

北京师范大学 心理学部

Developmental Population Neuroscience 发展人口神经科学（私人网络与社区）

左西年 (Xi-Nian Zuo)



Beijing Normal University
State Key Lab of Cognitive Neuroscience & Learning

National Basic Science Data Center
Chinese Data-sharing Warehouse for In-vivo Imaging Brain

PERSPECTIVES

Environmental influences on the pace of brain development

Ursula A. Tooley¹, Danielle S. Bassett² and Allyson P. Mackey³

Abstract | Childhood socio-economic status (SES), a measure of the availability of material and social resources, is one of the strongest predictors of lifelong well-being. Here we review evidence that experiences associated with childhood SES affect not only the outcome but also the pace of brain development. We argue

SCIENCE AND SOCIETY

Socioeconomic status and the brain: mechanistic insights from human and animal research

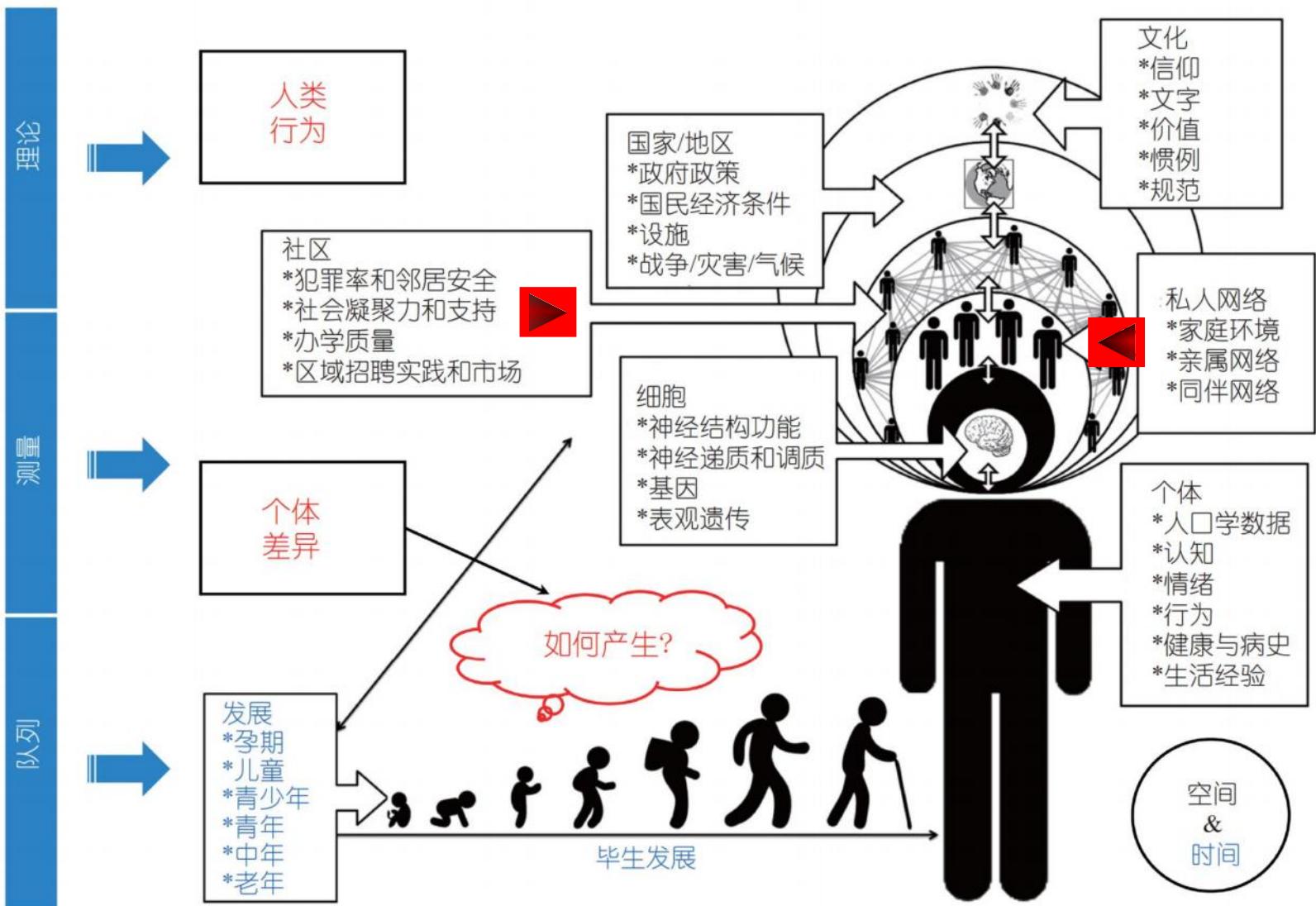
Daniel A. Hackman, Martha J. Farah and Michael J. Meaney

Abstract | Human brain development occurs within a socioeconomic context and childhood socioeconomic status (SES) influences neural development — particularly of the systems that subserve language and executive function. Research in humans and in animal models has implicated prenatal factors, parent–child interactions and cognitive stimulation in the home environment in the effects of SES on neural development. These findings provide a unique opportunity for understanding how environmental factors can lead to individual differences in brain development, and for improving the programmes and policies that are designed to alleviate SES-related disparities in mental health and academic achievement.

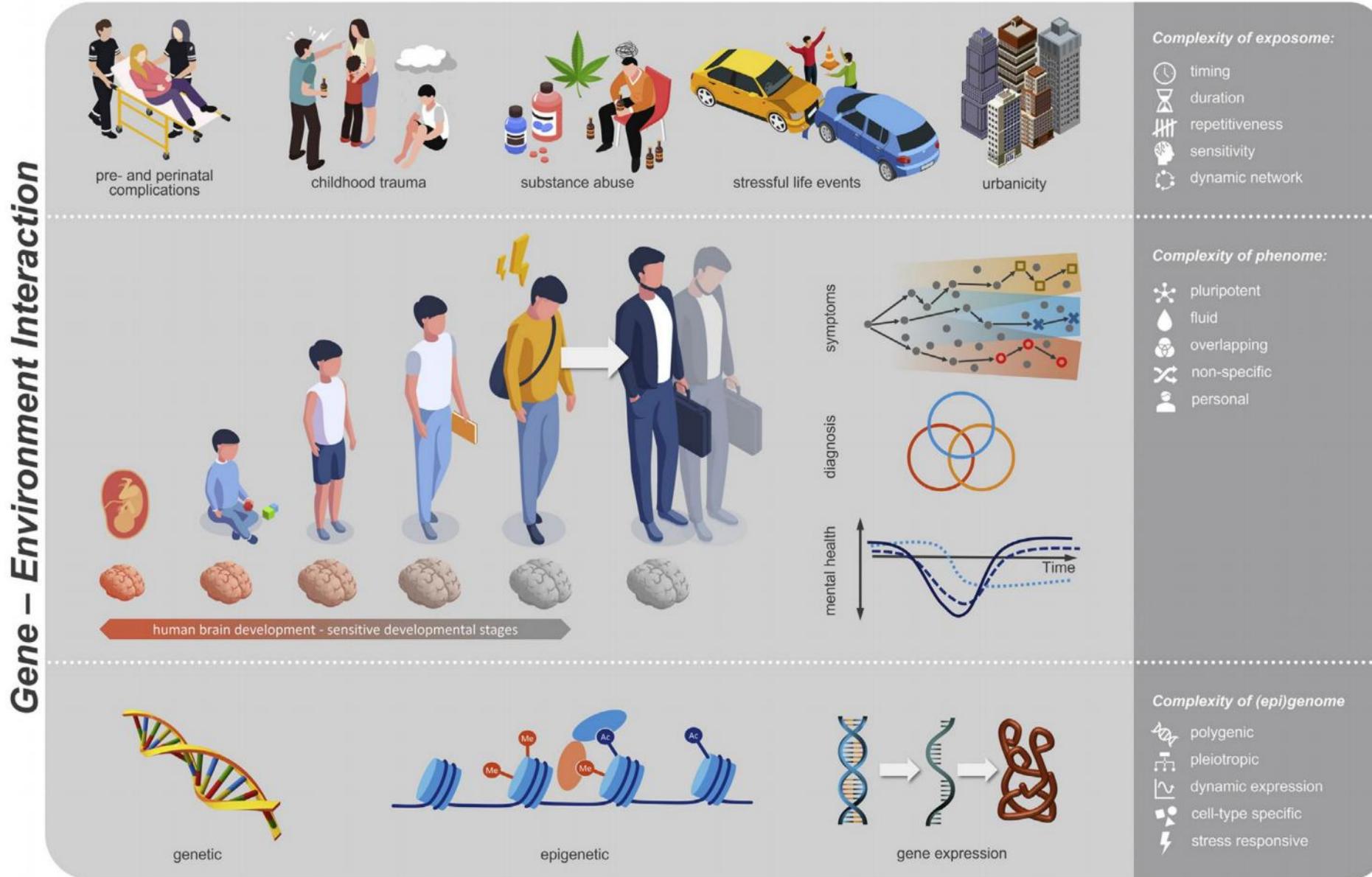
What is the expected human childhood? Insights from evolutionary anthropology

Willem E. Frankenhuys^{1,2} and Dorsa Amir^{3,4}

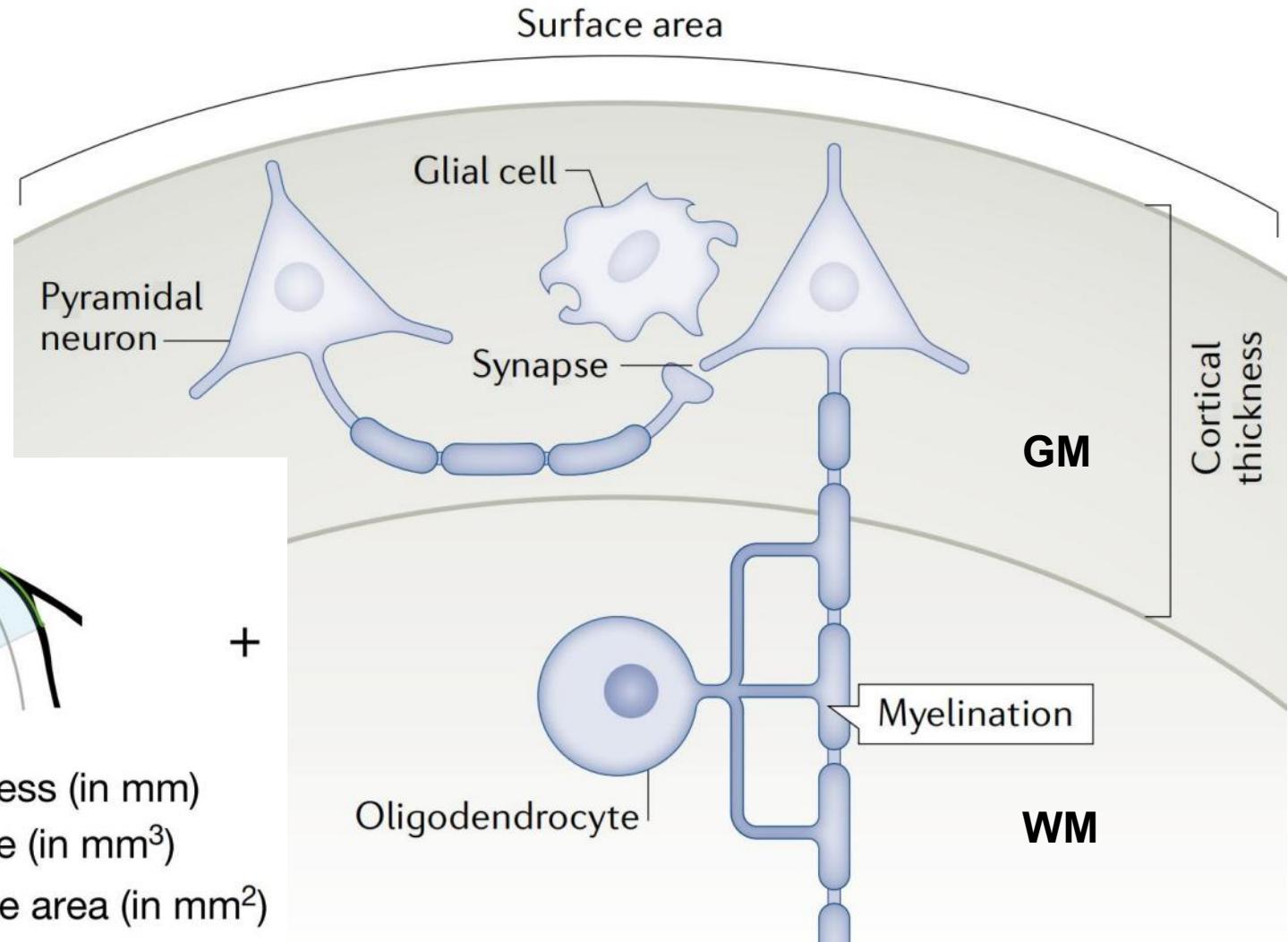
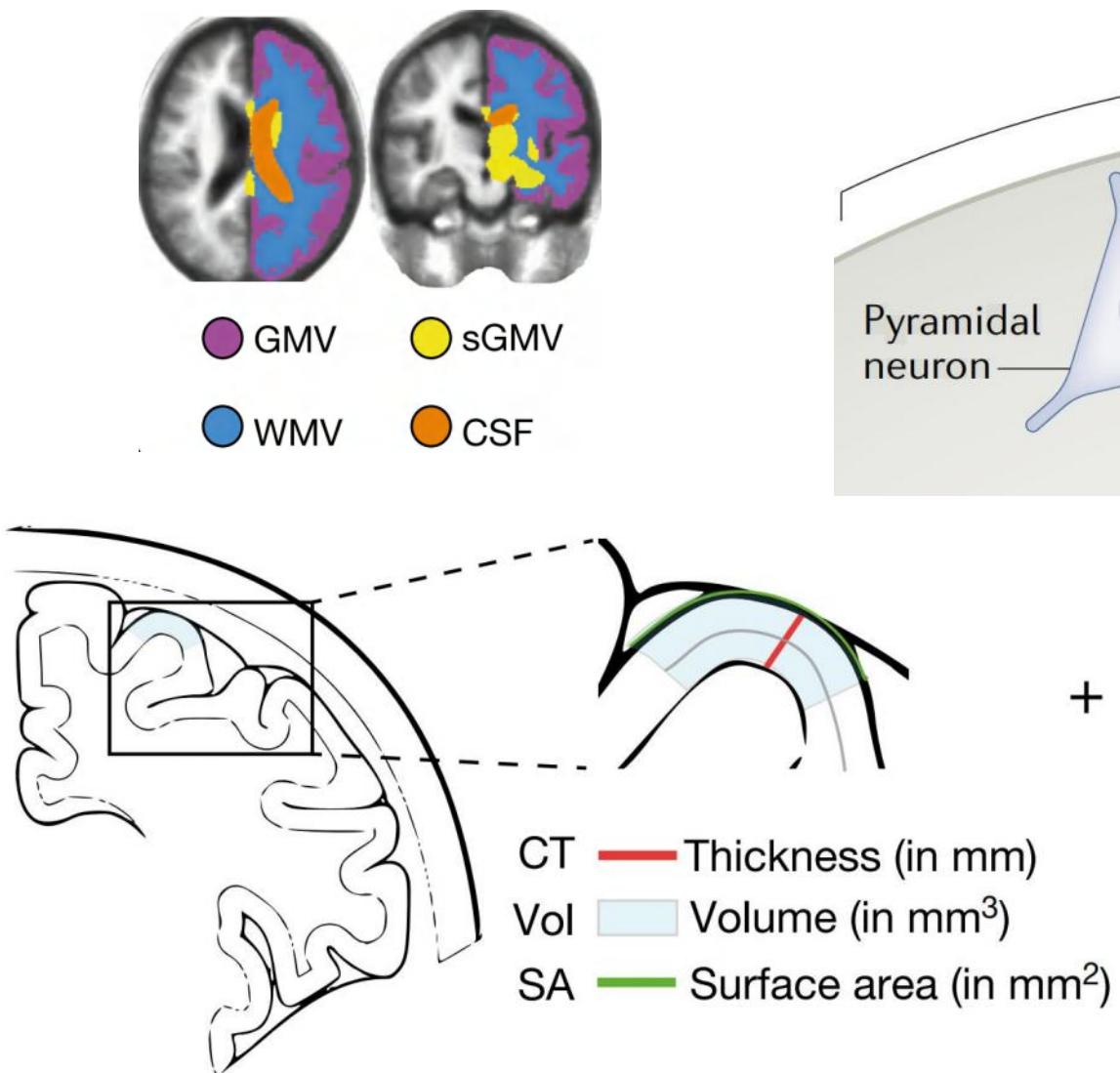
¹Department of Psychology, Utrecht University, Utrecht, the Netherlands, ²Max Planck Institute for the Study of Crime, Security and Law, Germany, ³Department of Psychology, Boston College, Chestnut Hill, USA and ⁴Department of Psychology, University of California, Berkeley, USA



