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Contextual & physiological markers for individual distress (CP-MIND). Brain health as a comprehensive framework for Mental-health equity☆

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ABSTRACT

Socioeconomic disadvantage shapes brain–mind health by intensifying exposures, resource scarcity, nutritional insecurity, violence, and weak social support, which dysregulate stress and immune systems. These conditions promote allostatic overload, whereby adaptive stress responses become maladaptive, degrading neural circuits for cognitive control and emotion regulation. In parallel, the microbiota–gut–brain axis links contextual adversity and diet quality to inflammation, barrier dysfunction, and neuroendocrine perturbations that further compromise resilience. Converging evidence connects these biological disruptions to structural and functional brain differences and higher risks of depression, anxiety, stress-related syndromes, and later neurodegeneration. While some sociocultural adaptations may bolster cooperation and communal coping, chronic physiological strain undermines durable resilience. This integrative review advances a combined framework, contextual & physiological markers for Individual distress, nested within a brain–mind health perspective, to organise how socioeconomic disadvantage-related exposures are embedded biologically via allostatic and microbiota–gut–-brain axis pathways and manifest as social-cognitive difficulties and affective symptoms. We synthesise evidence across behaviour, neural systems, and systemic physiology to identify leverage points for intervention. Priorities include early multi-domain strategies that reduce chronic stressors; strengthen sleep, nutrition, and social cohesion; and test mechanistic interventions (e.g., allostatic regulation, psychobiotic or dietary modulation) within equity-focused, life-course designs. Understanding how contextual and physiological markers interact is essential for designing effective, scalable policies and clinical approaches that mitigate adversity's neurobiological impact and reduce long-term disparities in brain–mind health.

Introduction

Brain–mind health (BMH) refers to the integrated biological and psychological processes that underlie mental function. Although brain health (the integrity and functioning of neural systems) and mental health (cognitive–affective well-being) are conceptually distinct, they are deeply interdependent. In this review we adopt CP-MInD (Contextual & Physiological Markers for Individual Distress) as an organizing framework to examine how contextual markers (e.g., socioeconomic conditions, social stressors) interact with physiological markers (e.g., allostatic load, sleep and inflammatory dysregulation, microbiota–gut–brain pathways) to shape individual distress and population-level disparities. Throughout, we treat brain and mental health as coupled domains within this CP-MInD framework.

Socioeconomic disadvantage (SED) refers to structural conditions of deprivation that constrain access to resources needed to meet basic needs (e.g., food, housing, healthcare) and to participate fully in society; it co-occurs with challenges such as pecuniary instability, food insecurity, housing instability, and educational inequality (Livingston et al., 2025). Within a health-inequity framework, SED functions as a key social determinant of preventable differences in morbidity and mortality observed between marginalized groups and reference groups, aligning with widely used definitions of “health disparities” (Livingston et al., 2025). In CP-MInD, SED is treated as a multidimensional exposure that

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operates through intersecting contextual pathways (e.g., housing, food access, discrimination and violence), progressively embedding as physiological dysregulation (e.g., stress biology, Microbiota–gut–brain axis [MGBA] alterations) across the life course and elevating risk for stress-related symptoms, social-cognitive difficulties, and mood–anxiety outcomes.

To ground biological embedding more explicitly in social context, we incorporate the sociobiome perspective, which asks how socioeconomic conditions shape gut-microbiota composition and function in ways that heighten disease susceptibility (Nobre and Costa, 2022). In this view, socioeconomic status (SES), at both individual and neighbourhood levels, can exert a stronger influence on microbiota profiles than heritability, with early-life exposures (e.g., diet, crowding, environmental xenobiotics) leaving durable signatures into adulthood and contributing to health disparities; the framework also outlines community-level levers (nutrition access, breastfeeding support, targeted microbiota modulation) to mitigate these effects. Positioned alongside CP-MInD, sociobiome offers a tractable bridge from contextual adversity to physiological markers (MGBA and allostatic processes) relevant to individual distress (Nobre and Costa, 2022).

The consequences of SED manifest across psychological (e.g., emotional regulation, personality) (Evans and Kim, 2007; Kim et al., 2013; McLaughlin et al., 2019), sociocognitive (e.g., empathy, altruism, decision-making) (Corrigan et al., 2014; Farah, 2017; Mezzina et al., 2022), and physiological domains (e.g., brain health, cortisol levels, and features of the MGBA) (Belfiore et al., 2024; Cryan et al., 2019; Johnson et al., 2016; Kraft and Kraft, 2021; Kirkbride et al., 2024; Merlo et al., 2024; Noble et al., 2015; Ortega et al., 2023; Remes et al., 2021; Vera-Urbina et al., 2022), ultimately contributing to adverse mental-health outcomes (e.g., depression, anxiety, stress) (Hamza et al., 2024; Kirkbride et al., 2024; Knifton and Inglis, 2020; Lund et al., 2011; Marmot et al., 2012; Patel et al., 2018; Rojas-Thomas et al., 2023).

Concrete features of disadvantaged contexts, such as resource scarcity (Evans and Kim, 2007; Kurbatfinski et al., 2024; Lund et al., 2010), disrupted sleep (Besedovsky et al., 2019; Iesanu et al., 2022; Lorton et al., 2006), limited access to sports/leisure opportunities (Biddle and Asare, 2011; Hawkley and Capitanio, 2015; Stults-Kolehmainen, 2023), reduced availability of healthy food (Jakubowska et al., 2024; McLaughlin et al., 2012a), and exposure to neighborhood violence or crime (Del Portillo et al., 2024; Fowler et al., 2009; Mollica et al., 2004), collectively undermine brain–mind health and dysregulate physiological processes (e.g., cortisol dynamics, the microbiota) (Besedovsky et al., 2019; Cryan, 2016; Del Portillo et al., 2024; Iesanu et al., 2022; Jakubowska et al., 2024; Kurbatfinski et al., 2024; Lorton et al., 2006; Stults-Kolehmainen, 2023). Moreover, violent contexts and social deprivation alter social cognition, empathy, altruism, and decision-making, often heightening arousal in ways that further harm brain–mind health in vulnerable populations (Carriedo et al., 2024; Fowler et al., 2009; Kupferberg and Hasler, 2023; Mollica et al., 2004).

These exposures intersect with sex- and gender-based inequalities and with experiences of discrimination, shaping patterns of stressor exposure, resource access, and trajectories of health vulnerability. A sex/gender lens clarifies how structural norms and roles compound contextual adversity across settings, including the Global South, strengthening the case for equity-focused prevention and intervention (Baez et al., 2024). In parallel, discrimination (from everyday unfair treatment to lifetime exposures) has been consistently linked to higher allostatic load in adults, underscoring a pathway through which social inequities become biologically embedded and contribute to downstream cognitive, affective, and health outcomes (Miller et al., 2021).

We refer to this broad range of environmental, psychological, and physiological factors as CP-MInD. Consistent with sociobiome, CP-MInD links social adversity to MGBA-related and allostatic pathways, providing a coherent scaffold for integrating context, biology, and social cognition.

The study of brain–mind health, especially in socioeconomically vulnerable sectors of society, is a complex task that requires a multidimensional perspective (Alegría et al., 2018; Kirkbride et al., 2024), observing individual personality traits, social cognition, emotional skills, and day-to-day behavior (Alegría et al., 2018; Carriedo et al., 2024; Santamaría-García et al., 2020). A multidomain view is also necessary, integrating behavioral information, brain functional models, and body physiology (Gautam et al., 2024).

Our goal is to integrate contextual stressors and physiological markers, specifically allostatic load and the MGBA, within the CP-MInD framework to inform targeted prevention and intervention. We posit that these domains jointly shape social cognition in vulnerable groups. CP-MInD enables assessment of the collective impact of markers, understood as supra-additive, intersectional interactions among coexisting markers, effects greater than the sum of individual contributions, rather than a simple additive model. See Fig. 1 for the conceptual framework and Table 1 for the evidence summary.

Allostasis, brain–mind health and socioeconomic disparities

SED shape brain trajectories across the life course, amplifying burdens of chronic stress and mood disorders and accelerating neuropathological cascades linked to dementia risk (Hackman et al., 2010; Hazzouri et al., 2011a; Ibáñez et al., 2023). In low–socioeconomic status (low-SES) groups, SED contributes to a cycle of neural and systemic dysfunction characterized by faster neurodegeneration (Ibáñez et al., 2023), stress-exacerbated proteinopathy (e.g., amyloid-β/tau) (Donovan et al., 2016; Sierra-Fonseca and Gosselink, 2018), and maladaptive neuroplasticity (e.g., stress-related atrophy in prefrontal–limbic circuits) (Noble et al., 2012; Zannas et al., 2015). Conceptually, homeostasis denotes feedback-based maintenance of internal stability within narrow physiological ranges (e.g., temperature, pH, glucose) (Billman, 2020). Allostasis describes stability through change, anticipatory, brain-mediated adjustments across neuroendocrine, autonomic, immune, and behavioral systems to meet environmental demands (Bobba-Alves et al., 2022). Repeated or chronic recruitment of these responses accumulates as allostatic load (multisystem "wear and tear" and energetic costs), and when regulatory capacity is exceeded, allostatic overload ensues with maladaptive outcomes (Bobba-Alves et al., 2022; Durán et al., 2024; McEwen and Gianaros, 2010; Rojas-Thomas et al., 2023; Thanaraju et al., 2024).

