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RESEARCH PAPER

A MULTI-METHOD META-ANALYSIS OF NEURO-BEHAVIORAL MOTOR INHIBITION OUTCOMES ACROSS SUBSTANCE-USE PROBLEMS AND RELATED DEVELOPMENTAL PSYCHOPATHOLOGICAL CONDITIONS: A SELF-REGULATION APPROACH

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Abstract

Objectives: Alterations in motor inhibition (MI) may be involved in the homotypic and heterotypic developmental pathways of substance use disorders (SUDs) and related conditions (e.g., binge drinking). However, no studies have clarified the neuro-behavioral correlates of MI across the various conditions (e.g., ADHD and major depressive disorder in childhood/adolescence) constituting developmental trajectories of SUDs and related problems, especially within a self-regulation conceptual framework.

Method: A multi-method meta-analysis (i.e., voxel-based, network, and multi-level) of MI tasks (i.e., Go/No-Go; Stop Signal) was conducted. Neuroimaging (fMRI) and event-related potential (ERP) data were analyzed. The study included children and adolescents with ADHD, adolescents with major depressive disorder, and subjects with SUDs and related conditions.

Results: Sixty-eight studies (fMRI: 42; ERPs: 26) were included. The analysis showed a shared increased responsiveness of the rostral anterior cingulate cortex among conditions of interest during "No-Go" trials compared to healthy controls (HCs). Reduced N200/P300 waves, together with increased reaction times in response to MI task experimental conditions, were found among conditions of interest compared to HCs. Network meta-analysis results showed that distinct patterns of neural activity linked to different self-regulation domains differentiated these clinical conditions from one another.

Conclusions: Common and distinct patterns of neuro-behavioral responses associated with self-regulation mechanisms, especially regarding MI, should be considered key latent dimensions involved in the homotypic and heterotypic developmental pathways of SUDs and related problems.

Key words: substance-related disorders, developmental pathways, homotypic and heterotypic continuity, motor inhibition, self-regulation

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Introduction

Substance use disorders (SUDs) represent one of the most prevalent mental disorders worldwide (Castaldelli-Maia & Bhugra, 2022) leading cause of global morbidity and functional impairment (Rehm & Shield, 2019). Characterized by behavioral dysregulation, SUDs are prototypic conditions within the externalizing spectrum (Ringwald et al., 2023). From a developmental perspective, SUD onset is prospectively linked to both externalizing and internalizing manifestations. Specifically, childhood externalizing disorders — such as deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD) and conduct disorder (CD) — predict later SUDs (Mustonen et al., 2023). Similarly, adolescent internalizing symptoms, particularly depression, significantly predict substance-related conditions independently of externalizing traits (Hussong et al., 2017). These pathways reflect two core developmental phenomena: homotypic continuity (within-spectrum progression, e.g., ODD to SUDs) and heterotypic continuity (between-spectra progression, e.g., depression to SUDs) (Costello et al., 2003). Despite these established associations, the underlying neuro-behavioral mechanisms driving these developmental trajectories remain to be fully elucidated.

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One of the most relevant underlying dimensions potentially involved in these developmental pathways is motor inhibition (MI). Indeed, deficits in motor inhibition (MI) are the core features of SUDs compared to attentional or decision-making dimensions (Cavicchioli, Movalli, Bruni, et al. 2023). Specifically, MI problems are robust predictors of adolescent substance-use emergence (Tarter et al., 2003) and the transition to problematic use in adulthood (Thomsen et al., 2018), playing a key role in disorder maintenance (Koob & Volkow, 2016). Regarding developmental pathways, MI deficits may underpin the longitudinal associations between childhood externalizing/internalizing conditions and later SUDs. For instance, severe MI problems in children with ADHD increase the risk of adult SUDs (Ortal et al., 2015) and represent the primary factor explaining the retrospective link between childhood ADHD symptoms and SUD severity (Cavicchioli et al., 2022). Similarly, MI deficits exacerbate substance-use behaviors in youth with conduct problems (Blair, 2020). Notably, MI also mediates the relationship between depressive symptoms and early substance-use onset (Pang et al., 2014) and moderates the longitudinal association between depression and substance-use severity during adolescence (Felton et al., 2020).

Neuro-behavioral mechanisms of MI

MI comprises a set of mechanisms — motor preparation, finalization, and response inhibition— temporally organized to meet internal and external demands (Aron, 2011; Wright et al., 2014). As one of the earliest regulatory functions to emerge (6–9 months), MI matures through adolescence, providing a foundational scaffold for complex executive functions (EFs) like cognitive flexibility and planning (Diamond, 2013; Nigg, 2017).

The Go/No-Go (GNG) and Stop Signal Task (SST) are the gold standards for assessing MI across development. Behavioral outcomes from these tasks provide specific indices of MI (Aron, 2011; Wright et al., 2014): i) *hits and correct rejections* reflect effective motor execution and the ability to refrain from prepotent responses, respectively; ii) *commission errors* indicate a failure to restrain non-pertinent actions, while *omission errors* typically correlate with fluctuations in sustained attention; iii) *reaction times* (RTs) serve as an index of motor preparation efficiency.

The neural underpinnings of mechanisms linked to MI are frequently investigated using Event-Related Potentials (ERPs) due to their high temporal resolution and non-invasive nature. The N200 and P300 waves are robust developmental markers of MI (Albert et al., 2013; Brydges et al., 2012). The N200 is associated with conflict monitoring, while the P300 supports the finalization of motor inhibition (Groom & Cragg, 2015). Developmental data suggest a progressive specialization, where the P300's role in top-down behavioral regulation increases linearly with age (Johnstone et al., 2007).

Topographically, fMRI data (Aron, 2011; Hikosaka, 2011) identify an extended network sustaining MI, including the right inferior frontal gyrus (rIFG) for response inhibition, the pre-supplementary motor area (pre-SMA) for action selection, and the subthalamic nucleus (STN) and striatum for effective inhibitory signaling.

While meta-analyses (Qiu & Wang, 2021; Zhang, Peng, Eickhoff, et al. 2021) have reported MI alterations in adults with SUDs — such as reduced N200 amplitudes and hypoactivation in the IFG — there is a lack of comprehensive evidence focusing on MI paradigms from a dimensional and developmental perspective. Such a synthesis is critical to clarifying the neuro-behavioral trajectories that characterize SUDs and related conditions from childhood through adulthood.