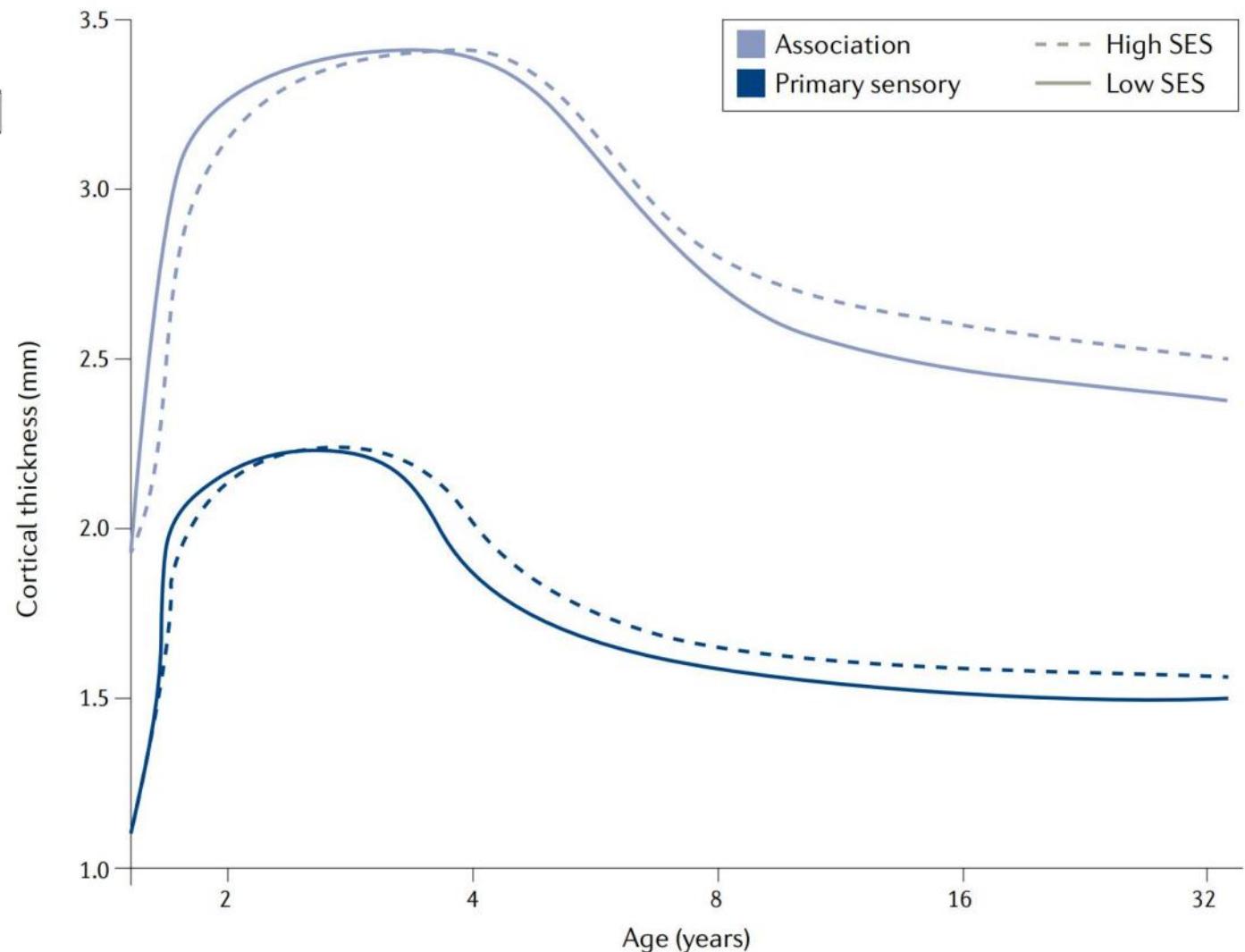
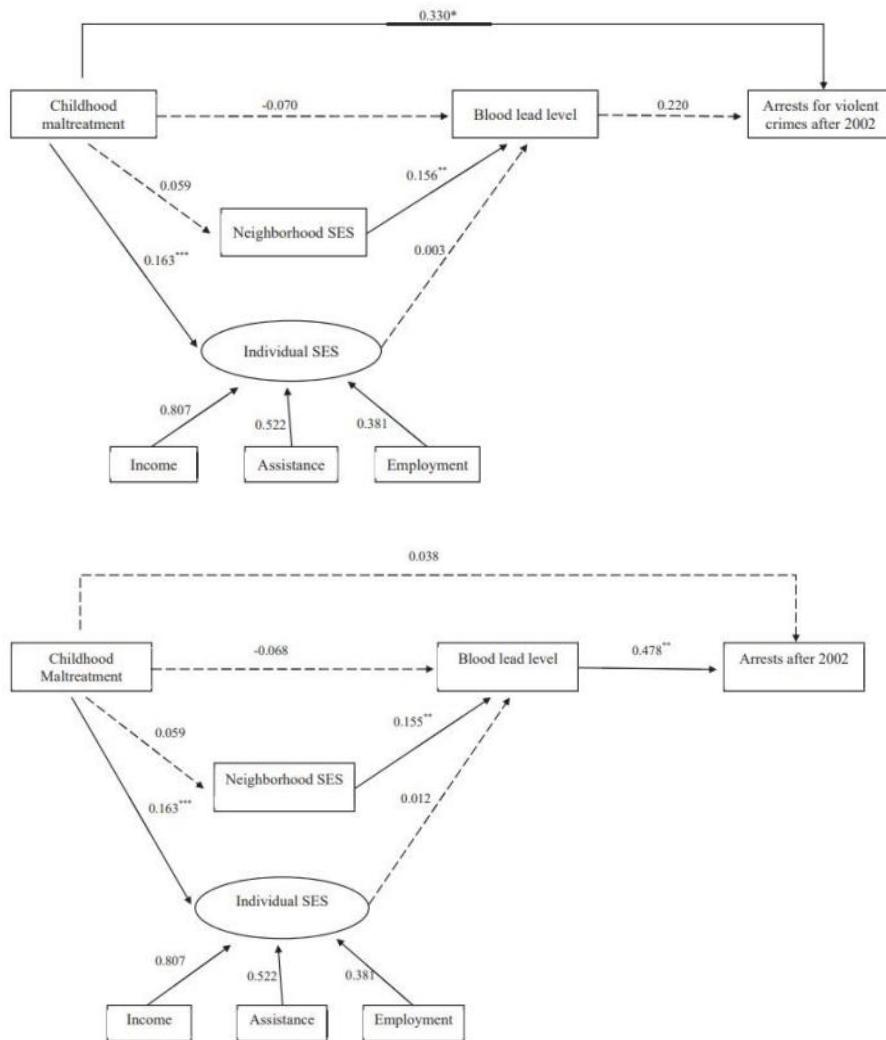
The Complexity: Genome-Phenome-Exposome



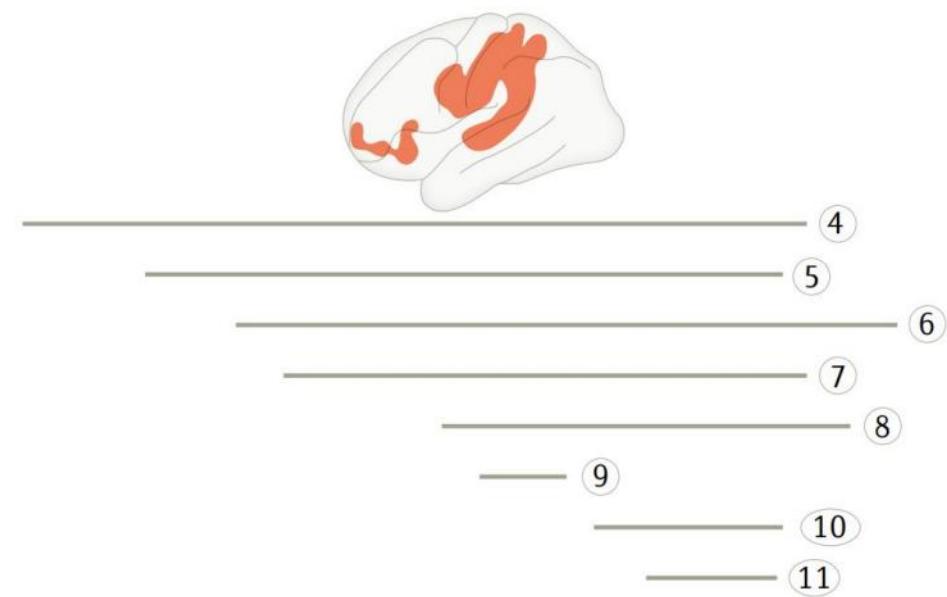
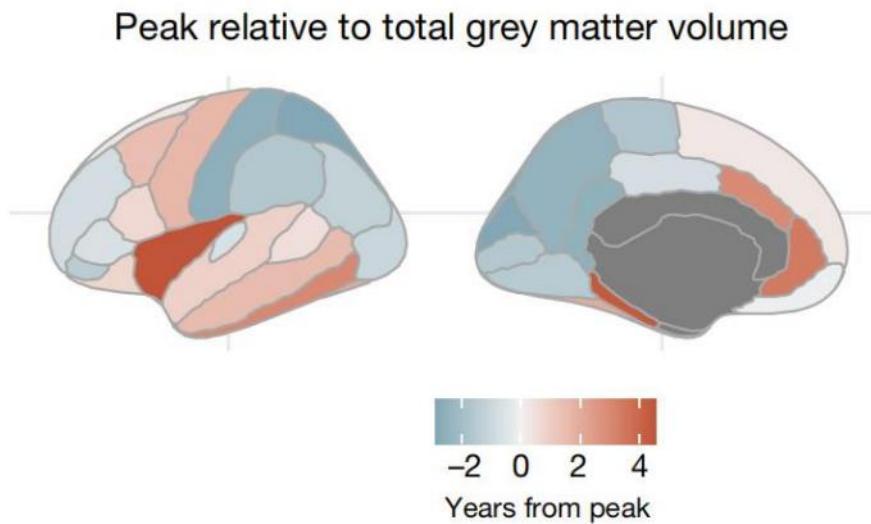
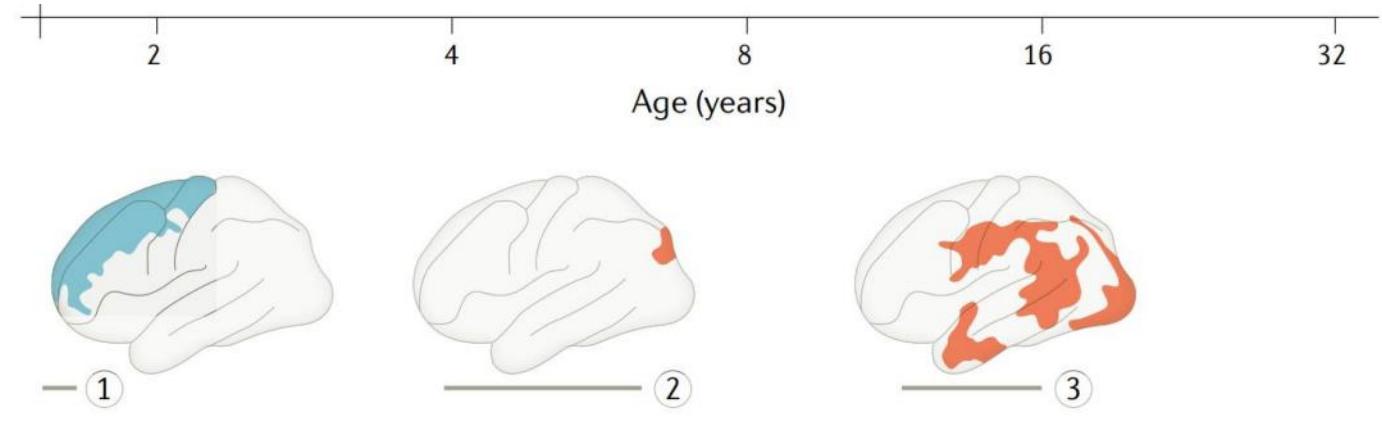
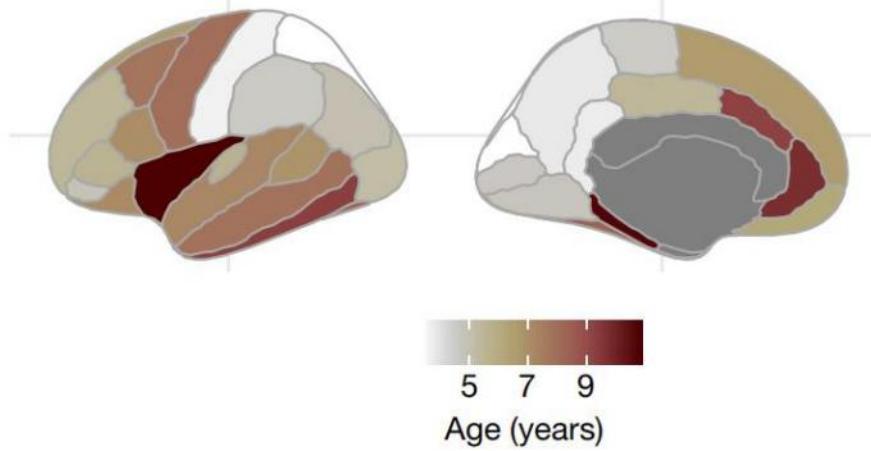
Brain Imaging Phenotype: Cortical Thickness



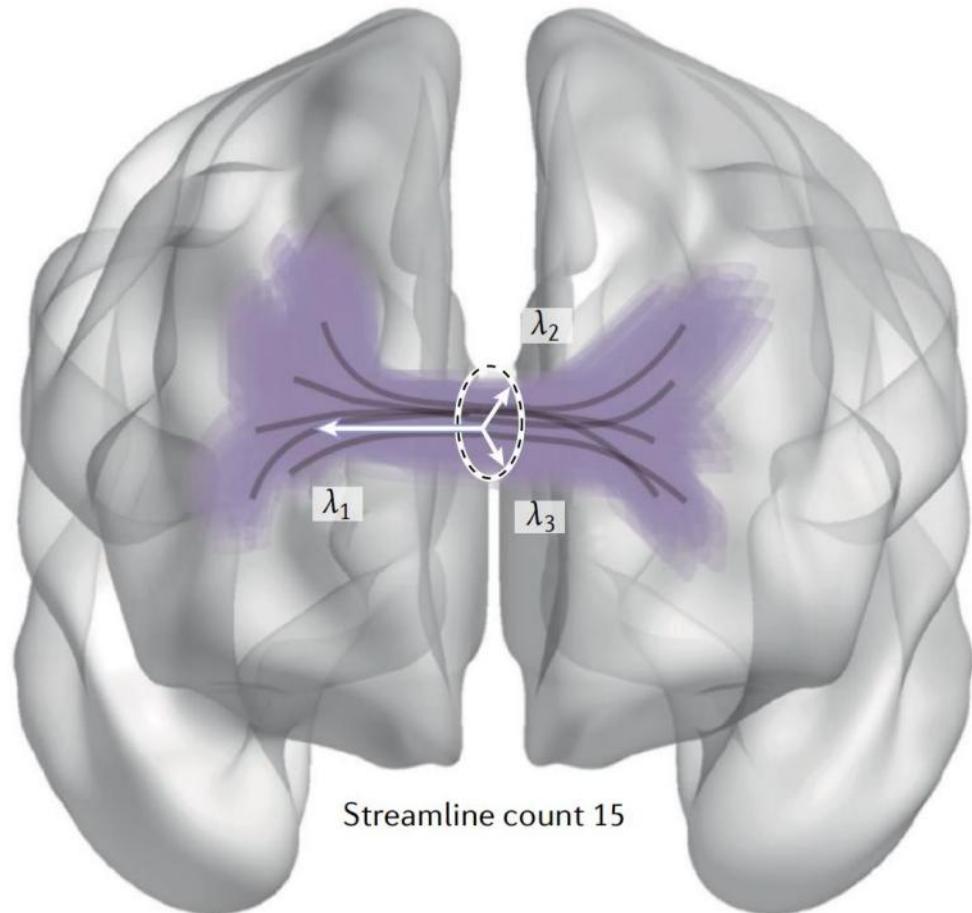
Cortical Thickness: Regional Variation and SES



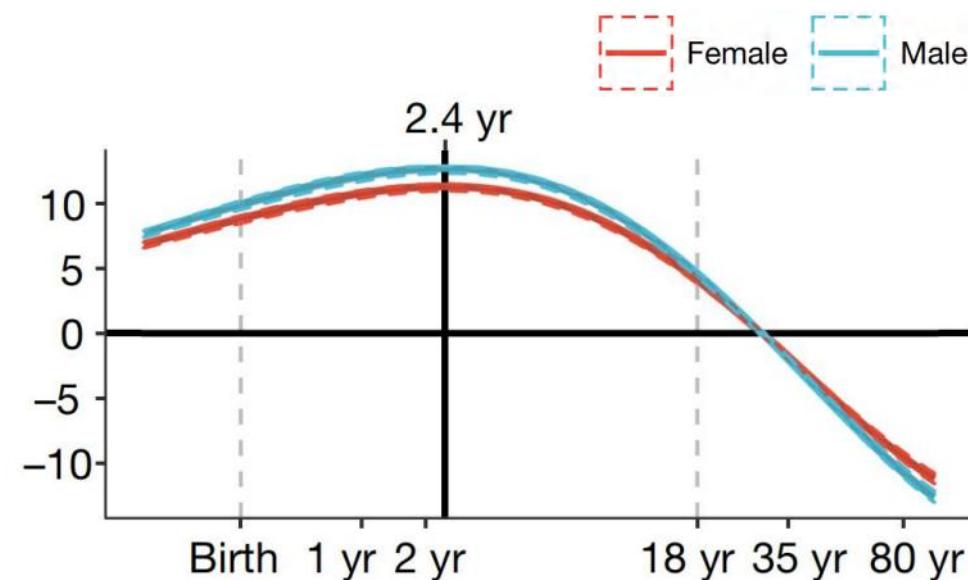
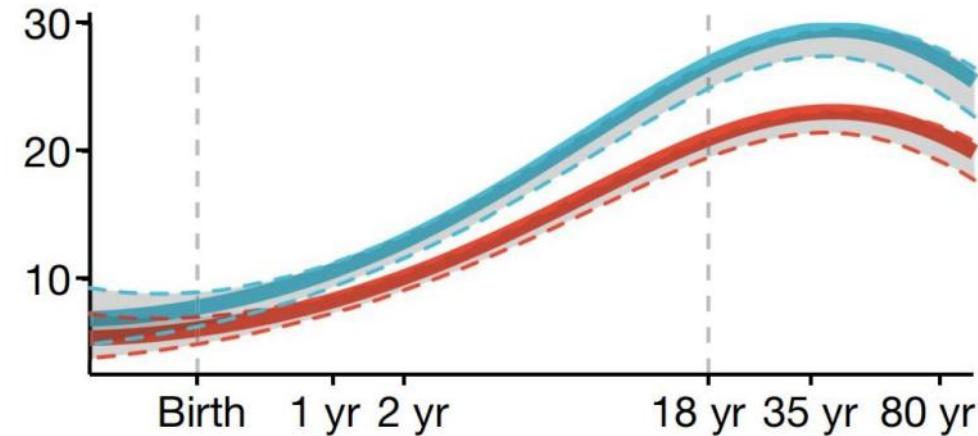
Cortical Thickness: Associations with SES



