



Continuity versus change in latent profiles of emotion regulation and working memory during adolescence[☆]

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ARTICLE INFO

Keywords:

Adolescence
Emotion regulation
Working memory
Latent Transition Analysis

ABSTRACT

Significant structural and functional brain development occurs during early adolescence. These changes underlie developments in central neurocognitive processes such as working memory (WM) and emotion regulation (ER). The preponderance of studies modeling trajectories of adolescent brain development use variable-centered approaches, omitting attention to individual differences that may undergird neurobiological embedding of early life stress and attendant psychopathology. This preregistered, data-driven study used latent transition analysis (LTA) to identify (1) latent profiles of neural function during a WM and implicit ER task, (2) transitions in profiles across 24 months, and (3) associations between transitions, parental support, and subsequent psychopathology. Using two waves of data from the ABCD Study (Mage T1 = 10; Mage T2 = 12), we found three unique profiles of neural function at both T1 and T2. The Typical, Emotion Hypo-response, and Emotion-Hyper response profiles were characterized by, respectively: moderate amygdala activation and fusiform deactivation; high ACC, fusiform, and insula deactivation; and high amygdala, ACC, and insula response to ER. While 69.5 % remained in the Typical profile from T1 to T2, 27.8 % of the sample moved from one profile at T1 to another at T2. However, neither latent profiles nor transitions exhibited associations between parental support or psychopathology symptoms.

1. Introduction

Throughout late childhood and adolescence, emotion regulation (ER) and working memory (WM) undergo significant changes, matched by underlying structural and functional developments in the brain. Although developmental science has documented normative adolescent brain development trajectories, the focus on average change may obscure inter-individual differences and intra-individual change in brain development. Indeed, adolescents' brain development varies (Foulkes and Blakemore, 2018), particularly in response to risky and promotive environmental inputs. For example, positive and supportive parenting behaviors are critical in shaping children's neurocognitive development and resulting WM and ER capacity (Borelli et al., 2021; Clark and Frick, 2018; Deane et al., 2020; Oshri et al., 2021; Schroeder and Kelley, 2010; Whittle et al., 2016). Moreover, variability of brain development underlies neurobiological vulnerabilities and attendant risk for the development of psychopathology (Beauchaine and McNulty, 2013). This

preregistered, data-driven study aimed to: 1) derive latent profiles of neural function during working memory and implicit emotion processing task in a priori ROIs, 2) identify latent statuses of neural function across 24 months (Mage, baseline = 11, Mage, T2 = 13), and 3) explore parental support and demographic covariates as predictors of statuses, and 4) evaluate between-status differences in the development of psychopathology.

1.1. Working memory and emotion regulation during adolescence

Adolescence comprises a period of significant neurocognitive growth. Early maturation of motivation and reward circuitry (e.g., ventral striatum, medial frontal and orbitofrontal cortices) paired with more protracted maturation of cognitive control systems (e.g., lateral prefrontal, parietal, and anterior cingulate cortices) precipitates greater attendance to and salience of emotional information (Casey et al., 2008; Pfeifer and Allen, 2012; Shulman et al., 2016; Steinberg, 2017).

[☆] The data that will be used for this study are available from the NIMH Data Archive (NDA). Restrictions apply to the availability of these data, which will be used under license for this study. Data are available at https://nda.nih.gov/edit_collection.html?id=2573 with the permission of the NDA.

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<https://doi.org/10.1016/j.dcn.2022.101177>

Received 6 June 2022; Received in revised form 15 November 2022; Accepted 16 November 2022

Available online 19 November 2022

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However, these neurocognitive developments also underlie gradual increases in adolescents' capacity for EF, or goal-directed control of thought and behavior, and ER, or modulation of emotional reactions in order to accomplish goals. Despite these increases in EF and ER, a maturational imbalance can occur between quickly-developing motivational systems and slowly-developing EF/ER systems in adolescence. This imbalance, in turn, can predispose adolescents to inconsistent behavioral regulation and heightened vulnerability for psychopathology (Carlson and Zelazo, 2011; Fuhrmann et al., 2015; Luna et al., 2010).

Growing evidence suggests increasing differentiation of EF components by late childhood and early adolescence, necessitating research among adolescents that focuses on specific EF measures. Above and beyond other EF components, working memory (WM) – the process of maintaining and manipulating information for a short period of time in order to guide behavior (Baddeley, 1998) – is a central mechanism underlying self-regulation and consequent adaptation throughout adolescence (Huang-Pollock et al., 2017; Vuontela et al., 2013). However, day-to-day task demands rarely require “cold” WM, or that which occurs in isolation from emotion processing (Banich, 2009; Blair et al., 2007; Pessoa, 2008; Pessoa and Ungerleider, 2004). Given that emotion receives priority in neural processing of stimuli (Pessoa and Ungerleider, 2004), ER is critical to successful WM and overall EF (Banich et al., 2009; Levens and Phelps, 2008; Mikels et al., 2008). Although ER takes many forms, it can be generally categorized into implicit ER, which involves passive, automatic, and often unconscious processing of emotional information, and explicit ER, which involves conscious cognitive effort aimed at modifying the emotional response (Gyurak et al., 2011).

Paradigms such as the emotional N-back (EN-back) are designed to elicit brain function at the intersection of implicit ER and WM. The EN-Back task presents emotionally-salient stimuli (typically emotional faces) while prompting the subject to hold information in an active cognitive state for use in a working memory task (Rougier et al., 2005). Given that the EN-back requires management of automatic emotional responses at varying degrees of working memory load, it is also considered an implicit ER task. The confluence of WM and ER processes elicits increases in activity across multiple functional domains. EN-back studies in adolescence and adulthood reveal working memory-related activations within frontoparietal regions (e.g., middle and superior frontal gyrus, inferior parietal cortex) and deactivations in motivation-oriented regions such as the cingulate cortex and insula (Chaarani et al., 2021; Liu et al., 2021; Rosenberg et al., 2020; Vetter et al., 2017). Implicit emotion processing tasks such as the EN-back recruit areas underlying visual and somatosensory processing (primary and secondary somatosensory cortices, insula, supramarginal gyrus, and basal ganglia), executive control (medial orbitofrontal cortex, superior frontal gyrus, anterior cingulate, posterior cingulate, precuneus, and superior temporal sulcus), valuation and motivation (vmPFC, striatum), and memory (hippocampus, amygdala; Chaaya et al., 2018; Frank et al., 2014; Fusar-Poli et al., 2009; Ghashghaei et al., 2007; Haxby et al., 2000; Hiser and Koenigs, 2018; Kanwisher et al., 1997; Kohn et al., 2014; Kropf et al., 2018; Lindquist et al., 2012; Richler and Gauthier, 2014; Sel et al., 2014; Sergerie et al., 2008; Vuilleumier and Pourtois, 2007; Vetter et al., 2020).

Several studies have also examined the developmental trajectories of neural function underlying WM and ER throughout adolescence. According to a meta-analysis of 10 WM imaging studies among adolescents and young adults (ages 10–30, $n = 382$), WM-related function increased with age within the rostral middle frontal, precuneus, inferior parietal, and premotor cortices and decreased with age within the superior frontal, postcentral, and posterior cingulate cortices (Andre et al., 2016). However, in an accelerated longitudinal study of 8–30 year-olds, Simmonds et al. (2017) found decreases in the middle frontal cortex, anterior cingulate, insula, and basal ganglia, as well as increases in the primary visual, visual association, and inferior temporal cortices. These group-level changes were associated with improved WM performance

over time. Meta-analyses and reviews of emotion regulation studies among adolescents also reveal somewhat mixed findings. For example, in a review of 24 neuroimaging studies by Del Piero et al. (2016), changes in neural reactivity to emotion from childhood to adulthood were characterized by linear decreases in amygdala, insula, and fusiform gyrus response and increases in medial prefrontal/anterior cingulate response from childhood to early adulthood. Taken together, these findings suggest that neural specialization, and subsequent efficiency, during WM processing increases during adolescence, as reflected by decreased recruitment of the medial PFC (e.g., the middle frontal gyrus and anterior cingulate) and increased activation within visual cortices. On the other hand, neural function underlying ER is characterized by increasing prefrontal influence, as the medial PFC (particularly the anterior cingulate) increases in activity and regions underlying threat, motivation, and face processing (the amygdala, insula, and fusiform gyrus, respectively) decrease in activity.

Although these average group-level trends are significant, they are limited by their variable-centered methodology, which focuses on mean-level associations between variables (for example, average magnitude of brain function predicting average level of behavior). Person-centered methods, on the other hand, characterize heterogeneity between and within individuals by identifying subgroups of people based on their multivariate similarities (Howard and Hoffman, 2018; Muthén and Muthén, 2000). These approaches are warranted, as a growing body of evidence suggests high variability of structural brain development (e.g., cortical thickness and gray matter volume) across adolescence (Lebel and Beaulieu, 2011; Mills et al., 2021; Paus et al., 2008; Tamnes et al., 2013; Wierenga et al., 2014). Preliminary evidence from person-centered studies using fMRI, EEG, and neurocognitive tasks indicate substantial within- and between-person variability in neural function as well (Kjelkenes et al., 2022; Ordaz et al., 2013; Tang et al., 2018). Ordaz et al. (2017) used mean growth curve modeling to characterize high within-person variability in neural function underlying EF in an accelerated longitudinal sample of 123 participants ages 9–26. Using latent class growth analysis within a sample of 43 12–16-year-old females, Tang et al. (2017) detected several unique trajectories of frontal alpha symmetry, suggesting both intra- and inter-individual variability of functional neural risk for psychopathology (Coan and Allen, 2004). Finally, Kjelkenes et al. (2022) used a normative modeling framework to identify inter-individual deviations from the norm in neurocognitive ability among youths ages 12–16.

Latent profile analysis (LPA) and its longitudinal extension latent transition analysis (LTA) form another branch of person-centered methods that may be especially advantageous for examination of neural function over time (Bray et al., 2010; Collins and Lanza, 2009; Lanza et al., 2013). LPA is a dimension reduction technique that characterizes heterogeneity across multiple variables into unobserved homogenous subgroups at a single time point. LTA extends LPA across time by (1) characterizing latent profiles at multiple time points, and (2) estimating the probability of individual movement from one profile to another across time points. Unlike growth mixture modeling (GMM) and latent class growth analysis (LCGA), in which subgroups of individuals are characterized by their level and shape of change over time, LTA examines both between-person differences at static points and within-person continuity or discontinuity across time. As such, LTA has the potential to create a more comprehensive picture of brain function by characterizing inter-individual variability first at static points and then modeling development of intra-individual change (Bray et al., 2010).

LTA differs from other person-centered approaches in its capacity for multivariate modeling. Whereas LCGA/GMM models often encounter issues of convergence and under-identification when modeling trajectories of more than 4 variables at a time, LPA and LTA models allow for inclusion of multiple indicators (extant studies have included between 4 and 24; Scotto Rosato and Baer, 2012; Wurpts and Geiser, 2014) and typically improve in performance with increases in number of high-quality indicators (Wurpts and Geiser, 2014). This multivariate

capacity means that several brain regions may be included in an LTA model simultaneously, which allows for better characterization of brain-wide function and interaction between multiple regions during tasks. Given that WM and ER processes recruit numerous brain regions simultaneously (Ahmed et al., 2015; Andre et al., 2016), a method such as LTA may be ideal for modeling their neural underpinnings across time.

1.2. Parenting and neurocognitive development

Warm and supportive parenting behaviors are potent predictors of positive youth development, whereas a lack of parental support is tied to development of psychopathology in adolescence (Huffman and Oshri, 2022; Meeus, 2016; Oshri et al., 2021; Waller et al., 2013; Weitkamp and Seiffge-Krenke, 2019). This link between the parenting context and youth psychopathology is mediated by the development of central neurocognitive processes, namely ER and EF, that underlie behavioral adaptation (Butterfield et al., 2021; Reuben et al., 2016). Growing research suggests that parental support is central to the formation of effective emotion regulation (Kerr et al., 2019; Morris et al., 2017) and working memory (Hughes and Devine, 2019) at both the behavioral and neural levels. The parent-child relationship fosters development of emotion regulation abilities primarily through modeling, socialization, and family emotional climate. Parents can model for their children effective emotion processing and regulation; they can also teach their children how to manage their emotions via discussion, transmission of ER strategies, and encouragement (Meyer et al., 2014). Use of these strategies is linked to heightened ER abilities during late childhood and adolescence (Morelen et al., 2016; Morris et al., 2017). Similarly, the emotional climate of the family lays the foundation for attachment security between the parent and child, which is closely linked to development of emotion reactivity and regulation throughout the lifespan (Morris et al., 2017).

Above and beyond genetic influence, parental support and sensitivity to children's affective states is also a central predictor of child executive functioning, including working memory (Hughes and Devine, 2019; Lucassen et al., 2015; Towe-Goodman et al., 2014). Caregivers act as "external regulators" of their child's affect, especially during infancy and early childhood, which facilitates development of the child's self-regulation and executive function (Gunnar and Donzella, 2002). Moreover, consistent caregiver sensitivity and support allow for the child to interact with their immediate environment in a way that elicits positive, encouraging, and/or effective responses from the caregiver (Bernier et al., 2010), further promoting the internalizing of constructive self-regulatory strategies (Bernier et al., 2012). To this point, a recent meta-analysis spanning 2000–2016 confirmed consistent associations between positive parenting (characterized by warmth, responsiveness, and sensitivity) and overall executive function among children ages 0–8 (Valcan et al., 2018). Similarly, Sosis-Vasic et al. (2017) found that greater parental involvement was associated with improved executive functioning, including working memory, response inhibition, and cognitive flexibility, among both children and adolescents.