Unity and diversity of executive functions and its implication for psychopathological conditions

While MI is distinct from other attentional mechanisms (Nigg, 2017), the bifactor model of Executive Functions (EFs) posits a high-order common/inhibitory factor alongside independent factors for updating and shifting (Diamond, 2013, Friedman & Miyake, 2017). Developmentally, EFs are indistinguishable before age 5 (Wiebe et al., 2011). From mid-childhood through adolescence, the common factor explains most variance, though subcomponents begin to differentiate behaviorally (RTs, error rates) and electrophysiologically via N200 and P300 amplitudes (Brydges et al., 2014). In adulthood, the three-factor structure — inhibition, updating, and shifting— is consistently observed (Karr et al., 2018). Neuroimaging data support this trajectory. Indeed, Zhang, Peng, Eickhoff et al. (2021) demonstrated that while adolescents show a broad common EF network, adults exhibit clear topographical separation between inhibition (rIFG, insula), updating (parietal lobule), and shifting (precuneus).

The balance between common and specific EF factors may reflect a general liability to psychopathology, often termed the *p-factor* (Caspi et al., 2014). Meta-analyses indicate that diverse conditions — including schizophrenia, SUDs, and mood disorders — share gray matter loss in the dorsal anterior cingulate cortex (ACC) and insula, correlating with poor EF performance (Goodkind et al., 2015). Furthermore, a "multiple-demand network" (IFG, pre-SMA, ventromedial prefrontal cortex [VLPFC]) appears commonly recruited — and often abnormally so — across clinical conditions (McTeague et al., 2017). However, disorder-specific patterns exist; for instance, psychotic disorders show pronounced left lateral PFC hypoactivation related to updating (Nee & D'Esposito, 2015), whereas internalizing disorders show specific hippocampal/amygdala gray matter loss (Goodkind et al., 2015).

Therefore, a common inhibitory latent factor might represent a transdiagnostic dimension linked to developmental trajectories of SUDs. Nevertheless, the precise interplay between this general factor and the specific MI processes central to SUDs requires a further clarification.

From MI to self-regulation

Alterations of MI and their implications for executive functioning and developmental psychopathology could be understood within the broader framework of self-regulation — the ability to modulate internal states and behaviors (Nigg, 2017). Specifically, Barkley's (1997, 2001) model provides a comprehensive developmental perspective, viewing EFs as "behaviors-to-the-self" that internalize with age. This system coordinates four specialized networks to support effective MI:

I) Sensing-to-the-self and related non-verbal working memory processes are guided by the Executive Control (Witt et al., 2021) and Dorsal Attention (Silver & Kastner, 2009) networks, inclusing dorsolateral prefrontal cortex (DLPFC), VLPFC, intraparietal sulcus areas (IPS) and posterior parietal cortex (PPC) (Segal & Elkana, 2023; Ptak et al., 2017)

II) Speech-to-the-self and verbal working memory involve the internalization of speech, localized in the left IFG and bilateral superior (STG) and middle (MTG) temporal gyri (Langland-Hassan, 2021);

III) Emotion/Motivation-to-the-self integrates sensory-motor and verbal processes with affective and motivational states via a network including the ventromedial PFC, amygdala, and insula (Poppa & Bechara, 2018);

IV) Play-to-the-self and EFs linked to fluency, flexibility and cognitive generativity share neural correlates with the sensing-to-self domain (DLPFC, VLPFC).

This model extends the EF bifactor framework by incorporating emotional regulation — significantly impaired in SUDs and related psychopathology (e.g., Cavicchioli, Tobia, & Ogliari, 2023; Wilcox et al., 2016; Stellern et al., 2023) — and linguistic processes, which may represent a burgeoning transdiagnostic dimension (Fradkin et al., 2024). By addressing both "unity and diversity," Barkley's model might offer a robust background for investigating the homotypic and heterotypic developmental pathways of substance-related disorders.

Table 1 summaries the topographical organization of self-regulation system proposed by Barkley's model together with their overlaps and diversities with common/inhibition factor of EFs

The current study

According to empirical evidence previously discussed, the study employs a multi-method approach across three levels of functioning: i) topographical (fMRI) examining neural networks linked to common inhibitory factors (Zhang, Peng, Eickhoff, et al., 2021) and transdiagnostic liabilities (McTeague et al., 2017); ii) temporal utilizing ERP waves as neurophysiological markers of self-regulation and psychopathological liability (Bernat et al., 2020); iii) behavioral analyzing RTs and error rates to clarify how MI alterations are overtly expressed across the developmental spectrum. Departing from these considerations, we hypothesized that:

I) specific topographical patterns within self-regulation networks could differentiate internalizing from externalizing conditions associated with SUDs;

II) SUDs and related psychopathology might share common topographical and temporal brain responses during MI tasks;

III) these neural patterns should be replicated in behavioral performances

We utilized a region-of-interest (ROI) and voxel-based approach for fMRI data to isolate self-regulation networks. For temporal and behavioral data, multi-level meta-analyses were conducted to account for the entire developmental spectrum, identifying both shared and distinct mechanisms underlying SUD trajectories.