Low-SES environments intensify exposure to psychosocial stressors (e.g., financial strain, trauma) that epigenetically prime stress-sensitive pathways, hypothalamic–pituitary–adrenal (HPA) axis and inflammatory signalling, linking SED to brain–mind health decline and neurodegeneration (Madsen et al., 2017; Szanton et al., 2005). Chronic adversity often includes precarious employment (Madsen et al., 2017) and reduced social support (Brooks et al., 2014; Carriedo et al., 2024; Franco-O'Byrne et al., 2023a,b; Rojas-Thomas et al., 2023), which synergistically elevate allostatic burden (Szanton et al., 2005). The brain orchestrates these stress responses via distributed circuitry, prefrontal cortices, hippocampus, and amygdala, integrating threat appraisal with peripheral effectors (Herman et al., 2003; McEwen and Gianaros, 2010; Ulrich-Lai and Herman, 2009). Acute stress adaptively engages neuroendocrine and autonomic systems (sympathetic–adreno–medullary activation), transient cardiovascular responses, and immune mediators (rapid cytokine release and leukocyte redistribution), supporting short-term allostatic adjustments such as energy mobilization and heightened vigilance (Arnsten, 2009; Dhabhar, 2014; Joëls and Baram, 2009; McEwen, 2007; McEwen and Gianaros, 2011; Sapolsky et al., 2000). However, the chronic activation common in low-SES settings can convert otherwise protective mechanisms into drivers of neural deterioration (Cohen et al., 2012; Gianaros and Manuck, 2010; Lau et al., 2016; Lawson et al., 2017).

Prolonged stress exposure fosters allostatic overload (McEwen, 2004; McEwen and Akil, 2020), glucocorticoid-receptor desensitization (Corwin et al., 2013; Saaltink and Vreugdenhil, 2014), heightened

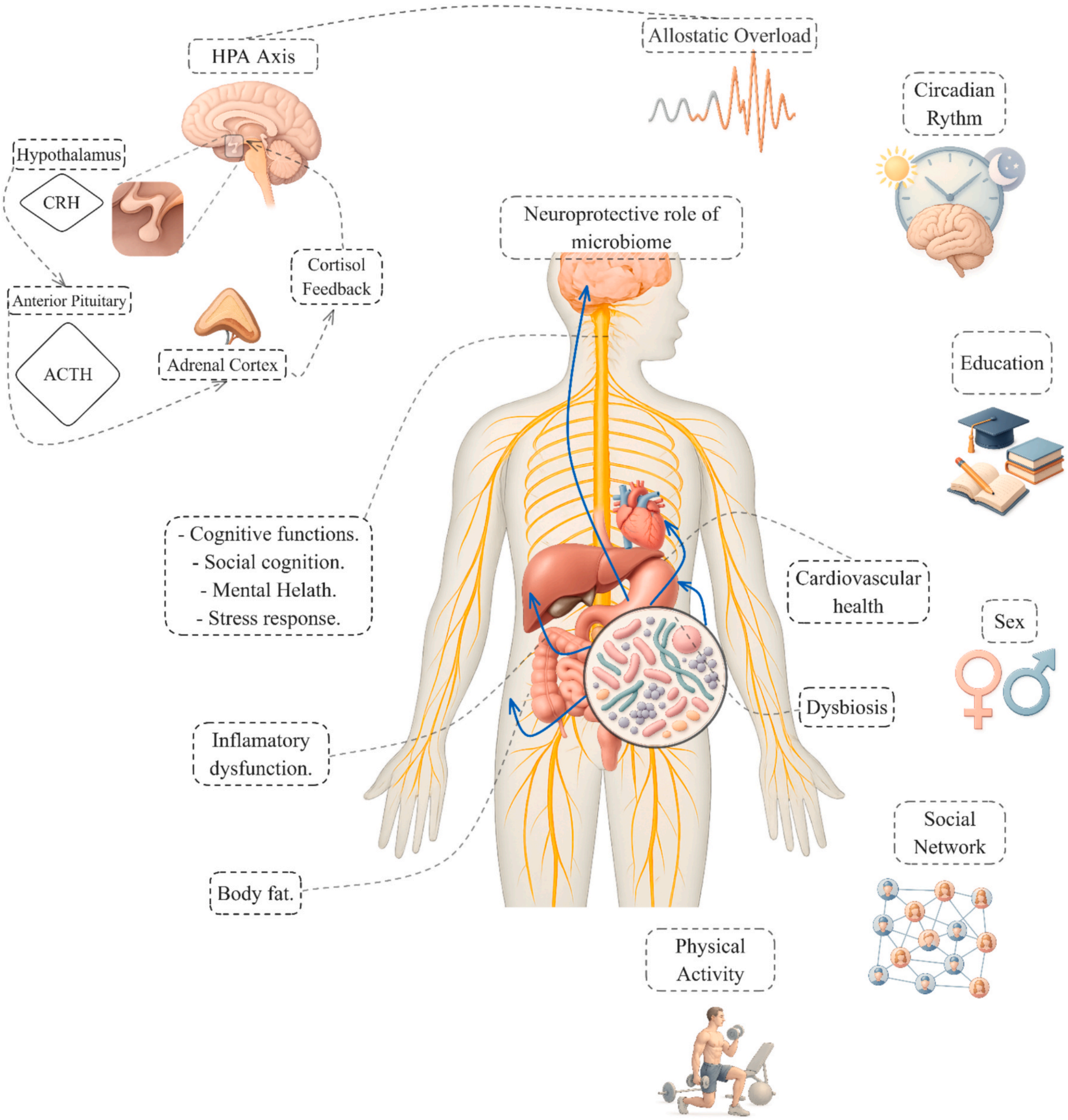

Fig. 1. CP-MInD conceptual model linking contextual and physiological markers of individual distress. The diagram depicts how contextual exposures interface with bodily systems to shape brain–mind health. Right panel (contextual markers): education; social network/loneliness; sleep quality; physical activity; and sex differences, conceptualised as external inputs that modulate internal regulation. Centre, neurocognitive role of microbiome. Right panel, stress system. It represents a dynamic feedback between contextual inputs and physiological factors, underscoring the interdependence of psychosocial and biological processes in the emergence, maintenance, and mitigation of distress. This model motivates integrated assessment and intervention across contextual and physiological domains to explain individual variability in stress responsivity and brain–mind health outcomes.

neuroinflammation, and structural/functional changes in stress-sensitive regions. These include prefrontal dysfunction that impairs executive control (Arnsten, 2009; Arnsten et al., 2014; Alexandra Kredlow et al., 2022; Rojas-Thomas et al., 2023), amygdala hyperreactivity to threat (Alexandra Kredlow et al., 2022; Maren and Holmes, 2015; Rojas-Thomas et al., 2023), and hippocampal disruptions that weaken negative feedback of the HPA axis (Godsil et al., 2013; Maren and Holmes, 2015). At the microstructural level, hippocampal atrophy and reduced dendritic complexity in prefrontal circuits erode cognitive reserve and heighten vulnerability to stress-related cognitive decline (Cohen et al., 2012; Lau et al., 2016; Sumowski et al., 2013).

In parallel, these stress-biology alterations aggravate proteinopathic cascades, multistep processes of misfolding, oligomerization, and accumulation of pathogenic proteins such as tau (neurofibrillary tangles in Alzheimer's disease) (Goedert, 2020), α-synuclein (Lewy bodies in Parkinson's disease) (Wong and Krainc, 2017), and TDP-43 (cytoplasmic inclusions in amyotrophic lateral sclerosis and frontotemporal dementia) (Wood et al., 2021). The resulting proteostatic failure disrupts synapses and accelerates neurodegeneration (Hipp et al., 2019; Soto and Pritzkow, 2018; Ibáñez et al., 2023). Together, these convergent disruptions form a self-reinforcing cycle in which neural dysregulation hastens dementia pathogenesis, elevating population burden, and, in turn, entrenches the very socioeconomic–biological gradients that initiated the cascade (Galvin et al., 2021; Gaugler et al., 2023; Ibáñez et al., 2023; Wilson et al., 2013; Zsoldos et al., 2018).

Allostatic load (AL) operationalises the cumulative "wear and tear" of chronic stress as a multisystem composite spanning neuroendocrine, cardiovascular, metabolic, and immune markers (Durán et al., 2024; Guidi et al., 2020; McEwen and Akil, 2020; Rojas-Thomas et al., 2023). Canonical features include altered HPA-axis activity, such as chronically

Table 1

CP-MInD Dimensions: Integrated Evidence Map of Contextual and Physiological Markers for Individual Distress (Brain–Mind Health Framework).