1.3. ER and WM as predictors of psychopathology

Disruptions in both ER and WM are strong predictors of psychopathology in adolescence and adulthood. Low WM capacity underlies lack of self-regulation (Huang-Pollock et al., 2017; Vuontela et al., 2013), and is often implicated in externalizing, internalizing, ADHD, and poor academic achievement (Ahmed et al., 2015; Beck et al., 2010; Cassidy et al., 2016; Matthews et al., 2008). ER is a similarly powerful risk factor for psychopathology. A child with low ER may be less able to modify their emotional response in the face of daily challenges and as a result is more likely to develop internalizing and externalizing behaviors throughout their lifespan (Aldao et al., 2016; Halligan et al., 2013; Kim-Spoon et al., 2013; Shapero et al., 2016; Sheppes et al., 2015).

A number of studies have examined interactions between EF/WM process and ER at the neural level. In two early studies, Gray and Braver (Gray and Braver, 2002) and Herrington et al. (Herrington et al., 2005) found that affective stimuli modulated activity in EF regions during a WM task: positive emotional face stimuli increased dlPFC activation, whereas negative emotional stimuli decreased dlPFC activation. In a meta-analysis of 33 fMRI studies, Schweizer et al. (2019) found that vlPFC, amygdala, temporal, and occipital activation increased during a WM task when visual stimuli were emotionally salient, indicating the greater cognitive (and thus metabolic) demand on EF regions in the context of emotion. When interference in EF by emotional information is excessive, problems of self-regulation often ensue (Mueller, 2011). Indeed, among those exhibiting psychopathology, affective information disrupts executive function more frequently than healthy individuals (Ochsner and Gross, 2005). However, no studies have examined the development of WM-ER interactions over time, nor during adolescence – a period in which developing cognition-emotion interactions exert a particularly salient influence on behavior and psychopathology (Luna et al., 2010; Paus et al., 2008).

1.4. The current study

The current study aimed to (1) derive latent profiles of neural function during working memory and implicit emotion processing task in a priori ROIs, (2) identify latent statuses of neural function across 24 months (M_{age} , baseline = 10, M_{age} , T2 = 12), and (3) explore parental support and demographic covariates as predictors of latent statuses, and (4) evaluate the differences in youth internalizing and externalizing symptoms across latent statuses.

Hypotheses. Due to the complexity of study aims, we summarize all research questions, hypotheses, and analytic plans in Table 1.

1a) We hypothesized that neural activation during working memory and emotion regulation (e.g., the 2 back vs. 0 back and Faces vs. Places conditions of the EN-back, respectively) would form homogenous subgroups, or latent profiles, characterized by distinct patterns of task-activated regional function within regions delineated in Table 2. We anticipated that each profile would diverge in levels of activation, especially within the amygdala and medial prefrontal regions. We also hypothesized at least one "low regulation" profile at baseline that evinces particularly high amygdala and/or low ACC activation during the ER and low rostral middle frontal activation during the WM condition. Additionally, we hypothesized a "high regulation" profile at baseline that shows high anterior cingulate activation during ER and visual cortex activation during WM. Although we originally proposed to include biological sex and age as indicators alongside relevant ROIs during the model building process (given their relevance to organization and function of neural circuits underlying WM and ER [Hill et al., 2014; Stevens and Hamann, 2012; Ullsperger and Nikolas, 2017; Zahn-Waxler et al., 2015]), inclusion in this way caused convergence issues (more details in Results section). As such, we included them as covariates (see number 3 below).

1b) Given the relatively short time frame between the two waves, we hypothesized the number of latent profiles yielded by the LPA to remain consistent from baseline to T2 (24 months after baseline).

2) We anticipated that most individuals in the sample would remain in or transition into the "high regulation" profile between baseline and T2. Conversely, we also hypothesized that very few youths would transition from a high to low regulation profile; rather, some youths who began in a low regulation profile would remain at T2.

3) Low parental emotional support and low family income would significantly affect LTA parameters by changing probability of latent statuses and/or decreasing probability for transition into in a high regulation class. We hypothesized that family history of mental illness would similarly impact LTA parameters, given the documented effect of parental symptoms on adolescent psychopathology (Schulz et al., 2021). Although we initially proposed to account for potential effects of

Table 1
Hypotheses and analytic plan.

Research question	Hypothesis	Analysis plan	Effect of interest	Threshold for determining support of hypothesis
Are there qualitatively distinct subgroups of adolescents who evince unique patterns of neural response underlying ER and WM?	Inter-individual (between-person) differences in neural response during an ER and WM task will be evident at both T1 and T2 ¹ within relevant ROIs ² . Optimal class solution will yield between 3 and 5 profiles. We hypothesize at least 1 "high regulation" profile (high ACC [ER] and rMFG/visual cortex [WM]) and 1 "low regulation" profile (high amygdala [ER], low ACC [ER], low rMFG [WM]).	We will use 2 separate LPAs to characterize homogenous subgroups of varying regional activation during the EN-back task 0- vs. 2-back (WM) and Faces vs. Places (ER) conditions. Indicators will include relevant ROIs (Table 2) as well as youth biological sex and age.	To determine optimal number of classes: BIC, A-BIC, class size; To identify characteristics of and differences between groups: probabilities of profile membership, item response probabilities ³ , size of each profile	Hypotheses will be confirmed if we identify an optimal profile solution of 3–5 profiles that show differences in mean ROI activation within each task condition.
Is there change between latent profiles across time? If so, what is the probability that an adolescent's distinct pattern of neural function underlying WM and ER will change between ages 10 and 12?	1) The LPA at T1 and T2 will show measurement invariance. 2) Probability of transitioning from one latent profile at T1 to another profile at T2 will range from low to moderate ($\tau = .2-.5$), with highest probabilities for transition observed among those moving into a "high regulation" profile.	We will conduct 1) repeated-measures LPA to determine measurement invariance, and 2) LTA to determine individual probability of transitioning from one latent profile at T1 to another 24 months later.	Difference in model fit of nested models using likelihood ratio difference test, BIC, a-BIC; Probability of latent statuses ⁴ (δ); Probability of transitioning from k profile at T1 to k profile at T2 (τ); item response probabilities	Hypotheses will be confirmed if we detect 1) a significant LRDT of nested models and lower BIC and a-BIC of constrained model, which indicate measurement invariance, and 2) more than one unique latent status (δ) and probability for transition (τ).
How do parental emotional support, family history ⁵ , and family income impact latent statuses and transitions of neural function underlying WM and ER?	Low parental emotional support, low family income, and family history of mental illness will significantly affect profile transition probabilities, by changing probability of latent statuses and/or increasing probability for transition into in low-regulation class(es).	To test whether the slope of these covariates impacts latent statuses from T1 to T2, we will conduct two subsequent models: 1) parental support and family income will be included as covariates, and 2) family history of mental health problems will be added subsequently.	Changes in model fit parameters (significant LRDT of nested models); Probability of latent statuses (δ); Probability of transitioning from k profile at T1 to k profile at T2 (τ); item response probabilities	Hypotheses will be confirmed if we detect changes in model fit, model parameters (probability of latent statuses (δ), probability of transitioning from k profile at T1 to k profile at T2 (τ), and/or item response probabilities) after adding parent emotional support, family income, and family history of mental illness as covariates.
How do youth internalizing and externalizing symptoms at T2 differ across latent statuses?	Profile transition probabilities will significantly affect rates of internalizing and/or externalizing symptoms at T2, such that latent statuses evincing low neural regulation will show increased mean values of youth internalizing and externalizing symptoms.	We will estimate the mean values of parent- and youth-reported internalizing and externalizing symptoms (derived by latent factor scores) and conduct separate Wald tests to determine if symptoms differ significantly across statuses.	Mean values of internalizing and externalizing symptom scores per each latent status; Significance ($p < .05$) of each Wald test	Hypotheses will be confirmed if any Wald test shows significant differences in internalizing and/or externalizing symptoms across latent statuses ($p < .05$) after correcting for multiple comparisons using Benjamini-Hochberg method (number of tests depends on number of profiles ⁶).

Notes. ¹T1 = Time 1, T2 = Time 2, measured 24 months after T1; ²ROIs = Region(s) of interest. See Table 2 for specific ROIs and directional hypotheses; ³Item response probabilities are mean ROI activation per profile; ⁴Latent statuses are latent profiles that remain consistent over time; Family history = sum score of parental history of alcohol use, drug use, depression, mania, hallucinations, trouble with the law, nervous disorders, hospitalization for mental health problems, and suicidality; ⁶In 3-profile estimation: 2 outcomes* 3 comparisons per outcome = 6 tests; 4-profile: 2 * 6 = 12 tests; 5-profile: 2 * 8 = 16 tests

Table 2
Hypotheses regarding function and change of ROIs.

Emotion regulation (<i>faces vs. places</i> condition)			
ROI	Function	Activity at T1	Change
Amygdala	Emotion & threat detection	Increase	Decrease
Insula	Somatosensory, salience	Increase	Decrease
Fusiform gyrus	Visual face processing	Decrease	Decrease
Anterior cingulate	Integration, regulation	Increase	Increase
Working memory (<i>0-back vs. 2-back</i> condition)			
Rostral middle frontal gyrus	Executive function, memory	Increase	Decrease
Anterior cingulate	Integration, regulation	Decrease	Decrease
Lateral occipital	Visual processing	Increase	Increase
Inferior parietal	Visual processing	Increase	Increase

Note. Hypotheses are sourced primarily from systematic reviews and meta-analyses: Del Piero et al. (2016), Andre et al. (2016). Simmonds et al. (2017) was also used to inform WM hypotheses change over time. Chaarani et al. (2021) was used to inform task-based activation specific to the ABCD sample.

scanner type on imaging data by also including it as a covariate (McCormick et al., 2021; McNeish and Kelley, 2019), this caused convergence issues (more information in Results section). As such, we included it as a clustering variable alongside family ID, which has also been used frequently within the ABCD sample (Bernanke et al., 2022; Lees et al., 2020a, 2020b; Pagliaccio et al., 2020; Saragosa-Harris et al., 2022).

4) We anticipated that latent status and transition probabilities would significantly affect rates of parent- and youth- reported internalizing and externalizing symptoms at T2, such that latent statuses and/or transitions evincing low neural regulation will show increased mean values of parent- and youth-reported internalizing and externalizing symptoms.

2. Method

To address our questions, we used data from the Adolescent Brain Cognitive Development (ABCD) Study. Launched in 2015, the ABCD Study recruited over 11,500 adolescents aged 9–10 at baseline. Participants were sampled in such a way to be representative of the population

of the United States. The ABCD Study will continue to follow these youths every 6 months for a total of 10 years. Questions and tasks are similar across waves but may be adjusted as is age appropriate. Questions include demographic information, socioeconomic background, family history of physical and mental disorders, physical and mental health, and subjective experiences. We used a subset of participants with complete behavioral and functional neuroimaging (Emotional N-Back) data at baseline and 24 months after baseline. All subjects included passed the ABCD Study’s quality control measures for functional imaging to ensure evaluation of interpretable data. [Table 3](#), [Table 4](#).

2.1. Measures

2.1.1. EN-back task

The EN-back is an implicit facial emotion and memory processing task eliciting short-term WM while presenting equal numbers of happy, fearful, and neutral facial expressions, as well as places, in each run ([Barch et al., 2013](#)). When presented with a happy, fearful, or neutral face, or a place, participants were asked to respond as to whether the picture presented is a “Match” or “No Match.” Conditions alternated between 0-back and 2-back conditions, for which youth were instructed to respond “Match” when the current stimulus corresponded to that presented at the beginning of the block or two trials back, respectively. Participants completed two runs of the task, with eight blocks per run and 10 trials (2.5 s each) per block. The current study employed two task conditions: *faces vs. places*, which was modeled to remove the effects of working memory and isolate effects of ER, and *0-back vs. 2-back*, which was modeled to remove the effects of ER and isolate effects of WM.

2.1.2. Image preprocessing and calculation region-of-interest data

Preprocessing and analysis of MRI data was completed by the ABCD Data Analysis and Informatics Center and is outlined elsewhere ([Casey et al., 2018](#); [Hagler, 2019](#)). In short, parcellated cortical regions used in the study analyses were derived from Desikan atlas cortical surface reconstruction and subcortical segmentation performed in FreeSurfer 5.3.0 ([Desikan et al., 2006](#)). Estimates of task-related activation strength were computed for each individual using general linear modeling (GLM) in AFNI’s 3dDeconvolve and released as contrast beta weights. The present study uses GLM beta coefficients averaged across both runs.

2.1.3. Working memory (indicators of LPA; IV and DV)

WM ROIs were measured at T1 and T2 (24 months after T1) and include the rostral middle frontal gyrus (rMFG), rostral and caudal anterior cingulate (rACC, cACC), lateral occipital cortex (LOC), and inferior parietal cortex (IPC). To decrease computational burden and risk of convergence issues, mean values of bilateral activation were computed for each ROI.