Methods

Criteria for selecting studies

This meta-analysis was conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines (Page et al., 2021); the study selection flow chart is presented in **figure 1**. To ensure high empirical quality, the analysis specifically targeted studies published in peer-reviewed scientific journals. The literature search

Table 1. *Topographical organization of brain responses related to common inhibition factor and self-regulation system*

Common inhibitory factor		Self-regulation system	
Topographical organization	***Developmental Stage***	***Domain***	***Topographical organization***
rIFG	Adulthood	Motor	rIFG
I-MC	Adulthood		I-MC
pre-SMA	Adolescence, Adulthood		pre-SMA
			Striatum
			Cerebellum
DLPFC	Adulthood	Sensing and Play	DLPFC
VLPFC	Adulthood		VLPFC
			IPS
			PPC
		Speech	STG
			MTG
			IIFG
Insula	Adolescence	Emotion/motivation	Insula
			Amydgala
			AMPFC/VMPFC/OFC
ACC	Adolescence		
PCC	Adolescence		

ACC= Anterior Cingulate Cortex; IFG = inferior frontal gyrus I-MC = Primary Motor Cortex; IPS = intraparietal sulcus; DLPFC = Dorsolater Prefrontal Cortex; MTG = Middle Temporal Gyrus; PCC = posterior cingulate cortex PPC = posterior parietal cortex; pre-SMA = pre-supplementaty Motor Area; STG = Superior Temporal Gyrus; VLPFC = Ventrolateral Prefrontal Cortex; VMPFC = ventromedial prefrontal cortexu; OFC = orbitofrontal cortex

was performed across Scopus, PubMed, PsycINFO, and ISI Web of Knowledge. Specific keywords used for each database are provided in the supplementary materials. Authors M.C. and A.S. independently conducted the online research.

To ensure a reliable initial sample, a double-blind screening process was employed. M.C. and A.S. screened articles based on their abstracts, identifying studies that involved the administration of MI tasks (e.g., Go/No-Go, SST) alongside fMRI or EEG data collection. Inter-rater reliability for the final study selection was assessed using Cohen's kappa (*k*) (Cohen, 1960). Comprehensive inclusion and exclusion criteria for the meta-analytic procedures are detailed in the supplementary materials.

Data analyses

Topographical organization of brain responses: ROI- and voxel-based meta-analysis of fMRI data

A Bayesian hierarchical network meta-analysis was applied for the ROI-based approach. The {gemtc} R package (van Valkenhoef et al., 2012) was used to estimate the pooled effect size (ES) (d_{pooled}) and its 95% credible interval (CrI) for each brain network associated to self-regulation subsystems. A random-effect model was applied. The nodesplit method (Dias et al., 2010) was adopted in order to assess the inconsistency of results within the network, which is represented by one or more significant differences between estimations based on direct and indirect evidence. In presence of inconsistency, separate network meta-analyses for each condition of interest were conducted. The Surface Under the Cumulative Ranking (SUCRA) score (Salanti et al., 2008) was calculated to highlight which self-regulation brain networks could be the most representative for capturing specific responses of each condition of interest.

We conducted a voxel-based meta-analysis using GingerALE 3.0.2 (http://www.brainmap.org/), which enables coordinate-based meta-analysis of fMRI data (Eickhoff et al., 2009; Laird et al., 2005; Turkeltaub et al., 2002). The ALE algorithm models activation foci as 3D Gaussian probability distributions to account for spatial uncertainty. For each study, these distributions were merged into a Modeled Activation Map (MAMap). These maps were then combined to yield voxel-wise ALE scores representing spatial overlap across experiments.

We performed separate analyses based on trial types and groups. Lookig at No-Go Trials, we analyzed increased brain responses in clinical groups versus healthy controls (HCs), and vice versa. This was replicated for specific populations (SUDs, ADHD, and MDD). The same comparative approach was applied to neuroimaging data for Go trials.

Talairach coordinates were converted to MNI space using GingerALE's built-in algorithm. Statistical significance was set at a voxel-level $p < .05$ (1000 permutations, minimum volume 200 mm^3), with Family-Wise Error (FWE) correction applied at the cluster level ($p < .05$) (Eklund et al., 2016).

Temporal organization of brain responses and behavioral performances

A multi-level approach was adopted to meta-analyze findings related to ERP components in responses to experimental conditions (i.e., "No-Go" and "Go" trials). The multilevel meta-analytic procedures were performed through the {metafor} *R* package. This allowed to estimate pooled ESs (d_{pooled}) controlling for interrelationships among multiple ESs calculated within the same study (Viechtbauer, 2010). The estimation of model parameters was based on the restricted maximum likelihood method (Harrer et al., 2021). The 3-level meta-analysis posited that ESs (level 2) were aggregated within clusters composed of each study (level 3).

The *Q* statistic (Hedges & Olkin, 1985) and multi-level I^2 index (Cheung, 2014) were estimated in order to evaluate the heterogeneity in ESs. According to the multi-level version of I^2 index, the total heterogeneity was splitted into a within- (i.e. level 2) and between-study (level 3) variability. Following a multi-level approach, the Akaike (AIC) and Bayesian Information Criterion (BIC) indexes were used to compare the fit to data of the 2-level with the 3-level model through the application of a likelihood ratio test (LRT).

Three-level mixed-effects meta-regressions were conducted to evaluate the impact of several moderators on ESs. Regarding temporal brain responses, we examined specific ERPs (negative: N100 vs. N200; positive: P100, P200, P300, or late waves) and experimental conditions (No-Go vs. Go). Additional moderators included: study characteristics (i.e., publication year , sample size), gender, age, and clinical population (SUDs, ADHD, MDD), task type (GNG vs. SST), % Go trials, stimulus duration, and inter-stimulus interval. Finally, publication bias was assessed using Egger's regression (Egger et al., 1997), with significance estimated via bootstrap procedures

Behavioral outcomes

The multi-level procedures used for temporal brain responses were replicated to analyze behavioral outcomes, specifically RTs, error rates, and correct response rates. In the three-level meta-regression, we explored the moderating effects of: EEG vs. fMRI, commission and omission errors (analyzed both combined and separately) and all other variables previously tested for the temporal organization of brain responses (e.g., age, task type, and clinical population). We applied Z-tests (Borenstein et al., 2011) to compare ESs across distinct behavioral indices: RTs (motor preparation), error rates (motor inhibition), and correct response rates (motor execution). Bonferroni's correction was applied for multiple comparisons.

