Introduction

Obsessive-compulsive disorder (OCD) is recognized as a prevalent and persistent neuropsychiatric condition, impacting an estimated 2% to 3% of individuals worldwide (de Mathis et al., 2013). The disorder commonly arises in early life and is characterized by the presence of compulsions – ritualized behavioral or mental acts, and obsessions – intrusive and unwanted thoughts and worries (Karno et al., 1988). OCD is unique among mental illnesses in that it exhibits both externalizing and internalizing symptoms (Guzick et al., 2019). Externalizing features, like compulsivity and repetitive actions, are often outwardly disruptive and align with disorders such as attention-deficit/hyperactivity disorder (ADHD) and disruptive behavior disorders. In contrast, internalizing aspects, including anxiety, concerns, and obsessions, cause internal distress and align with conditions like depressive and anxiety disorders, often leading to avoidance or withdrawal (Achenbach, 2001). Understanding OCD within this dual framework enhances our grasp of its complexity and informs more effective therapeutic strategies. These frameworks are not only therapeutically beneficial but are also supported by empirical research (Kessler et al., 2011; Slade & Watson, 2006).

The prevalence of OCD in childhood varies between 0.1% and 4% (Douglass et al. 1995; Fleitlich-Bilyk and Goodman 2004) and previous studies report that functional impairment (de Bruijn et al. 2010; Canals et al. 2012) and several psychiatric comorbidities are associated not only with full blown OCD, but also with subclinical OCD and obsessive-compulsive symptoms (OCS) as well (Flament et al. 1988; Zohar et al. 1992; Heyman et al. 2001; Shams et al. 2011; Alvarenga et al. 2015). Moreover, longitudinal studies suggest that OCS can persist over time (Fullana et al. 2009) and increase the risk of developing OCD (Black and Gaffney 2008; Fullana et al. 2009), which supports a dimensional approach during evaluation. (Saad et al., 2017)

While OCD was historically considered a singular nosologic entity, increasing evidence highlights its clinical and etiological heterogeneity (3–7). This reconceptualization suggests that OCD comprises multiple symptom dimensions, potentially overlapping and rooted in distinct neural systems, such as the cognitive control circuit (frontal cortex, thalamus, and striatum) (10W,11W), attention circuit (parietal cortex) (12,13), and emotional circuit (amygdala) (14W). (Wu et al., 2022)

Among 1938 healthy adolescents from the IMAGEN study, compulsive behaviors (including OCD and eating disorder symptoms) were associated with greater ventral striatal, orbitofrontal, and dorsolateral prefrontal cortex gray matter volume (16P). While much work suggests that disruptions in corticostriatal-thalamo-cortical circuits are central to OCD pathophysiology, [e.g., (17P)], leading to deficits in controlling obsessions and urges to perform rituals, emerging evidence highlights broader circuit alterations, [e.g., (18P)]. Particularly, meta-analytic work suggests OCD-related alterations in connectivity within and between the executive network, salience network, and default mode network (DMN) (19P), in line with a proposed triple network model of psychopathology (20P). Furthermore, reviews examining research on pediatric OCD highlight structural, functional, and connectivity alterations in both frontostriatal and task-control neural circuits (21–23P). For example, findings from our group suggest alterations in cingulo-opercular structural connectivity (24P) and functional connectivity between DMN and task-positive network regions in pediatric OCD (25P). The ENIGMA (Enhancing Neuro Imaging Genetics through Meta Analysis) OCD consortium noted larger thalamic volumes (26P) (201 healthy control subjects vs. 103 unmedicated subjects with OCD) and thinner parietal cortices (27P) in youths with OCD compared with healthy control subjects. (Pagliaccio et al., 2021)

Subclinical obsessive-compulsive symptoms (OCSs) in childhood predict increased risk for obsessive-compulsive disorder (OCD) diagnosis in adulthood (odds ratio [OR] w5–10) (1P). Critically, OCSs are common in children and adolescents and, even below clinical threshold, are associated with impairment and comorbidity (1–7P). Childhood OCSs exhibit moderate stability, with some ebb and flow within individuals over the course of development (4,8P). Cross-sectional research generally suggests greater OCS prevalence in middle versus early childhood, (3P) whereas longitudinal work indicates weak persistence of OCSs during this developmental period (4,8P). Uncovering neural and cognitive mechanisms underlying OCSs and their variable course over childhood could help improve early identification of children most at risk for OCD. . (Pagliaccio et al., 2021)