Table 3
LPA fit indices by time point.

T1			
Class solution	BIC	a-BIC	Smallest class size
1	48473.898	48410.432	7928 (100)
2	42551.489	42452.977	1403 (17.7)
3	36,369.347	36,235.879	495 (6.3)
4	33149.873	32981.45	418 (5.3)
5	29385.337	29181.957	363 (4.6)
6	26538.885	26300.55	4 (.05)
T2			
Class solution	BIC	a-BIC	Smallest class size
1	27496.889	27433.334	6164 (100)
2	22629.873	22531.363	499 (8.1)
3	17,955.246	17,821.781	381 (6.2)
4	15296.427	15128.007	219 (3.6)
5	12084.908	11881.533	209 (3.4)
6	10607.348	10369.018	20 (.3)

Table 4
T1 and T2 item response probabilities.

Profile	Working Memory				
	Rostral middle frontal	Inferior parietal	Caudal anterior cingulate	Rostral anterior cingulate	Lateral occipital
T1					
Emotion hypo-response	-0.051	-0.017	-0.093	-0.201	-0.144
Emotion hyper-response	0.044	0.023	0.027	-0.087	-0.034
Typical T2	0.099	0.065	0.061	-0.064	-0.023
Emotion Hypo-response	0.131	0.09	0.056	-0.123	-0.01
Emotion Hyper-response	0.133	0.094	0.072	-0.088	0.043
Typical Emotion Regulation	0.146	0.084	0.073	-0.104	-0.022
	Amygdala	Caudal anterior cingulate	Rostral anterior cingulate	Fusiform	Insula
T2					
Emotion hypo-response	-0.214	-0.688	-0.646	-0.886	-0.526
Emotion hyper-response	0.595	0.509	0.531	0.238	0.48
Typical T2	0.183	-0.024	-0.005	-0.316	0.005
Emotion Hypo-response	-0.097	-0.44	-0.406	-0.714	-0.34
Emotion Hyper-response	0.61	0.61	0.603	0.356	0.555
Typical	0.209	0.012	0.043	-0.275	0.035

2.1.4. Emotion Regulation (Indicators of LPA; IV and DV)

ER ROIs were measured at T1 and T2 (24 months after T1) and included the amygdala, insula, fusiform gyrus, rACC, and cACC. To decrease computational burden and risk of convergence issues, mean values of bilateral activation were computed for each ROI.

2.1.5. Parenting behaviors (IV)

Parental support was measured at T1 using the CRPBI-Short Parental Acceptance subscale ([Schaefer, 1965](#)). Youths reported on the emotional support of their caregiver who participated in the study with them at baseline. This subscale is the mean of 5 items, such as “makes me feel better after talking over my worries with him/her” and “believes in showing his/her love for me,” which youths evaluated using a Likert scale (1 = *Not like them*; 3 = *A lot like them*).

2.1.6. Childhood psychopathology (DV)

Child psychopathology was measured at T2 using symptom subscales of the parent-reported Child Behavior Checklist (CBCL; 119 items) and youth-reported CBCL Brief Problem Monitor (BPM; 19 items; ([Achenbach et al., 2017](#))). The CBCL is 119 items and the BPM is 19 items. In the CBCL, parents report on the presence of youths’ behaviors over the last 6 months using a Likert scale (0 = *Not True*, 1 = *Somewhat or Sometimes True*, 2 = *Very True or Often True*), such as rule-breaking (“Breaks rules at home, school, or elsewhere”), aggression (“Cruelty, bullying, or meanness to others”), anxious-depressed symptoms (“Too fearful or anxious”), and withdrawn-depressed symptoms (“Withdrawn, doesn’t get involved with others”), and somatic complaints (“Headaches, nausea”). Internalizing problems are a sum of the anxious-depressed,

withdrawn-depressed, and somatic complaints subscales, and externalizing problems are a sum of the rule-breaking and aggressive behavior subscales. The BPM has been designed as a brief counterpart to the CBCL. It is structured with the same Likert responses and produces youth-reported item ratings and scale scores for internalizing, externalizing, and attention problems that can be directly compared to the CBCL. Both the CBCL and BPM have been studied and validated in many different cultures among youths ages 6–18. The current study will employ raw scores of youths' anxious-depressed, withdrawn-depressed, rule-breaking, and aggressive symptoms (parent report), as well as youth-reported internalizing and externalizing symptoms.

2.1.7. Family history of mental health

Family history of parental mental health problems were assessed at T1 via the participating caregiver. Inclusion of family history data in the ABCD study is dependent on the participating caregiver verifying they had knowledge about the child's biological parents. Participating caregivers reported on whether the child's biological parents had ever (in their lifetime) evinced drug problems, alcohol problems, depression, mania, hallucinations, problem behavior (e.g., fighting, not holding a job, trouble with the law), nerve problems or nervous breakdowns, suicidality, or hospitalization due to these problems. Each answer is coded dichotomously, such that 1 = Yes and 0 = No. A sum score of parent mental health problems was computed (minimum = 0, maximum = 9) prior to inclusion in the LTA.

2.1.8. Covariates

In addition to parental support and family history of mental health problems, we tested whether baseline covariates – annual family income, child age in months, and child biological sex (coded dichotomously as 0 = Female, 1 = Male) – were significant predictors of class membership, latent statuses, and likelihood for transition.

2.2. Analysis plan

2.2.1. Missing data

All imaging data were filtered using quality control measures as outlined by Hagler (2019) and in ABCD Release Notes 4.0, MRI Quality Control Recommended Inclusion (<https://doi.org/10.15154/1523041>). Briefly, quality control metrics include imaging protocol compliance, mean head motion, framewise displacement, presence of artifacts, irregularities, or incidental findings, and behavioral task performance. Per ABCD data release 4.0, a single quality control index (abcd_imginc101) has been added to indicate those who pass all quality control filtering measures. The current sample was filtered using this quality control index (0 = passing QC, 1 = not passing QC).

2.2.2. Inclusion/exclusion criteria

T1 (baseline) data included all imaging data present at baseline that passed quality control (n = 7930) and all parental support and demographic data of those who have complete imaging data. T2 data include all imaging data available at T2 that passes quality control (estimated n = 6184) and all CBCL and YSR data of those who have complete imaging data.

2.2.3. Statistical outliers

Outliers can bias results of multivariate analyses such as latent profile and latent transition analysis (Fidell, 2001). During pre-processing of tabulated ABCD imaging data, beta values with greater than 5% signal change are censored (replaced with empty cells), accounting for less than 0.5% of the sample (Hagler (2019)). For imaging variables with skewness greater than 2, we will Winsorize the top and bottom 25%, which has also been recommended by Hagler (2019). Both LPA and LTA will be conducted with Winsorized and non-Winsorized imaging variables to test robustness of the models to normality.

2.2.4. Sampling weights

The ABCD Study includes propensity weights, which are weighted estimates derived from the American Community Survey used to calibrate ABCD distributions to nationally representative controls (Heeringa and Berglund, 2020).

2.3. Statistical models

Structural equation modeling in Mplus version 8.1 will be used to test all study hypotheses (Muthén and Muthén, 2014). All models were estimated using maximum likelihood with robust standard errors (Klein and Moosbrugger, 2000). Multilevel modeling was used to account for clustering effects of participants within families and scanner type (Saragosa-Harris et al., 2022). Although scanner was originally proposed as a covariate, Mplus software is currently unable to include categorical covariates with more than 10 categories. Given this, we instead used scanner as a nesting variable alongside family ID. Propensity weights were also used to calibrate distributions of the current sample to nationally representative controls from the American Community survey, thus mitigating potential selection bias in the ABCD sampling process (Heeringa and Berglund, 2020; Saragosa-Harris et al., 2022). Prior to conducting the LTA, we create two separate measurement models of child internalizing and externalizing symptoms. Using confirmatory factor analysis (CFA), we created a latent factor of internalizing symptoms using parent-reported subscales of youth anxious-depressed and withdrawn-depressed symptoms, as well as youth-reported internalizing symptoms. We also created a latent factor of externalizing symptoms using parent-reported subscales of youth rule-breaking and aggressive behavior and youth-reported externalizing behaviors. We saved the resulting factor scores to a separate data file and include them as distal outcomes in Step 4 of the LTA.

2.3.1. Latent transition analyses

LTA Step 0. We built separate LPAs with T1 and T2 imaging data using the aforementioned ROIs within Mplus version 8.1. For both baseline and T2 indicators, we fit separate LPAs starting with a null 1-class model and increasing the number of profiles by one until stopping criteria were reached. In a simulation by Whittaker and Miller (2021), BIC and adjusted BIC (a-BIC) were found to predict number of correct classes with significantly greater accuracy than any fit indices, including AIC, entropy, VLMRT, and BLRT. As such, we used BIC and a-BIC to determine whether the k class solution was better than the $k-1$ class solution, as indicated by decreases in both criteria. Scree plots were also used to visualize where BIC values began to display diminishing value for each additional class. The class number at the “elbow” of the plot where BIC values level out provides an indicator of best fitting number of classes (Nylund-Gibson and Choi, 2018). We also included as a stopping criterion a class size of less than 5% of the sample. This criterion was included to prevent fitting a model with so many classes that making comparisons between them becomes unwieldy and their qualitative differences (in both item response probabilities, probabilities of latent status, and probabilities of transitions) become meaningless. Finally, we planned not to estimate the $k + 1$ model if the best loglikelihood did not replicate or the model did not converge. As such, the following 4 questions comprised our selection criteria; if the answer to any one of these questions was no for models at either T1 or T2, we planned to stop and select the $k-1$ class solution.

- Did the model converge?
- Was the best loglikelihood replicated?
- Are the BIC and the adj-BIC lower than the $k-1$ model?
- Are all class sizes greater than approximately 5% of the sample?

LTA Step 1. If finding the number of classes derived at each time point to be the same, we tested longitudinal measurement invariance by comparing model fit of a constrained and unconstrained model. In the

constrained model, all item response probabilities are constrained to be equal across T1 and T2 (see Appendix for Mplus syntax). We then compared model fit between the constrained and unconstrained LTA models by comparing BIC, a-BIC, and conducting a log likelihood ratio difference test (LRDT). Measurement invariance may be concluded if the BIC and a-BIC are lower in the constrained model, and if the LRDT is significant. We planned to fit a repeated measures-LTA if the number of classes between T1 and T2 were not consistent or if the LTA did not evince longitudinal measurement invariance, based on the recommendation of Bray et al. (2010). After failing to establish measurement invariance, however, we conducted exploratory LTA of the non-invariant model, based on the recommendation of Nylund et al. (2022). *LTA Step 2.* We used the results of the LTA to characterize probability of latent statuses (e.g., latent profiles that remain consistent from T1 to T2), probability of transition (e.g., moving from k profile at T1 to k profile at T2), and item-response probabilities (e.g., mean ROI activation by profile). *LTA Step 3.* We tested whether parental support, family history of mental health problems, family income, child biological sex, and child age impacted LTA parameters (see Appendix for Mplus syntax). To do so, we ran the same LTA model repeatedly in a stepwise manner, including one additional covariate each time. In this way, we aimed to disentangle the unique effects of each covariate on the model. For each covariate model, we conducted an LRDT to determine whether inclusion of these covariates significantly changed the model fit compared to the previous model (Ryoo et al., 2018). Using odds ratios, we also determined whether inclusion of these covariates conferred a greater likelihood of belonging to one latent status over another. *LTA Step 4.* We then tested whether psychopathology symptoms differed across latent statuses by estimating the mean values of internalizing and externalizing factors (see Statistical Models, above) within each T1 profile. We conducted Wald tests to determine whether these differences were statistically meaningful (Nylund, Muthén et al., 2007). Across all instances of multiple comparisons (e.g., comparing odds ratios of covariates and mean values of outcome variables), we corrected for family-wise error using the Benjamini-Hochberg method (Thissen et al., 2002).

2.3.2. Reliability and robustness testing

Although a number of LPA, LCA, and LTA analyses have been conducted using cross-validation techniques, an LCA simulation study by Whittaker and Miller (2020) indicated that the most accurate cross-validation methods perform less accurately than BIC and a-BIC in a large single sample ($n = 800$). Considering our use of BIC and a-BIC to fit the proposed models, and also the large sample size and number of indicators of the current study (both of which increase estimation accuracy), we conducted our analyses using the full sample without cross-validation. We also tested if our findings were robust without controlling for clustering effects of family and scanner and no longer weighting estimates by propensity scores.

2.3.3. Power analysis

A previous simulation study by Nylund et al. (2007) indicated that for an 10-item complex LPA model with 4 unequal classes, a sample size of 1000 provided excellent coverage values for all parameters, including for the smallest class of 5%. This simulation also showed that for both LMR and BLR tests, a sample size of 1000 provided sufficient power (greater than .80) to detect the k class model for a 10-item, 4 class complex LPA model. Given that the proposed sample is significantly larger than 1000, we anticipated adequate power to detect 3 and 4 class models. We confirmed this by conducting a Monte Carlo simulation study for a complex LPA model with 13 indicators, 5 covariates, and 4 outcomes. For both 3 and 4 class solutions, LMR and BLR tests were significant ($p < 0.01$) and the proportion of replications at the 5% level for the BLRT, indicating that the proposed sample size of roughly 7000 provided adequate power to correctly identify the k class model. The simulation also indicated that the proposed sample size provided enough power to reject the null for each of the 4 outcome variables

100% of the time.