CP-MInD Dimension	Core Findings	Key Mechanisms / Markers	Implications for Research & Practice	Authors
Context—Sleep	Socioeconomic adversity is linked to shorter/fragmented sleep and diurnal disruption; sleep problems co-travel with higher AL and may mediate SED → BMH gradients.	Multi-night actigraphy; diurnal cortisol slope/CAR; evening cortisol elevation; integration with AL subsystems.	Combine actigraphy (≥7 nights) with repeated diurnal cortisol in high-disadvantage cohorts; treat sleep improvement as a mechanistic AL-reduction target.	(St-Onge et al., 2016; Besedovsky et al., 2019; Iesanu et al., 2022; Lorton et al., 2006).
Context—Physical activity	Low-SES contexts show constraints on PA; reduced PA co-occurs with higher AL and autonomic/immune imbalance.	Reduced HRV; elevated resting BP/catecholamines; inflammatory tone.	Community-feasible PA programs; track HRV/inflammatory markers alongside symptoms and AL composites.	(Gruenewald et al., 2009; Stults-Kolehmainen, 2023; Biddle and Asare, 2011; Hawkley and Capitanio, 2015).
Context—Diet quality / free & added sugars	Higher free/added sugars and SSBs associate with adverse neuropsychiatric outcomes; guidance converges on < 25 g/day sugars and < 1 SSB/week.	Glycemic/inflammatory load; MGBA dysbiosis; HPA/circadian coupling.	Preregistered nutrition trials linking sugar reduction to AL/MGBA and mood outcomes; stratify by baseline diet/microbiome.	(Huang et al., 2023; Xiong et al., 2024).
Context—Ultra-processed foods (UPFs)	Greater UPF intake associates with higher odds of depressive/anxiety symptoms and prospective depression risk; UPF growth is steepest in poorer/early-life contexts.	Low nutrient density, additives, and processing → dysbiosis; barrier dysfunction; low-grade inflammation → ↑AL.	Early-life UPF reduction; harmonized diet–microbiome–mental-health protocols including LMIC/UMIC sites.	(Lane et al., 2022; Lane et al., 2024; Popkin and Laar, 2025).
Context—Environmental safety & opportunity	Educational opportunity, neighborhood safety, violence exposure, food access and other SED facets plausibly shape GM and network organization via chronic stress/inflammation.	Long-term stressors impacting prefrontal–limbic circuits; HPA/inflammatory mediation.	Pair structural policies (education, safety) with mechanistic endpoints (MRI/EEG + AL panels) in longitudinal designs.	(Brito and Noble, 2014; Noble et al., 2015; Del Portillo et al., 2024).
Context—Perceived control / stressor load	Low-SES combines higher stressor frequency with lower perceived control; both amplify AL and downstream BMH risk.	Recurrent uncontrollable stress → HPA hyperactivity, immune dysregulation.	Incorporate perceived-control measures; test buffering interventions (cash transfers, social support).	(Szanton et al., 2005; Adler et al., 2008).
Physiology—Allostasis / Allostatic Load (AL)	AL captures multisystem "wear-and-tear" of SED-related chronic stress; higher AL is observable from adolescence and predicts worse cardiometabolic/mental outcomes.	Composite across HPA (flattened diurnal slope/CAR), SAM (BP/catecholamines), inflammatory/metabolic markers (e.g., CRP, insulin resistance).	Use AL for risk stratification and as a primary mechanistic endpoint; report subsystem scores; integrate with contextual measures.	(Juster et al., 2010; Seeman et al., 2001; Guidi et al., 2020; Williams et al., 2022; Lucente and Guidi, 2023).
Physiology—HPA axis / cortisol	Experimental and clinical literatures show consistent HPA hyper-/dys-reactivity under chronic stress; lowering stress burden may attenuate later cognitive decline.	Flattened diurnal slope; elevated evening cortisol; blunted/augmented TSST responses (sex-dependent); diminished CAR at higher SED.	Field diurnal sampling with repeated measures; evaluate HPA-targeted adjuncts (e.g., in treatment-resistant depression).	(Vreeburg et al., 2009; McEwen and Gianaros, 2010; Adam et al., 2017).
Physiology—Immune–inflammatory tone	Stress- and diet-linked cytokine cascades/glial regulation provide routes by which context/MGBA influence neural systems and BMH.	Peripheral cytokines (e.g., CRP, ILs); microglial activation; intestinal/BBB permeability.	Include standardized inflammatory panels with neural/endocrine endpoints; test anti-inflammatory adjuncts in stress-related comorbidity.	(Loh et al., 2024; Perry and Holmes, 2014).
Physiology—Microbiota–gut–brain axis (MGBA)	Bidirectional gut–brain signaling links diet/context to neural and social-cognitive outcomes; dysbiosis impairs axis function; stress and circadian processes couple to MGBA.	SCFAs; vagal/enteric signaling; endocrine/immune routes; gut/BBB integrity; circadian–HPA coupling; oscillatory taxa (e.g., L. reuteri).	Mechanistic, preregistered multi-omics trials (diet/psychobiotics) with neural/endocrine endpoints; stratify by baseline microbiome/diet.	(Cryan et al., 2019; Foster and McVey Neufeld, 2013; Sharvin et al., 2023; Zhou and Foster, 2015).
Neural systems—Structure & large-scale networks	SED associates with reduced GM and altered control/salience/default-mode networks; effects are heterogeneous and developmentally patterned; stress/inflammation likely target dlPFC circuitry.	DLPFC-centered control networks; prefrontal–limbic coupling; EEG markers: P1/N1/N2/ERN/N400/P3 modulation differences.	Combine MRI/EEG with physiological/context panels in longitudinal cohorts; model nonlinear/heterogeneous effects.	(Merz et al., 2023; Baysarowich et al., 2025; Katus et al., 2020).
SED facet—Nutritional disadvantage	SED increases probability of poor diet quality; global UPF shifts are steepest in poorer households and from early life, shaping taste and metabolic risk.	Diet → MGBA dysregulation → inflammation/stress → ↑AL and mood risk.	Policy levers improving access to minimally processed, nutrient-dense foods; early-life nutrition supports.	(Popkin and Laar, 2025; Lane et al., 2022, 2024; De Sequeira et al., 2022).

(*continued on next page*)

Table 1 (*continued*)

CP-MInD Dimension	Core Findings	Key Mechanisms / Markers	Implications for Research & Practice	Authors
SED facet—Educational opportunity / cognitive reserve	SES-linked educational gaps contribute to differences in brain structure and later social-cognitive outcomes via stress and reserve pathways.	Lower reserve → greater vulnerability of control circuits under chronic stress.	Early enrichment/schooling quality as upstream, population-level intervention.	(Brito and Noble, 2014; Noble et al., 2015).
SED facet—Neighborhood safety / instability	Safety/instability/violence are components of cumulative SED exposures that shape BMH and social cognition, partly via vigilance and AL.	Chronic vigilance; altered PFC–limbic coupling; ↑AL trajectories.	Improve neighborhood safety/ cohesion; measure downstream AL and neural changes.	(Del Portillo et al., 2024; Fowler et al., 2009; Mollica et al., 2004).
Social cognition under SED	Lower SES is often associated with poorer empathy/ToM/ prosociality; EEG shows attenuated modulation of attention/conflict/semantic/ outcome components; measurement/task heterogeneity and moderators (stress biology, comorbidity) explain mixed findings.	Distributed “social brain” (mPFC/OFC, ACC/insula, TPJ, amygdala); stress/ inflammation-sensitive control networks (dlPFC).	Longitudinal, mechanistic studies; test mediation by neural/ physiological pathways; harmonize tasks/culture-fair measures.	(Beaudoin et al., 2020; Migeot et al., 2022; Katus et al., 2020; Carriedo et al., 2024).
SED → neurodegeneration risk	Chronic SED sustains allostatic overload and accelerates neural senescence; stress-exacerbated proteinopathy implicated in dementia pathways.	HPA hyperactivity; neuroinflammation; proteostatic failure (Aβ/tau, α-syn, TDP-43).	Combine structural policies (poverty/discrimination reduction) with biological targets (inflammation/cortisol) to modify risk trajectories.	(Noble et al., 2012; Sierra-Fonseca and Gosselink, 2018; Zannas et al., 2015; Ibáñez et al., 2023).
Cross-cutting—Discrimination & structural adversity	Everyday and lifetime discrimination consistently predict higher AL, evidencing biological embedding of inequities; intersects with sex/ gender inequalities.	AL subsystems (cardio-metabolic, inflammatory) mediate disadvantage → BMH; cumulative stress exposure.	Model intersecting exposures (discrimination, violence) together with AL/MGBA; equity-focused prevention and policy.	(Miller et al., 2021; Williams et al., 2022; Baez et al., 2024).
Developmental windows & early embedding	Elevated AL is already detectable by middle childhood in low-income samples; early SES predicts cortical/ subcortical growth and later cognition/risk.	Coupled trajectories: ↑AL ↔ internalizing; stress-sensitive neurodevelopment (hippocampus, prefrontal, amygdala).	Early, tailored supports (stress regulation, sleep, nutrition, stability); trauma-informed care; poverty alleviation.	(Tyrell et al., 2023; Barch et al., 2022).
Sex/gender differences in stress biology	Sex steroids modulate HPA/ SAM; men often show larger ACTH/cortisol to achievement-type stress (TSST), whereas responses vary by paradigm and hormonal milieu; staging matters for diurnal/acute profiles.	Sex-specific ACTH/cortisol slopes; hormone-phase effects; links to depression/anxiety differ by sex/gender.	Explicit sex/gender modeling; repeated diurnal sampling; stage hormones in AL/HPA studies; tailor interventions to life-course windows.	(Stephens et al., 2015; Liu et al., 2017; Albert and Newhouse, 2019; Zorn et al., 2016; Traustadóttir et al., 2003).