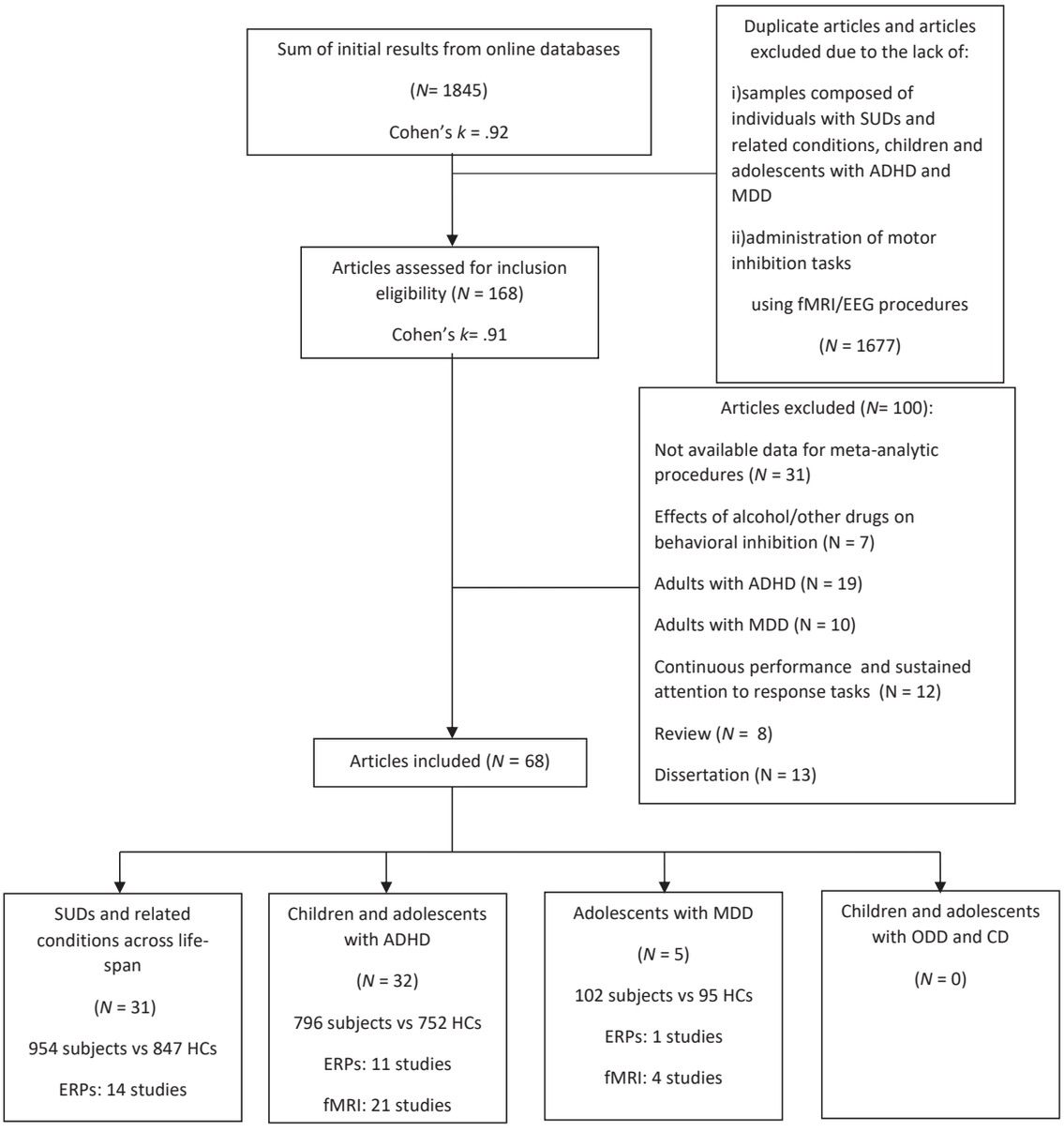
Results

Descriptive statistics

Figure 1 illustrates the study selection process. Sixty-eight independent studies (detailed in **table S1**) were included, involving 3,546 participants. Clinical samples comprise: 954 individuals with SUDs and related conditions, 796 children/adolescents with ADHD, and 102 adolescents with MDD.

Table S2 summarizes sample characteristics, procedures of brain activity acquisition (fMRI, ERPs), and behavioral tasks (GNG, SST). Briefly, 31 studies focused on SUDs and related conditions: 20 on SUDs (*N* = 20; 29.4%; mean age: 33.74), 9 on binge/heavy drinking (*N* =9; 13.4%; mean age: 21.70) and 2 on positive family history (*N* = 2; 2.9%, mean age: 12.55), using fMRI (*N* =17) and ERPs (*N*=14). Thirty-two studies (47.1%) assessed ADHD (mean age: 12.49) via fMRI (*N*=21) and ERPs (*N*=11). The remainder evaluated MDD (mean age: 17.40) using fMRI (n=4) and EEG (n=1). No studies focused on ODD or CD. Regarding behavioral

Figure 1. *CONSORT flow chart of studies inclusion process*



measures, 54 studies (79.4%) employed GNG tasks and 14 (20.6%) used SST. Detailed brain response data (topographical and temporal) for No-Go and Go trials across all groups are reported in **tables S3–S6**.

Topographical organization of brain responses

No-Go trials

ROI-based network meta-analysis

Table 2 details the ROI-based meta-analysis. Initial nodesplit analysis across all conditions vs. HCs revealed significant inconsistency, prompting separate network meta-analyses for each group.

For SUDs and related conditions, nodesplit analysis confirmed consistency. Primary responses to No-Go trials included a marked deactivation of motor self-regulation areas (d_{pooled} = -2.10; 95% CrI: [-3.00 – -1.10]; SUCRA: .99) and heightened dorsal attention network activity (sensing-to-the-self/non-verbal WM: (d_{pooled} = 1.30; 95% CrI: [.87 – 1.70]; SUCRA: .87).

In ADHD, results were consistent. No-Go trials were characterized by large deactivation in the dorsal attention network (d_{pooled} = -1.80; 95% CrI: [-2.40 – -1.30]; SUCRA: .99) and activation of the speech processing network (verbal self-regulation; (d_{pooled} = 2.50; 95% CrI: [1.50 – 3.40]; SUCRA: .98).

Among adolescents with MDD, consistent findings showed a non-significant deactivation of the executive control network (sensing-to-self/non-verbal WM: d_{pooled} = -.77; 95% CrI: [-2.60 – 1.10]; SUCRA: .79) and significant activation of the speech processing network (d_{pooled} = 1.60; 95% CrI: [.14 – 3.10]; SUCRA: .86). **Figure 2** summarizes these representative brain responses by condition.