3. Results

3.1. Descriptive analyses

Descriptive analyses and distributions of all study variables are included in Tables 5, 6, and 7. Only participants with EN-back imaging data that passed quality control measures were included in our analyses. As such, the imaging sample at T1 and T2 consisted of 7930 and 6183 participants, respectively. Because some participants participated in only one wave of imaging data collection, the full sample was 9552, which included those with at least one time point of imaging data (T1 and/or T2). A correlation analysis (Fig. 1) indicated that youth externalizing and internalizing symptoms were moderately and negatively correlated with parental support and household income, and positively correlated with family history of mental illness. Parent- and youth-reported psychopathology symptoms were moderately and positively correlated. Tables 8,9,10,11.

3.2. CFA of T2 child- and parent-reported psychopathology

A two-factor model of child internalizing and externalizing behavior exhibited an acceptable fit to the data (see Fig. 2). Child-reported internalizing and externalizing were covaried due to high correlation and corresponding recommendation of modification indices. Resulting factor scores (e.g., individual Z-scores indicating factor-level standard deviations above and below the sample mean) were saved in a separate data file and used in subsequent analyses (see Step 4). However, given the low factor loading of child-reported measures (< 0.40) and the large disparity between parent- and child-reported factor loadings, we chose to follow all analyses with exploratory analyses of separate parent- and child-reported psychopathology.

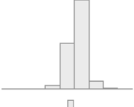
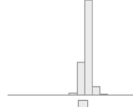
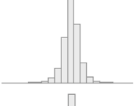

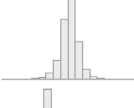
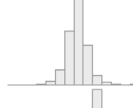
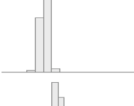


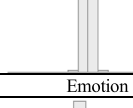
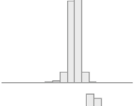
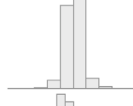
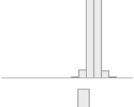
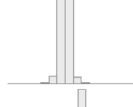
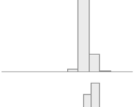
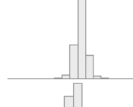
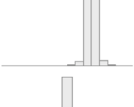
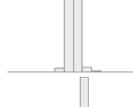
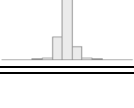
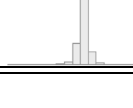
3.3. Step 0: Repeated measures LTA

At both T1 and T2, we began with a null 1-class model and increased the profile number by 1 until model fit criteria were reached. For both T1 and T2 data, the 3-profile model indicated optimal fit (Table 3, Fig. 3). Although we initially proposed to include child age and child biological sex as indicators of profiles alongside imaging variables, T2 models that included age and sex indicated convergence difficulties. Beginning at the 3-profile solution for T2, several parameters were automatically fixed to prevent singularity of the information matrix, indicating that the 3- to 6-profile models were not identified. However, this issue was resolved when removing both child age and biological sex as indicators. As such, we re-ran all profile solutions for both T1 and T2 data to test whether they were sensitive to inclusion of child age and biological sex. Because the optimal profile solution and item response probabilities remained consistent without inclusion of child age and biological sex as indicators, we chose to fit all models without these indicators. Details on fit and profile solutions for models including child age and biological sex as indicators may be found in the Appendix.

3.4. Comparing T1 and T2 profile solutions

At both T1 and T2, the profile solution indicated three distinct profiles largely differentiated by neural response to emotion regulation; neural response to working memory was relatively similar across profiles at both time points (Fig. 4, Fig. 5, Table 4). The “Typical” profile ($n_{T1} = 6661, 84\%$; $n_{T2} = 5059, 82.1\%$) was characterized by moderate amygdalar activation and fusiform deactivation to the ER (*faces vs. places*) condition at both T1 and T2. All other ER-related ROIs within this profile exhibited low to negligible response. Within this profile, rostral middle frontal and caudal ACC response to the WM (*0 vs. 2-back*) condition were slightly higher than other profiles. The “Emotion hypo-

Table 5
Descriptive statistics of imaging variables.

Variable	Mean (SD)	Graph	Valid	Missing	Mean (SD)	Graph	Valid	Missing
Working Memory T1								
Rostral middle frontal	0.1 (0.3)		7929 (83.0%)	1623 (17.0%)	0.1 (0.3)		6183 (64.7%)	3369 (35.3%)
Rostral anterior cingulate	-0.1 (0.3)		7929 (83.0%)	1623 (17.0%)	-0.1 (0.3)		6183 (64.7%)	3369 (35.3%)
Caudal anterior cingulate	0 (0.3)		7929 (83.0%)	1623 (17.0%)	0.1 (0.2)		6183 (64.7%)	3369 (35.3%)
Inferior parietal	0.1 (0.2)		7929 (83.0%)	1623 (17.0%)	0.1 (0.2)		6183 (64.7%)	3369 (35.3%)
Lateral occipital	0 (0.4)		7929 (83.0%)	1623 (17.0%)	0 (0.4)		6183 (64.7%)	3369 (35.3%)
Emotion Regulation T1								
Rostral anterior cingulate	0 (0.4)		7930 (83.0%)	1622 (17.0%)	0 (0.3)		6184 (64.7%)	3368 (35.3%)
Caudal anterior cingulate	0 (0.3)		7930 (83.0%)	1622 (17.0%)	0 (0.3)		6184 (64.7%)	3368 (35.3%)
Fusiform gyrus	-0.3 (0.4)		7930 (83.0%)	1622 (17.0%)	-0.3 (0.4)		6184 (64.7%)	3368 (35.3%)
Insula	0 (0.3)		7930 (83.0%)	1622 (17.0%)	0 (0.3)		6184 (64.7%)	3368 (35.3%)
Amygdala	0.2 (0.4)		7930 (83.0%)	1622 (17.0%)	0.2 (0.4)		6184 (64.7%)	3368 (35.3%)

response” profile ($n_{T1} = 495, 6.3\%$; $n_{T2} = 724, 11.8\%$) was characterized by moderate amygdalar deactivation and high cACC, rACC, fusiform, and insula deactivation at T1 to the ER condition, with slight decreases in deactivation across these regions at T2. The “Emotion hyper-response” profile ($n_{T1} = 772, 9.7\%$; $n_{T2} = 380, 6.2\%$) was characterized by high amygdala, cACC, rACC, fusiform, and insula activation at T1 to the ER condition, with slight increases in activation across these regions at T2. Individuals in this profile also exhibited slightly greater T1 deactivation during WM, particularly within the caudal and rostral ACC. However, WM activation within this profile increased at T2 to levels similar to other profiles. At both T1 and T2, the WM condition elicited slight deactivation in the rACC across profiles, which became more pronounced at T2.

We conducted exploratory Wald tests to establish whether item response probabilities within the same ROIs (for example, amygdala response within the Typical profile and amygdala response within the Hypo-response profile) were significantly different between profiles. All ER-related item response probabilities were significantly different between profiles at both T1 and T2 ($p = 0.000$) after correcting for family-wise error. At T1, rostral middle frontal and anterior cingulate WM response were significantly different between the Typical and Hypo-response profiles (all p -values < 0.05); however, these p -values did not remain significant after correcting for family-wise error. No other

ROIs exhibited significant WM-related differences between profiles at either T1 or T2. See [Appendix](#) for full results.

3.5. Step 1: Measurement invariance of LTA

After establishing that the 3-profile solution was optimal at both time points, we compared two different 3-profile LTA models ([Table 14](#)). The first model tested measurement invariance (MI) by constraining item response probabilities of all indicators across T1 and T2. For example, rostral middle frontal activation within Profile 1 at T1 was constrained to be equal to that within Profile 1 at T2, and this was done for all indicators that corresponded across both profiles and time points. Fit of the MI model was compared to that of a non-MI model, wherein no item response probabilities were constrained. Using a likelihood ratio difference test, we established that the non-MI model exhibited better fit than the MI model, and thus we could not conclude measurement invariance across time points. We followed this comparison with an exploratory comparison of a partial-MI model, in which we constrained only WM indicators that evinced relatively consistent item response probabilities across time and/or profiles. Specifically, we constrained WM items within the Typical and Emotion Hyper-response to be equal at T1, and WM items with all profiles to be equal at T3. After comparing this partial-MI model to the unconstrained non-MI model and fully

Table 6
Descriptive statistics of covariate/predictor variables.

Variable	Mean (SD)	Frequencies (%)	Graph	Valid	Missing
Parental support	2.7 (0.4)	-		8883 (93.0%)	669 (7.0%)
Household income	7.5 (2.2)	< 5K: 224 (2.5)		8814 (92.3%)	738 (7.7%)
		5K - 11,999: 263 (3.0)			
		12K - 15,999: 198 (2.2)			
		16K - 24,999: 370 (4.2)			
		25K - 34,999: 489 (5.5)			
		35K - 49,999: 726 (8.2)			
		50K-74,999: 1227 (13.9)			
		75K - 99,999: 1355 (15.4)			
		100K - 199,999: 2877 (32.6)			
> 200K: 1085 (12.3)					
Family history of mental illness	2.6 (2.3)	0: 2169 (22.7)		9552 (100.0%)	0 (0.0%)
		1: 1661 (17.4)			
		2: 1489 (15.6)			
		3: 1218 (12.8)			
		4: 1006 (10.5)			
		5: 765 (8.0)			
		6: 571 (6.0)			
		7: 382 (4.0)			
		8: 197 (2.1)			
		9: 94 (1.0)			
Youth biological sex	-	Female: 4615 (48.3)		9552 (100.0%)	0 (0.0%)
		Male: 4937 (51.7)			
Youth age in months	119.2 (7.5)	-		9552 (100.0%)	0 (0.0%)

constrained MI models, significant LRDTs once again indicated a superior fit of the non-MI model.

In our preregistration, we proposed to abandon the LTA in the case that longitudinal MI was not established. However, after investigating the results of the repeated measures LPA and non-MI LTA, we found that T1 and T2 profiles still exhibited strong evidence for stability from T1 to T2, making them readily comparable. As stated by Nylund-Gibson et al. (2022), although longitudinal MI is advantageous in reducing bias (Nylund, 2007) and increasing clarity and ease of LTA interpretation, it is not a required prerequisite to fitting an LTA model. In the case of a non-MI LTA model, researchers must take care to interpret transition probabilities specific to their respective classes (Nylund-Gibson et al., 2022). Considering these points, we proceeded with our proposed analyses using a non-MI LTA. Given that the following steps were preregistered only for an LTA exhibiting longitudinal MI, the following analyses are considered exploratory.

3.6. Step 2: Interpreting LTA profiles and transitions

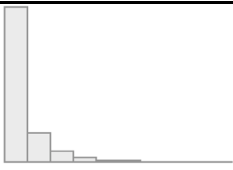
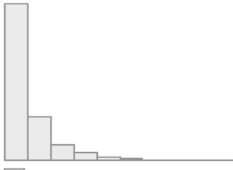
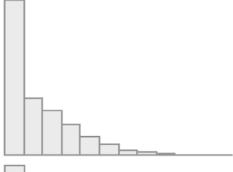
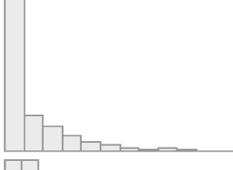
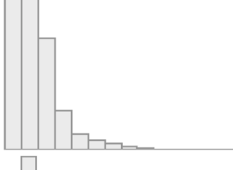
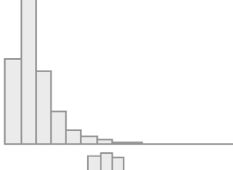
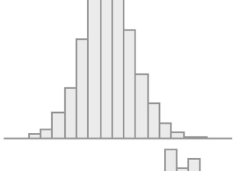
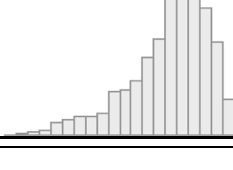
As specified above, the three profiles were similarly characterized at T1 and T2 by patterns of moderate response (the Typical profile), hypo-response (Emotion Hypo-response profile), and hyper-response

(Emotion Hyper-response profile). Results of the LTA yielded probabilities of latent statuses and transitions based on posterior probabilities respective to each profile and time point (Fig. 6, Table 14). Individuals most commonly began and remained in the “Typical” profile (n = 6622, 69.5 %). The second most common transition was characterized by movement from the Typical profile to the Emotion Hypo-response profile (n = 900, 9.4 %). The third most common transition was characterized by movement from the Emotion Hyper-response profile to the Typical profile (n = 698, 7.3 %). Those in the atypical profiles were most likely to transition into the Typical profile at T2. All other transitions involving less than 5 % of the sample are listed in Table 14.