This table synthesizes the reviewed evidence for each CP-MInD dimension in four decision-relevant columns: (i) Core Findings, distilling the most robust results with attention to effect direction, magnitude (when available), and developmental sensitivity; (ii) Key Mechanisms/Markers, detailing pathways and biomarkers through which exposures influence Brain–Mind Health (e.g., HPA/SAM activity, inflammatory indices, HRV, microbiota–gut–brain axis features, and structural/functional neural metrics); (iii) Implications for Research & Practice, outlining design and policy levers, measurement guidance (e.g., multi-night actigraphy; diurnal cortisol with CAR and slope; harmonized multi-omics; longitudinal MRI/EEG), and translational considerations for high-disadvantage settings; and (iv) Authors, providing canonical and recent sources formatted for immediate manuscript use. All rows are explicitly framed as CP-MInD dimensions spanning context (sleep, physical activity, diet/UPF, safety/opportunity, perceived control), physiology (allostasis/HPA, immune–inflammatory tone, MGBA), neural systems, SED facets (nutrition, education, neighborhood), social cognition, neurodegeneration risk, cross-cutting discrimination/structural adversity, developmental windows, and sex/gender differences. Abbreviations: SED = socioeconomic disadvantage; AL = allostatic load; HPA = hypothalamic–pituitary–adrenal axis; SAM = sympathetic–adreno–medullary; MGBA = microbiota–gut–brain axis; HRV = heart-rate variability; GM = gray matter; ToM = theory of mind; UPF = ultra-processed food; SSB = sugar-sweetened beverage; LMIC/UMIC = low-/upper-middle-income countries.

elevated cortisol or blunted diurnal slopes (Adam et al., 2017; Staufenbiel et al., 2012), sustained sympathoadrenal activation (e.g., higher blood pressure or catecholamines; Lambert and Lambert, 2011; Ross and Van Bockstaele, 2021), and immunometabolic dysregulation (e.g., elevated C-reactive protein and insulin resistance; Furman et al., 2019; Steptoe et al., 2014). By integrating these indicators, AL quantifies how prolonged stress perturbs multiple systems and, at higher levels, progresses toward allostatic overload (Juster et al., 2010; Seeman et al., 2001; Szanton et al., 2005).

Contemporary cohort data strengthen the socioeconomic gradient in AL in the National Health and Nutrition Examination Survey (NHANES) analyses. Black women with higher educational attainment show substantially lower prevalence of high AL than those with less education, even after adjustment (Williams et al., 2022). In rural Chinese adolescents, longer exposure to neighbourhood poverty predicts higher AL, which in turn forecasts earlier pubertal timing and greater internalising symptoms (Ren et al., 2023). Nationally representative U.S. data likewise indicate that adolescents from lower-income households have two- to three-fold higher prevalence of high AL, with income-related protection most evident among non-Hispanic White youth (Mathis et al., 2025). These findings align with recent syntheses of adolescent AL (Lucente and Guidi, 2023) and large studies linking socioeconomic adversity and discrimination to elevated AL trajectories (Cuevas et al., 2024; see also Williams et al., 2022). Together, this literature supports the central claim that low-SES contexts are associated with higher AL, reflecting both direct biological stress effects and indirect pathways, such as stress-related sleep disruption and reduced physical activity, that compound physiological burden (Cortes-Canteli et al., 2010; Gruenewald et al., 2009; St-Onge et al., 2016).

These allostatic mechanisms are further shaped by SES-linked differences in perceived control and stressor frequency, with low-SES groups facing greater cumulative exposure than higher-SES peers (Szanton et al., 2005). AL is therefore a useful lens for understanding brain–mind health disparities because it captures stress-responsive intermediary biomarkers, such as a diminished cortisol awakening response, through which socioeconomic gradients translate into biological vulnerability (Adler et al., 2008; Chen et al., 2006; Dowd et al., 2009; Pampel et al., 2010). Preclinical work converges with human evidence: chronic stress–induced glucocorticoid neurotoxicity in the hippocampus mirrors SES-related grey-matter atrophy observed in neuroimaging studies (Brito and Noble, 2014; Noble et al., 2015; Sapolsky, 2000; Wingenfeld and Wolf, 2014). This translational alignment underscores the relevance of targeting neuroendocrine dysregulation, particularly in mood disorders, to reduce downstream neurodegenerative risk (Perry and Holmes, 2014; Picard et al., 2014). Consistent patterns of HPA hyperactivity in experimental and clinical contexts (Vreeburg et al., 2009) reinforce the plausibility that interventions lowering stress burden may attenuate later cognitive decline, including dementia (McEwen and Gianaros, 2010; Hazzouri et al., 2011b).

SED perpetuates biological gradients by sustaining allostatic overload (Dufford et al., 2018; Noble et al., 2012). In low-SES populations, prolonged exposure to systemic stressors accelerates neural senescence, evidenced by stress-related hippocampal atrophy, a hallmark of depression, and amplifies proteinopathic cascades that intersect with affective dysregulation (Chan et al., 2018; Garrett and Wellman, 2009; Zhan et al., 2018). This bidirectional cycle, in which structural inequities exacerbate biological vulnerability, highlights the urgency of interventions that address both upstream drivers (e.g., poverty, discrimination) and stress-related molecular pathways (e.g., neuroinflammation, cortisol dysregulation) that underlie disparities in brain–mind health and neurodegenerative risk (Chan et al., 2018; Garrett and Wellman, 2009; Zhan et al., 2018).

Pharmacological strategies that target stress and immune pathways, such as modulation of the HPA axis in treatment-resistant depression and anti-inflammatory approaches for stress-related comorbidities, are being evaluated as adjuncts to reduce affective burden and may also modify downstream neurocognitive risk (McEwen and Gianaros, 2010). In parallel, non-pharmacological interventions have accruing support: structured physical activity, mindfulness-based programs, biofeedback, neurostimulation (e.g., TMS, VNS), virtual-reality supported exposure, and trauma-focused psychotherapies can reduce anxiety symptoms and complement standard care, with effect sizes contingent on modality, dose, and population (Antos et al., 2024). At the social level, global evidence shows that stronger bonding with close networks, particularly family, and membership in multiple groups are associated with greater engagement in health-promoting behaviours, lower anxiety and depression, and higher well-being, underscoring the value of interventions that strengthen social cohesion alongside individual treatments (Tunçgenç et al., 2023). Integrating these modalities with precision public-health measures that address socioeconomic determinants (e.g., education, income support, access to care) offers a pragmatic route to disrupt links between socioeconomic disadvantage and brain health (Ibáñez et al., 2023; Marmot, 2005). Prioritising developmental windows, through trauma-informed care, poverty alleviation, and sleep and nutrition supports, may prevent stress-sensitive biological changes from becoming entrenched across the life course (McEwen and Gianaros, 2010; Rojas-Thomas et al., 2023).

Childhood is a pivotal period for the biological embedding of socioeconomic adversity. In a neurodevelopmental cohort of 491 low-income children aged 8–12 years (52 % male; 68 % Black; 21 % Latino), Tyrell et al. (2023) used latent profile analysis to jointly model cumulative SES risk, AL, and mental-health functioning, identifying six profiles ranging from “low risk” to combined mental–physical burden. Profile membership varied with maltreatment history, emotion regulation, affect, and personality, consistent with resilience theory’s emphasis on multilevel adaptation. Notably, physiological dysregulation (elevated AL) was already detectable by middle childhood, highlighting a critical opportunity for early, tailored interventions in low-SES populations (Tyrell et al., 2023).

Stress reactivity and AL exhibit clear sex/gender differences relevant to mood-disorder disparities: men tend to show higher global AL, whereas women display sex/gender-specific pathways linking AL to mental-health outcomes (Juster et al., 2016; Kerr et al., 2020). Because AL is a dynamic, multisystem index that varies with sex, gender, age, stressor type, and coping, observed differences should be interpreted within life-course and contextual moderators (Volarić et al,. 2024). Mechanistically, acute stress recruits the sympathetic–adreno–medullary system and the HPA axis, which ordinarily restore homeostasis; dysregulation of these systems is closely implicated in affective psychopathology (Rubinow and Schmidt, 2018). Sex steroids further shape stress biology: experimental manipulation of peripheral hormones produces sexually dimorphic central and peripheral stress-response patterns, indicating that estrogen/testosterone signalling modulates HPA-related processes and reactivity (Guo et al., 2018).

Cortisol, the HPA end product, coordinates metabolic, immune, and neural responses, and alterations in diurnal slope or stress reactivity are implicated across stress-related disorders (Stephens et al., 2015). In acute psychosocial paradigms, men typically exhibit larger ACTH/cortisol responses than women, a pattern replicated in a large single-site Trier Social Stress Test (TSST), a *meta*-analysis, and samples of healthy older adults (Liu et al., 2017; Stephens et al., 2015; Traustadóttir et al., 2003). However, effect direction and magnitude depend on the probe and hormonal milieu: after pharmacological HPA activation with naloxone, women can show greater cortisol responses than men, and endogenous/contraceptive hormones modulate reactivity (Uhart et al., 2006; Zorn et al., 2016). Sex steroids interface with stress circuitry and depression risk across the female life course, and large samples indicate that testosterone (in men) and progesterone (in women) inversely relate to TSST responsiveness (Albert and Newhouse, 2019; Stephens et al., 2015). Estrogen fluctuations further modulate networks for reactivity, cognition, and emotion, contributing to elevated vulnerability during reproductive transitions (Albert and Newhouse, 2019). Across the menstrual cycle, basal cortisol shows small but reliable phase differences (slightly higher in the follicular than the luteal phase), underscoring the need for careful hormone staging and repeated diurnal sampling (Klusmann et al., 2022). Complementing these baseline effects, large TSST datasets suggest sex-linked dynamics in acute HPA responses: men show steeper ascending/descending ACTH–cortisol slopes (faster onset and recovery), whereas women display attenuated cortisol responses to achievement-type stressors and may respond more to social-rejection paradigms (Stephens et al., 2015).