Voxel-based meta-analysis

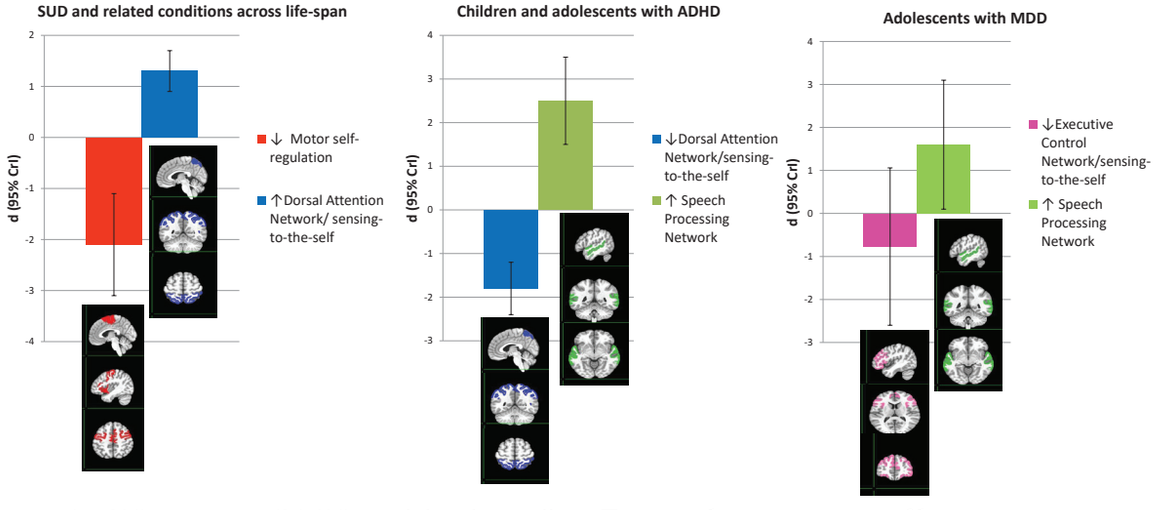
ALE meta-analysis (cluster-based FWE; $p < .05$) revealed that, compared to HCs, clinical groups shared increased responses in the rACC (74.3%) and VMPFC (20.4%; **table S8**). Sub-group analyses (**tables S9–S11**) identified the following recurrent findings: i) SUDs and related conditions showed heightened activity in the

Table 2. *Network meta-analysis results for No-Go trails*

Brain Network	All conditions		SUDs and related conditions across life span		Children and adolescents with ADHD		Adolescents with MDD	
	d (95% CrI) ↑ vs HCs [SUCRA]	*d* (95% CrI) ↓ vs HCs [SUCRA]	*d* (95% CrI) ↑ vs HCs [SUCRA]	*d* (95% CrI) ↓ vs HCs [SUCRA]	*d* (95% CrI) ↑ vs HCs [SUCRA]	*d* (95% CrI) ↓ vs HCs [SUCRA]	*d* (95% CrI) ↑ vs HCs [SUCRA]	*d* (95% CrI) ↓ vs HCs [SUCRA]
Motor	**1.70 (.94 – 2.50) [.93]**	-1.20 (-1.60 – -.82) [.79]	-	**-2.10 (-3.00 – -1.10) [.99]**	1.60 (.81 – 2.40) [.84]	-1.10 (-1.60 – -.64) [.71]	-	-
Emotion / motivation	1.30 (1.00 – 1.60) [.78]	-1.20 (-1.40 – -.96) [.79]	1.10 (.77 – 1.30) [.70]	-1.10 (-1.40 – -.84) [.76]	1.60 (1.20 – 2.00) [.85]	-1.20 (-1.60 – -.88) [.78]	.90 (-1.00 – 2.80) [.69]	-.73 (-2.60 – 1.20) [.76]
Executive Control	1.20 (.82 – 1.60) [.70]	-1.20 (-1.40 – -.96) [.77]	1.30 (.80 – 1.80) [.85]	-1.10 (-1.40 – -.84) [.75]	1.10 (.57 – 1.60) [.67]	-1.20 (-1.60 – -.85) [.77]	-	**-.77 (-2.60 – 1.10) [.79]**
Dorsal Attention	1.30 (.90 – 1.70) [.78]	**-1.40 (-1.60 – -1.10) [.94]**	**1.30 (.87 – 1.70) [.87]**	-1.10 (-1.40 – -.80) [.74]	1.30 (.20 – 2.40) [.75]	**-1.80 (-2.40 – -1.30) [.99]**	1.10 (-.81 – 3.00) [.73]	-.71 (-2.50 – 1.20) [.75]
Speech Processing	1.40 (1.10 – 1.70) [.84]	-1.20 (-1.40 – -.94) [.78]	1.10 (.80 – 1.40) [.73]	-1.10 (-1.50 – -.80) [.73]	**2.50 (1.50 – 3.40) [.98]**	-1.20 (-1.50 – -.84) [.76]	**1.60 (.14 – 3.10) [.86]**	-.80 (-2.70 – 1.11) [.78]

Bold: The most representative brain responses according to SUCRA values

Figure 2. *The most representative topographical brain responses to No-Go trials for each condition of interest (ROI-based network meta-analysis)*



rACC (55.2%), VMPFC (24.4%), and basal ganglia (16.1%); ii) ADHD highlighted increased responses in the cingulate gyrus (67.2%; rostral/dorsal ACC) and motor areas (23.7%; I-MC and SMA): iii) MDD adolescents showed elevated activity in the dorsal ACC (62.5%) and rIFG (57.2%). The ALE algorithm yielded no significant deactivations, neither across all conditions nor within individual groups.

Go trials

Due to the small number of studies (N = 3) and limited conditions of interest (i.e., children with ADHD or a positive family history for SUDs), ROI- and voxel-based meta-analytic results for Go trials are provided as supplementary materials (**tables S11–S12**, **figure S1**).

Temporal organization of brain activity

Table S13 details the multi-level meta-analysis for ERP components in No-Go and Go conditions. Regarding negative waves, the primary finding was a significant, frontal N200 amplitude reduction across all clinical groups (d_{pooled} = .29 [.06 – .52]; p <.01). Results were heterogeneous ($Q_{(115)}$ = 365.15; p< .001; $I^2_{\text{Level 3}}$ = 75.88%) with detectable publication bias.