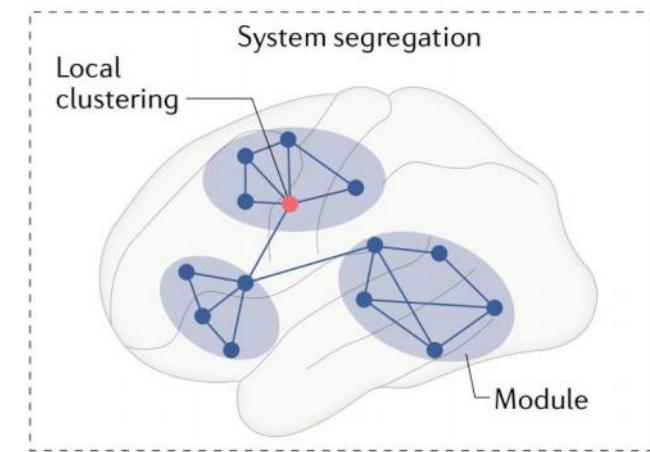
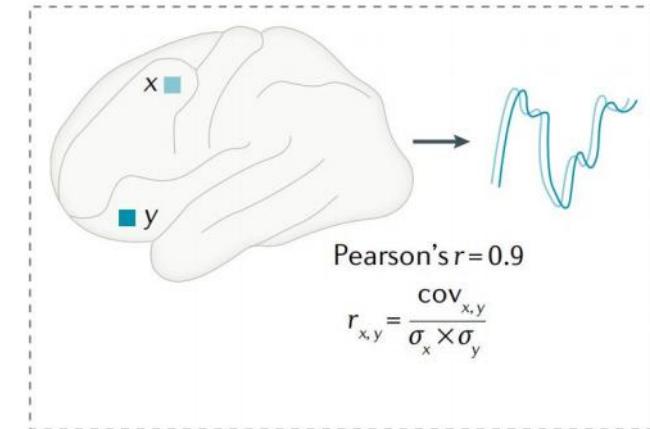
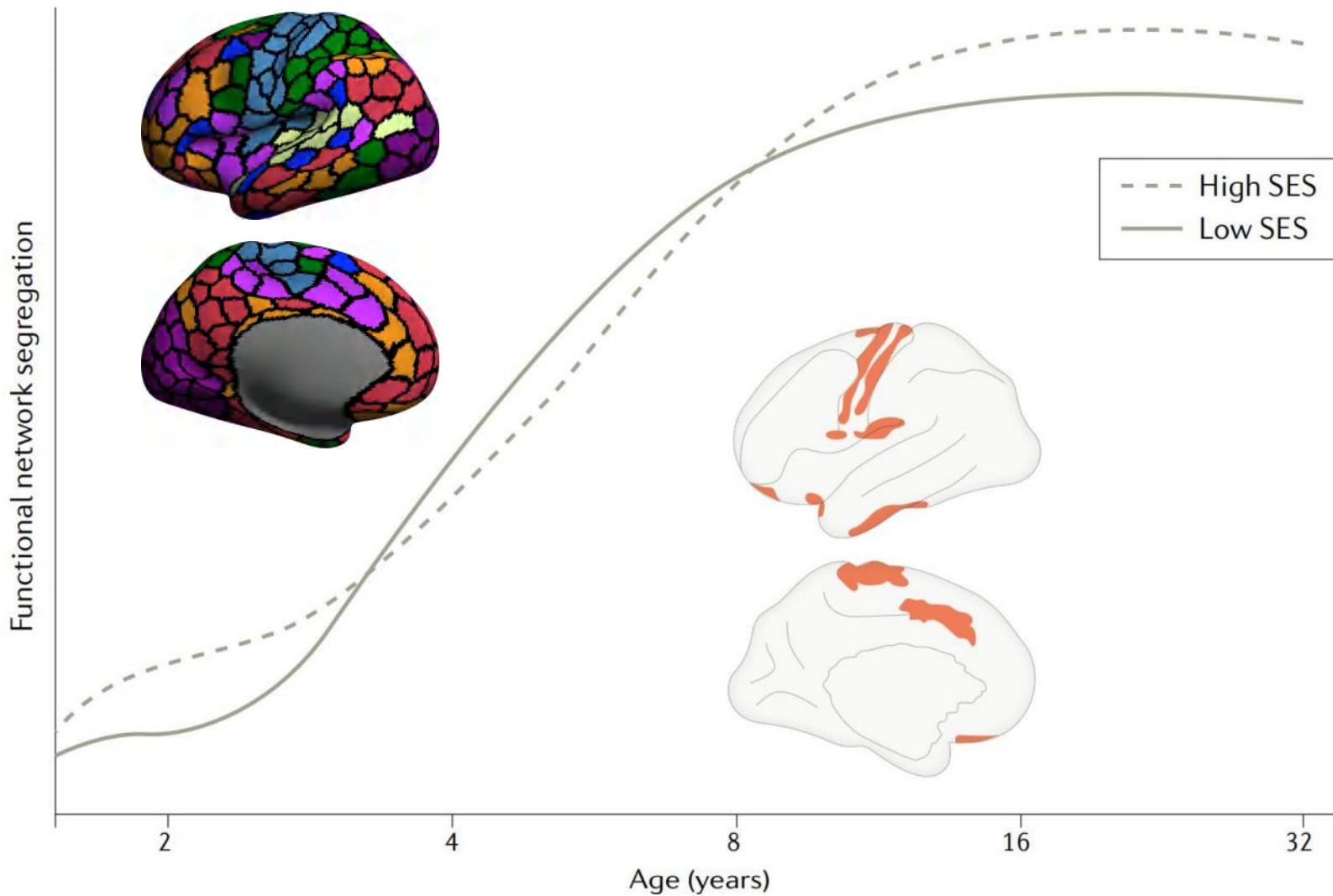
Brain Imaging Phenotype: Network Connectivity



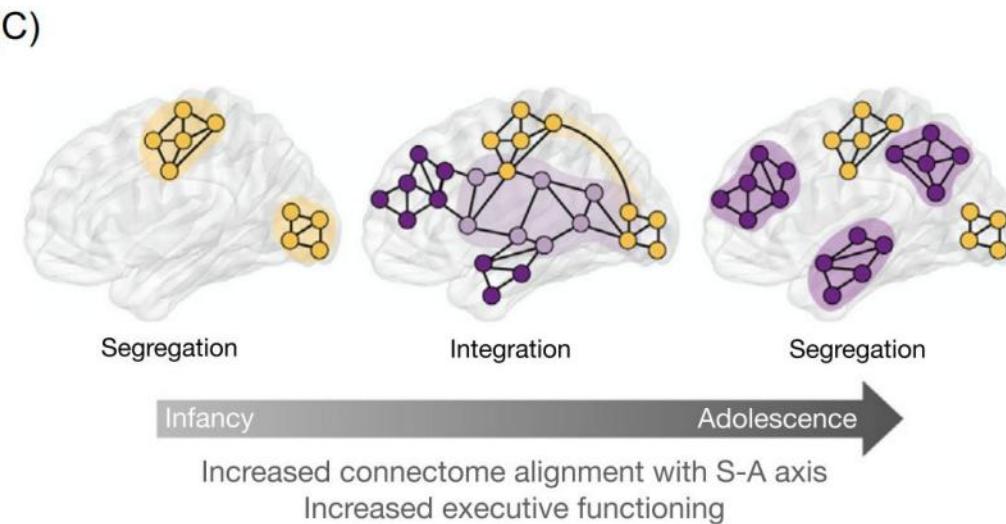
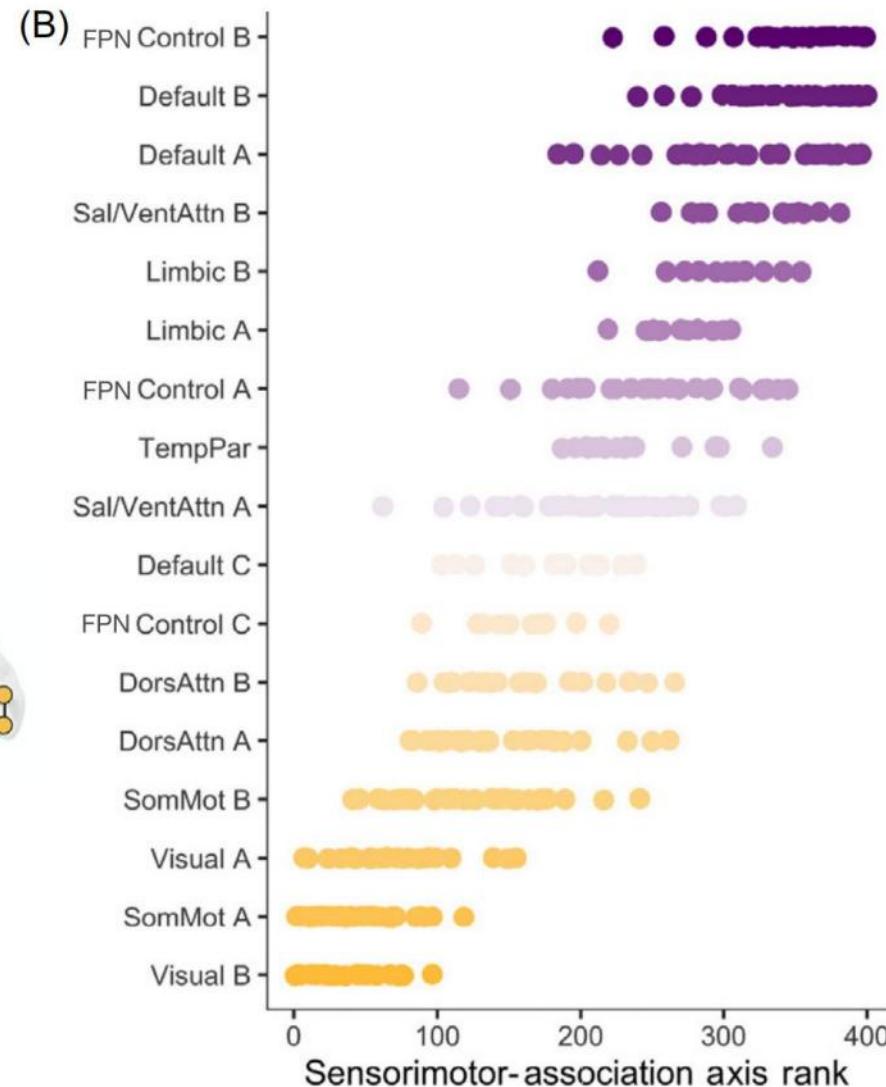
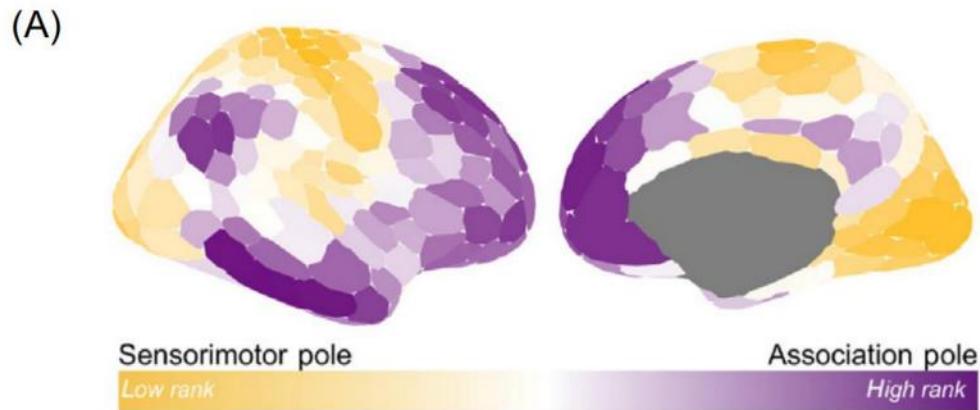
$$FA = \sqrt{\frac{(\lambda_1 - \lambda_2)^2 + (\lambda_2 - \lambda_3)^2 + (\lambda_1 - \lambda_3)^2}{2(\lambda_1^2 + \lambda_2^2 + \lambda_3^2)}}$$



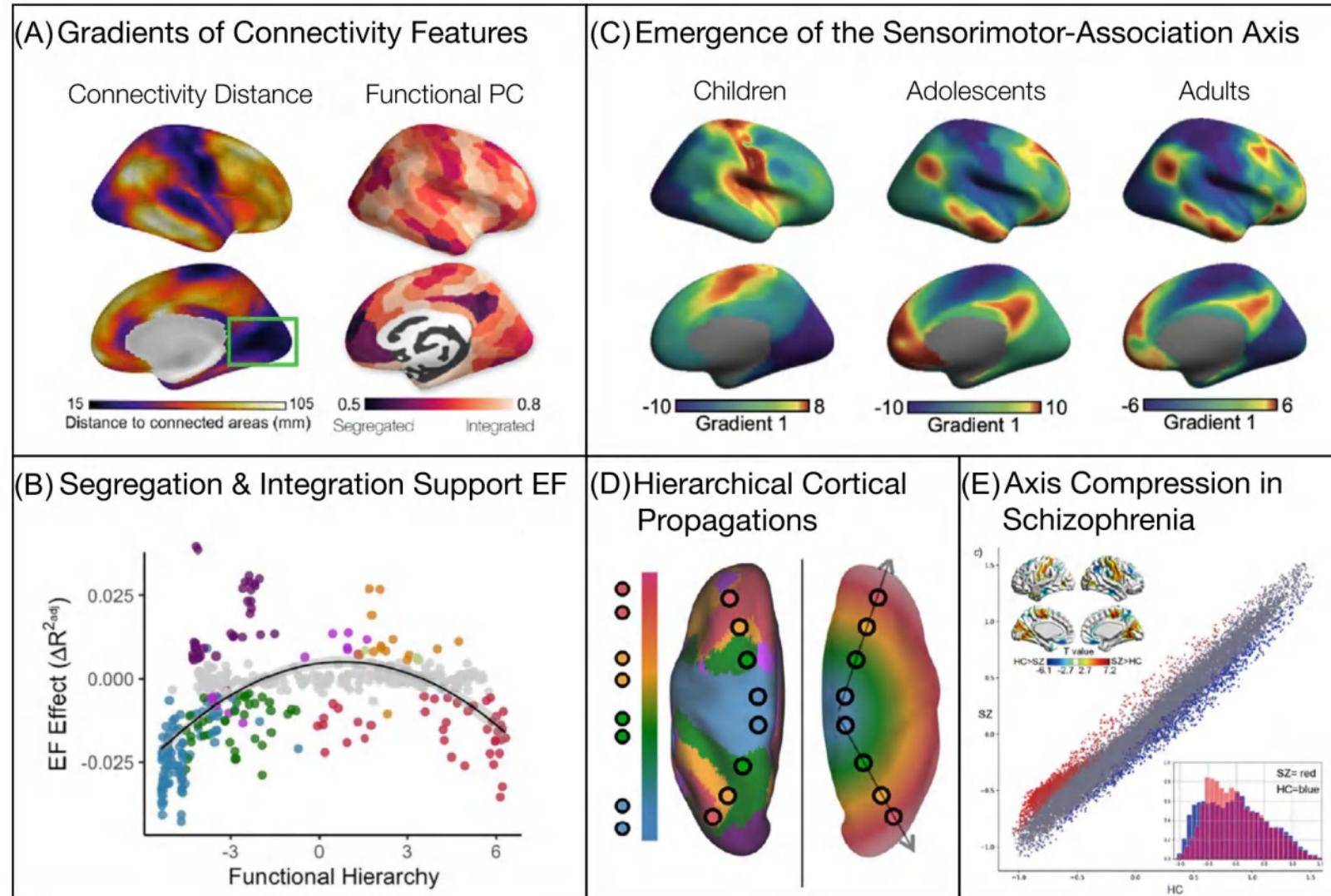
Brain-SES Associations: Network Function