Clinically, sex-specific alterations are evident: women with current major depression/anxiety often show blunted cortisol responses to psychosocial stress, whereas men with current major depression or social anxiety frequently exhibit exaggerated responses (Zorn et al., 2016). Developmentally, the female HPA axis appears more susceptible to early-life programming of reactivity, and adolescent data reveal sex-specific coupling between HPA activity, redox balance, internalising symptoms, and white-matter microstructure (Carpenter et al., 2017; Schilliger et al., 2024). Beyond biology, sociocultural gender norms shape appraisal and coping: in a population sample, endorsement of masculine roles related to lower distress, whereas a composite of gendered norms related to higher depressive symptoms and perceived stress—reinforcing the need to distinguish biological sex from sociocultural gender in mental-health research (Mommersteeg et al., 2023). Converging neuroendocrine and neuroimaging evidence indicates that stressor type matters: men tend to mount greater HPA responses to achievement-evaluation stressors, whereas women show stronger responses to social-relational stress, with sex steroids shaping these patterns across development and adulthood (Handa et al., 2022). In line with this, imaging studies report stressor-specific sex differences in amygdala–prefrontal coupling and broader tendencies for men to engage prefrontal control circuits and women limbic/striatal circuits during stress, with opposite associations to subjective reactivity and regulation (Bürger et al., 2023; Goldfarb et al., 2019). Interpersonal context also modulates physiology in sex-specific ways: during standardised psychosocial stress, brief physical partner contact attenuates women's cortisol and heart-rate responses, whereas verbal support alone does not (Ditzen et al., 2007). Because AL is dynamic and multisystemic, analyses of stress-related disparities should model these moderators explicitly rather than assume uniform effects (Volarić et al,. 2024).

Low-SES populations face compounded risks because socioeconomic disadvantage both amplifies exposure to chronic stressors and constrains access to protective resources (Tyrell et al., 2023). The demonstration of early physiological dysregulation in low-income children underscores the urgency of interventions that target stress physiology, emotion regulation, and environmental stability in childhood (Tyrell et al., 2023). More broadly, enduring health disparities across socioeconomic gradients reflect SES as a structural driver of outcomes through intertwined biological and psychosocial pathways (Ibáñez et al., 2023; Marmot, 2005). Within this dynamic, AL emerges as a critical target for intervention because it mediates links between socioeconomic adversity and declines in metabolic, cardiovascular, and immune function (Durán et al., 2024; Juster et al., 2010; Seeman et al., 2004; Szanton et al., 2005).

Finally, resources that buffer stress appear context-sensitive. Using biomarker data from 2,096 adults, Podber and Gruenewald (2023) tested whether the frequency of positive life experiences (PLEs) mediates links between cumulative SES and AL, and whether effects vary by SES. They observed a small indirect effect of PLEs at the population level, but moderation analyses showed that PLEs predicted lower AL among individuals with lower SES, not among those with average or higher SES, yielding a moderated-mediation pattern. Supplementary analyses indicated unique contributions of childhood and adult SES to both PLEs and AL, with cardiovascular, lipid–metabolic, glucose, and inflammatory subsystems driving the conditional indirect effects (Podber and Gruenewald, 2023).

Together, these findings position AL as a central mechanism through which socioeconomic disadvantage, chronic stress, and neurobiological vulnerability jointly shape brain–mind health disparities, while highlighting that buffers such as PLEs are most protective under conditions of disadvantage (Cohen et al., 2012; Durán et al., 2024; Ibáñez et al., 2023; Podber and Gruenewald, 2023; Rojas-Thomas et al., 2023). Even so, AL is only one dimension of the broader puzzle that includes cognitive reserve and neurodegenerative risk (Hackman et al., 2010; Noble et al., 2012; Zannas et al., 2015). Accordingly, emerging work is widening the lens to multisystem determinants, such as the microbiota–gut–brain axis, advancing integrative, systems-level approaches to preventive neuroscience and precision public health (Molinuevo et al., 2022).

Microbiome, brain–mind health and socioeconomic disparities

Social and economic inequities increase the likelihood of poor diet quality, which in turn undermines BMH. One explanatory pathway is the influence of the intestinal microbiota on the central nervous system (CNS) (De Sequeira et al., 2022). Extending the earlier "gut–brain axis", Cryan et al. (2019) formalised the MGBA to denote bidirectional communication between gut microbes and the brain. Beyond core roles in metabolism and immunity, the microbiota signals to the brain via endocrine, immune, neural, and metabolic routes (e.g., microbially derived metabolites), ultimately modulating neuronal and glial function (Chang et al., 2022; Cryan et al., 2019; De Sequeira et al., 2022).

Mechanistically, converging evidence shows that gut-derived cues shape brain function through coordinated immune, neural, endocrine, and circulatory pathways (Loh et al., 2024). Microbiota-produced metabolites (notably short-chain fatty acids), microbially influenced neurotransmitters and gut hormones, and cytokine cascades regulate glial physiology (microglia, astrocytes, oligodendrocytes) and synaptic plasticity; rapid afferent signals travel via the vagus and enteric nervous systems; and changes in intestinal and blood–brain barrier integrity alter central exposure to peripheral mediators (Loh et al., 2024). Together, these multiscale interactions offer a biologically plausible bridge from environmental/contextual adversity to neurobiological vulnerability and align with systems-level syntheses linking microbiota–brain mechanisms to cognition (Castells-Nobau et al., 2024).

Within this axis, host–microbe mutualism is fundamental: the intestine provides a niche for microbial growth, and microbes contribute to host homeostasis (Zhou and Foster, 2015). Because MGBA dynamics support both physical and mental dimensions of health, dysbiosis can impair axis function and contribute to adverse outcomes; alterations in microbial community composition and function have been linked to MGBA dysregulation (Foster and McVey Neufeld, 2013; Gomez-Eguilaz et al., 2019; Malan-Muller et al., 2018; Sampson et al., 2016; Sharon et al., 2016; Zhou and Foster, 2015).

Region-specific modulation further clarifies how microbial signals intersect with social cognition. A recent review maps microbiota-sensitive circuits (including prefrontal–limbic, insular, and hippocampal networks) implicated in empathy, theory of mind, and social decision-making, drawing on translational models and emerging human neuroimaging (Sharvin et al., 2023). Complementarily, the microbiota regulates stress responsivity via the circadian system: microbial depletion perturbs suprachiasmatic clock-gene rhythmicity, shifts diurnal glucocorticoid profiles, and drives time-of-day–specific stress phenotypes, with oscillatory taxa (e.g., Lactobacillus/Limosilactobacillus reuteri) highlighted as candidates (Leone et al., 2015; Thaiss et al., 2016). These region- and rhythm-level mechanisms situate MGBA pathways within the CP-MInD framework that links diet, stress biology, and social-cognitive outcomes, particularly under low-SES conditions.

Dietary transitions magnify these issues. Globally, many low- and middle-income settings are experiencing rapid growth in ultra-processed food (UPF) availability and intake (including among infants and toddlers) alongside slower declines in stunting and faster rises in overweight/obesity, with the steepest increases now concentrated in poorer households; early exposure appears to shape long-term taste preferences toward high-sugar/sodium products (Popkin and Laar, 2025). Quantitative syntheses connect higher UPF consumption with adverse mental-health outcomes: a *meta*-analysis of 17 observational studies (~385,000 participants) associated greater UPF intake with higher odds of depressive and anxiety symptoms and increased prospective risk of depression, with directionally consistent effects across populations (Lane et al., 2022). An umbrella review spanning ~ 9.9 million participants rated evidence linking UPF exposure to common mental disorders and anxiety as "convincing" or "highly suggestive" and outlined plausible mechanisms (including poor nutrient profiles, additives, and processing effects) that implicate inflammation and the microbiome (Lane et al., 2024). While observational designs limit causal inference, these results reinforce a coherent behavioural–biological pathway by which high-sugar/UPF patterns can erode microbial diversity and barrier function, amplify inflammation, and adversely affect BMH.

Stress states also reshape the gut ecosystem. Stress hormones and sympathetic neurotransmitters alter gastrointestinal physiology and, by extension, the microbial habitat; preclinical data link such stress-related dysbiosis to anxiety- and stress-like behaviors, providing a bidirectional account of stress–microbiome–brain interactions (Bauer et al., 2022; Foster and McVey Neufeld, 2013; Gomez-Eguilaz et al., 2019; Luna and Foster, 2014; Ma et al., 2021; Malan-Muller et al., 2018; Zhou and Foster, 2015).

Focusing specifically on anxiety, Ma et al. (2021) conducted a randomised, placebo-controlled trial in an adult population to evaluate the efficacy of *L. plantarum P-8* probiotics. Over 12 weeks, participants received either a daily oral supplement of *L. plantarum P-8* or a placebo. Their findings revealed that certain microorganisms, such as *Bifidobacterium adolescentis*, *B. longum*, and *Faecalibacterium prausnitzii*, increased in prevalence, while *Roseburia faecis* and *Fusicatenibacter saccharivorans* decreased significantly. These shifts point to a possible association between probiotic-induced modulation of the gut microbiota and reductions in stress and anxiety. The study also highlighted changes in the gut metagenome at the level of species-level genome bins (GBS) and functional genes (Ma et al., 2021).

