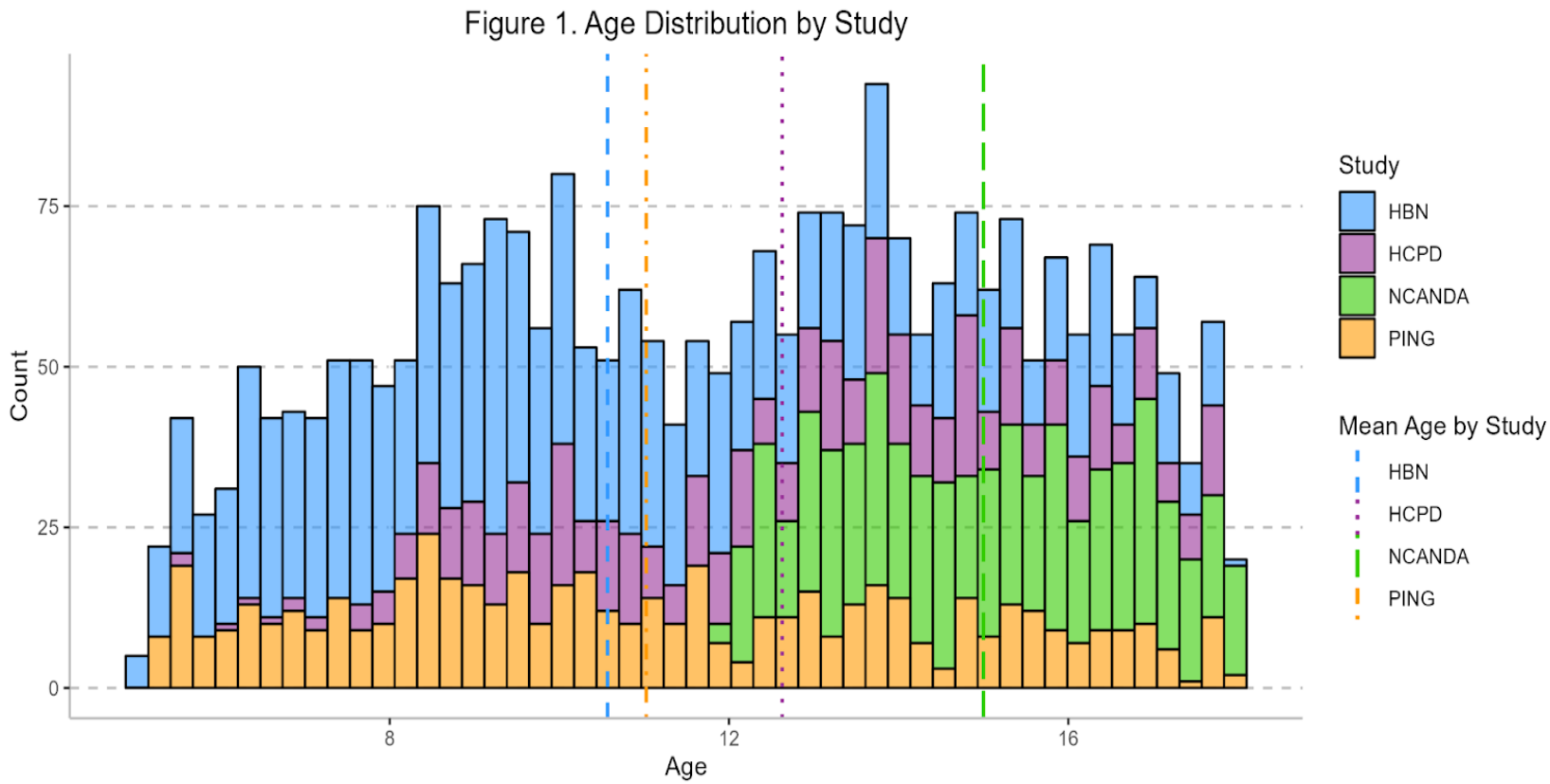
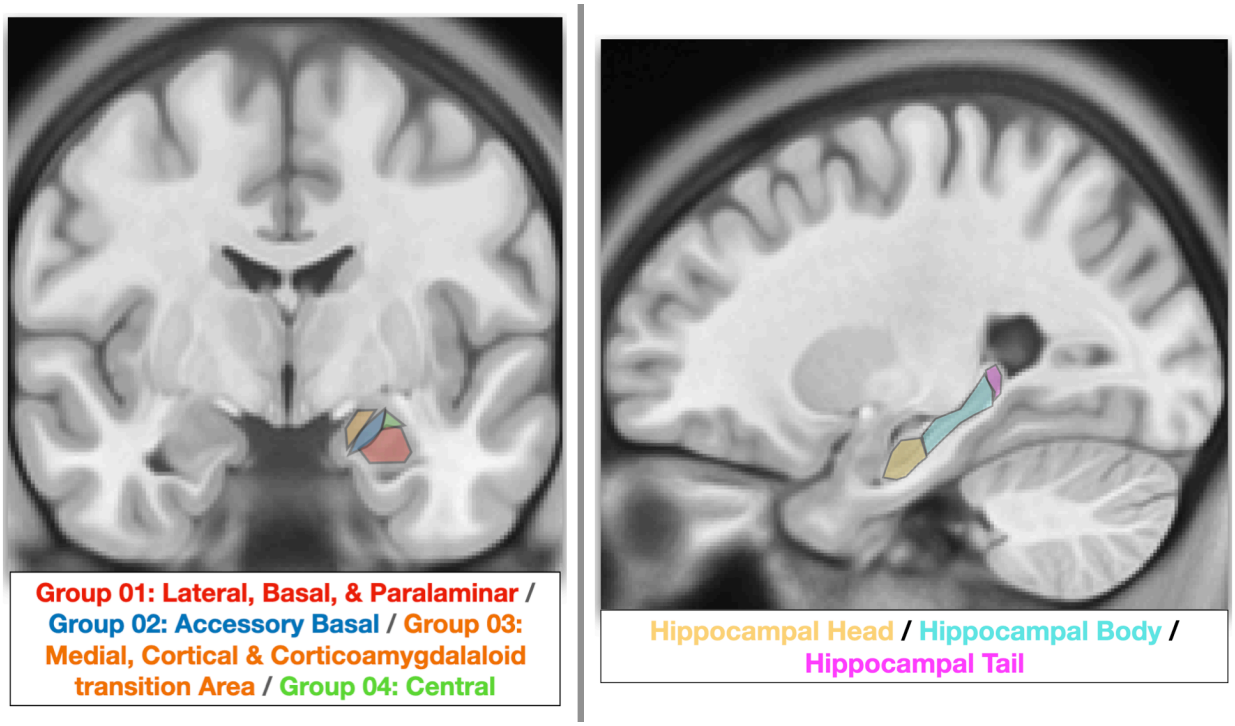
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