3.7. Step 3: Investigating impacts of covariates on LTA parameters

We then investigated the impact of parental support, family history of mental illness, and family income on LTA parameters. Because issues with model identification prevented us from fully probing the effects of child biological sex and age on profiles, we conducted exploratory analyses of two additional covariate models that included child biological sex and age at baseline. In order to assess the unique contributions of each covariate to model fit and LTA parameters, we included them in the model in a stepwise manner (using the order described above),

Table 7
Descriptive statistics of outcome variables.

Variable	Mean (SD)	Graph	Valid	Missing
Externalizing (parent report)	3.7 (5.2)		5332 (55.8%)	4220 (44.2%)
Internalizing (parent report)	4.8 (5.5)		5332 (55.8%)	4220 (44.2%)
Externalizing (youth report)	2.1 (2)		5918 (62.0%)	3634 (38.0%)
Internalizing (youth report)	1.7 (2.1)		5972 (62.5%)	3580 (37.5%)
Externalizing (factor score)	0 (0.7)		6173 (64.6%)	3379 (35.4%)
Internalizing (factor score)	0 (0.7)		6173 (64.6%)	3379 (35.4%)
Mean WM response time	884.3 (111)		6184 (64.7%)	3368 (35.3%)
WM accuracy rate	0.9 (0.1)		6184 (64.7%)	3368 (35.3%)

culminating in a final model with all three covariates included. Model fit was compared between each model using a likelihood ratio test.

Reduced G^2 , BIC, and a-BIC values paired with significant LRDTs ($p < .0001$) indicated significantly improved model fit after including parental support, family history of mental illness, income, and child biological sex (Table 12). The addition of child age, however, did not confer significant changes in model fit, LTA parameters, or prediction of profile membership, and thus was not included in the final model. All LTA parameters, including item response probabilities and likelihood for transition, exhibited relative stability after inclusion of each covariate compared to the baseline model (Table 13, Table 14). However, both T1 and T2 Emotion Hyper-response profiles exhibited slight changes in item

response probabilities after including parental support and family income in the model (Fig. 7). In the T1 Emotion Hyper-response profile, activation across WM ROIs increased slightly after including parental support; subsequently, activation of all ROIs decreased as a result of including income in the model. In the corresponding T2 profile, activation during WM decreased slightly in the rostral middle frontal cortex, inferior parietal cortex, and cACC, and deactivation during WM increased slightly in the rACC and lateral occipital cortex, after including parental support and income in the model.

When using the T1 and T2 Typical profiles as reference classes, child biological sex predicted profile membership (Table 12). Namely, male youths were significantly more likely to belong to the Emotion Hypo-

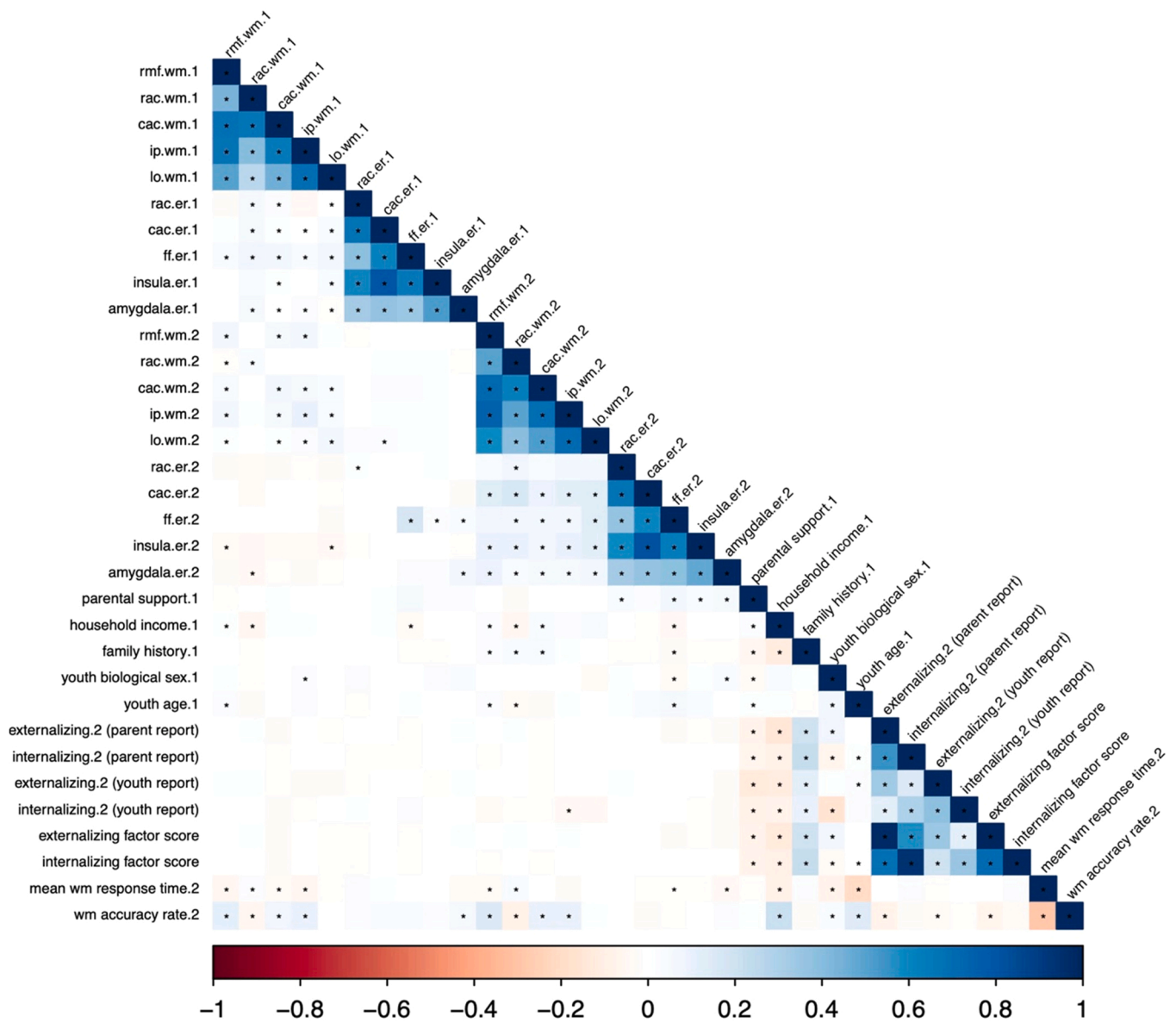


Fig. 1. Bivariate correlations of all study variables. Asterisks indicate significant correlation at or below $p = 0.05$.

Table 8
Comparison of model fit after implementing measurement invariance.

Measurement invariance?	G^2*	BIC	α -BIC	DF	G^2 diff	DF diff	LRDT	p-value
Comparison 1: MI v. non-MI								
Yes (fully constrained)	-32655.13	65,841.68	65,657.36	58				
No (unconstrained)	-26676.16	54,158.60	53,878.95	88	-5999.27	30	11,998.53	< .0001
Comparison 2: Partial MI v. non-MI								
Partial* *	-26692.01	54,052.88	53,820.90	73				
No (unconstrained)	-26676.16	54,158.60	53,878.95	88	-15.86	15	31.71	0.007
Comparison 3: MI v. partial MI								
Yes (fully constrained)	-32655.13	65,841.68	65,657.36	58				
Partial* *	-26692.01	54,052.88	53,820.90	73	-5963.12	15	11,926.23	< .0001

Note. MI = measurement invariance. *Likelihood ratio statistic. * *T1 profile 1 & profile 3 WM items constrained to be equal; T2 all WM items across all profiles constrained to be equal.

response profile than the Typical profile at T2 (OR = 1.449, SE = 0.217, $p = .038$). However, after correcting all p-values for family-wise error (16 tests total), these ORs were no longer significant (adjusted p-value = 0.229).

3.8. Step 4: Evaluating mean differences in T2 psychopathology across latent transitions

In the final step of model building, we used several Wald tests to determine whether mean differences in T2 internalizing and

Table 9
Comparison of model fit after stepwise addition of covariates.

Covariates?	G^2 *	BIC	a-BIC	DF	G^2 diff	DF diff	LRDT	p-value
Comparison 1								
None	-26676.16	54,158.60	53,878.95	88				
Support	-24741.69	50,319.64	50,027.28	92	-1934.46	4	3869.93	< .0001
Comparison 2								
Support	-24741.69	50,319.64	50,027.28	92				
Support, family history*	-24736.68	50,345.97	50,040.90	96	-5.01	4	10.03	0.04
Comparison 3								
Support, family history	-24736.68	50,345.97	50,040.90	96				
Support, family history, family income	-20015.99	40,918.38	40,600.60	100	-4720.69	4	9441.39	< .0001
Comparison 3								
Support, family history, family income	-20015.99	40,918.38	40,600.60	100				
Support, family history, family income, sex	-20000.88	40,923.63	40,593.14	104	-15.10	4	30.20	< .0001
Comparison 4								
Support, family history, family income, sex	-20000.88	40,923.63	40,593.14	104				
Support, family history, family income, sex, age	-19996.24	40,949.80	40,606.60	108	-4.64	4	9.28	0.06

Note. *Family history = family history of mental health problems.

externalizing symptoms were significantly different across T2 latent profiles (Table 14, Fig. 8). Three comparisons were conducted for both internalizing and externalizing symptoms (six comparisons in total). According to results of each Wald test, factor scores of internalizing and externalizing did not differ significantly based on profile membership at T2. Although those belonging to the Emotional Hyper-response profile at T2 exhibited the highest factor scores of both internalizing and externalizing, the non-significant Wald test indicates that the difference between psychopathology of the Emotion Hyper-response profile and other profiles was not significantly different from zero. Exploratory analyses that considered parent- and child-reported psychopathology separately yielded similar results. Although mean rates of child- and parent-reported internalizing and externalizing symptoms suggested slight differences across T2 profiles, they were not significantly different according to Wald tests (see Appendix).

We followed this step with an exploratory analysis of mean differences in T2 EN-back task behavior (Table 14, Fig. 8). Specifically, we tested whether total accuracy rate (reported as a percentage) and response time (in milliseconds) differed significantly across T2 profiles. We found that total accuracy but not response time differed between the Typical and non-typical profiles at T2, such that those in the Emotion Hypo- and Hyper-response profiles showed significantly lower accuracy (86.8% and 86.5%, respectively) than those in the Typical profile (89%). These significant differences remained after correcting for multiple comparisons.

3.9. Sensitivity analyses

Finally, we conducted sensitivity analyses to determine whether LPA parameters were robust to several model changes (see Appendix). We compared two models to the final model: one with non-winsorized LPA indicators (e.g., indicators evincing a skew greater than 2) and one without any clustering or propensity score weighting. When using T1 non-winsorized LPA indicators, all profile solutions beyond a 1-profile solution failed to converge. However, T2 indicators, which did not exhibit convergence difficulties, exhibited an optimal 3-profile solution consistent with the 3-profile solution of the final model in both item response probabilities and likelihood for profile membership. Because we were unable to establish a non-winsorized T1 profile solution, we did not explore a latent transition model with these data.

The model without clustering and weighting also showed similar profile solutions, item response probabilities, and transition probabilities compared to the final model, both within the baseline (no covariate) model and with all final covariates included (parental support, family history, income, and child biological sex).

4. Discussion

Extant research suggests that neural maturation evinces high inter-individual variability and intra-individual change, and this variability may underlie vulnerability for psychopathology in adolescence. Using latent transition analysis, the current preregistered study employed a large, two-wave sample of early adolescents to test whether neural underpinnings of working memory and emotion regulation may be characterized by distinct patterns of function both at fixed time points and across 24 months. Moreover, we explored whether parental emotional support and other demographic variables at the family and individual levels significantly contributed to inter- and intra-individual variability across time (e.g., latent transitions). Finally, we investigated the relevance of homogenous subgroups of neural function to behavior by testing mean differences of internalizing and externalizing psychopathology and (in a follow-up analysis) EN-back task performance across T2 latent profiles.

Both latent profile and latent transition analyses yielded three distinct profiles of neural function at T1 and T2, which were characterized primarily by high, low, and moderate neural response (respectively labeled Emotion Hyper-response, Emotion Hypo-response, and Typical) during an ER task (e.g., the *faces* vs. *places* condition of the EN-back). Although our results did not fully support our hypotheses, many aspects of them were confirmatory. We anticipated a “low-regulation” profile characterized by attenuated WM response of the rostral middle frontal and ER response of the ACC. Although the Emotion Hypo-response profile did exhibit these qualities, most other regions in the profile also exhibited low response (e.g., greater deactivation). It should be noted that BOLD activation to *faces* stimuli within this profile was calculated in contrast to the *places* stimuli. As such, it could be that individuals within this profile are showing particularly attenuated neural responses to images of faces or particularly elevated neural responses to images of places. Nonetheless, healthy individuals tend to show increased attentional bias and neural activation in response to viewing human facial stimuli compared to non-face stimuli (Reynolds and Roth, 2018; Ro et al., 2007; Royuela-Colomer et al., 2022). Individual differences in attention and neural response to emotional and facial stimuli often underlie development of psychopathology. While attentional bias for and increased neural response to emotional faces is linked to internalizing symptoms (Jenness et al., 2021; Royuela-Colomer et al., 2022), low attention and neural response to emotional faces is linked to psychopathic-like traits, including CU traits and antisocial behavior (Dargis et al., 2018; Huffman and Oshri, 2022; Kaseweter et al., 2020). Although we did not find those within this profile to evince greater frequency of psychopathology, this profile may highlight a group of youths particularly vulnerable to future maladaptation stemming from attenuated response to facial stimuli.