For positive components, clinical groups showed significant P300 amplitude reductions at central (d_{pooled} = -.33 [-.64 – -.03]; p <.05) and parietal (d_{pooled} = -.37 [-.57 – -.17]; p <.001) sites, regardless of experimental condition. Heterogeneity was significant and large ($Q_{(132)}$ = 464.27; p< .001; $I^2_{\text{Level 3}}$ = 79.14%), though no publication bias was detected. **Figure 4** summarizes these findings.

Figure 3. *Topographical brain responses to No-Go trials (ALE meta-analysis)*

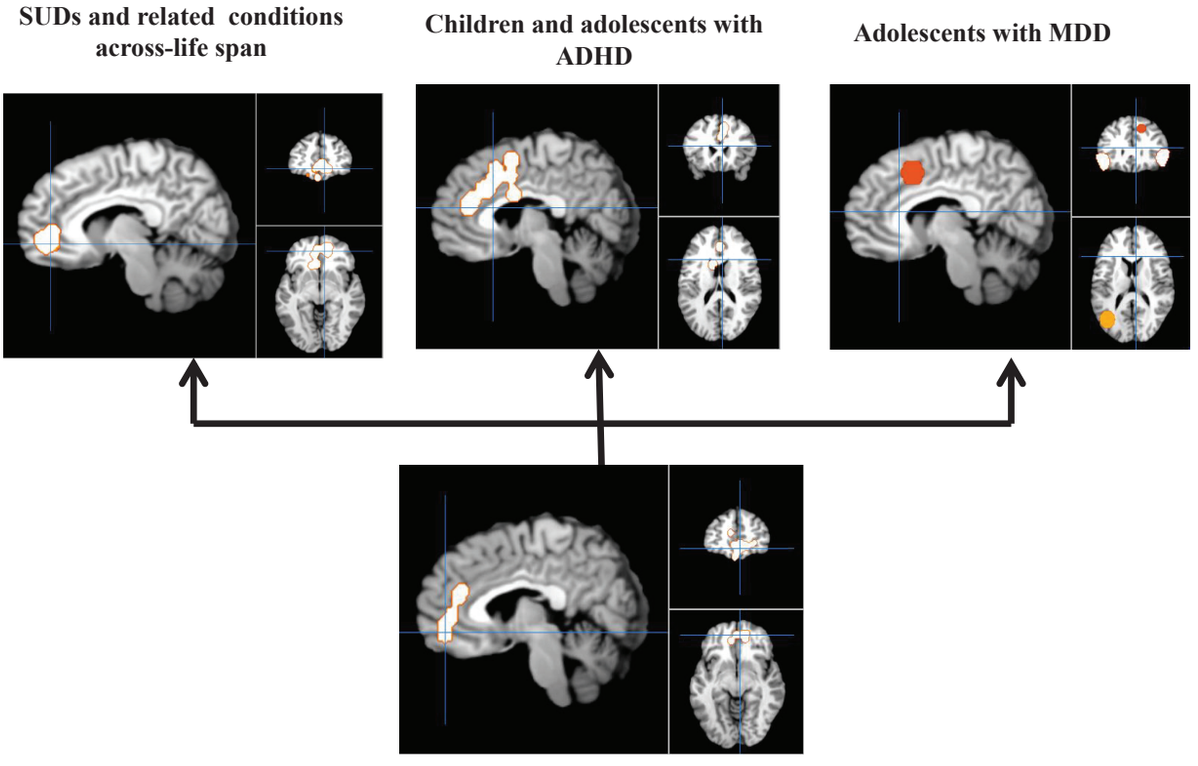
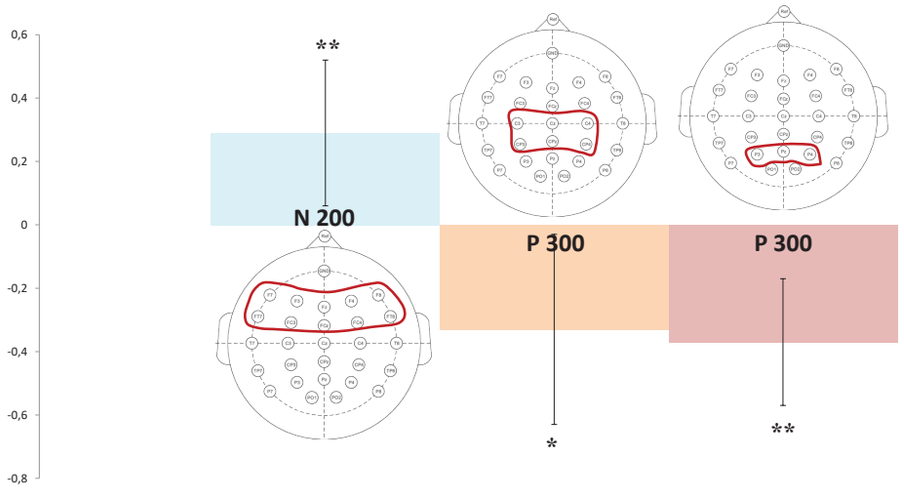


Figure 4. *Temporal organization of brain activity in response to No-Go and Go trials among conditions of interest compared to HCs*