Few pediatric studies have examined neural or cognitive mechanisms underlying OCSs or subsequent OCD risk. . (Pagliaccio et al., 2021)

Several relatively small studies in healthy adults associate OCSs with executive function deficits, e.g., cognitive flexibility (9,10P), spatial problem (11,12P), and response inhibition (13P) [also see (14P)]. . (Pagliaccio et al., 2021)

Likewise, few pediatric studies have related OCSs to brain structure and function. Among 255 healthy 8- to 12-year-olds, overall OCS scores did not associate with voxelbased morphometry, whereas subscales showed differential associations, e.g., ordering symptoms versus doubt-checking symptoms (15P). (Pagliaccio et al., 2021)

Diagnosis of Children With OCD-like Symptoms by the CBCL OCS Scale The CBCL is a parent-reported questionnaire in widespread use to measure behavioral problems for school-aged children (27–29) in large-scale population studies such as the ABCD Study (30,31). The OCS scale is a subset of the CBCL items that is associated with OCD symptoms, which has been shown to be effective in distinguishing subjects with OCD from subjects without OCD (32–34) and demonstrates good internal consistency and longitudinal stability (33,35). There are four OCS scale versions extracted from CBCL, named OCS-2, OCS-6, OCS-8, and OCS-11 and containing several overlapping subitems (Table 1) (32).(Wu et al., 2022).A close-up of a medical report

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Gathering information about a child’s functioning typically involves input from multiple informants, including the child and parents (Achenbach, 2006). Mental health issues can vary across different contexts (Bauducco et al., 2024; Beesdo et al., 2009). Children and adolescents may exhibit mental health concerns in certain environments, such as at home or school, but not in others, like during peer interactions. These contextual variations are evident across various domains, including conduct problems, attention, hyperactivity, and anxiety (Beesdo et al., 2009).

However, the reliability of parent reports for assessing children's experiences, especially for non-observable functions like emotions, has been questioned (Eiser & Morse, 2001). Parental assessments often differ from children’s self-perceptions, potentially due to biases, superficial observations, or the nature of the parent-child relationship. Conversely, children frequently lack objective self-perception (Barrett et al., 1991; Martin et al., 2004). Research indicates discrepancies and varying accuracy in symptom reporting, with no clear consensus on which group reports internalized symptoms more accurately, while parents tend to be more precise in identifying externalized (Silverman & Eisen, 1992).

As a secondary analysis, we examined the relationship between obsessive-compulsive symptoms as assessed by the CBCL and our OCD diagnostic constructs. We found that, in general, there were strong associations between all symptom-based and diagnostic constructs, suggesting that these constructs are indeed capturing the same underlying clinical phenomena. However, there were a few differences for associations between the CBCL, which is a parent-completed questionnaire, and nOCD or bnOCD, suggesting that the CBCL, while potentially useful as a dimensional measure of obsessive-compulsive symptoms (OCS), does not help to further discriminate “true” OCD from subclinical symptoms. The exception to this was the obsessions question on the CBCL, which was much more strongly correlated with nOCD than with bnOCD. This finding may suggest that obsessions, which are internalizing symptoms and often only identified by the affected child, may, when prominent enough to be recognized by parents, be a key discriminating feature of OCD versus OCS. (Ivankovic et al., 2024)

Research question: To what extent does structural brain data explain the variation in OCD symptoms as reported by youths versus their parents?

Hypothesis:  There will be a significant difference in the prediction accuracy of structural brain data between self-reported and parent-reported anxiety symptoms in adolescents with OCD, with an expectation of higher accuracy for self-reported symptoms.

1. **Integrative Assessment of OCD Symptoms: Concordance and Discrepancies Across Informant Reports and Structural MRI Correlates**
   * **Objective**: Examine the agreement and discrepancies among parent (CBCL), child (BPM), and teacher (BPM) reports of OCD symptoms, while exploring their associations with structural brain markers.
   * **Approach**: Analyze the consistency of symptom reporting across three informant perspectives and correlate these with neural metrics, such as cortical thickness or subcortical volume, especially in regions implicated in OCD.
   * **Expectation**: Greater alignment between specific reports and certain brain regions, offering insights into which informant reports may reflect the underlying neural dysfunction more accurately.