Table 10
Changes in profile item response probabilities after stepwise inclusion of covariates.

T1	Working memory					Emotion regulation				
	Rostral middle frontal	Inferior parietal	Caudal anterior cingulate	Rostral anterior cingulate	Lateral occipital	Amygdala	Caudal anterior cingulate	Rostral anterior cingulate	Fusiform	Insula
Emotion Hypo-response (6.6 %)										
Baseline	-0.051	-0.017	-0.091	-0.198	-0.144	-0.213	-0.686	-0.644	-0.884	-0.523
+ Support	-0.036	-0.016	-0.083	-0.183	-0.155	-0.208	-0.703	-0.665	-0.904	-0.542
+ Family history	-0.036	-0.016	-0.083	-0.183	-0.155	-0.209	-0.704	-0.666	-0.904	-0.542
+ Family income	-0.035	-0.016	-0.079	-0.177	-0.174	-0.232	-0.708	-0.675	-0.891	-0.54
+ Sex	-0.037	-0.016	-0.079	-0.178	-0.174	-0.234	-0.709	-0.675	-0.891	-0.54
+ Age	-0.037	-0.016	-0.079	-0.178	-0.174	-0.234	-0.709	-0.675	-0.891	-0.54
Emotion Hyper-response (10.2 %)										
Baseline	0.044	0.023	0.028	-0.085	-0.033	0.597	0.51	0.532	0.238	0.481
+ Support	0.071	0.05	0.049	-0.068	0.005	0.597	0.505	0.539	0.242	0.482
+ Family history	0.071	0.05	0.049	-0.068	0.005	0.597	0.505	0.539	0.242	0.482
+ Family income	0.046	0.028	0.03	-0.089	-0.035	0.558	0.452	0.492	0.194	0.434
+ Sex	0.046	0.028	0.03	-0.089	-0.035	0.558	0.452	0.492	0.194	0.434
+ Age	0.046	0.028	0.03	-0.089	-0.035	0.558	0.452	0.492	0.194	0.434
Typical (83.2 %)										
Baseline	0.099	0.065	0.061	-0.064	-0.023	0.183	-0.023	-0.004	-0.315	0.005
+ Support	0.094	0.06	0.058	-0.067	-0.03	0.185	-0.025	-0.006	-0.315	0.005
+ Family history	0.094	0.06	0.058	-0.067	-0.03	0.185	-0.025	-0.006	-0.315	0.005
+ Family income	0.096	0.063	0.06	-0.065	-0.024	0.178	-0.035	-0.019	-0.322	-0.005
+ Sex	0.096	0.063	0.06	-0.065	-0.024	0.178	-0.035	-0.019	-0.322	-0.005
+ Age	0.096	0.063	0.06	-0.065	-0.024	0.178	-0.035	-0.019	-0.322	-0.005
T2										
Emotion Hypo-response (12.5 %)										
Baseline	0.13	0.09	0.056	-0.122	-0.011	-0.097	-0.439	-0.405	-0.713	-0.339
+ Support	0.121	0.084	0.047	-0.129	-0.023	-0.09	-0.437	-0.403	-0.705	-0.338
+ Family history	0.121	0.084	0.047	-0.129	-0.023	-0.089	-0.436	-0.402	-0.704	-0.337
+ Family income	0.115	0.084	0.037	-0.132	-0.027	-0.102	-0.435	-0.405	-0.699	-0.338
+ Sex	0.115	0.084	0.038	-0.132	-0.027	-0.1	-0.433	-0.403	-0.698	-0.337
+ Age	0.115	0.084	0.038	-0.132	-0.027	-0.1	-0.433	-0.403	-0.698	-0.337
Emotion Hyper-response (6.4 %)										
Baseline	0.133	0.093	0.071	-0.089	0.04	0.61	0.609	0.602	0.355	0.554
+ Support	0.092	0.064	0.038	-0.121	-0.003	0.612	0.607	0.598	0.349	0.551
+ Family history	0.09	0.063	0.036	-0.122	-0.005	0.612	0.607	0.597	0.35	0.552
+ Family income	0.071	0.041	0.015	-0.15	-0.035	0.589	0.585	0.576	0.329	0.532
+ Sex	0.07	0.041	0.014	-0.152	-0.036	0.591	0.587	0.577	0.331	0.534
+ Age	0.07	0.041	0.014	-0.152	-0.036	0.591	0.587	0.577	0.331	0.534
Typical (81.1 %)										
Baseline	0.146	0.084	0.073	-0.104	-0.022	0.21	0.013	0.043	-0.274	0.035
+ Support	0.149	0.087	0.078	-0.101	-0.016	0.209	0.012	0.043	-0.276	0.034
+ Family history	0.149	0.087	0.078	-0.101	-0.016	0.209	0.012	0.043	-0.276	0.034
+ Family income	0.155	0.091	0.08	-0.1	-0.014	0.206	0.011	0.036	-0.276	0.034
+ Sex	0.155	0.091	0.08	-0.1	-0.014	0.206	0.011	0.036	-0.275	0.034
+ Age	0.155	0.091	0.08	-0.1	-0.014	0.206	0.011	0.036	-0.275	0.034

Note. Bolded lines indicate item response probabilities of final model.

We anticipated that youth who evince relatively higher levels of amygdalar response to ER would be paired with lower levels of response within the ACC. Instead – and similar to the Emotion Hypo-response profile – we found within the Emotion Hyper-response profile that activity other ER-related ROIs corresponded closely to that of the amygdala. The ACC in particular nearly matched the level of activation of the amygdala at both T1 and T2. This may suggest that among youths with very elevated amygdala response to emotion, prefrontal regulatory regions such as the ACC have to “work harder” to modulate subcortical activity and facilitate emotion regulation (Ochsner and Gross, 2014;

Yang et al., 2020). However, the close positive association between amygdala and ACC function in the Hyper-response profile, as well as the relatively elevated amygdala function in other profiles, may be normative within the developmental period of the current sample (Gee et al., 2013; Silvers et al., 2017). In a study focusing on ER development from childhood to early adulthood, Silvers et al. (2017) found that increasing age predicted decreasing amygdala response and increased inverse coupling between the vmPFC and amygdala during a reappraisal task. Indeed, vmPFC-amygdala coupling did not become inverse in the cohort studied by Silvers et al. until approximately age 16. As more waves of

Table 11
Changes in latent transitions after stepwise inclusion of covariates.

		Model					
		Baseline	+ Support	+ Family History	+ Family Income	+ Sex	+ Age
T1	T2	%	%	%	%	%	%
Hypo	Hypo	1.4	1.4	1.4	1.5	1.5	1.5
Hypo	Hyper	0.9	1	1	1	1	1
Hypo	Typical	4.3	4	4	4	4	4
Typical	Hypo	9.4	9.4	9.5	9.3	9.3	9.3
Typical	Hyper	4.3	4.3	4.3	4.4	4.3	4.3
Typical	Typical	69.5	70	70	67.9	70	67.9
Hyper	Hypo	1.6	1.6	1.6	2	2	1.9
Hyper	Hyper	1.2	1.3	1.3	1.5	1.5	1.5
Hyper	Typical	7.3	7	7	8.6	8.6	8.6

Note. Bolded lines indicate top three most common transitions.

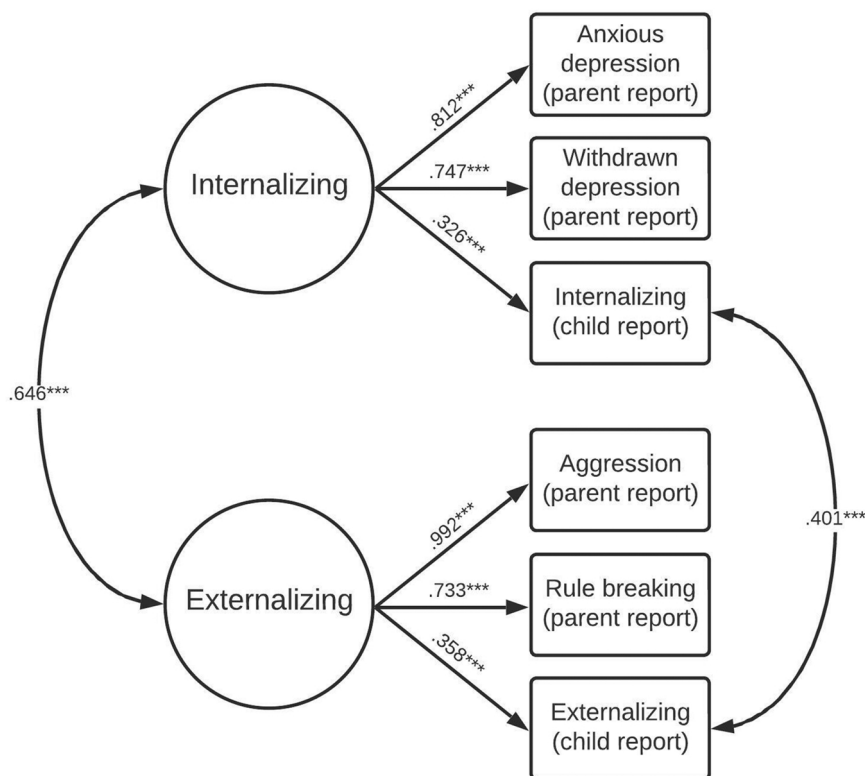


Fig. 2. Measurement model of T2 child internalizing and externalizing behaviors. All values are standardized. Model fit was acceptable: $\chi^2(7) = 191.697$, $p = 0.000$; RMSEA = .065; CFI = .969; TLI = .933; SRMR = .033).

ABCD data and other large longitudinal imaging cohorts are released, additional research will be necessary to identify the developmental periods at which high positive coupling between the amygdala and prefrontal regions is normative and, conversely, maladaptive.

Why is it that profiles exhibited such distinction in ER-related neural response but not WM-related response? This lack of heterogeneity in neural function underlying WM may be due to the relatively young age of the sample. Although WM, along with other EF processes, begins to exhibit marked increases in early adolescence, it is still in its nascency. Developmental studies encompassing late childhood and early adolescence suggest that WM function grows more specialized throughout adolescence and into adulthood (Andre et al., 2016; Del Piero et al., 2016; Simmonds et al., 2017). Therefore, the neural function underlying WM among those in the current sample may simply not be mature enough to evince notable variation between individuals. As more time points of functional neuroimaging data become available for the current cohort, follow-up studies employing LTA or similarly person-centered methods are crucial to determining whether this homogeneity is

indeed specific to early adolescence.

Our findings were largely dimensional in nature, with profiles that captured high, moderate, and low response to an ER condition. With this in mind, it may stand to reason that a method like LTA is unnecessary when outcomes seem continuous (e.g., ranging uniformly from low to high). We argue that the current method remains advantageous for two reasons. First and most importantly, LTA offers the utility of modeling likelihood for transition – in other words, continuity or discontinuity – in patterns of neural function across time. In the current study, there was indeed a high number of individuals (nearly 30 %) who moved from one profile to another, thus exhibiting notable change in their pattern of neural response to the task across 24 months of early adolescence. Our results were inconclusive regarding which factors contributed to this discontinuity; nonetheless, the current study offers compelling evidence that LTA can yield unique insights into brain development when compared to other longitudinal methodologies. Moreover, the multivariate capacity of LPA and LTA allowed us to investigate regional activation underlying both WM and ER simultaneously. As a result, we

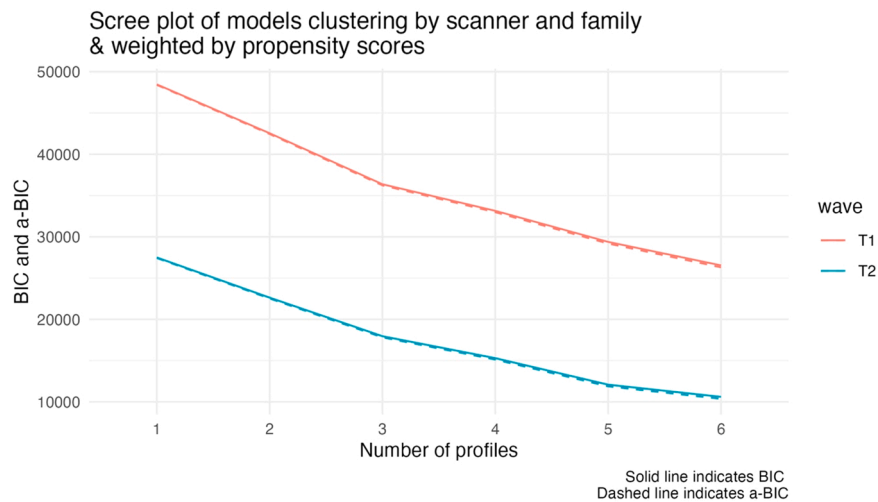


Fig. 3. Scree plots of T1 and T2 profile solutions indicate “elbows” at the 3-profile solution where BIC values begin to display diminishing value for each additional class. Although another “elbow” is observed at the 5-profile solution for T2, this solution exhibited profiles comprised of less than 5% of the sample, which did not align with our predetermined fit criteria.