In a broader review of microbiota and mental health, Järbrink-Sehgal and Andreasson (2020) identified eight studies published between 2018 and 2019 linking stress and anxiety to microbiota composition in adults. One randomised controlled trial they reference examined long-term use of *Lactobacillus gasseri* and found improvements in mental state and sleep quality and better maintenance of intestinal microbiota under stress conditions. Specifically, the intervention appeared to counteract the decline in *Bifidobacterium spp* and increased *Streptococcus spp* (Järbrink-Sehgal and Andreasson, 2020).

The role of *Bifidobacterium longum* in social stress has also been explored in a double-blind, placebo-controlled trial by Wang et al. (2019). Using the cyberball paradigm, they demonstrated that *Bifidobacterium* influences both resting neural dynamics and stress-related neural responses involved in regulating negative emotions (Wang et al., 2019). This aligns with broader evidence indicating that psychological, social, and chronic stress can all affect the gut microbiota (Bailey et al., 2010; Molina-Torres et al., 2019). Furthermore, both animal experiments (De Sequeira et al., 2022; Golubeva et al., 2015; Monnikes et al., 1992; Murakami et al., 2017) and preliminary human studies suggest that stress-related alterations, especially involving *Lactobacillus*, *Bacteroides*, and *Clostridium*, may begin as early as pregnancy due to prenatal stress.

Critically, the microbiota plays a key role in programming the HPA axis during the first years of life, thereby exerting a lifelong influence on stress reactivity (Malan-Muller et al., 2018; Papalini et al., 2018; Zhou and Foster, 2015). One preliminary human study specifically showed a relationship between the gut microbiota and the acute glucocorticoid response to stress in a clinical sample (Hantsoo et al., 2018).

Research on populations exposed to highly stressful events has also revealed characteristic longitudinal changes in the intestinal microbiota linked to mental health. In particular, *Faecalibacterium*, commonly found in the colon of healthy individuals, and *Eubacterium* eligens, noted for its strong anti-inflammatory activity, showed significant reductions in stressed populations (Chung et al., 2017; Gao et al., 2022).

Moreover, stress disrupts the integrity of the intestinal barrier and increases its permeability. This allows bacteria to migrate through the intestinal mucosa and directly interact with immune cells and neuronal cells within the nervous system (Gao et al., 2022; Molina-Torres et al., 2019). At the same time, stress activates the autonomic nervous system, which affects the secretion of gastric acid, bile, mucus, and intestinal motility, factors strongly tied to the composition and richness of the gut microbiota (Malan-Muller et al., 2018).

Such disruptions to the microbial community are not only associated with gastrointestinal disorders like irritable bowel syndrome, which can contribute to impaired immune function, neurodevelopment, and behavioural issues, but also with stress itself (Chang et al., 2022; Gao et al., 2022; Molina-Torres et al., 2019). In a recent study of post-traumatic stress among healthcare workers, Gao et al. (2022) observed a sustained decrease in alpha diversity (i.e., reduced bacterial variety) in the microbiota of these individuals, a pattern seen in multiple disease states.

Psychiatric disorders, including major depressive disorder and generalised anxiety disorder, have been associated with specific alterations in the gut microbiota (Gao et al., 2022; Huang et al., 2018). For instance, *Ruminococcus gnavus*, a bacterium linked to Crohn's disease, has been tied to diminished sleep quality and increased intestinal permeability. Meanwhile, *Lachnospiraceae* and *Roseburia* species have been implicated in both depression and post-traumatic stress (Bajaj et al., 2019; Gao et al., 2022).

Building on this connection between gut health and mental well-being, Papalini et al. (2018) used a randomised, double-masked longitudinal design to assess the neurocognitive effects of probiotics in healthy women under stress. Their findings suggest that these probiotics can mitigate the negative impact of stress on cognitive functioning, but only in stressful conditions, no benefit was observed under neutral conditions (Papalini et al., 2018). Beyond stress and anxiety, growing evidence points to associations between the microbiota and depression (Chang et al., 2022; Dash et al., 2014; Foster and McVey Neufeld, 2013; Gao et al., 2022; Huang et al., 2018; Zhou and Foster, 2015).

In individuals diagnosed with depressive disorders, fecal analyses have revealed notable correlations between altered gut microbiota profiles and the presence of depression. For example, an overall underrepresentation of *Bacteroidetes* (particularly *Alistipes*) was observed in those with depression (Zhou and Foster, 2015). Furthermore, as Foster and McVey (2013) describe in their review, changes in gut microbiota composition modulate neural signalling systems related to plasticity, as well as serotonergic and GABAergic pathways in the central nervous system (Foster and McVey Neufeld, 2013).

Social cognition and brain–mind health in socioeconomic disparities

Socioeconomically vulnerable populations consistently exhibit higher rates of mental health issues (Foubert et al., 2021; Lorant et al., 2003; McLaughlin et al., 2012b; Zhu et al., 2019), elevated stress levels (Farah, 2017), and compromised socio-cognitive processes (Kraus et al., 2010; Migeot et al., 2022; Salas et al., 2021; Stellar et al., 2012; Varnum et al., 2015). Although the theoretical framework explaining these connections is still evolving, current evidence points to SES as a key risk factor for depression (Lorant et al., 2003). Similar findings extend to children: parental stress can mediate the link between SES and depressive symptoms (Nagy et al., 2020), while parental anxiety levels have been shown to mediate the relationship between SES and childhood anxiety, ultimately affecting the child's morning cortisol response and reflecting the functionality of the HPA axis (Zhu et al., 2019).

From a cross-sectional standpoint, adults of lower SES often present with higher levels of depression than their higher-SES counterparts, primarily driven by exposure to stressors (Foubert et al., 2021). Notably, subjective SES (i.e., how individuals perceive their own socioeconomic standing) has also been linked to mood disorders, anxiety, substance abuse, and disruptive behaviours (McLaughlin et al., 2012b), suggesting that both objective and subjective dimensions of SES play critical roles in mental health outcomes. Moreover, lower SES is correlated with heightened chronic stress (McEwen and Gianaros, 2010) and is frequently identified as a potent stressor an individual can face (Farah, 2017). This stress has cascading effects on biological, psychological, social, and cognitive domains (Crielaard et al., 2021; Rimmele et al., 2022; Rojas-Thomas et al., 2023). For example, individuals experiencing financial hardship (Ankuda et al., 2021; Chen et al., 2022) show impairments in cognitive functions such as memory, executive functions, and socio-cognitive abilities like emotional processing and social behaviour. Corresponding alterations have also been observed in brain regions linked to these functions, including the amygdala, hippocampus, and prefrontal cortex, critical areas in social adequacy (Kim et al., 2013; Luby et al., 2013).

Large-scale and longitudinal work now links SES to differences in cortical development/structure and control networks, implicating weakened cognitive control in pathways of chronic stress and low-grade inflammation (Merz et al., 2023; McGlinchey et al., 2024; Baysarowich et al., 2025). Converging findings indicate that socioeconomic disadvantage shapes social cognition not only through contextual contingencies but also via biological routes that compromise frontoparietal and frontolimbic circuits supporting inhibition, working memory, emotion regulation, mentalizing, and social decision-making (Noble et al., 2015; Merz et al., 2023; Baysarowich et al., 2025). In children, lower SES is associated with altered amygdala–prefrontal connectivity via stress biology, specifically elevated cortisol, implicating this pathway in emotion regulation and recognition (Tian et al., 2021). In adults, SES–brain associations span structural, functional, and biomarker differences; importantly, what is detectable and how it is interpreted is conditioned by regional constraints in biomarker access and cross-cultural harmonization, issues underscored for the Global South, where infrastructure, funding, and tool availability remain uneven (McGlinchey et al., 2024). Mechanistically, chronic stress and inflammation appear to preferentially degrade dorsolateral prefrontal cortex circuitry that supports cognitive control, providing a biologically grounded route from socioeconomic adversity to executive dysfunction (Noble et al., 2015; Ren et al., 2023). Complementing this, recent syntheses of SES–brain development highlight heterogeneous, often nonlinear patterns (mixtures of deficit, adaptation, and resilience across tasks and developmental windows) rather than a single uniform effect (Baysarowich et al., 2025). Empirically, SES disparities have also been tied to differences in HPA-axis regulation and prefrontal structure, underscoring stress-sensitive pathways that connect context with neural health (Merz et al., 2023).

Against this backdrop, the social-cognition literature remains multifactorial: variability in SES indicators (income, education, neighborhood), task demands (e.g., affect recognition vs. theory of mind), and moderators such as stress physiology and comorbidity likely contributes to mixed findings, including reports of both enhanced sociocognitive capacities in some low-SES adults and age-related decrements in others (Carriedo et al., 2024; Kraus et al., 2010; Migeot et al., 2022; Salas et al., 2021; Stellar et al., 2012; Varnum et al., 2015). Overall, SES relates to social cognition through stress- and inflammation-sensitive control circuits, with observed effects conditioned by measurement context, regional research capacity, and stage of the life course (McGlinchey et al., 2024; Merz et al., 2023; Baysarowich et al., 2025). In the next section, we examine how these contextual, biological, and psychological factors intersect to clarify SES–social cognition links and to inform targets for intervention.

Low-SES, allostatic load and microbiota in social cognition

Individuals in socioeconomically vulnerable contexts face multiple barriers to developing and sustaining social-cognitive skills, and diet is a key, modifiable pathway linking context to brain–mind health. Beyond evidence that specific nutrients (e.g., tryptophan) can acutely modulate empathy and emotion recognition (Reuter et al., 2020), large syntheses show that high intakes of free/added sugars and sugar-sweetened beverages are broadly harmful and associated with neuropsychiatric outcomes; current guidance recommends keeping free/added sugars below 25 g per day and sugar-sweetened beverages to fewer than one serving per week (Huang et al., 2023). Complementing this guidance, a recent systematic review and *meta*-analysis pooling 40 studies ($n \approx 1.21$ million) found that higher total dietary sugar intake was associated with a 21 % greater odds of depression; the association for anxiety was not significant overall, with stronger effects in women and in studies using food-frequency questionnaires (Xiong et al., 2024).