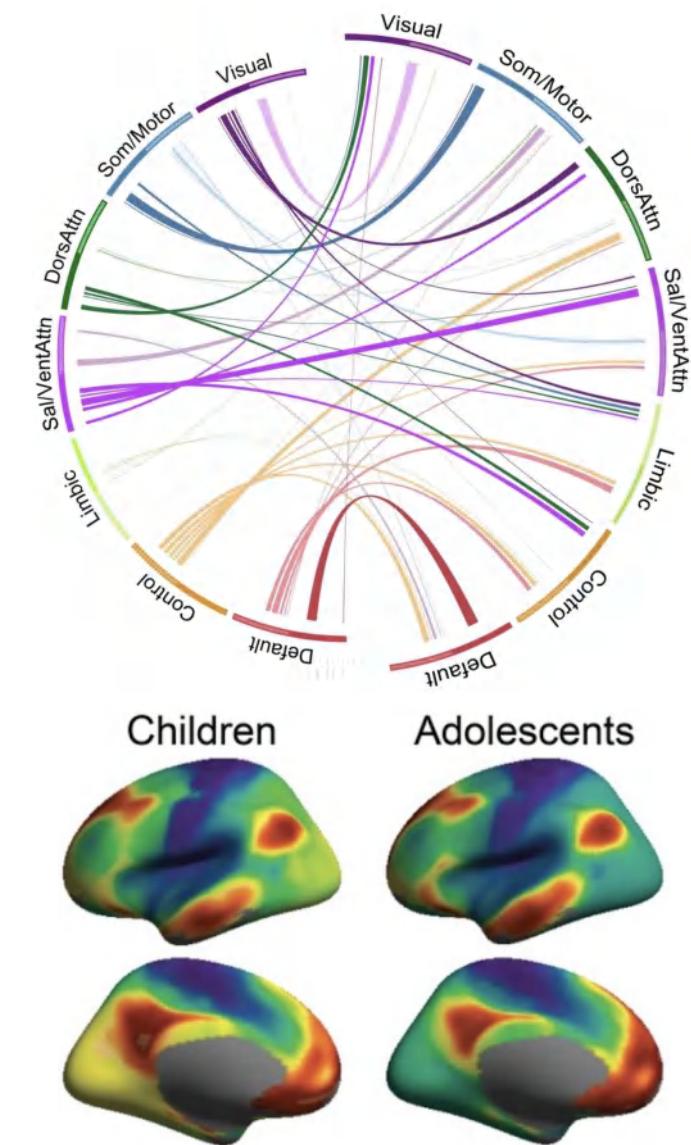
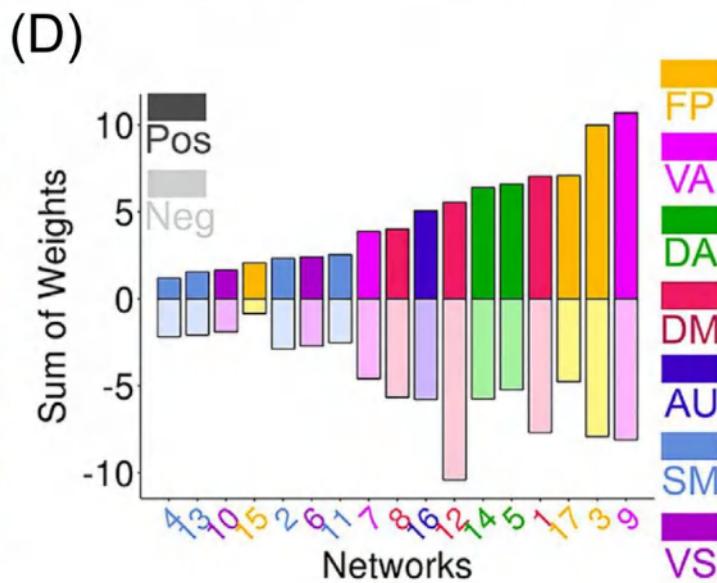
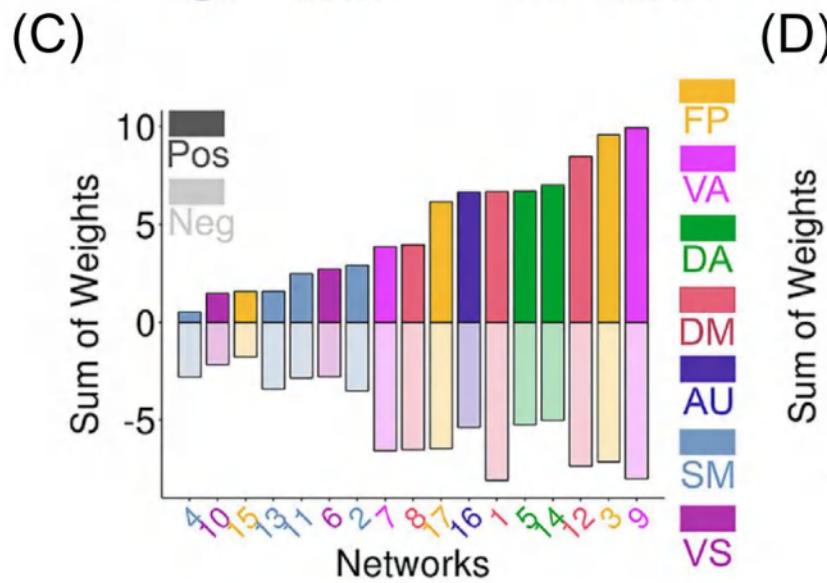
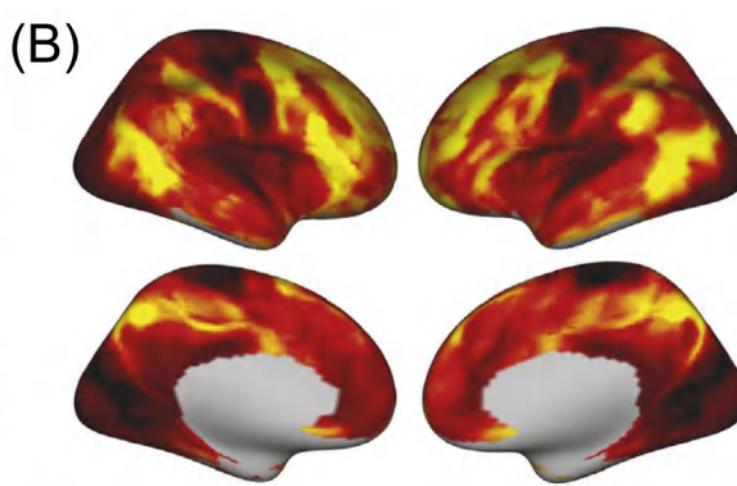
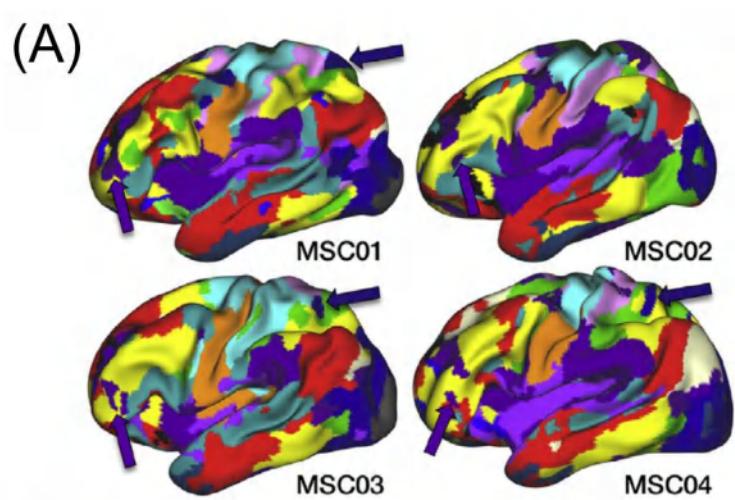
Brain Network Development: A Hierarchical Theory



Brain Network Development: A Hierarchical Theory



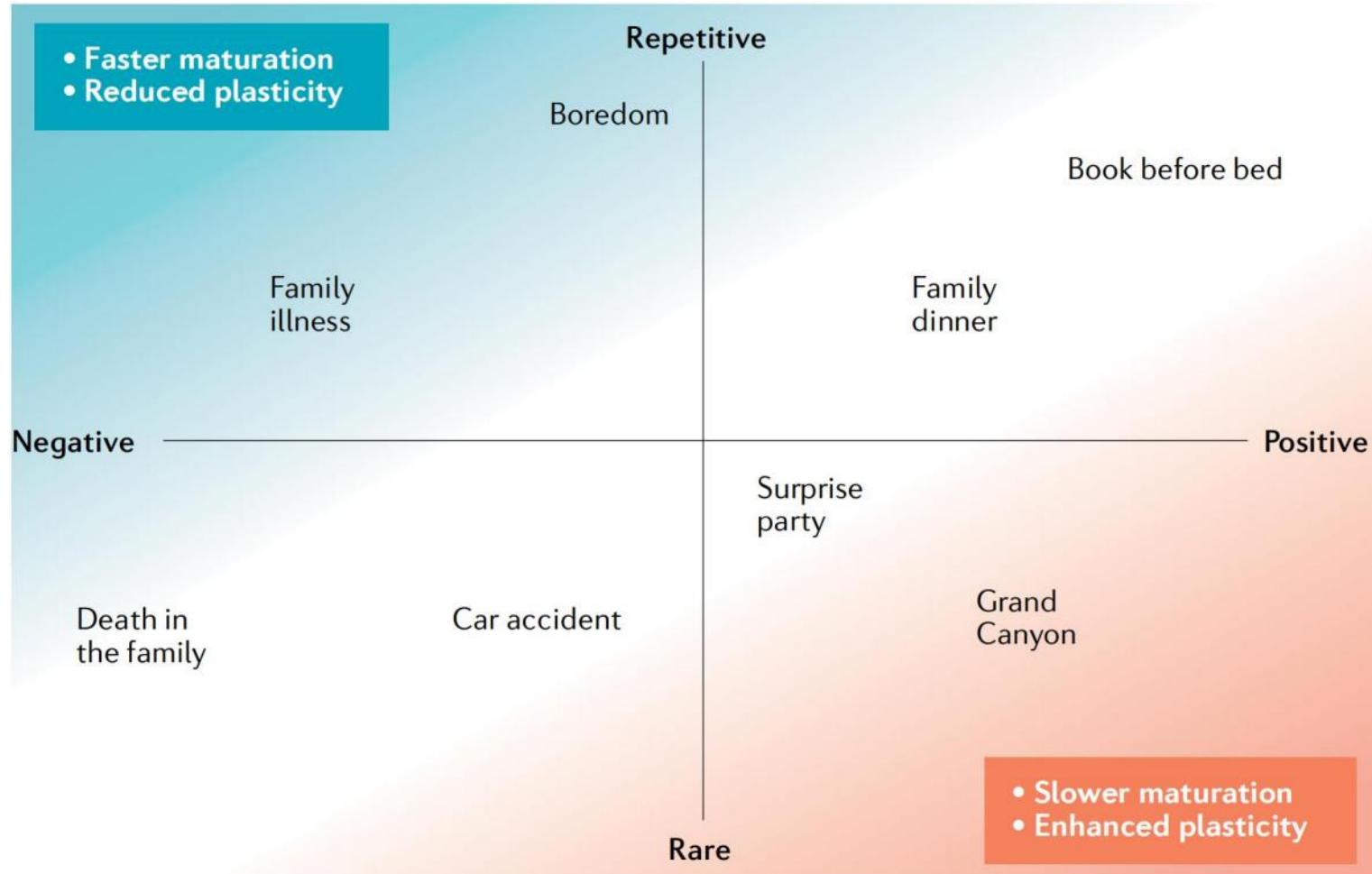
Brain Network Development: A Hierarchical Theory



Brain Plasticity and Cognitive Enrichment

Cellular or molecular measure	Neuroimaging measure
Excitation–inhibition balance	Magnetic resonance spectroscopy, glutamate chemical exchange saturation transfer, GABA chemical exchange saturation transfer
Extracellular matrix organization (including perineuronal nets)	Multicompartment diffusion imaging (for example, neurite orientation dispersion and density imaging or soma and neurite density imaging)
Myelin	Fractional anisotropy or mean diffusivity from diffusion imaging; multicompartment diffusion imaging; T1-weighted to T2-weighted ratio; magnetization transfer; quantitative MRI
Levels of neurotransmitters (such as dopamine, acetylcholine or serotonin)	Positron emission tomography, functional MRI or resting-state functional MRI of neuromodulatory nuclei

Brain Plasticity and Cognitive Enrichment



Integrative theory: childhood experiences affect the pace of brain development. According to our model, experiences that are chronic or repetitive and negative encourage faster maturation and increase allostatic load, potentially restricting plasticity.

Experiences that are rare and positive, triggering surprise and awe, are associated with strong neurochemical signals to delay maturational processes and enhance plasticity. Experiences in the other quadrants (rare and negative, or repetitive and positive) are predicted to

IMPORTANCE The association between poverty and unfavorable cognitive outcomes is robust, but most research has focused on individual household socioeconomic status (SES). There is increasing evidence that neighborhood context explains unique variance not accounted for by household SES.



Original Investigation | Pediatrics

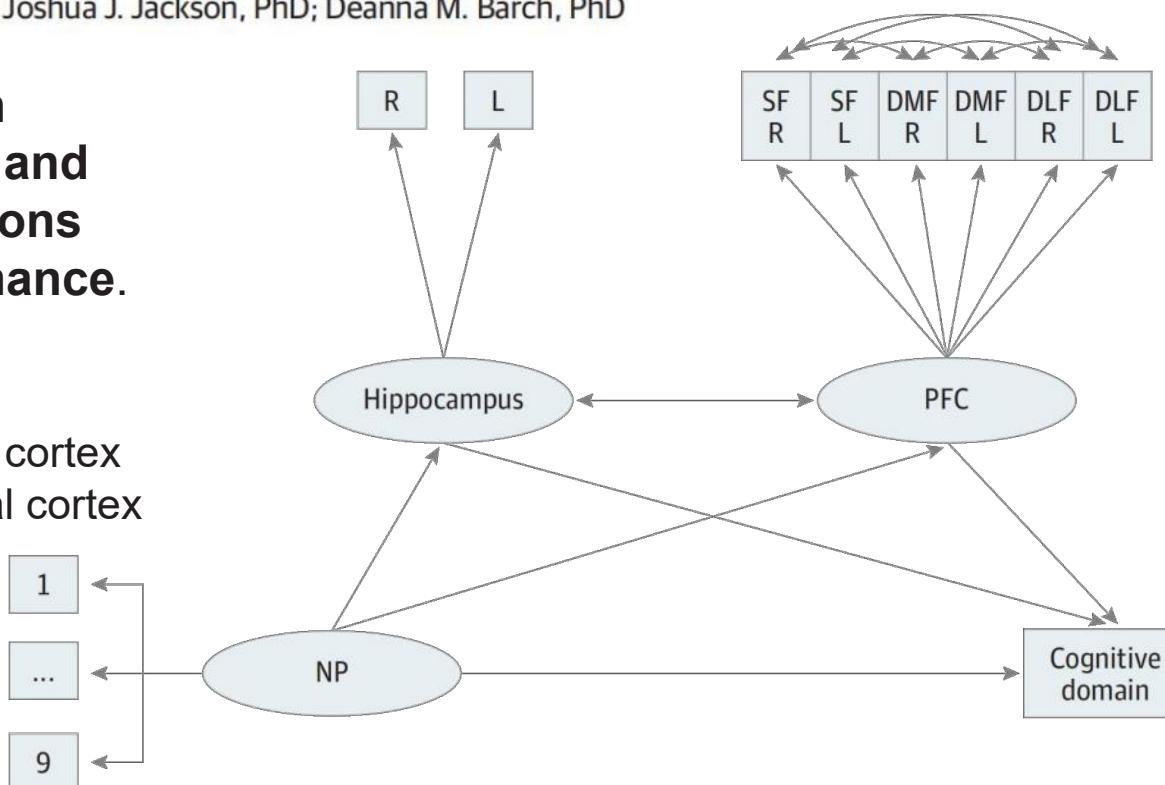
Assessment of Neighborhood Poverty, Cognitive Function, and Prefrontal and Hippocampal Volumes in Children

Rita L. Taylor, MA; Shelly R. Cooper, PhD; Joshua J. Jackson, PhD; Deanna M. Barch, PhD

Schematic of SEM With Neighborhood Poverty and Brain Region Associations With Cognitive Performance.

DLF: dorsolateral prefrontal cortex
DMF: dorsomedial prefrontal cortex
SF: superior frontal gyrus
NP: neighborhood poverty

L: left; R: right



Key Points

Question What are the associations between neighborhood poverty, child cognitive performance, and brain structure after accounting for household-level poverty?

Findings This cross-sectional study of 11 875 children aged 9 and 10 years found an association between neighborhood poverty, prefrontal and hippocampal volume, and performance on cognitive tasks. These results remained even after controlling for individual household income.

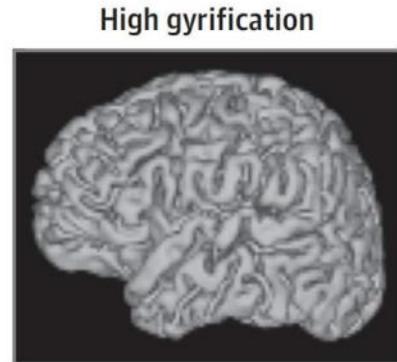
Meaning The findings of this study provide evidence that the broader neighborhood context uniquely contributes to prefrontal and hippocampal development and cognitive performance and should be considered in studies of early life poverty and adversity.

IMPORTANCE Exposure to early-life adversity alters the structural development of key brain regions underlying neurodevelopmental impairments. The association between prenatal exposure to adversity and brain structure at birth remains poorly understood.

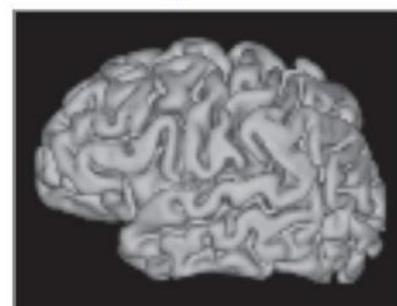


Original Investigation | Pediatrics

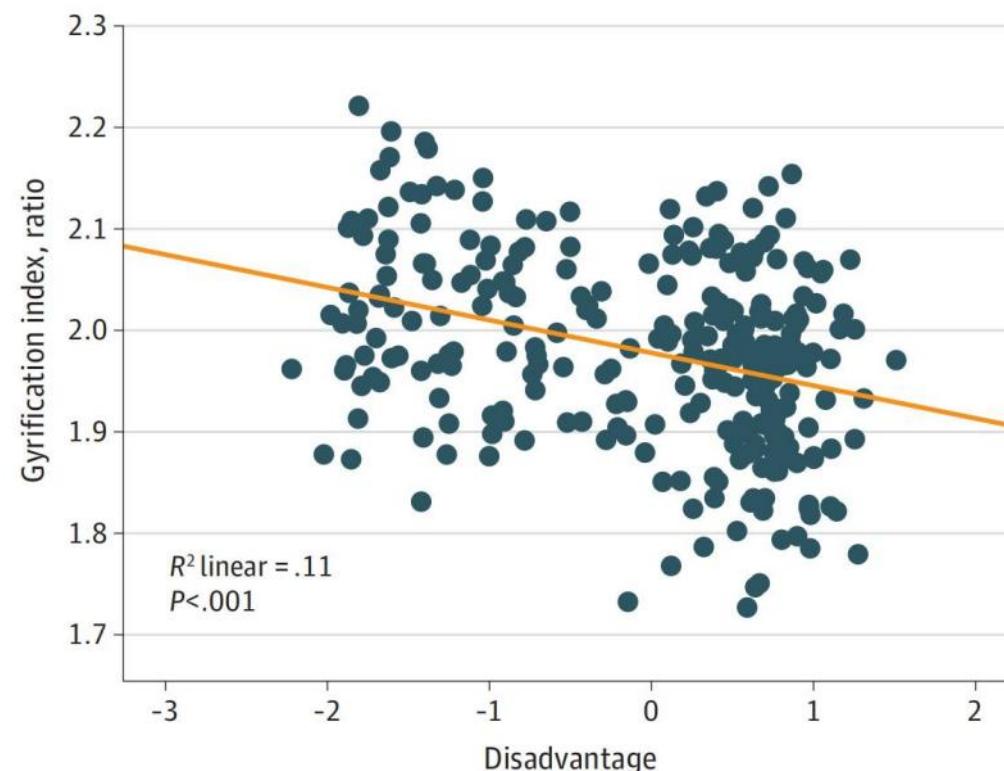
Association of Prenatal Exposure to Early-Life Adversity With Neonatal Brain Volumes at Birth