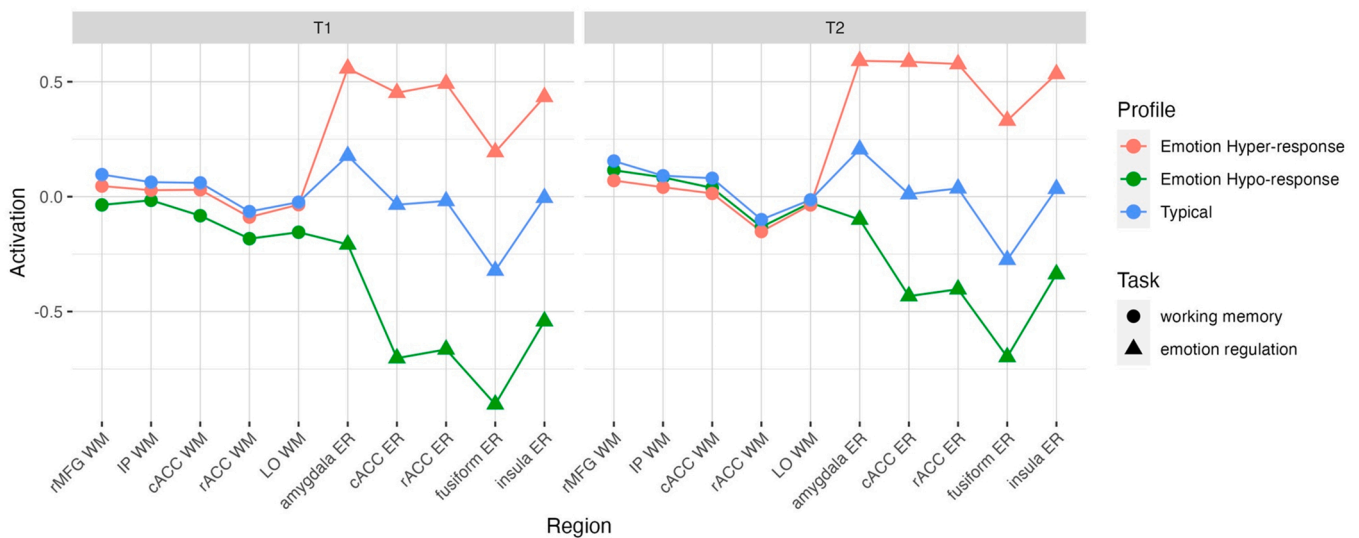


Fig. 4. T1 and T2 latent profile solutions.

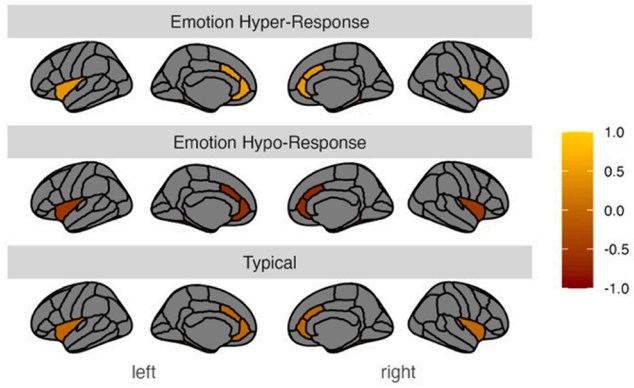
were able to observe a notable distinction in variation of neural response based on whether the task elicited WM or ER processes. Although several studies have investigated the role of WM and ER in early adolescence, no study to our knowledge has yielded results which simultaneously account for both WM and ER function in the same statistical model.

Counter to our hypotheses, parental support did not predict profile membership at either time point. However, inclusion of both parental support and family income in the model significantly impacted patterns of brain activation within the Emotion Hyper-response profile. Although the limitations of this statistical evidence prevent us from drawing directional conclusions between parental support and WM- and ER-related neural function, it may indicate that parental support and family income are particularly relevant to neural function among youth evincing heightened response to emotional stimuli, particularly within the amygdala and ACC. Indeed, a large body of evidence using both functional MRI and physiological data suggests that neurobiologically “reactive” youths may be more sensitive to contextual inputs via the rearing environment than their less reactive counterparts (Guyer, 2020; Huffman et al., 2020; Liu et al., 2021; Roberts and Lopez-Duran, 2019). Using data-driven longitudinal methods such as LTA may be a crucial

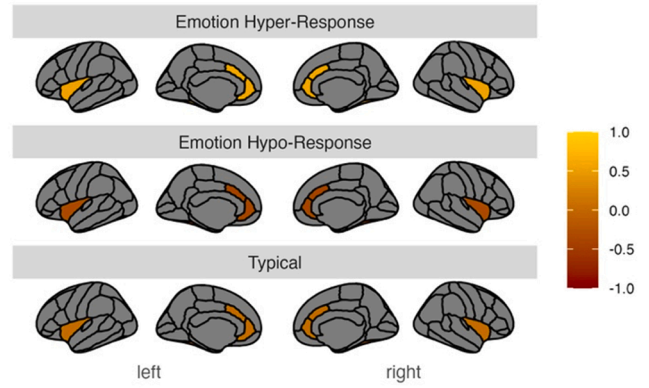
next step to identifying profiles of neurobiological sensitivity to environmental inputs and their relevance to developmental outcomes among youths.

There are numerous established links between normative variations in parenting behavior, including parental warmth and support, and youth emotion regulation (Kopala-Sibley et al., 2020; Tang et al., 2020). However, studies on parenting as a precursor to child neurobiological development have largely focused on parenting behaviors outside of the normative range (e.g., neglect, abuse, psychiatric illness, and addiction; Heeringa et al., 2016; McLaughlin, Peverill, et al., 2015; McLaughlin, Sheridan, et al., 2015; Teicher et al., 2016). In a scoping review, Farber et al. (2022) identified ten studies that investigated normative range parenting and functional neurodevelopment among youths. Several studies identified by the authors found significant associations between positive parenting behaviors and neural function, including amygdala response to threat (Farber et al., 2019; Romund et al., 2016), striatal response to reward (Telzer et al., 2013), and prefrontal response during cognitive control tasks (Kim-Spoon et al., 2017; McCormick et al., 2016; Telzer et al., 2013). Although invaluable, these studies are limited by a predominance of cross-sectional designs (six out of ten) and small

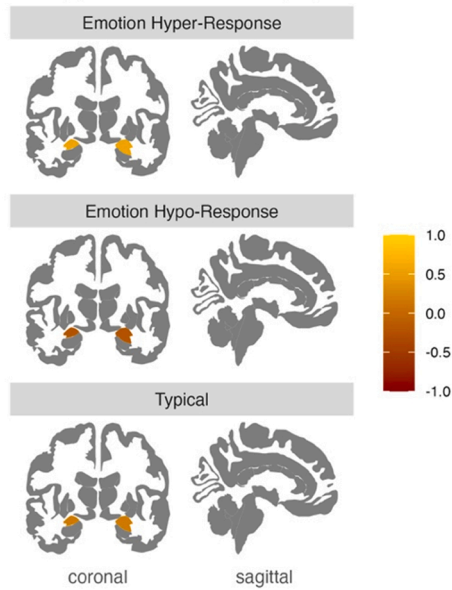
T1 Cortical Activation Underlying ER Across Profiles



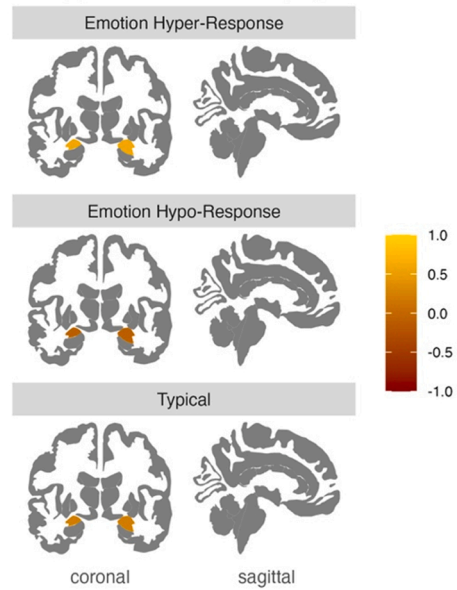
T2 Cortical Activation Underlying ER Across Profiles



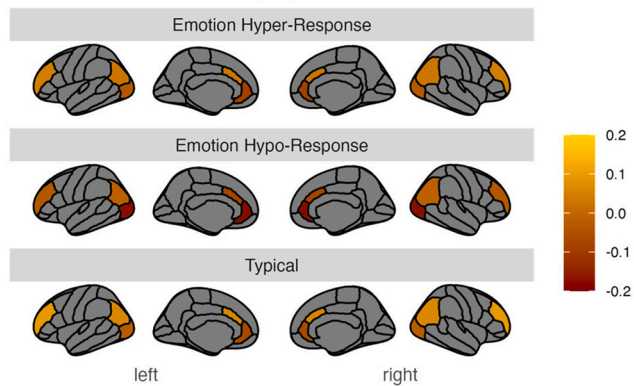
T1 Amygdalar Activation Underlying ER Across Profiles



T2 Amygdalar Activation Underlying ER Across Profiles



T1 Cortical Activation Underlying WM Across Profiles



T2 Cortical Activation Underlying WM Across Profiles

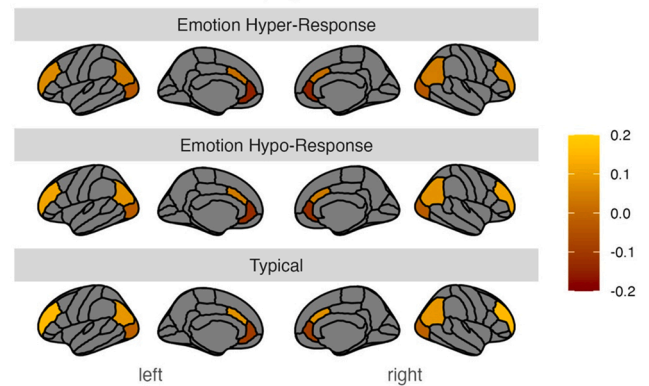


Fig. 5. Cortical and subcortical activation underlying ER and WM across profiles.

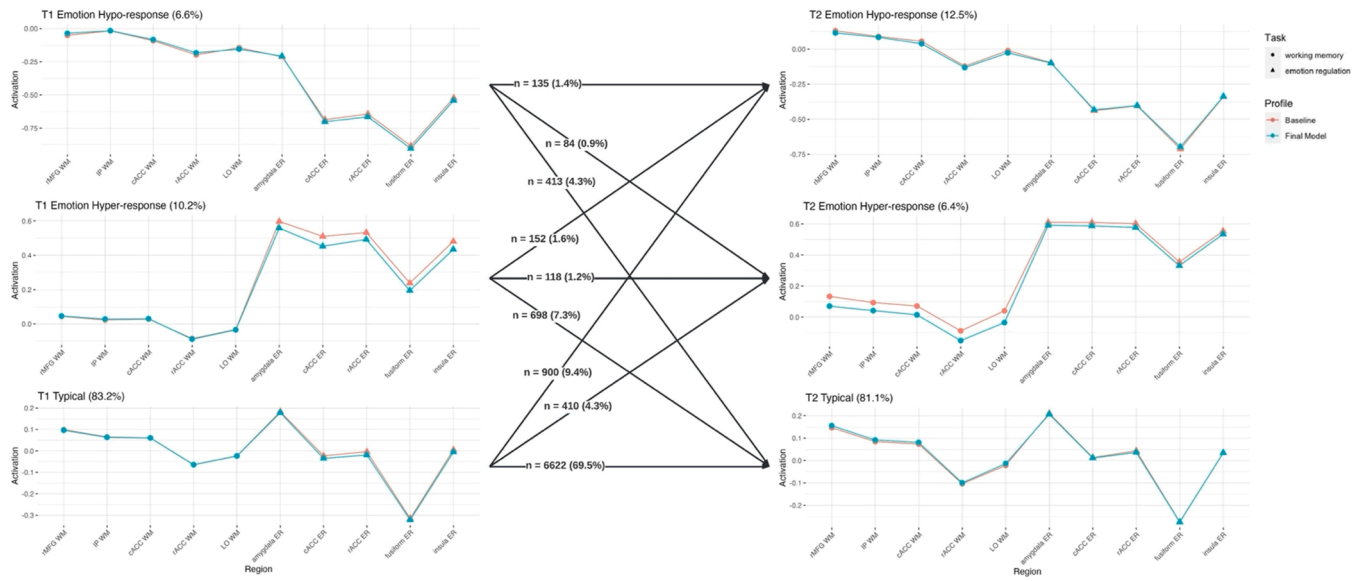


Fig. 6. Latent transition probabilities.

Table 12
Logistic regression odds ratio results (Reference class: Typical).