*$p < .05$; **$p < .01$

Behavioral outcomes

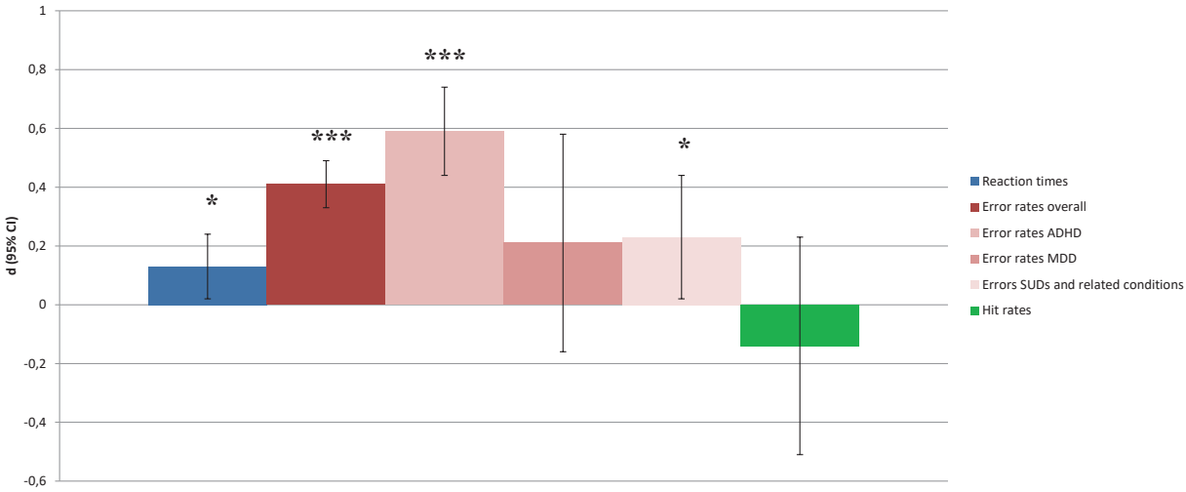
Table S14 details the multi-level meta-analysis of behavioral performances. Regarding RTs, clinical groups were significantly slower than HCs (d_{pooled} = .13 [.02 – .24]; p < .05) across both No-Go and Go trials. Results were moderately heterogeneous ($Q_{(75)}$ = 127.54; p < .001; $I^2_{Level\ 2}$ = .00%; $I^2_{Level\ 3}$ = 48.99%), with no significant moderators or publication bias detected.

Error rates showed a small-to-moderate increase in clinical groups vs. HC (d_{pooled} = .41 [.29 – .53]; p < .001). Heterogeneity was significant ($Q_{(66)}$ = 105.01; p < .001; $I^2_{Level\ 2}$ = .00%; $I^2_{Level\ 3}$ = 42.09%), explained by clinical condition as a moderator. Accordingly, sub-group analyses revealed: i) moderate ES for ADHD (d_{pooled} = .59 [.44 – .74]; p < .001); ii) small but significant ES for SUDs and related conditions (d_{pooled} = .23 [.02– .44]; p < .05); iii) non-significant pooled ES for MDD.

Direct comparison of absolute pooled ESs showed that error rates were significantly more affected than RTs (Z = 3.58; p < .001). Specifically, the ADHD group exhibited the poorest behavioral performance,

significantly exceeding both the SUD (Z = 2.76; $p < .01$) and and MDD groups (Z = 1.88; $p < .05$). No significant difference was found between SUD and MDD (Z = .09; *ns*). **Figure 5** summarizes these behavioral outcomes.

Figure 5. *Behavioral outcomes among conditions of interest compared to HCs*



*$p < .05$; ***$p < .001$

Discussion

The current study clarifies common and distinct neuro-behavioral dimensions of MI in SUDs and related conditions, ADHD, and MDD—conditions developmentally linked through shared self-regulation deficits (Barkley, 1997, 2001). This meta-analysis yields four primary findings:

I) brain responses to No-Go trials distinctively differentiate SUDs, ADHD, and MDD based on self-regulation domains (ROI-based meta-analysis)
II) heightened rACC and VMPFC responsiveness during MI demands is common across all conditions and developmental stages (voxel-based meta-analysis)
III) reduced N200 and P300 amplitudes are shared across clinical groups.
IV) all clinical groups show slower RTs; whereas, higher error rates are specific to externalizing conditions, namely ADHD and SUDs.

ROI-based results reveal that SUDs are characterized by motor self-regulation hypo-activity (e.g., rIFG, pre-SMA, basal ganglia) and increased recruitment of the dorsal attention network (non-verbal working memory). This aligns with literature linking rIFG dysfunction to loss of control on substance-use behaviors and relapse (Goldstein & Volkow, 2011), while the dorsal attention network relates to substance-specific attentional biases (Field et al., 2014). Conversely, ADHD involves a large deactivation of the dorsal attention network, consistent with core deficits in self-regulation of attention (Castellanos & Proal, 2012). Notably, both ADHD and MDD share an increased recruitment of the speech processing network (verbal self-regulation) during No-Go trials. This suggests that individuals with atypical development may compensate for MI deficits by relying on inner speech and semantic processing (Langland-Hassan, 2021). Because these high-order functions impose a heavy cognitive load (Carruthers, 2002), MI becomes highly demanding, creating a vulnerability factor for broader behavioral regulation failure (Baumeister & Vohs, 2007).

ALE meta-analysis revealed shared topographical responses across all conditions, specifically involving the rACC and VMPFC. These results align with prior findings (Goodkind et al., 2015; McTeague et al., 2017) identifying the anterior ACC as a neurobiological signature of psychopathological liability and their developmental pathways (Caspi et al., 2014; Smith et al., 2020). The joint recruitment of the rACC and VMPFC suggests that MI demands are processed with high emotional valence (Bush et al., 2000; D'Argembeau, 2013). This supports the hypothesis that inhibiting prepotent responses requires significant emotional and mental effort (Inzlicht et al., 2018), regardless of age or diagnosis. Such neural organization — linked to the emotional/motivational self-regulation domain — may provide the substrate for MI difficulties described by the ego depletion model (Baumeister, 2003). Within this framework, the high subjective effort needed to intentionally inhibit actions exhausts limited cognitive and emotional resources, thereby increasing the likelihood of automatic and impulsive behaviors (Baumeister, 2003).

Current findings also reveal a shared reduction in frontal N200 and centro-parietal P300 amplitudes across all clinical groups and developmental stages, regardless of task demands (Go/No and Go trials). These results suggest alterations in self-regulation mechanisms governing both motor inhibition and execution. The frontal N200 is linked to conflict monitoring and motor organization (Huster et al., 2013), while the P300 reflects the inhibition of task-irrelevant neural activity (Polich, 2007). Together, these temporal brain activity alterations represent a stable neurophysiological marker shared across internalizing and externalizing conditions (Bernat et al., 2020), potentially underpinning the diverse developmental trajectories toward SUDs.