High gyrification



Low gyrification



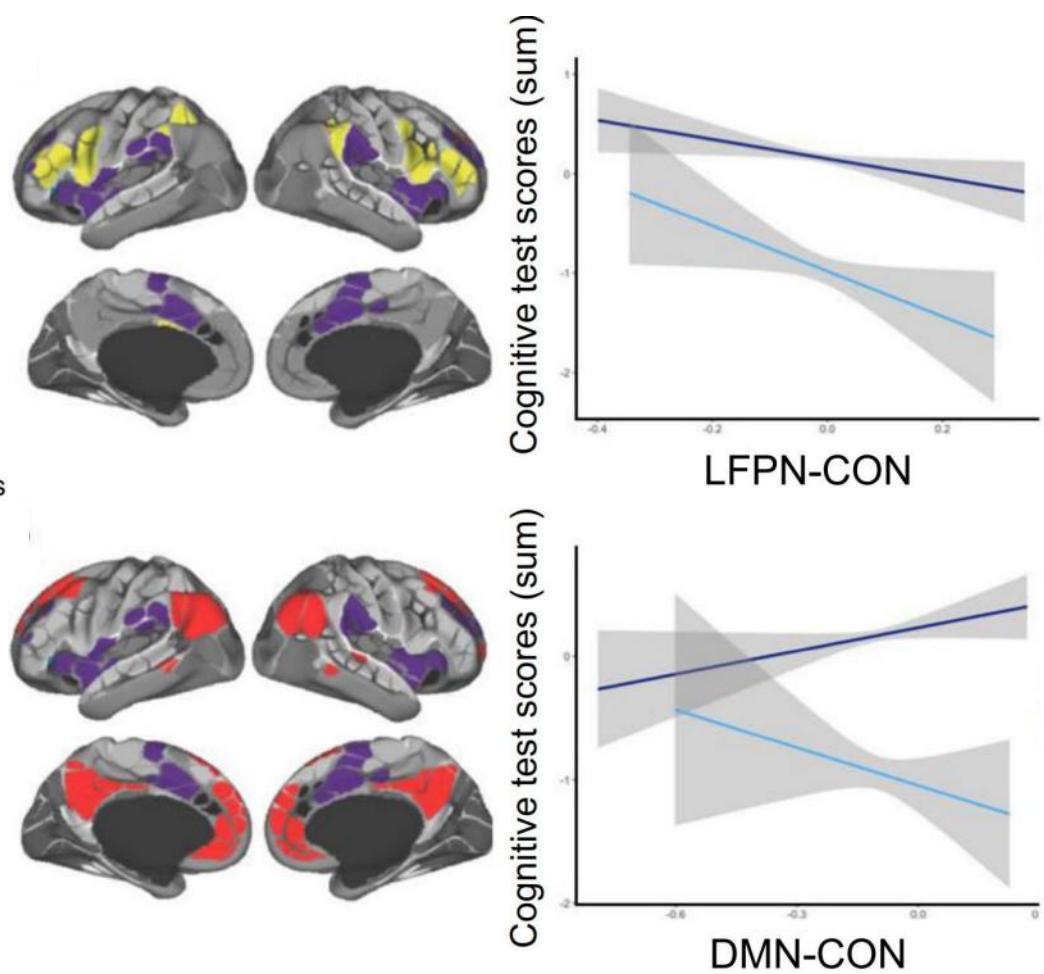
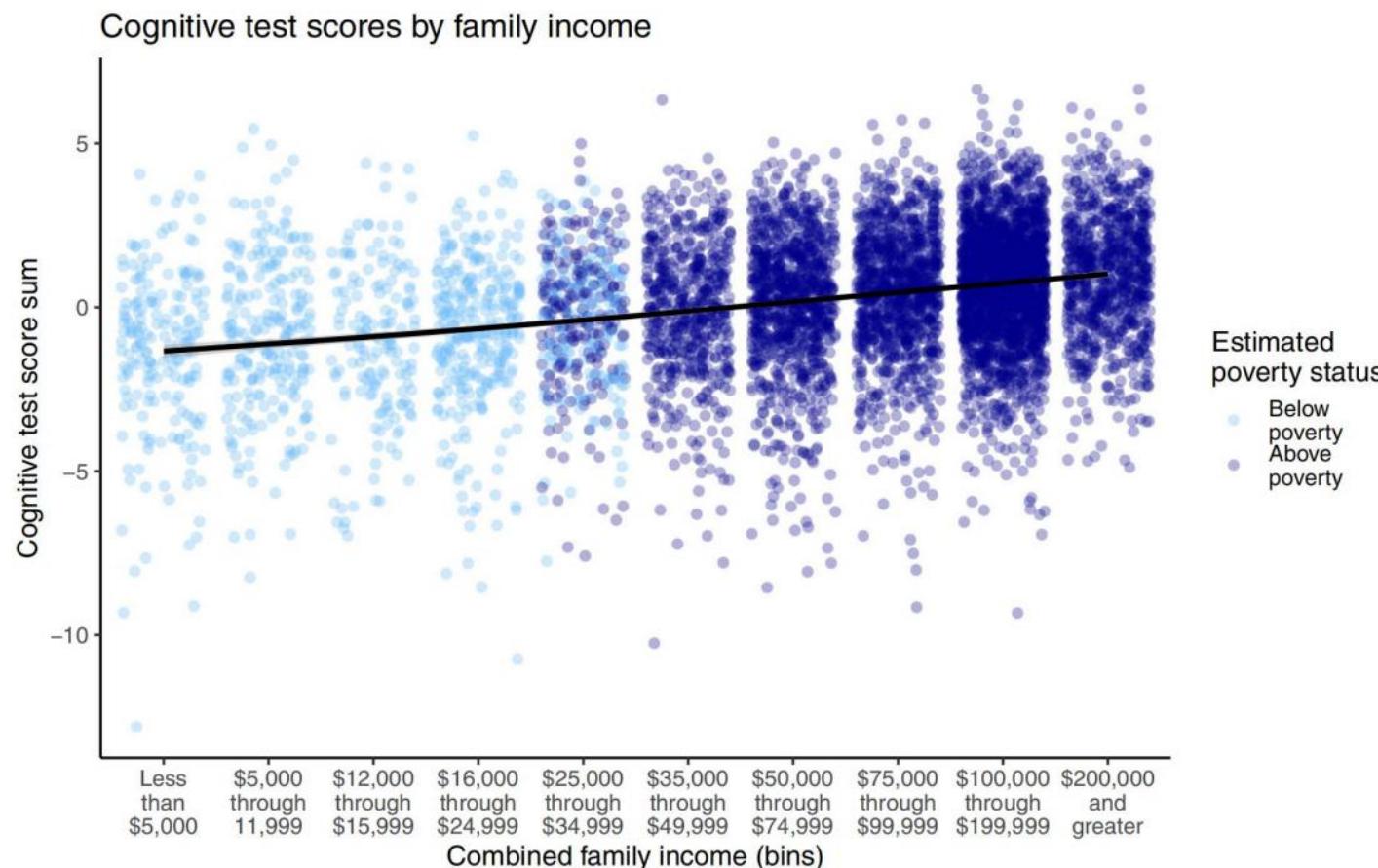
Key Points

Question Is prenatal exposure to maternal social disadvantage and psychosocial stress associated with global and relative infant brain volumes at birth?

Findings In this longitudinal, observational cohort study of 280 mother-infant dyads, prenatal exposure to greater maternal social disadvantage, but not psychosocial stress, was associated with statistically significant reductions in white matter, cortical gray matter, and subcortical gray matter volumes and cortical folding at birth after accounting for maternal health and diet.

Brain network coupling associated with cognitive performance varies as a function of a child's environment in the ABCD study

Monica E. Ellwood-Lowe¹✉, Susan Whitfield-Gabrieli² & Silvia A. Bunge^{1,3}



Early deprivation alters structural brain development from middle childhood to adolescence

Margaret A. Sheridan^{1*}, Cora E. Mukerji^{2,3}, Mark Wade⁴, Kathryn L. Humphreys⁵, Kathryn Garrisi¹, Srishti Goel^{1,6}, Kinjal Patel¹, Nathan A. Fox⁷, Charles H. Zeanah⁸, Charles A. Nelson^{3,9,10}, Katie A. McLaughlin¹¹

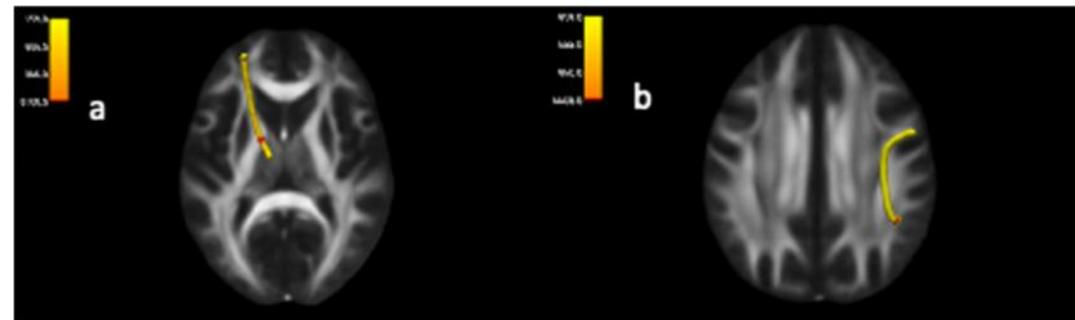
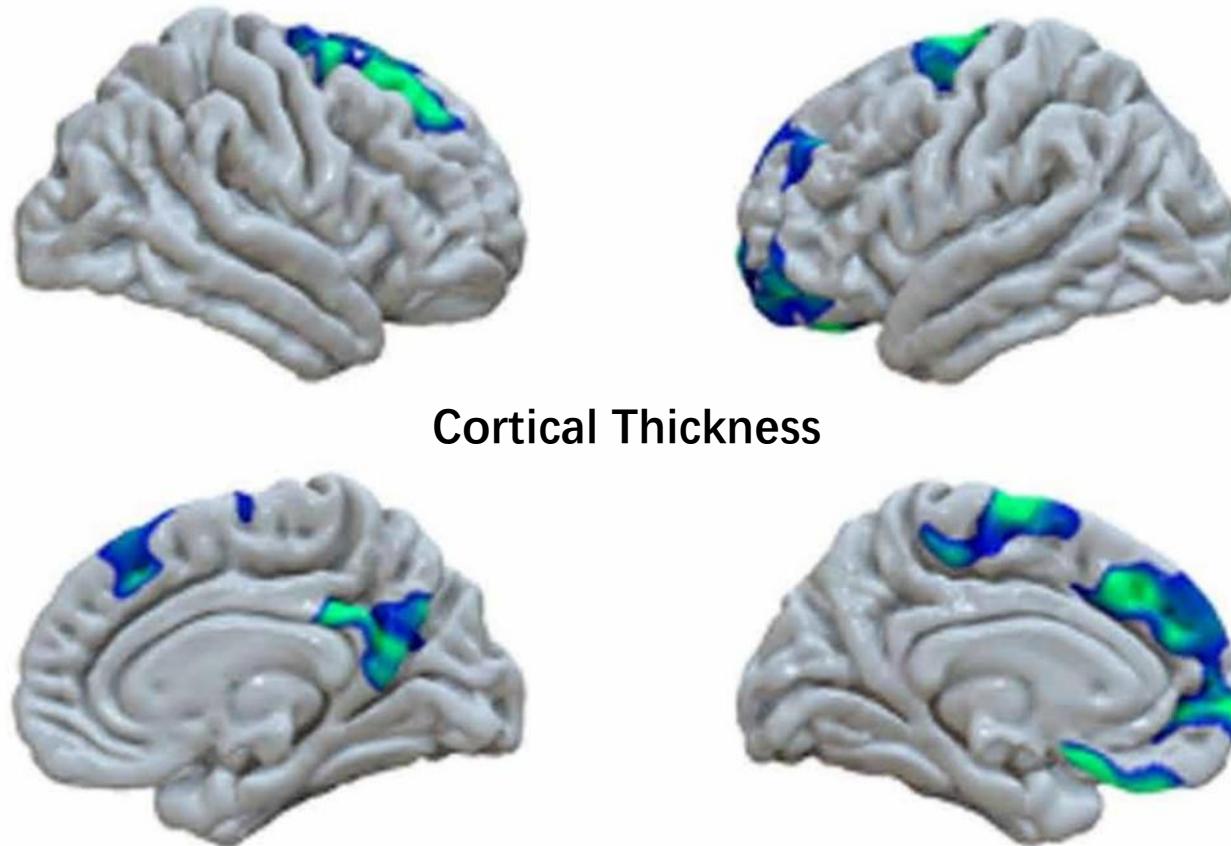
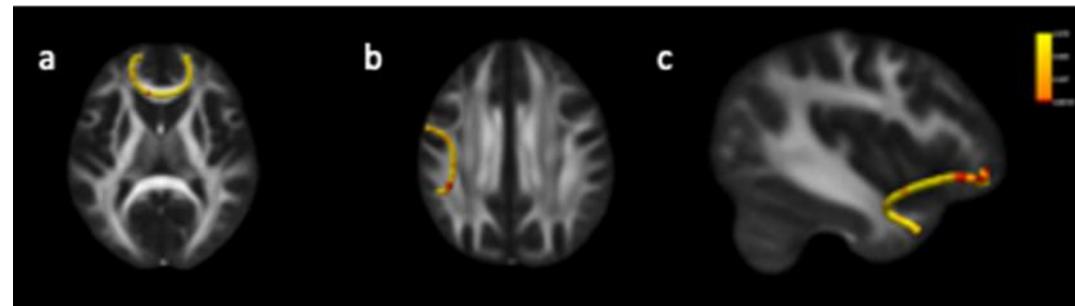
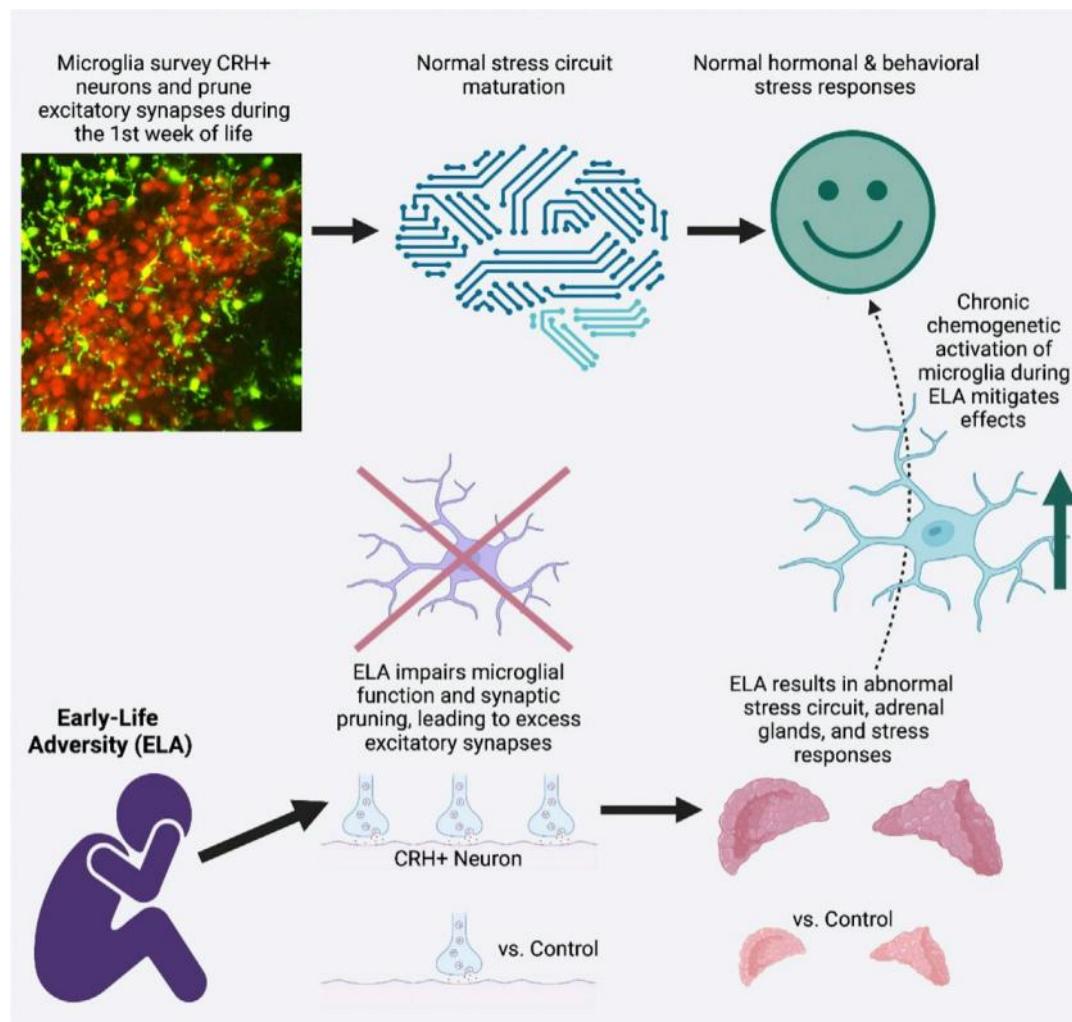


Table 3. Differences in white matter structure by group at 16 years. Significant differences in white matter integrity between children who had ever been exposed to institutionalization and those raised in families as well as between those randomized to foster care intervention and those assigned to remain as usual.