	OR	SE	p	Adjusted p
T1 Emotion Hypo-response				
Support	1.000	0.143	0.998	0.449
Family history	0.978	0.024	0.359	0.327
Family income	0.967	0.024	0.170	0.270
Biological sex*	1.168	0.122	0.169	0.128
T1 Emotion Hyper-response				
Support	1.365	0.261	0.162	0.838
Family history	0.936	0.034	0.055	0.449
Family income	0.944	0.029	0.055	0.505
Biological sex	1.342	0.193	0.076	0.327
T2 Emotion Hypo-response				
Support	0.936	0.174	0.712	0.888
Family history	0.953	0.031	0.134	0.449
Family income	0.957	0.033	0.188	0.327
Biological sex	1.449	0.217	0.038	0.229
T2 Emotion Hyper-response				
Support	1.014	0.139	0.101	0.229
Family history	0.975	0.023	0.292	0.537
Family income	0.981	0.030	0.618	0.537
Biological sex	1.136	0.165	0.412	0.229

Note. *Female = 0, Male = 1. Odds ratios that are greater than 1 indicate that the event is more likely to occur as the predictor increases. Odds ratios that are less than 1 indicate that the event is less likely to occur as the predictor increases.

sample sizes (eight out of ten under 200; all under 1000). Moreover, these studies focus on measurements of parenting and neural function in middle to late adolescence (13–18 years). The current study builds upon this nascent literature by highlighting a particular pattern of neural

Table 13
Mean values of outcome variables across latent trajectories in final model.

	n*	%	Internalizing	Externalizing	Accuracy rate	Response time
Hypo to Hypo	105	1.5	0.027	0.042	0.871	889.412
Hypo to Hyper	69	1	0.060	0.069	0.863	889.174
Hypo to Typical	280	4	0.012	0.012	0.890	882.827
Hyper to Hypo	138	2	0.027	0.042	0.871	889.412
Hyper to Hyper	106	1.5	0.060	0.069	0.863	889.174
Hyper to Typical	606	8.6	0.012	0.012	0.890	882.827
Typical to Hypo	658	9.3	0.027	0.042	0.871	889.412
Typical to Hyper	306	4.3	0.060	0.069	0.863	889.174
Typical to Typical	4805	67.9	0.012	0.012	0.890	882.827

Note. Estimated n based on posterior probabilities rounded up to nearest whole number.

response among early adolescents that may confer additional sensitivity to the parenting and socioeconomic contexts.

When assessing mean differences in T2 behavior, we found no significant difference in internalizing and externalizing symptoms across T2 latent profiles. However, exploratory analyses suggested modest but significant differences in EN-back behavioral performance between the Typical and non-typical profiles, indicating that neural response to WM and ER conditions within the Hypo- and Hyper-response profiles may

Table 14
Wald χ^2 tests of equality in outcome variables.

	χ^2 *	p	Adjusted p
Internalizing			
Hypo v. Hyper	0.240	0.624	–
Hypo v. Typical	0.100	0.752	–
Hyper v. Typical	0.751	0.386	–
Externalizing			
Hypo v. Hyper	0.174	0.676	–
Hypo v. Typical	0.415	0.520	–
Hyper v. Typical	1.037	0.309	–
Accuracy rate			
Hypo v. Hyper	1.077	0.299	0.449
Hypo v. Typical	9.274	0.002	0.006
Hyper v. Typical	21.601	0.000	0.000
Response time			
Hypo v. Hyper	0.001	0.979	0.979
Hypo v. Typical	1.217	0.270	0.449
Hyper v. Typical	0.689	0.407	0.488

Note. All tests have 1 degree of freedom. Hypo = Emotion Hypo-response, Hyper = Emotion Hyper-response.

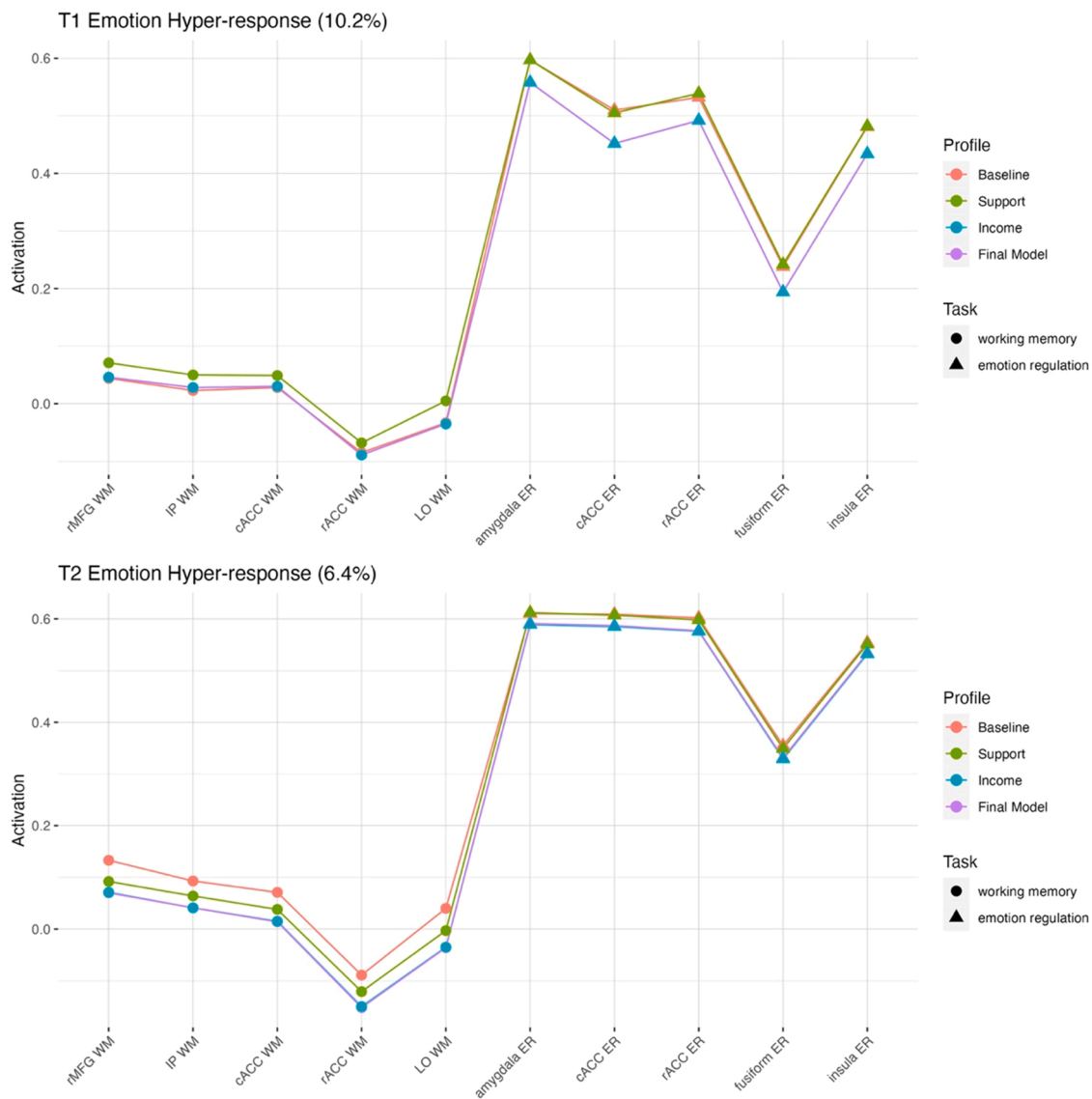


Fig. 7. Fluctuation in item response probabilities of Emotion Hyper-response profiles after adding covariates.

undermine performance during affective WM tasks. Although the current study did not find these same profiles to evince greater psychopathology, a large body of evidence suggests that WM performance in the context of affective information is itself a consistent correlate of psychopathology. Individuals exhibiting psychopathology often exhibit lower WM performance when simultaneously processing emotional information (Huang-Pollock et al., 2017; Schweizer et al., 2019). Conversely, interventions aimed at improving both affective and non-affective WM capacity are often effective in improving emotion regulation and mitigating psychopathological symptoms (Jopling et al., 2020; Xiu et al., 2018). Should later studies find that those evincing similar patterns of neural response found in the current study are at greater risk for psychopathology, affective WM may be a compelling target for preventive intervention.

Given the highly variable nature of associations between brain function patterns and psychological phenotypes, the fact that we did not detect significant differences between psychopathology symptoms is not entirely surprising. First of all, this may be an example of equifinality, in which unique mechanisms precipitate a similar outcome. Examples of equifinality have been previously documented in brain-behavior associations, such as between patterns of aberrant reward processing and multiple types of psychiatric outcomes (e.g., mood disorders,

schizophrenia, and addiction; Luijten et al., 2017; Nusslock and Alloy, 2017; Pine and Fox, 2015). However, the null finding may also stem from increased methodological rigor. Recent studies and commentaries (Marek et al., 2022) have pointed out that many extant studies identifying direct links between neural function and psychopathology are limited by small sample size and unreliable phenotypic measurements, which in turn inflate effect sizes and increase risk for irreproducibility (Nikolaidis et al., 2022). When sample size is large, as in the current study, brain-behavior associations fail to reproduce or at the very least, show smaller effect sizes than comparable less-powered studies (Marek et al., 2022). That we did not find strong associations between neural function and psychopathology might also be a picture of the complex pathways underlying not only psychopathology but resilience as well. Evidence that fails to support direct developmental links between atypical neural function and psychopathology arguably undergirds the idea of resilience: individuals and their behaviors are greater than a sum of their biological and contextual “parts,” however extensively those parts have been measured. Indeed, the complexity and inscrutability of the mediating and moderating processes that prevent inherent or inherited risk from developing into psychopathology cannot be overstated, as decades of developmental studies have confirmed.

The current study has several limitations. First, the use of only two

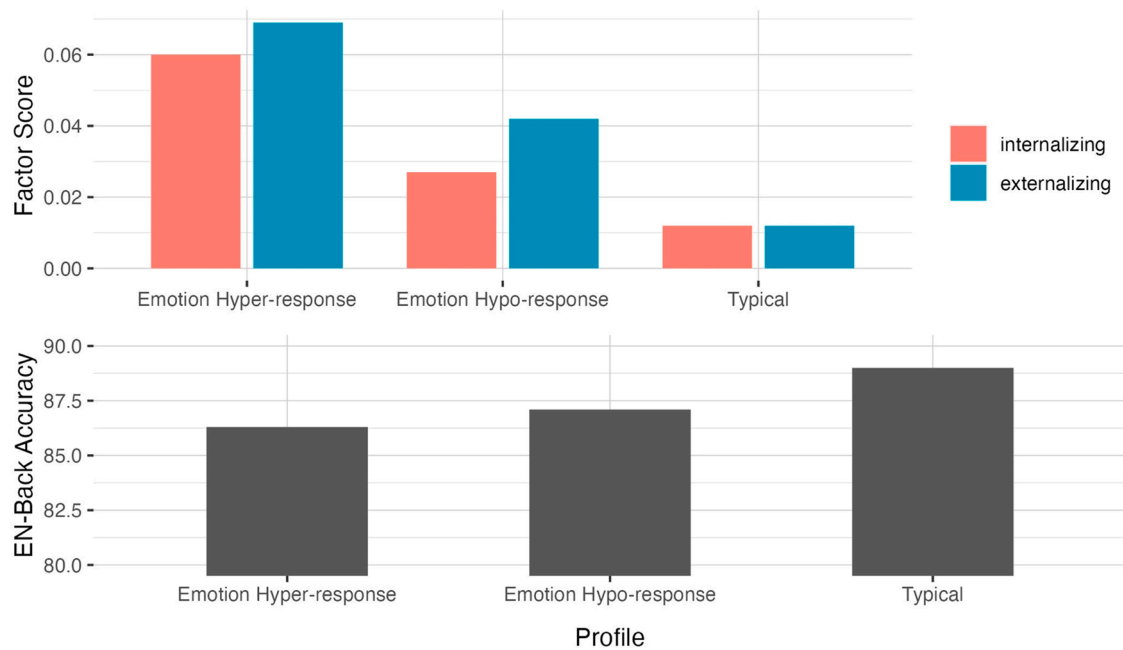


Fig. 8. Mean differences in psychopathology and WM accuracy across profiles.

time-points limits our ability to draw conclusions beyond the brief period studied. Considering our predictors and outcomes were assessed at T1 and T2, respectively, we were able to investigate cross-sectional associations only between covariates of interest and T1 neural function, as well as outcomes of interest and T2 neural function. Additionally, our use of mean values of regional activation across hemispheres prevented us from investigating lateralized contributions of neural response to LTA parameters. This choice was made after establishing within the current sample the high positive inter-hemispheric correlations of all ROIs to prevent model-overfitting and convergence difficulties. However, given that lateralization during WM and ER processes has been previously documented, additional research is necessary to determine how hemispheric differences impact the current profiles of neural function. Despite these limitations, our findings offer valuable insight into person-centered patterns of neural function underlying key cognitive processes across a crucial period of adolescent development. We hope that the current study establishes a framework by which others may investigate, replicate, and expound upon the profiles generated within later waves of the ABCD Study and other large neuroimaging cohorts.

Funding

This work was financially supported by National Institute on Drug Abuse, Grant/Award Number: F31DA053828. National Institute on Drug Abuse, Grant/Award Number: K01DA045219.

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.

Data availability

The authors do not have permission to share data.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at [doi:10.1016/j.dcn.2022.101177](https://doi.org/10.1016/j.dcn.2022.101177).

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