* **Discrepancy Score**:
  + **Objective**: Identify the degree of disagreement or discrepancy between the informant reports.
  + **Measure**: Compute a discrepancy score reflecting the divergence of scores between different informants (e.g., absolute difference or variance).
  + **Model Usage**: Use XGBoost to predict these discrepancies from neural markers and explore which brain features are associated with higher disagreements.
* **OCD Severity Prediction**:
  + **Objective**: Direct prediction of OCD severity as an aggregate measure derived from the various informant reports.
  + **Measure**: Create a composite severity score from CBCL, BPM-child, and BPM-teacher reports and analyze it as a continuous outcome.
  + **Model Usage**: Predict this composite score using brain structures and other covariates, interpreting brain features that contribute to perceived severity.

1. **Predictive Value of Multi-Informant Reports in Identifying Structural Brain Changes in Pediatric OCD**
   * **Objective**: Evaluate the predictive value of combined parent, child, and teacher reports in explaining variations in brain structure associated with OCD.
   * **Approach**: Use regression models to assess which combination of informant ratings best predicts structural changes in neural circuits associated with OCD, such as the frontal-striatal circuitry.
   * **Expectation**: Identification of a composite score that incorporates different perspectives, enhancing prediction of structural MRI findings related to OCD pathophysiology.

Methods

Used data from only year 2 follow up

Demographics Table:

Age

race

gender

SES

Usable MRI

CBCL OCS sum

OCS t score

Medicated/ Unmedicated

Clinical Presentation Table:

### Commonly Reported Demographic Variables:

1. **Age:**
   * Report the mean age and age range to provide a clear picture of the sample's developmental stage.
2. **Gender:**
   * Report the proportion or percentage of male and female participants. If your dataset includes non-binary or other gender categories, include those as well.
3. **Ethnicity/Race:**
   * Provide a breakdown of the racial and ethnic composition of your sample. This could include categories such as White, Black or African American, Asian, Hispanic or Latino, and others depending on the dataset's coding.
4. **Socioeconomic Status (SES):**
   * SES can be reported using variables like parental education level, household income, or an SES index if available.
5. **Geographical Location:**
   * If relevant, describe the geographical distribution of the sample, such as urban vs. rural residence or regions/states represented.
6. **Family and Household Structure:**
   * Include information on the family structure (e.g., single-parent households, guardianship) and household size if relevant.

### Additional Variables for Specific Samples (e.g., OCD):

1. **Duration or Age of Onset:**
   * If focusing on a clinical sample like OCD, you might report the average age of onset of symptoms and duration of disorder.
2. **Clinical Measures or Scales:**
   * Include any assessment scores, such as severity scales specific to OCD, if part of your dataset.
3. **Comorbid Conditions:**
   * Report on any other diagnosed mental health conditions that are prevalent within your sample.

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Sample

Limitations  
Narrow diagnostic constructs, which required the endorsement of psychiatric disorders at a majority of longitudinal assessments, demonstrated a better rate approximation of literature-reported prevalences for most disorders (e.g. the prevalence of broad obsessive-compulsive disorder (OCD) was 13.3% compared to narrow OCD at 2.6% and a literature-reported prevalence of 2.3%). Analysis of comorbidity, using OCD as a representative example, also showed a better approximation of literature-reported comorbidity rates using the narrow construct, with some exceptions. Conclusions: Self- or parent-report-based assessments tend to overestimate prevalences of psychiatric disorders in the ABCD Study, particularly when longitudinal data are summed to create lifetime prevalences. Such assessments should be accompanied by more in-depth assessments or clinician-administered structured interviews if using data where accurate disorder classifications are paramount (Ivankovic et al., 2024).

(Context: over reporting symptoms in ksads) Low positive agreement rates can be particularly impactful on statistical power in downstream analyses, especially in study designs or analyses where false-positive diagnoses can substantially dilute already small or weak effect sizes. (i.e. unaffected participants misclassified as affected), can drasƒitically increase the sample size requirements to meet the desired statistical power by increasing the noise of the case sample group (Manchia et al., 2013). (Ivankovic et al., 2024)