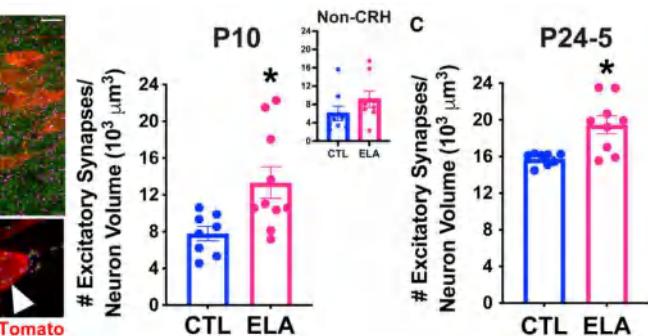
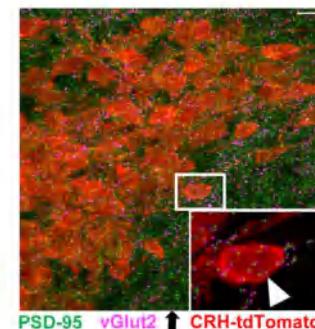
Tract	Metric	z value	FDR-corrected	Approximate coordinates of max vertex in MNI space		
				z	P	x
<i>Ever-institutionalized (n = 81) > never-institutionalized (n = 33)</i>						
L ant thalamic radiation	RD	3.663	0.022	-13.5	-2.7	7.9
	RD	3.452	0.028	-12.7	-4.4	7.5
	MD	3.541	0.042	-12.7	-4.4	7.5
	MD	3.16	0.054	-12.7	-4.4	7.5
R sup longitudinal fasciculus	MD	3.67	0.034	43.2	-46.3	32.3
<i>Prolonged institutionalization (n = 41) > foster care (n = 41)</i>						
Forceps minor	MD	-3.67	0.023	-9.5	28.8	4.5
L sup longitudinal fasciculus	FA	-3.29	0.031	-39.0	-40.2	30.3
	FA	-3.45	0.031	-39.9	-41.4	30.4
L uncinate fasciculus	AD	4.44	0.002	-19.4	27.3	-6.2
	AD	3.27	0.024	-19.3	29.6	-7.0
	AD	3.35	0.024	-19.6	25.9	-6.1
	AD	3.20	0.024	-19.9	24.4	-6.1



Early stress-induced impaired microglial pruning of excitatory synapses on immature CRH-expressing neurons provokes aberrant adult stress responses

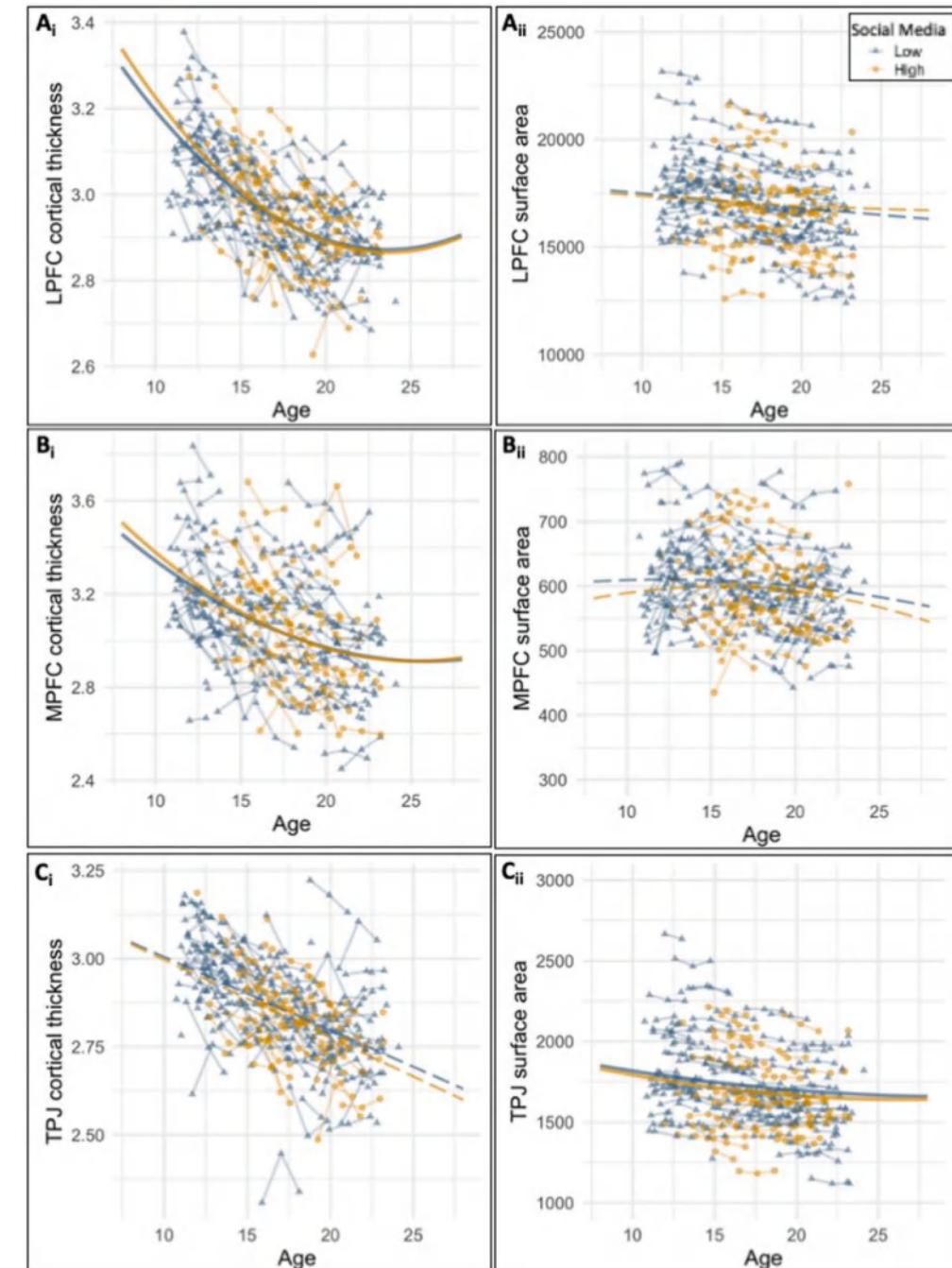
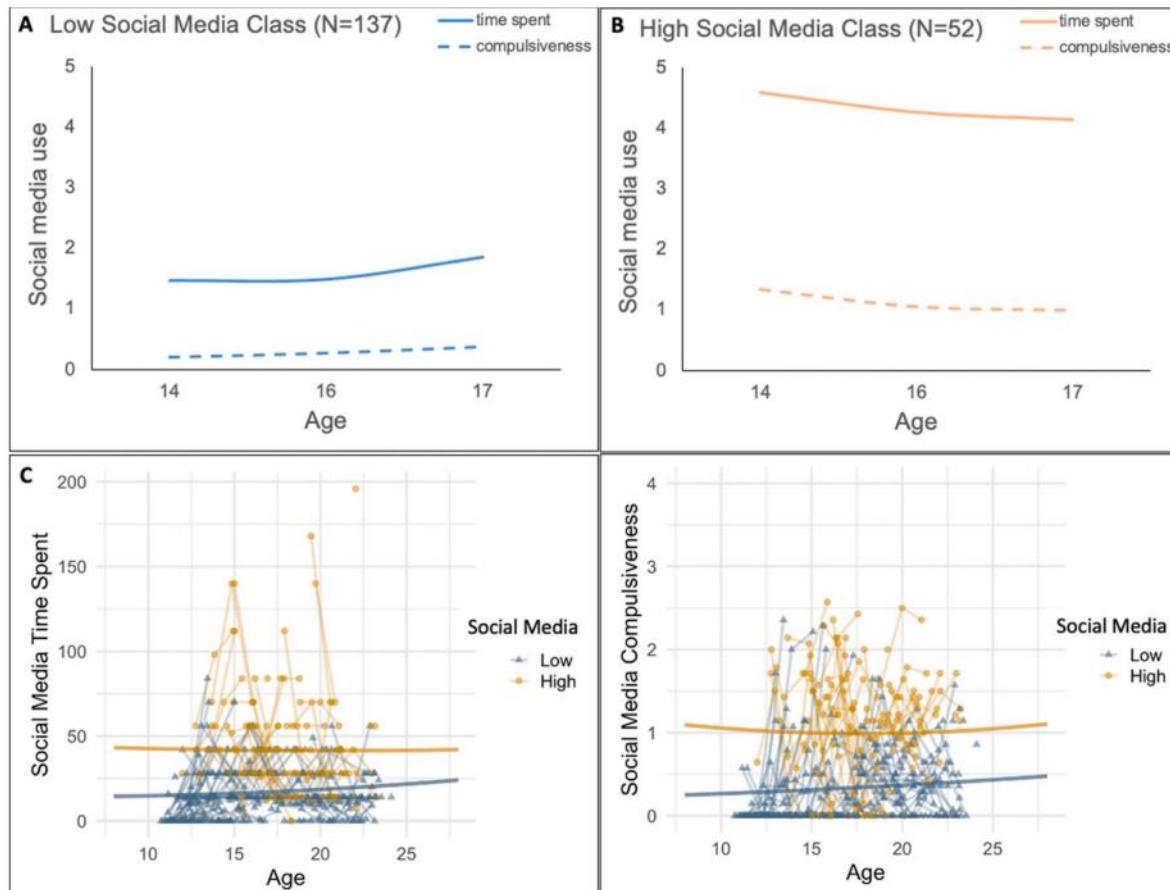


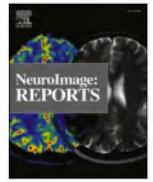
In brief



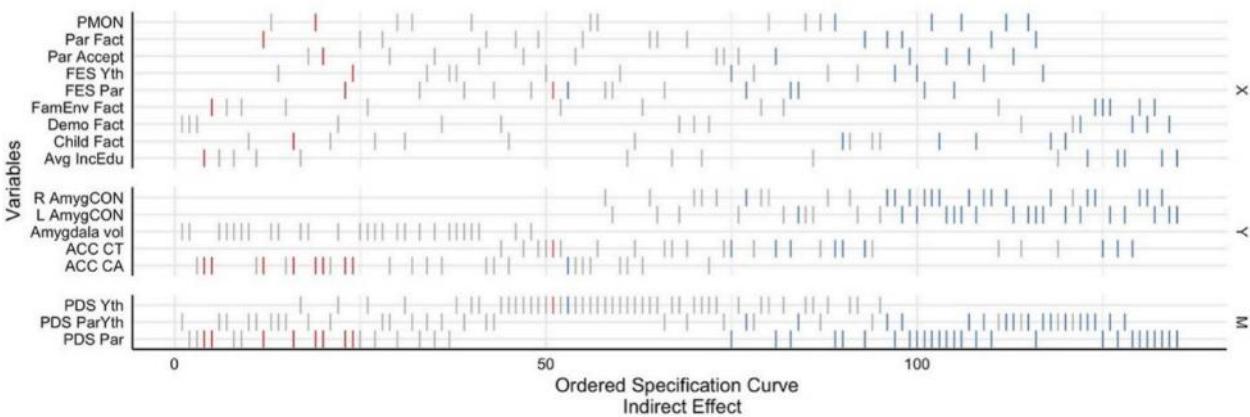
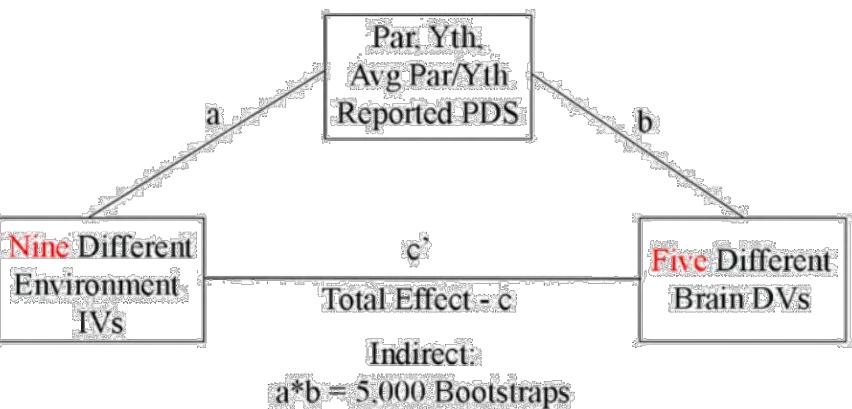
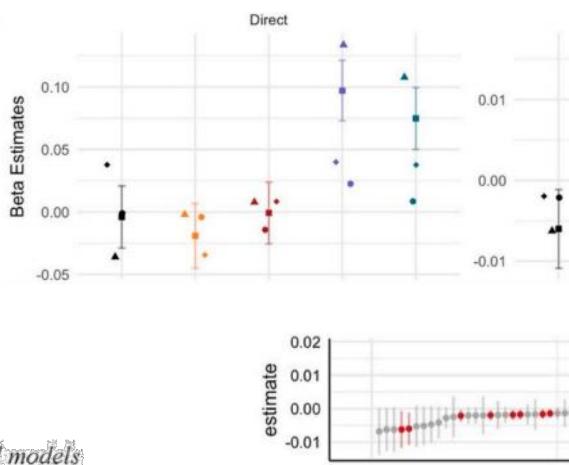
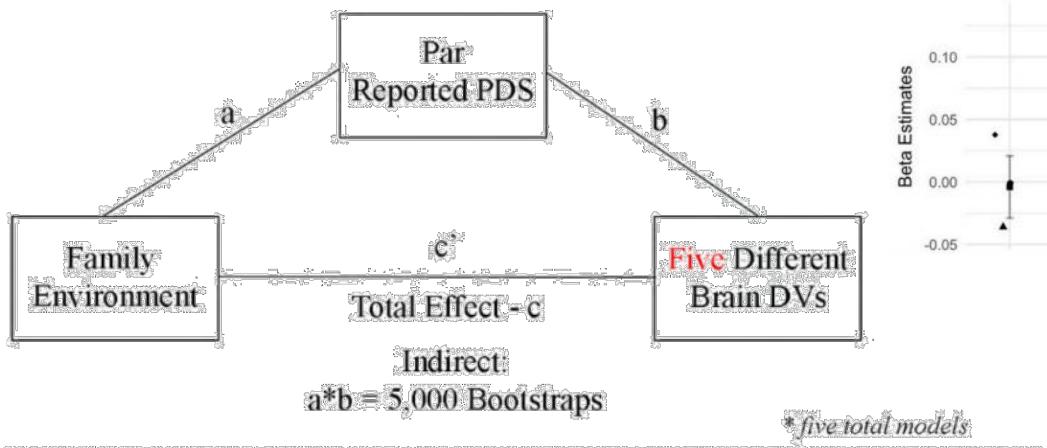
Early-life adversity (ELA) promotes lifelong aberrant stress responses and vulnerability to mental illnesses. Bolton et al. identify poor dynamics and hypothalamic CRH neurons' excitatory synapse pruning of ELA microglia, implicating microglial MerTK. Chronic chemogenetic activation of ELA microglia normalized process dynamics, synapse density, and adult hormonal and behavioral stress responses.

Longitudinal associations between social media use, mental well-being and structural brain development across adolescence[☆]



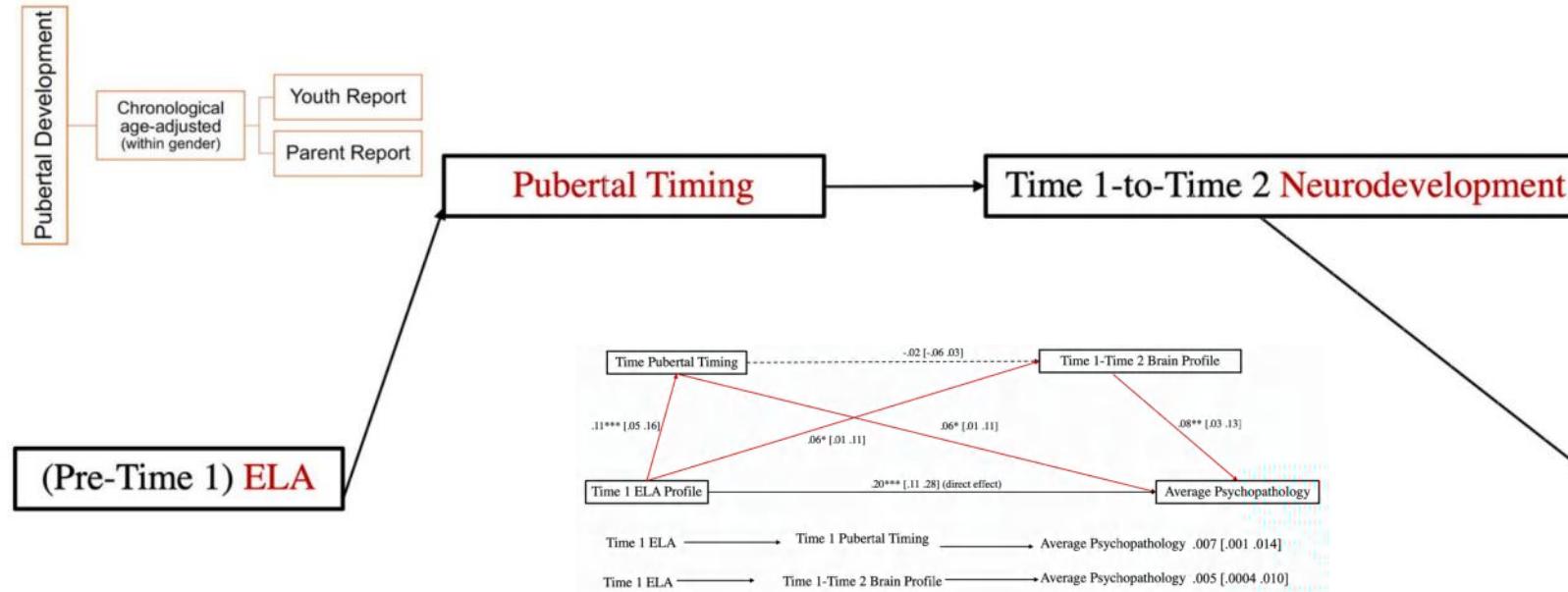


Mediating effect of pubertal stages on the family environment and neurodevelopment: An open-data replication and multiverse analysis of an ABCD Study®

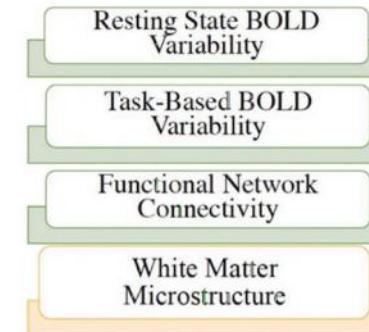


Puberty, Neurodevelopment, Family and Psychopathology

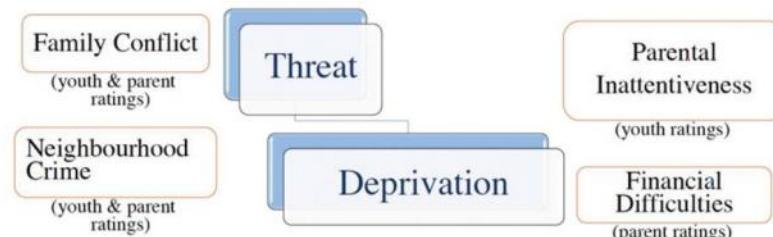
(b) *Measurement*



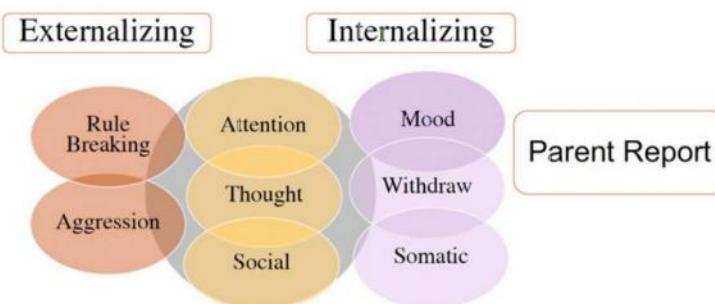
(c) *Measurement*



(a) *Measurement*



(d) *Measurement*



NEUROSCIENCE

Sexually divergent development of depression-related brain networks during healthy human adolescence

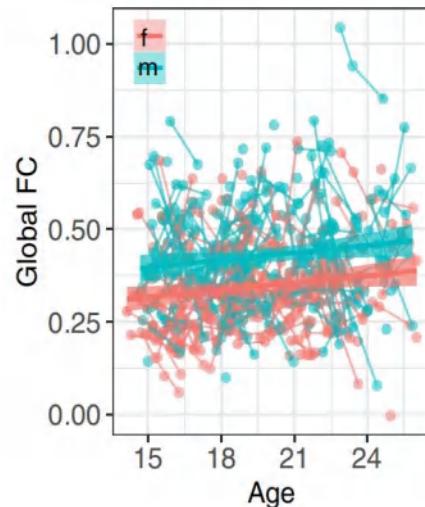
Lena Dorfschmidt^{1*}, Richard A. Bethlehem¹, Jakob Seidlitz^{2,3,4}, František Váša⁵, Simon R. White¹, Rafael Romero-García¹, Manfred G. Kitzbichler¹, Athina R. Aruldass¹, Sarah E. Morgan^{1,6,7}, Ian M. Goodyer¹, Peter Fonagy⁸, Peter B. Jones^{1,9}, Ray J. Dolan^{10,11}, NSPN Consortium†, Neil A. Harrison^{12,13}, Petra E. Vértes^{1‡}, Edward T. Bullmore^{1‡}

Sexual differences in human brain development could be relevant to sex differences in the incidence of depression during adolescence. We tested for sex differences in parameters of normative brain network development using fMRI data on $N = 298$ healthy adolescents, aged 14 to 26 years, each scanned one to three times. Sexually divergent development of functional connectivity was located in the default mode network, limbic cortex, and subcortical nuclei. Females had a more “disruptive” pattern of development, where weak functional connectivity at age 14 became stronger during adolescence. This fMRI-derived map of sexually divergent brain network development was robustly colocated with i prior loci of reward-related brain activation ii a map of functional dysconnectivity in major depressive disorder (MDD), and iii an adult brain gene transcriptional pattern enriched for genes on the X chromosome, neurodevelopmental genes, and risk genes for MDD. We found normative sexual divergence in adolescent development of a cortico-subcortical brain functional network that is relevant to depression.