In parallel, global diets have shifted toward ultra-processed foods (UPFs), with rapid increases documented among infants and toddlers in many low- and middle-income countries, patterns that may entrench early taste preferences and accelerate obesity risk, particularly in poorer households (Popkin and Laar, 2025). Meta-analytic evidence links higher UPF consumption to greater odds of depressive and anxiety symptoms and to increased prospective risk of depression (Lane et al., 2022), and an umbrella review identifies convincing or highly suggestive associations between UPF exposure and adverse cardiometabolic and mental-health outcomes (Lane et al., 2024). Together, these findings support a plausible behavioral–biological pathway whereby diets high in added sugars and UPFs erode gut-microbiome diversity and quality, amplify stress- and inflammation-related processes that contribute to allostatic load, and, in turn, hinder social–emotional functioning, especially where access to nutrition education and minimally processed, nutrient-dense foods is limited.

Despite this evidence, much mechanistic work remains preclinical. Rodent studies show that altering microbiota–gut–brain pathways can produce social behavior deficits and that targeted microbial interventions can reverse them (Buffington et al., 2016; Degroote et al., 2016; Desbonnet et al., 2013; Golubeva et al., 2017; Leclercq et al., 2017). For example, a maternal high-fat diet in mice induces offspring social deficits that are microbiota-mediated; co-housing or fecal microbiota transfer rescues behavior, and reconstitution with Lactobacillus reuteri restores oxytocin levels, ventral tegmental area (VTA) synaptic plasticity, and social behaviors (Buffington et al., 2016). These experiments establish a causal link in mice between maternal diet, microbial imbalance, VTA plasticity, and social behavior. In humans, related associations are consistent but remain non-causal; stronger inference will require quasi-experimental or interventional designs that integrate microbial, endocrine, immune, and neural measures (Russo and Williamson, 2007).

Translational work indicates that perturbing microbiota–gut–brain signaling affects the prefrontal cortex, amygdala, hippocampus, hypothalamus, and striatum through vagal, immune, endocrine, and metabolite/neurotransmitter pathways; converging human neuroimaging studies link microbial variation to differences in regional structure and functional connectivity relevant to affect regulation, mentalizing, and decision-making (Sharvin et al., 2023). In complementary experimental research, the gut microbiota regulates stress responsivity via the circadian system: microbial depletion disrupts core clock-gene rhythmicity in the suprachiasmatic nucleus, shifts diurnal glucocorticoid (corticosterone) rhythms, alters stress-pathway rhythmicity in the hippocampus and amygdala, and produces time-of-day–specific impairments in stress responses, with oscillations of taxa such as *Limosilactobacillus* reuteri emerging as candidate drivers (Tofani et al., 2024). Together, these region- and rhythm-level mechanisms outline biologically plausible bridges from context- and diet-driven microbial shifts to stress-sensitive control circuits that support empathy, altruism, and social decision-making.

Another approach has been to track the presence and concentration of bacteria in rat microbiota (Degroote et al., 2016; Desbonnet et al., 2013; Leclercq et al., 2017). Experiments using germ-free rats (Desbonnet et al., 2013) and antibiotic-treated rats (Degroote et al., 2016; Leclercq et al., 2017) demonstrate that the complete absence of gut microbiota leads to social impairments, including a lack of social interaction and diminished social preference. Intriguingly, administering certain *Lactobacillus* species, such as *L. reuteri* (Buffington et al., 2016) or *L. rhamnosus* (Golubeva et al., 2017), can reverse these social deficits in rats.

Although animal models have provided much of the available data, emerging human studies also underscore the role of the microbiome in socio-cognitive processes. For instance, women with higher concentrations of *Prevotella* bacteria tend to exhibit more inappropriate responses to affective imagery than those with lower levels (Barcik et al., 2021). Moreover, increased *Lactobacillus* spp. has been linked to lower affective empathy (Heym et al., 2019). According to the authors, this association may arise from the impact of *Lactobacillus* on brain regions, such as the prefrontal cortex and amygdala, that are integral to regulating and experiencing negative emotions.

An extensive review of how different microbiota microorganisms influence social cognition indicates that microbiota biodiversity shapes social-emotional skills by regulating the structures and dynamics of key brain regions involved in social behaviour, including the prefrontal cortex, orbitofrontal cortex, and amygdala (Sarkar et al., 2020). Additionally, it has been demonstrated that bacteria transferred during childbirth (primarily *Lactobacillus*) are critical for synaptogenesis and myelination in the frontal cortex during the first years of life (Sharon et al., 2016; Strandwitz et al., 2019). In adults, one study found that a higher abundance of *Prevotella* and *Bifidobacterium* correlated with stronger connectivity in frontoparietal circuits (Kohn et al., 2021), a key network in social behaviour. Given that prefrontal areas are associated with numerous sociocognitive functions, such as altruistic behaviour (Waytz et al., 2012), empathy (Iacoboni, 2007), social learning (Olsson et al., 2020), moral reasoning (Forbes and Grafman, 2010), social networks (Noonan et al., 2018), and decision-making (Aoi et al., 2020), these findings underscore the critical role of the microbiome in shaping social behaviour across developmental stages.

However, no studies to date have directly examined how human gut microbiota affects these aspects of social cognition and how their brain correlates with socioeconomically vulnerable populations, a significant challenge that remains unresolved. Another region seemingly influenced by gut bacteria is the amygdala (Fernandez-Real et al., 2015). A functional magnetic resonance imaging study showed that individuals who ingested probiotics (*Bifidobacterium animalis*, *Streptococcus thermophilus*, *Lactobacillus bulgaricus*, and *Lactococcus lactis*) exhibited reduced amygdala BOLD activity during an emotional attention task compared to those in a control group (Tillisch et al., 2013).

Further research has demonstrated that microbiota-derived metabolites also appear to modulate amygdala connectivity. In particular, 3-methylindole (skatole) is positively associated with anatomical connectivity between the amygdala and insula, while indoleacetic acid is positively linked to functional connectivity in this same circuit (Osadchiy et al., 2018). Given that the amygdala is a key structure in socio-affective processing and other socio-perceptual functions (Adolphs, 2003, 2010), these findings highlight a potential mechanism by which intestinal bacteria may influence social cognition.

Despite increasing evidence of the gut microbiota's influence on human social cognition, relatively few studies have directly investigated the microbiota–brain–sociocognitive interface. Consequently, critical questions remain about how shifts in microbiota composition and diversity may shape, or be shaped by, social behaviours and socio-emotional processes. While preliminary findings suggest that microbiota biodiversity plays a vital role in social cognition and socio-affective processing, further research is needed to clarify the underlying mechanisms, particularly involving vulnerable populations. The following section will build on these ideas by exploring the broader neural underpinnings of social behaviour and discussing their implications for overall mental well-being.

The social brain and brain–mind health

A growing *meta*-analytic and longitudinal literature links SES to differences in brain structure and functional organization that support social and emotional processing. A recent *meta*-analysis of structural MRI studies reports reliable SES-related differences in cortical and subcortical gray matter across regions implicated in cognition and affect (Qiu et al., 2025). Converging longitudinal evidence shows that lower preschool SES predicts smaller cortical and subcortical volumes and a shallower developmental slope of subcortical gray matter through adolescence; these trajectories partially mediate links from early SES to later cognitive function and risk behaviors. Regionally, lower early SES relates to reduced volumes in the hippocampus, caudate, putamen, and thalamus (with less consistent effects for the amygdala), and these findings persist after adjustment for early psychopathology, cumulative life events, and maternal mental health (Barch et al., 2021). Beyond gross morphology, large-sample work points to SES-related variation in frontoparietal, salience, and default-mode networks that scaffold executive and socio-affective functions (Merz et al., 2023). Global syntheses further note that measurement and access constraints, especially across the Global South, shape both detectability and interpretation of SES–-brain relations, and that effects are heterogeneous and often nonlinear, reflecting mixtures of deficit, adaptation, and resilience across tasks and developmental windows (Baysarowich et al., 2025; McGlinchey et al., 2024).

Within this neurobiological context, social cognition refers to the processes that allow people to perceive, interpret, and respond to others (Carriedo et al., 2024; Iacoboni, 2005). Core components include empathy (affective and cognitive; Decety and Jackson, 2004; Tousignant et al., 2017), altruism (incurring a personal cost to benefit others; Yang and Li, 2025), theory of mind (ToM; representing others' beliefs, intentions, and feelings; Beaudoin et al., 2020), and social decision-making (weighing one's own and others' payoffs under uncertainty and social norms; Falkenstein et al., 2024).