These temporal deficits are mirrored behaviorally. All clinical groups exhibited significantly slower RTs, likely reflecting impaired action preparation and the failure to inhibit internal or external distractions (Wright et al., 2014). Conversely, error rates varied by clinical condition, with impairments significantly more pronounced in externalizing conditions, particularly during early stages of development (ADHD). This

behavioral gradient aligns with the previously discussed ROI-based results, confirming that while some self-regulatory deficits are common dimensions among SUDs, ADHD and MDD during childhood and adolescence; on the other hand, the severity and quality of MI impairment differentiates specific clinical from each other.

Despite these findings, several limitations warrant discussion. First, the cross-sectional nature of the meta-analyzed data precludes definitive conclusions regarding the causal transition from childhood conditions to later SUDs. Longitudinal neuroscience studies are required to empirically validate the developmental considerations discussed herein.

Second, the small number of studies on adolescents with MDD (N=5) and the total absence of MI research on ODD and CD — critical externalizing precursors to SUDs (Colder et al., 2018) — limit the generalizability of these results. Future research must target these populations to clarify how their specific self-regulation profiles predict substance-use progression.

Third, significant methodological heterogeneity remains. Variations in experimental control conditions (e.g., No-Go vs. baseline or Go) were not controlled for due to insufficient study counts. Similarly, the scarcity of fMRI data regarding Go trials (N=3) highlights the need for research that balances the study of behavioral inhibition with goal-oriented execution.

Fourth, comorbidity effects could not be isolated. The clinical samples exhibited high heterogeneity, and SUD studies often lacked lifetime psychiatric histories, complicating the identification of specific neuro-behavioral underpinnings for homotypic versus heterotypic developmental pathways.

Finally, while current literature focuses on task-dependent responses, future research should integrate resting-state organization with task-related activity (Northoff et al., 2023). Investigating how baseline brain activity influences MI responses across development remains a relevant frontier for understanding self-regulation and its implications for SUDs.

Nevertheless, this is the first study to delineate the topographical and temporal neuro-behavioral alterations underlying self-regulation in the context of homotypic and heterotypic developmental pathways to SUDs. Specifically, shared patterns — such as ACC recruitment and reduced N200/P300 amplitudes — may represent the neural substrates of a common liability to these conditions. Conversely, our ROI-based analysis demonstrates that condition-specific self-regulation domains support the phenomenological differences observed across clinical groups (**figure 6**). These distinct processes should be prioritized as key targets for clinical intervention. Furthermore, SUD prevention programs should focus on reorganizing these self-regulatory networks to mitigate the emergence of substance-use behaviors starting in early adolescence.

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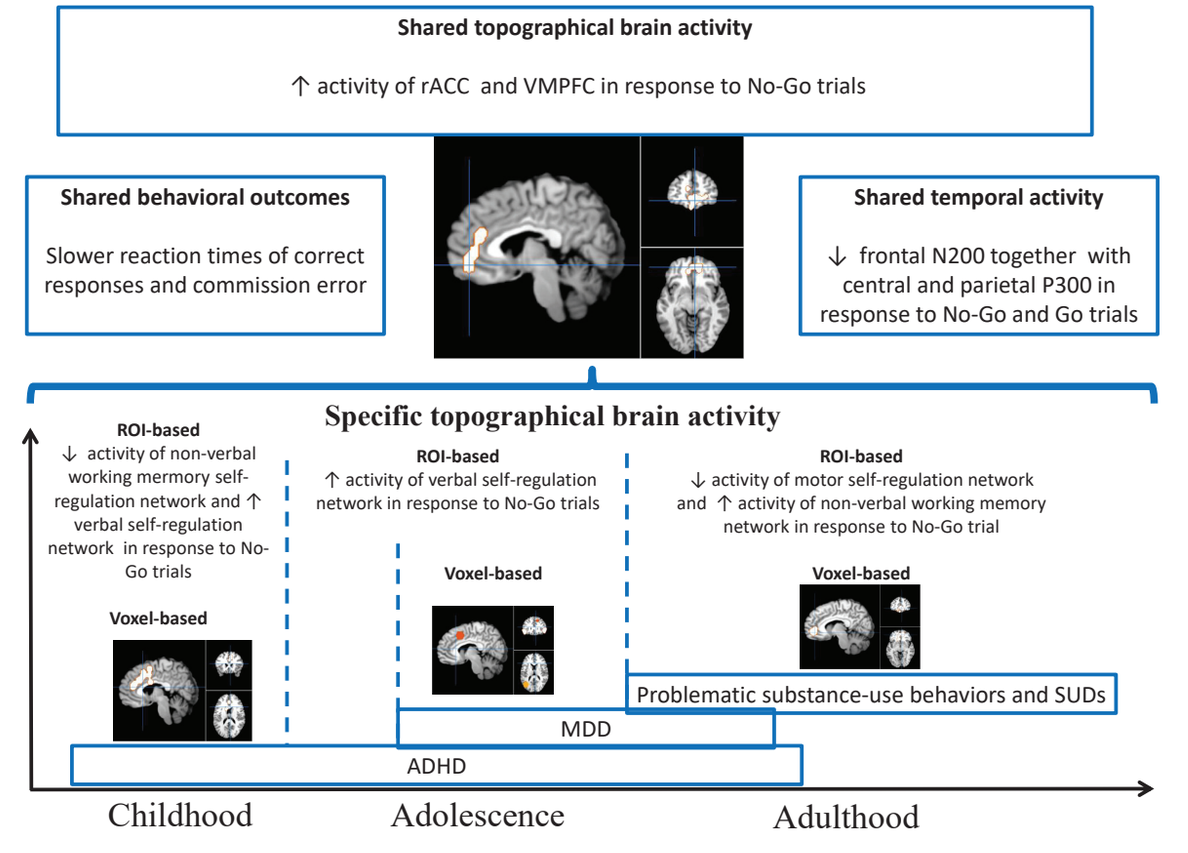
Figure 6. *Summary of neuro-behavioral findings associated to self-regulation and developmental pathways of SUDs and related problems in response to No-Go trials. Bottom of the figure show the specific topographical brain activity for ADHD, MDD and SUD during different stages of development. Top of the figure shows common neuro-behavioral dimensions*



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