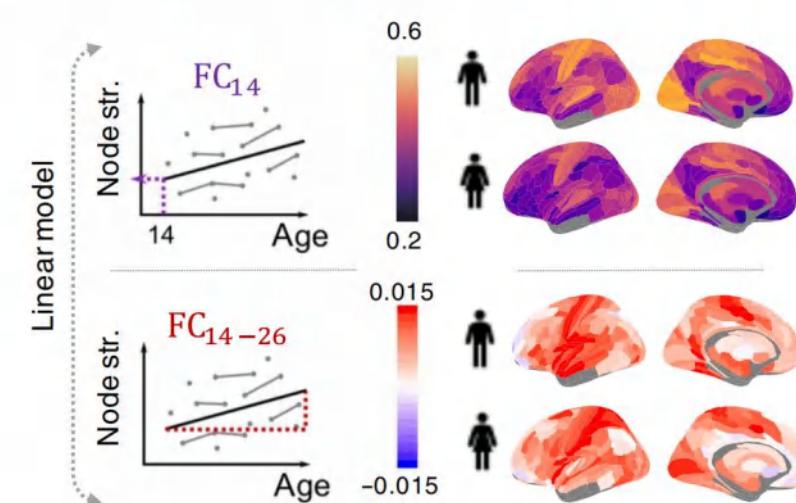
Table 1. Adolescent developmental MRI sample. Total $N = 298$ healthy young people participated in an accelerated longitudinal MRI study, with recruitment balanced for sex in each of five age-defined strata and each subject scanned between one and three times (with follow-up scans taking place approximately 6 and 18 months after baseline). FD, a measure of head movement in millimeters, was significantly greater in males compared to females on average over all ages and in the youngest two age strata specifically ($P < 0.05$, uncorrected; fig. S3).

Sex	Age stratification					All ages
	14–15	16–17	18–19	20–21	22–25	
<i>N</i> subjects	Female	22	151	24	32	22
	Male	32	33	24	35	147
FD (mm)	Female	0.13*	0.10*	0.12	0.10	0.13
	Male	0.15*	0.13*	0.12	0.14	0.13*
<i>N</i> scans/subject (1 2 3)	Female	9 22 3	11 25 3	6 16 2	14 16 2	54 86 11
	Male	7 24 1	8 24 1	7 16 1	11 20 4	41 98 8

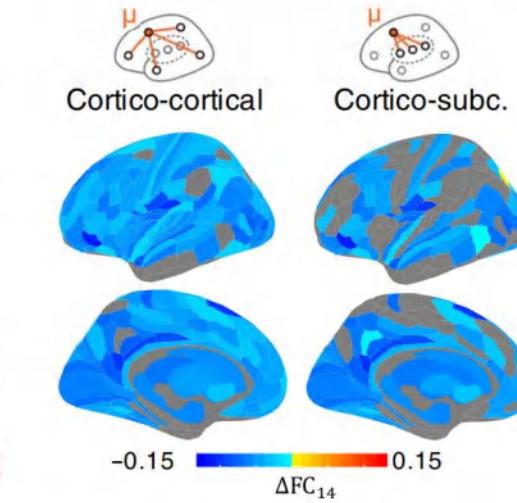
A I Global FC development



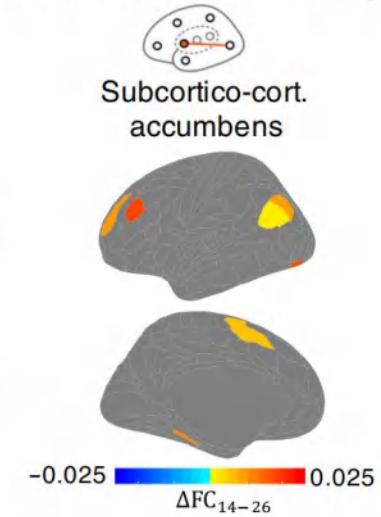
B I Estimators of baseline and adolescent change in FC



C I Sex diff. baseline connectivity



D I Sex diff. rate of change



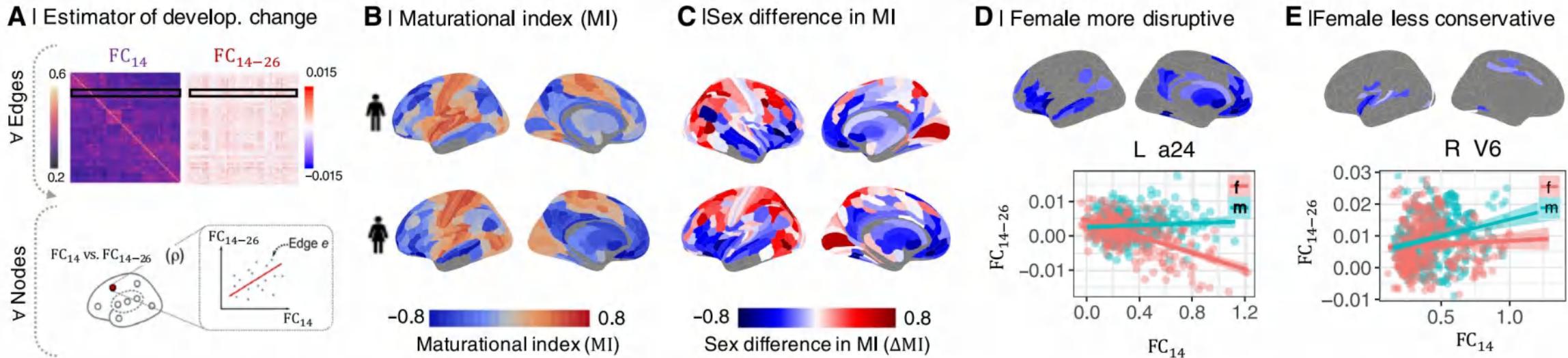


Fig. 2. Sex differences in MI. (A) The MI was estimated as the correlation between edgewise baseline connectivity at age 14 (FC₁₄) and the adolescent rate of change in connectivity per year (FC_{14–26}) at each regional node. (B) MI maps for males and females separately. MI was generally negative (blue) in frontal and association cortical areas and positive (orange) in primary motor and sensory cortices. (C) The sex difference in MI, $\Delta MI = MI_{\text{female}} - MI_{\text{male}}$, was significant in 230 of 346 regional nodes ($P_{\text{FDR}} = 0.05$). ΔMI was significantly negative in the ventral and medial prefrontal gyrus, ventrolateral prefrontal cortex, anterior and posterior cingulate gyrus, medial temporal gyrus, and subcortical nuclei (table S3), indicating sex differences in adolescent development of connectivity of these regions. More specifically, negative ΔMI defined a set of brain regions where adolescent development was either more disruptive (weak connections at 14 years became stronger during adolescence, and strong connections became weaker) or less conservative (strong connections at 14 years became stronger or weak connections became weaker during adolescence) in females compared to males. (D) Map of brain regions where development was more disruptive in females. As exemplified by the left area 24 (L a24), functional connections of disruptively developing nodes that were strong at 14 years (high FC₁₄, x axis) became weaker over the period 14 to 26 years (FC_{14–26} < 0, y axis), and edges that were weakly connected at 14 years became stronger over the course of adolescence, especially in females. (E) Map of brain regions where development was less conservative in females. As exemplified by right visual area V6 (R V6), connections that were strong at baseline become stronger over the period 14 to 26 years, especially in males.

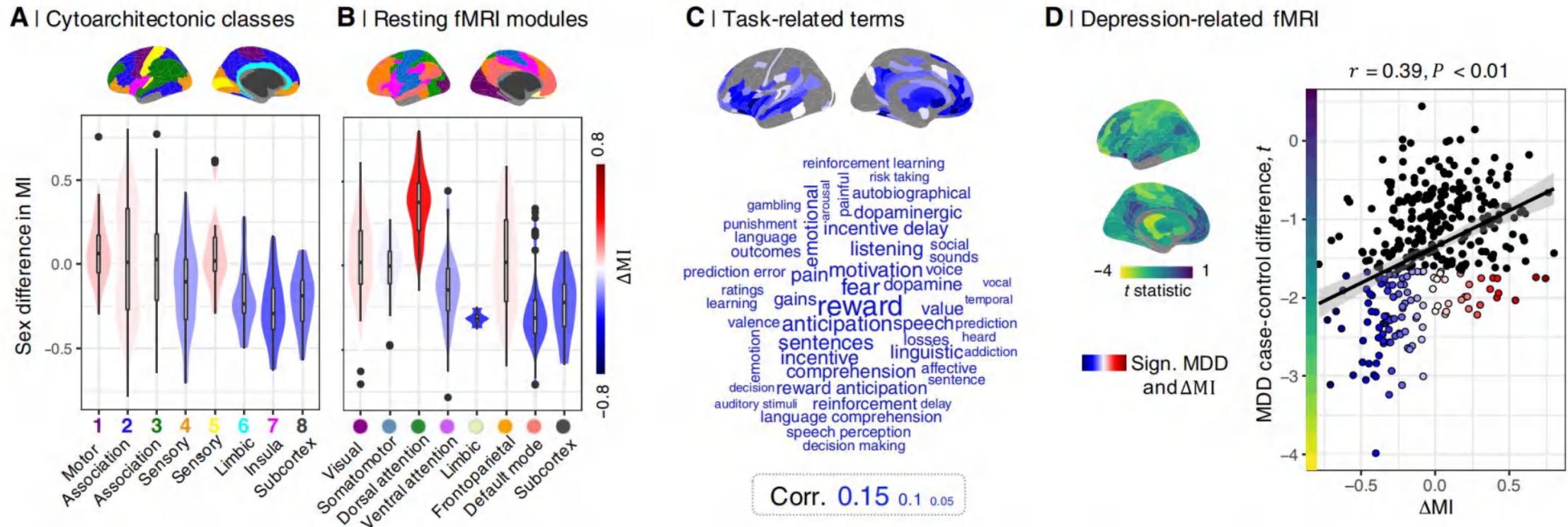
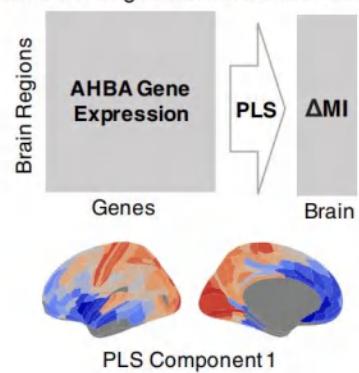
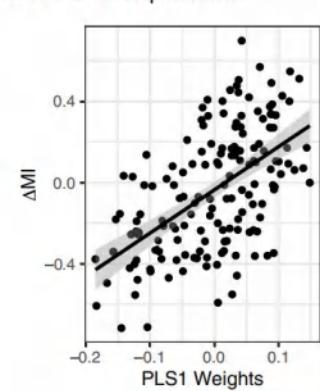
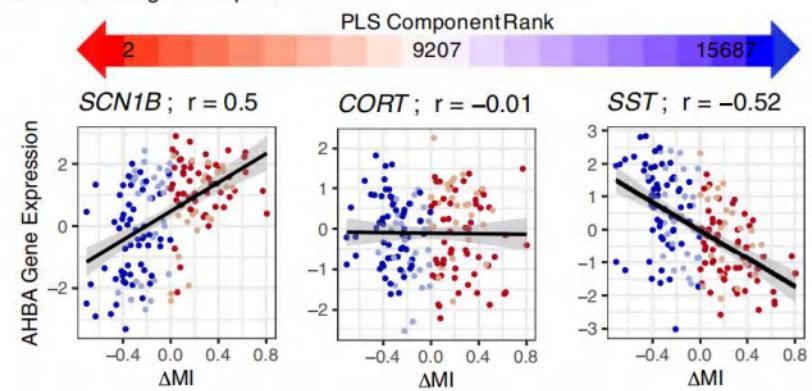
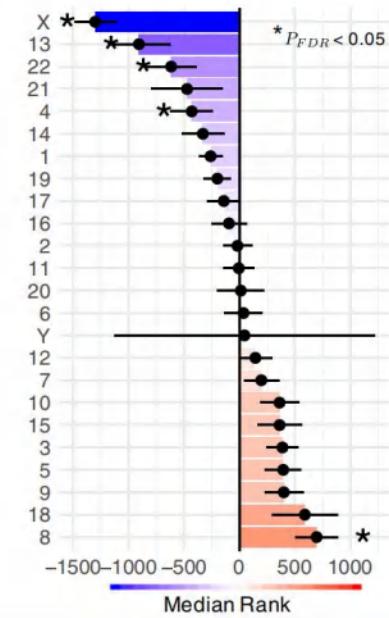
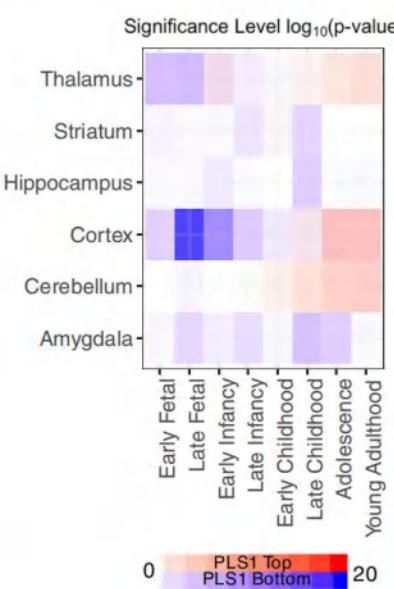
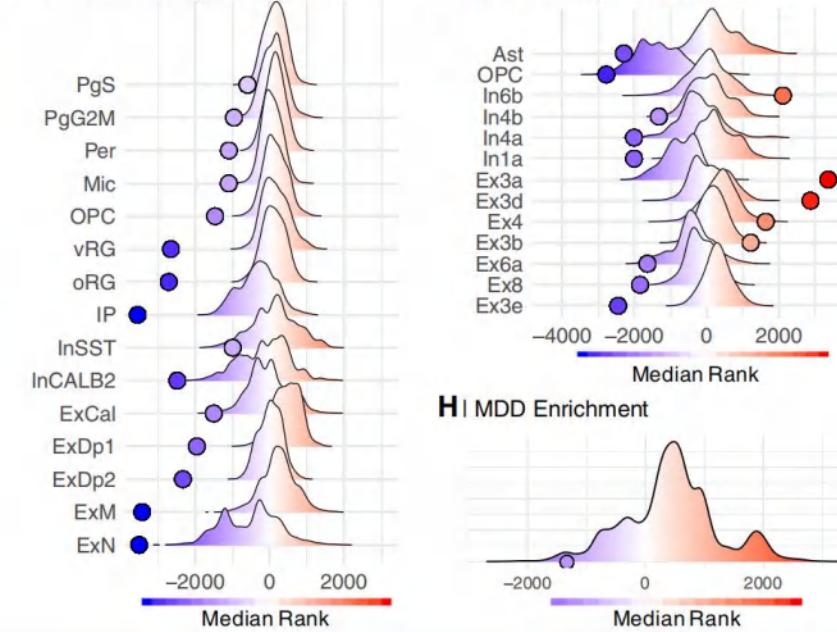
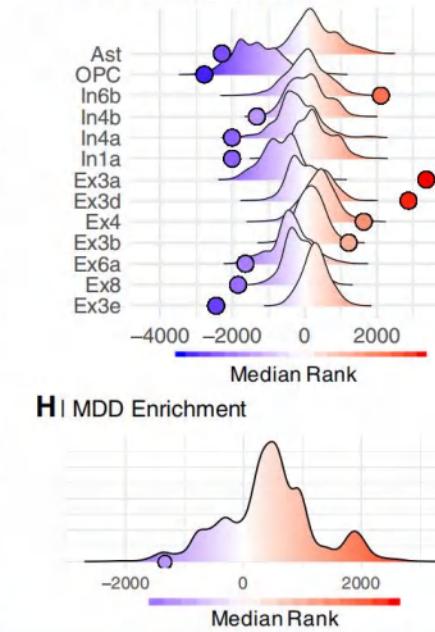
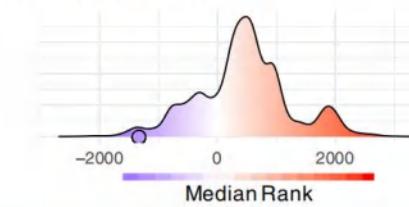


Fig. 3. Sex difference in MI in psychological and psychiatric context. (A) ΔMI was most negative in cytoarchitectonically defined secondary sensory, limbic, and insula cortex and subcortical structures (B) as well as functionally defined (fMRI) DMN, ventral attention network, limbic systems, and subcortical structures. (C) Word cloud of NeuroSynth meta-analytical cognitive terms scaled according to their strength of association with the disruptively developing brain regions (cortical map of $\Delta MI < 0$). (D) Scatterplot of MDD case-control t statistics (y axis) versus ΔMI (x axis). Each point represents one of 346 cortical or subcortical regions; regions of interest that show a significant MDD case-control difference, $t \neq 0$, and a significant sex difference in MI, $t \neq 0$, are highlighted. The fitted line and 95% confidence interval indicate the positive correlation ($r = 0.4, P < 0.001, P_{\text{spin}} < 0.001$) between the spatial maps of MDD case-control differences, t , and ΔMI , shown alongside the y and x axes, respectively. Regions with sexually divergent disruptive development in adolescence (negative ΔMI) had reduced degree of connectivity (negative t) in adult MDD cases.