Against this backdrop, we foreground three SES-sensitive constructs (altruism, ToM, and social decision-making) and link them to their neural substrates. Altruism engages a distributed network spanning ventromedial/dorsomedial prefrontal cortex, temporoparietal junction, and striatum; *meta*-analytic work differentiates nodes supporting empathic concern from those mediating value computation during prosocial choice (Yang and Li, 2025). Context matters: in some settings greater altruism covaries with negative affect and anxiety (Feng et al., 2020), particularly during prolonged stressors such as the COVID-19 pandemic (Morales et al., 2024), whereas in other contexts altruistic behavior is protective and associated with higher well-being (Giovanis and Ozdamar, 2022). Theory of mind relies on the medial prefrontal cortex, temporoparietal junction, and posterior superior temporal sulcus, and is best treated as multidimensional (e.g., belief reasoning vs. affective inferences) with heterogeneous task demands. Comprehensive reviews highlight notable psychometric challenges, especially in younger samples (Beaudoin et al., 2020). Charness et al. (2019) show that subtle social-salience cues improve ToM performance among low-income children, suggesting early ToM gaps are malleable via low-cost interventions. They also find that higher ToM predicts more accurate belief-based reasoning, better strategic coordination, and greater prosociality (even controlling for age and basic cognitive skills), underscoring ToM's ecological relevance in low-resource contexts. Clinically, individuals with depression frequently perform worse on ToM tasks, underscoring links between social-cognitive processes and affective symptoms (Pentaraki, 2017; Wang et al., 2008). Social decision-making integrates valuation and control under social uncertainty. Experimental evidence suggests that time pressure and information load selectively tax control networks during social choices, constraints that are likely exacerbated in disadvantaged contexts (Falkenstein et al., 2024). Decision style also matters: avoidant tendencies are associated with poorer mental-health indicators (Bavol'ár and Orosová, 2015), illustrating multiple routes by which social cognition intersects with psychological well-being (Carriedo et al., 2024; Franco-O'Byrne et al., 2023a,b). Complementing these mechanistic accounts, SES-linked cues of scarcity, instability, and low status can adaptively bias choices toward immediacy, altering self-regulation and cognitive priorities in ways that are locally rational yet potentially costly for longer-term goals (Sheehy-Skeffington, 2020).

These social-cognitive functions are supported by a distributed "social brain" including prefrontal and orbitofrontal cortices, anterior cingulate and insula, temporoparietal junction, and limbic structures such as the amygdala (Allain et al., 2019; Arioli et al., 2021; Blakemore and Choudhury, 2006; Evans et al., 2015; Kelly et al., 2017; Lee et al., 2004; Van Overwalle, 2009). Consistent with SES-related differences in control networks, recent work shows that chronic stress and low-grade inflammation can preferentially weaken dorsolateral prefrontal circuitry central to cognitive control, a pathway with downstream consequences for emotion regulation, mentalizing, and value-based choice (Noble et al., 2015).

Electroencephalography (EEG) complements the structural and network evidence by indexing SES-related differences in neural processing. Compared with higher-SES peers, individuals from lower-SES backgrounds often show attenuated modulation of classic event-related potentials (P1, N1, N2, ERN, N400, and P3) components linked to early visual attention, selective attention, conflict monitoring, error processing, semantic integration, and outcome evaluation (Katus et al., 2020; Perera et al., 2021). Many of these same signatures are reliably elicited in social-cognition tasks (e.g., empathy, mentalizing, value-based choice), including P1/P3/N1/N2 and the late positive potential (LPP), which jointly track affective arousal and learning-related cognitive operations (Billeke et al., 2013; Chen et al., 2020; Coll, 2018; Duan et al., 2021; Galang et al., 2020; Mercedes Perez-Rodriguez et al., 2015; Song et al., 2019; Weightman et al., 2014). Consistent with their clinical relevance, lower perspective-taking and empathic concern correlate with depressive symptoms (Huang et al., 2020).

Altruism engages valuation and control networks (vmPFC, striatum, dlPFC) together with mentalizing hubs (TPJ); *meta*-analytic work shows that contextual moderators (e.g., cost, social distance) systematically shape effect sizes, mechanisms plausibly sensitive to SES-linked stress burden and control-network differences (Yang and Li, 2025). ToM draws on a partially overlapping network (mPFC, TPJ, precuneus), is multidimensional, and faces well-documented measurement challenges, particularly in development (Beaudoin et al., 2020). ToM capacity predicts cooperative and fair behaviour in economic games (Charness et al., 2019), offering a bridge to social decision-making, where scarcity and instability can adaptively bias choices toward immediacy (Sheehy-Skeffington, 2020) and where group-norm processes bear on equity-relevant outcomes (Falkenstein et al., 2024). Clinically, perturbations in perspective-taking/ToM and empathic concern are associated with depressive symptoms, and ToM deficits are frequently observed in depression (Huang et al., 2020; Pentaraki, 2017; Wang et al., 2008).

Lower SES is often associated with poorer performance across socio-cognitive domains (Migeot et al., 2022), underscoring environmental contributions. Although links between social cognition and mental health are well supported, whether social-cognitive processes and their neural correlates mediate the SES–mental health relationship remains an open question, and a priority for future longitudinal and mechanistic research.

Conclusions and limitations

Conclusions

SED is consistently associated with differences in brain–mind health. Individuals from lower-SES backgrounds, on average, show reduced grey-matter volume and altered large-scale network organisation in regions supporting executive and socio-affective functions, patterns plausibly shaped by cumulative exposures such as diet quality, educational opportunity, neighbourhood safety, and chronic stress/inflammation. Complementary EEG evidence of diminished modulation in components such as P1, N1, N2, ERN, N400, and P3 aligns with disruptions to attention, conflict monitoring, semantic integration, and outcome evaluation that are relevant to social cognition. Framing these neural differences within core social-cognitive constructs (empathy, altruism, theory of mind, and social decision-making) clarifies links to mental-health outcomes. Taken together, the literature indicates that SES shapes socio-cognitive capacities in ways that affect emotional well-being, even as some mediating neural and physiological pathways remain to be fully specified. The CP-MInD framework integrates contextual stressors with physiological markers (particularly allostatic processes and the MGBA) to organise evidence across behaviour, neural correlates, and systemic physiology, and to motivate multidomain strategies that target both structural determinants and biological pathways.

Limitations

Several constraints temper these conclusions and guide priorities for future work. First, microbiome research remains disproportionately concentrated in high-income settings, which limits generalizability and risks obscuring context-specific dietary, infectious, and environmental exposures in low- and middle-income regions; recent overviews detail the sampling biases, platform heterogeneity, and under-representation that complicate cross-study aggregation (Arif et al., 2025; Andreu-Sánchez et al., 2025). Second, although preclinical work has illuminated candidate mechanisms, species differences, inflated effects, and ecological validity limits complicate translation to population health; state-of-the-art reviews outline the methodological standards and cross-species alignment needed to strengthen inference (Metwaly et al., 2025). Third, early human intervention evidence is promising but sensitive to how outcomes are measured; for example, a randomized, double-blind trial found that a multispecies probiotic reduced negative mood when assessed with high-frequency daily ratings, whereas conventional pre/post assessments missed change, underscoring the importance of measurement resolution and the choice of endpoints in MGBA-targeted trials (Johnson and Steenbergen, 2025). Finally, heterogeneity in the definition and measurement of socioeconomic status (spanning income, education, occupation, neighbourhood indices, and subjective SES) impedes synthesis and weakens causal inference; a recent critical review highlights the lack of clear definitions, insufficient variable reporting, and limited measures that hamper comparability across studies (Zaneva et al., 2024). Additional design issues, predominantly cross-sectional samples, residual confounding (e.g., comorbidities, medications, diet, sleep), publication bias, and outcome heterogeneity across social-cognition tasks and neural endpoints, can mask age, sex/gender, and cultural moderation and inflate apparent effects.

Future directions

Progress will depend on broadening representation and tightening methods. Representative, longitudinal cohorts that include low-/upper-middle-income countries (LMIC/UMIC) sites and employ harmonized CP-MInD protocols for diet, infection, environmental toxicants, and social adversity are essential to close global evidence gaps and enable valid cross-site comparisons (Arif et al., 2025; Andreu-Sánchez et al., 2025). Translational pipelines should pair preclinical MGBA/allostasis models with human experimental medicine (challenge paradigms, mechanistic RCTs, and validated biomarkers) to bridge bench-to-bedside gaps, with preregistration and adequate power for mechanistic endpoints (Metwaly et al., 2025; Johnson and Steenbergen, 2025). In parallel, rigorously controlled trials of psychobiotics/probiotics and dietary interventions that target MGBA pathways should adopt theory-driven, preregistered outcomes spanning mood, stress physiology, and social cognition, and incorporate stratification by baseline symptoms, diet, and microbiome features to identify likely responders (Kamal et al., 2025; Johnson and Steenbergen, 2025). Field-wide adoption of consensus SES batteries that combine objective and subjective indicators and capture life-course trajectories, alongside transparent reporting of SES construction and level (individual vs. neighborhood), will improve comparability and strengthen causal modeling (Zaneva et al., 2024). Finally, studies should integrate multimodal methods (neuroimaging, endocrine/immune panels, microbiome multi-omics, digital phenotyping, and validated social-cognition tasks) within developmental and intersectional designs to identify who benefits most from which interventions; pairing individual-level supports (stress reduction, sleep, nutrition) with structural policies (income support, housing, education access) offers the best prospect for durable, population-level gains in brain–mind health.

CRediT authorship contribution statement

Juan Pablo Morales: Conceptualization, Investigation, Methodology, Project administration, Supervision, Visualization, Writing – original draft, Writing – review & editing. **Fiorella Macchiavello:** Writing – review & editing, Writing – original draft, Methodology, Conceptualization. **Felipe Rojas-Thomas:** Writing – review & editing, Writing –

original draft, Resources, Methodology, Investigation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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