Sexually divergent, disruptive brain systems are colocated with brain tissue transcripts enriched for X chromosome, neurodevelopmental, and MDD risk genes. (A) We used PLS regression to map gene expression data onto MI. (B) PLS1 was positively correlated with MI; thus, low PLS1 scores were colocated with low MI or predominantly female more disruptive regions. (C) Relationship of MI to expression of exemplary genes: sodium voltage-gated *SCN1B*, a positively weighted gene near the top of the ranked PLS1 weights list; *CORT*, a near-zero weighted gene in the middle of the list; and *SST*, a negatively weighted gene near the bottom. Negatively weighted genes were more strongly expressed in regions of negative MI, that is, predominantly female > male disruptive regions, whereas positively weighted genes were more strongly expressed in regions with female > male conservative development indicated by positive MI. (D) Enrichment analysis for chromosomal genes. Plot of median ranks of genes from each chromosome on PLS1. (E) Enrichment analysis for neurodevelopmental genes. Negatively weighted genes were enriched for genes expressed in cortex during late fetal and early postnatal development and for genes expressed in the amygdala, hippocampus, and striatum during late childhood and adolescence. Positively weighted genes were enriched for genes typically expressed in cortex and cerebellum during adolescence and early adult life. (F) Enrichment analysis for prenatal cell type-specific genes. Negatively weighted genes (blue) were significantly enriched for genes expressed by prenatal vRG and oRG, microglia (Mic), oligodendrocyte progenitor cells, and excitatory neurons. (G) Enrichment analysis for adult cell type-specific genes. Negatively weighted genes were significantly enriched for genes expressed by adult astrocytes, OPC cells, and excitatory neurons. (H) Enrichment analysis for MDD-related genes.

A IPLS Regression AHBA on Δ MI**B** I PLS1 Interpretation**C** I PLS1 Weight Interpretation**D** Chromosome Enrichment**E** I Developmental Enrichment**F** I Prenatal Cell Type Enrichment**G** I Adult Cell Type Enrichment**H** I MDD Enrichment

Neurotoxicants, the Developing Brain, and Mental Health

Carlos Cardenas-Iniguez, Elisabeth Burnor, and Megan M. Herting

Biological Psychiatry Global Open Science

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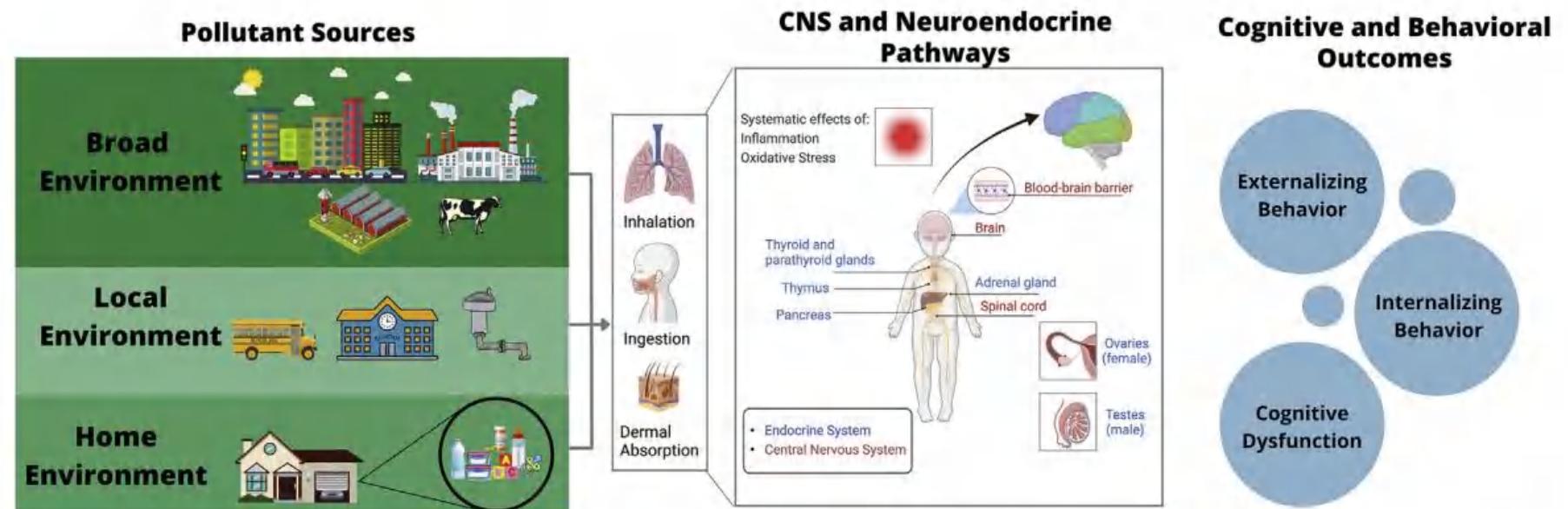
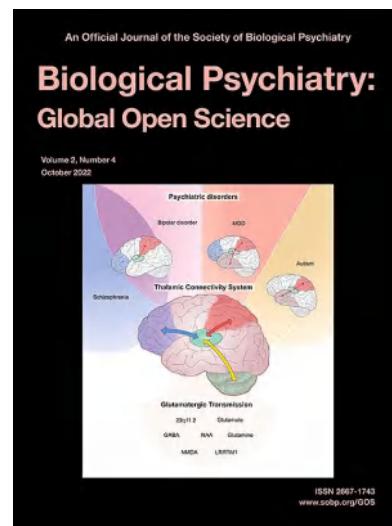
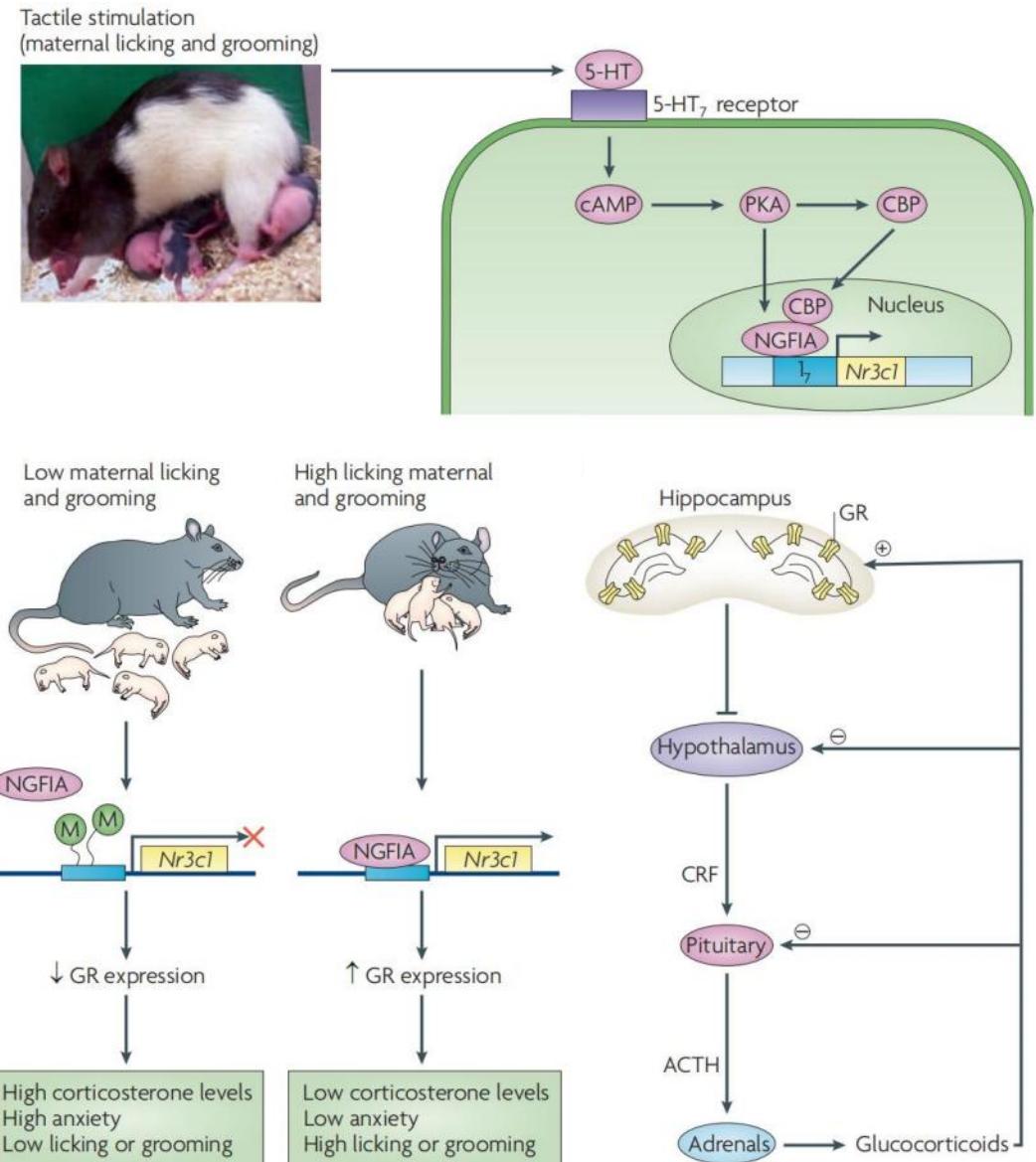
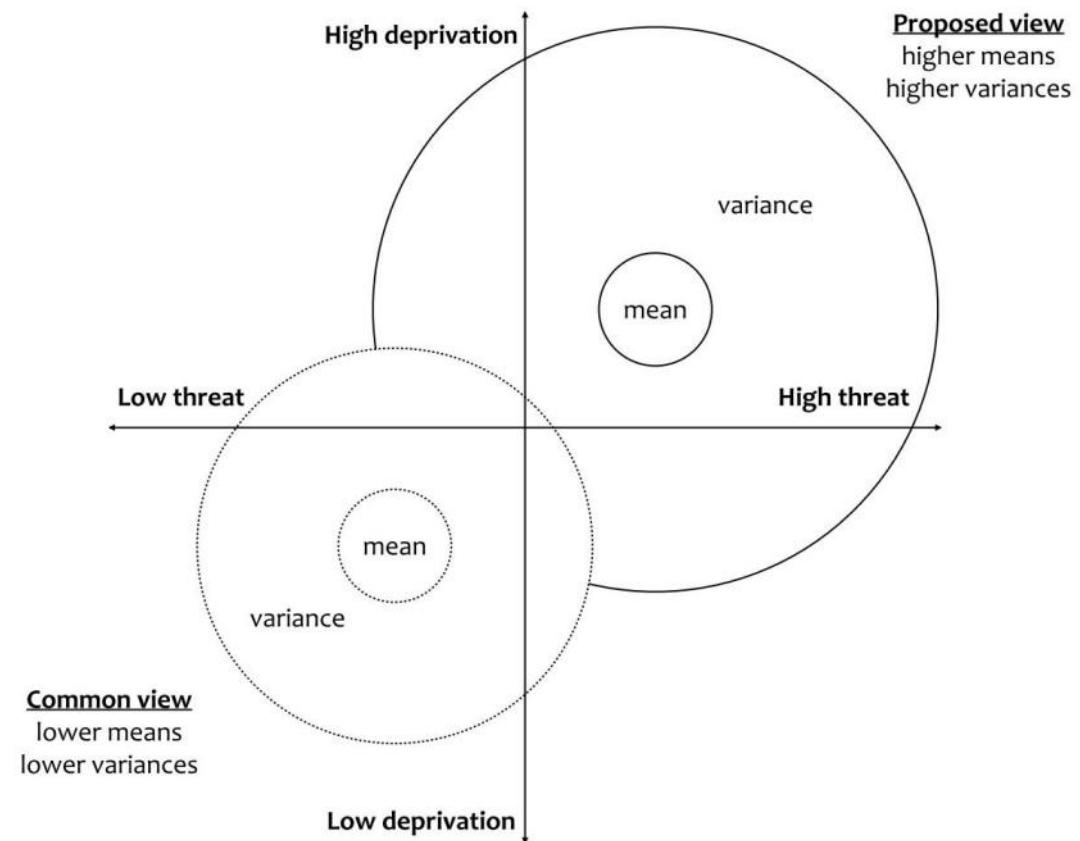


Figure 1. Sources of major neurotoxicants and proposed physiological targets. Exposure to the 3 classes of neurotoxicants discussed in this article (lead, outdoor particulate matter air pollution, and endocrine-disrupting chemicals) can occur through multiple avenues, because the sources for these toxicants include broad environmental pollution from traffic, agriculture, industrial activities, etc.; local pollution in areas, such as parks and schools, where children spend time; and home pollution, where children may be exposed to household items containing neurotoxic chemicals. These pollution sources, encountered in children's broad and local environments, may enter the body through inhalation, ingestion, and/or dermal absorption (see Table S1 for further details). Once in the bloodstream, these toxicants may cross the blood-brain barrier and directly act upon components of the central nervous system (CNS) or indirectly affect the brain by altering other systems, including air pollution-related increases in inflammation and oxidative stress or endocrine-disrupting chemicals acting to disrupt the endocrine system. A causal arrow is not drawn between the physiological diagram and the behavioral and cognitive outcomes, because no causal link has been confirmed. However, it is hypothesized that disruption of normal neurodevelopment and damage to neural and endocrine pathways may lead to changes in behavioral symptoms, cognitive development, and mental health in children.

REVIEWS AND OVERVIEWS

Early Adversity and Development: Parsing Heterogeneity and Identifying Pathways of Risk and Resilience

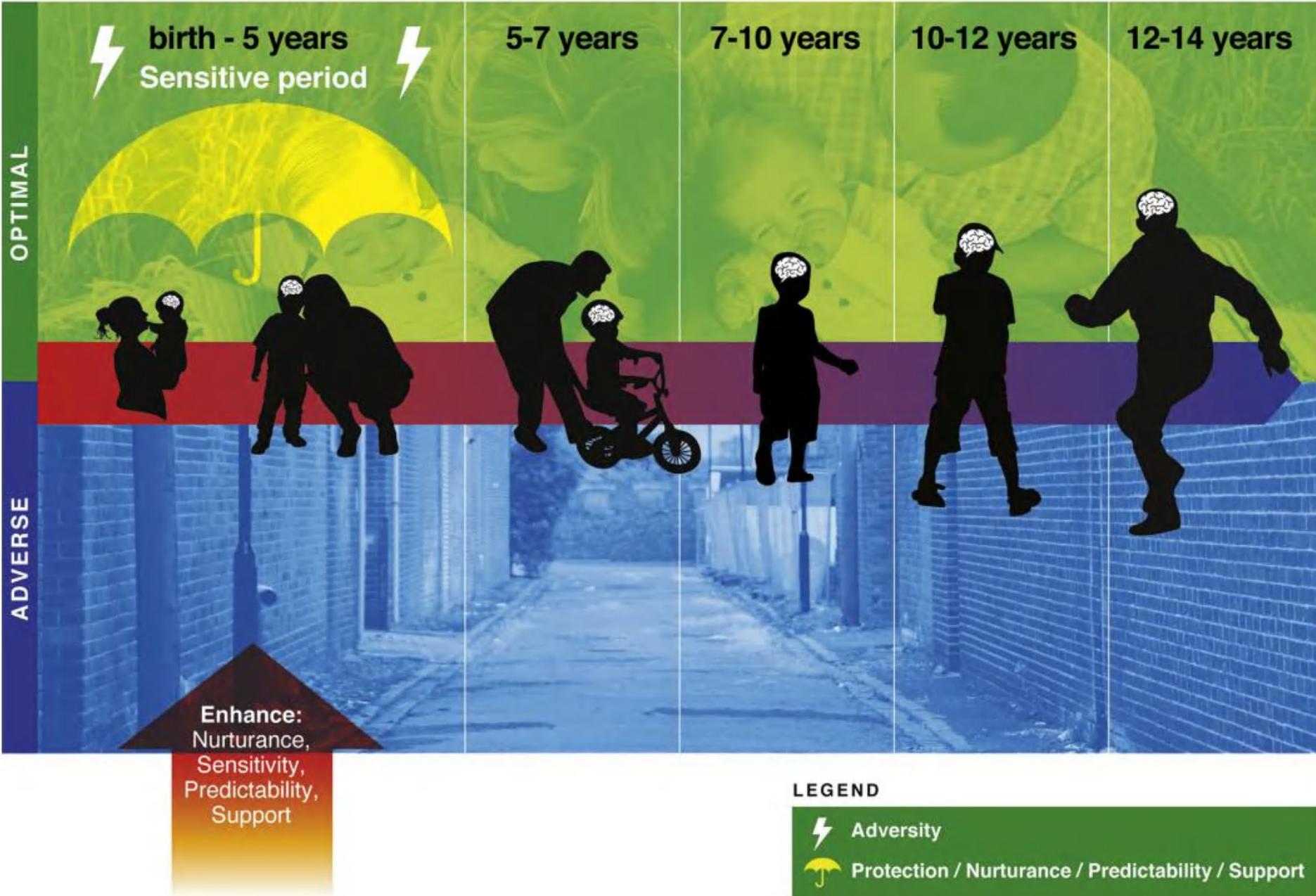
Dylan G. Gee, Ph.D.



Commentary

Introduction to the Special Issue on the Exposome—Understanding Environmental Impacts on Brain Development and Risk for Psychopathology

Deanna M. Barch



Perspective | Published: 28 April 2021

Environmental influences on the pace of brain development

Ursula A. Tooley, Danielle S. Bassett & Allyson P. Mackey 

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The environment influences brain development, experts say

Experts at a North Carolina State University meeting examined how toxicant may affect brain development.

BY JESSE SAFFRON

The NIEHS-funded [Center for Human Health and the Environment](#) (CHHE) at North Carolina State University (NCSU) held its fourth annual science symposium, "Interactions Between the Brain and the Environment," on Feb. 20. Academic researchers, government scientists, postdoctoral trainees, and undergraduate and graduate students met to listen, learn, and share their latest findings.

Too many chemicals, not enough time

"There's been a staggering rise in neurodevelopmental disorders in the United States and globally," noted National Toxicology Program (NTP) Toxicologist Mamta Behl, Ph.D., during her keynote talk. "At NTP, we're asking what could be some environmental causes."

She cited evidence showing increased prevalence of conditions such as [attention-deficit hyperactivity disorder](#) (ADHD) and [autism spectrum disorder](#). And she drew attention to industrial and commercial chemicals, such as [pesticides](#) and [flame retardants](#).

"There are tens of thousands of compounds in our environment that remain untested," she

