Eur J Psychiat. 40 (2026) 100337

Contents lists available at ScienceDirect

The European Journal of Psychiatry

journal homepage: www.elsevier.com/locate/ejpsy

Late-Onset ADHD symptoms in the general population: A scoping review of longitudinal trajectories in population-based cohorts

Sophie Bayard [a,*], Clarisse Madiouni [a], François Radiguer [b], Maëva Roulin [c], Sébastien Henrard [d]

[a] Univ Paul Valéry Montpellier 3, Univ Montpellier, EPSYLON EA 4556, F34000, Montpellier, France
[b] Urgences Cérébro-vasculaires, Université Paris-Sorbonne, AP–HP, CHU Pitié-Salpètrière, Paris, France
[c] Centre Genepsy, Romans-sur-Isère, France
[d] Payrac, France

ARTICLE INFO

Keywords:
ADHD, late onset
Trajectories, scoping review
Longitudinal
Population-based
Sleep
Developmental psychopathology

ABSTRACT

Background and objectives: The developmental validity of late-onset ADHD remains debated. This scoping review aimed to synthesize evidence from longitudinal trajectory studies in the general population to (1) examine the existence and characteristics of late-onset ADHD symptom trajectories, and (2) identify associated factors.
Methods: Following PRISMA-ScR guidelines, we systematically reviewed longitudinal studies published up to May 2025 that applied person-centered trajectory modeling to ADHD symptoms in population-based cohorts. Twelve eligible studies were included.
Results: Among the twelve studies included in this scoping review, ten identified at least one symptom trajectory consistent with late-onset ADHD, with prevalence estimates ranging from 3 % to 17.5 %, and were analyzed to examine associated factors. These late-onset trajectories were predominantly inattentive and less disruptive than persistent profiles, but consistently observed across cohorts but were reported in the majority of cohorts (10/12), not all. The most robustly supported associated factors included higher childhood cognitive functioning, fewer early externalizing problems, emerging internalizing psychopathology during adolescence, lower family socio-economic status, female gender, and polygenic liability for ADHD. However, methodological variability, particularly in informant source, symptom measurement and model specification, significantly influenced identification. Notably, no study assessed sleep-wake disturbances, treatment history, contextual supports or neurological antecedents.
Conclusion: Late-onset ADHD trajectories are commonly identified in general population cohorts (10/12 studies). Their interpretation calls for a dimensional, developmentally sensitive framework. Future studies should incorporate overlooked domains such as sleep-wake functioning, treatment exposure, and neurological risk factors to refine clinical characterization and diagnostic criteria.

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is classified as a neurodevelopmental condition that typically emerges in childhood, usually before the age of 12, according to the DSM-5.[1] However, growing evidence in the literature points to the existence of *late-onset* ADHD, defined as the emergence of symptoms during adolescence or early adulthood.[2] This alternative presentation challenges current nosological frameworks and raises important questions about whether late-onset cases represent a previously unrecognized variant of ADHD or a diagnostically distinct condition.[2,3]

At the heart of this controversy lies the issue of diagnostic validity. Do such cases constitute a genuine developmental subtype of ADHD, or are they better explained by measurement artifacts, recall bias, or diagnostic misclassification? [4,5] Some researchers argue that symptoms emerging later in development may reflect earlier subclinical difficulties that were masked, undervalued, or effectively compensated for during childhood.[6] This "compensation" or "scaffolding" hypothesis is supported by findings linking late-emerging ADHD symptoms to higher intellectual functioning and greater childhood resources.[5,7] The

* Corresponding author at: Laboratoire Epsylon EA 4556, Université Montpellier 3, Rue du Pr. Henri Serre, 34000 Montpellier, France.
E-mail address: sophie.bayard@univ-montp3.fr (S. Bayard).

https://doi.org/10.1016/j.ejpsy.2025.100337
Received 17 August 2025; Accepted 30 November 2025
Available online 9 January 2026

developmental transition from parental to self-assessment also introduces methodological challenges, as discrepancies between informants may artificially suggest a later onset.[2]

In parallel, it remains possible that some apparent cases of late-onset ADHD reflect alternative psychiatric conditions, such as depression or anxiety, remains under consideration. These disorders share overlapping symptoms (e.g., inattention, restlessness) and may confound diagnostic assessments, especially when based on self-report questionnaires.[8,9] However, the strict exclusion of comorbid conditions from late-onset definitions has been criticized for being overly simplistic, given the high rates of comorbidity observed in ADHD across the lifespan.[10,11] Despite rigorous control for comorbidities, approximately 2–3 % of participants in the Dunedin Multidisciplinary Health and Development Study still exhibited a late-onset ADHD syndrome emerging in adolescence or early adulthood.[12] These individuals may therefore represent valid cases of late-onset ADHD.

Given this complexity, some authors advocate or more flexible, data-driven approaches to ADHD classification that account for the heterogeneity of developmental trajectories, rather than relying on rigid age-of-onset thresholds.[5,13–15] From this perspective, late-onset symptoms may reflect the broader variability of ADHD rather than a distinct clinical entity. Contextual factors in adolescence, such as increased academic or social demands, may reveal previously masked vulnerabilities. More broadly, the debate raises questions about whether ADHD is a fixed childhood-onset disorder or a dynamic condition that evolves across development. Clarifying these issues will require integrative approaches that move beyond rigid age-of-onset criteria and incorporate multiple sources of information, contextual factors, and dimensional models of symptom expression.[2,16]

In this context, longitudinal trajectory analyses in population-based cohorts have become essential for understanding ADHD as a developmentally heterogeneous and dynamic condition.[16,17] Unlike clinical samples or cross-sectional designs—often biased by retrospective reporting or referral patterns, trajectory-based approaches provide a more ecologically valid view of symptom emergence, persistence, and remission over time.[18,19] Methods such as latent class growth analysis (LCGA) or growth mixture modeling (GMM) identify subgroups based on symptom progression, enabling a nuanced examination of atypical profiles such as late-onset ADHD.[12] The use of representative samples improves generalizability and supports the exploration of diverse cognitive, psychosocial and environmental correlates.

To date, no systematic synthesis has mapped the various ADHD symptom trajectories that emerge during adolescence or early adulthood in the general population. Given the heterogeneity of cohort designs, symptom definitions, and modeling strategies, a scoping review was deemed appropriate to comprehensively map existing evidence and explore the definitional contours and variability of the late-onset ADHD construct.[20] More specifically, this review aimed to (1) synthesize evidence from population-based longitudinal trajectory studies reporting symptom profiles compatible with late-onset ADHD; and (2) identify and organize the associated factors reported across these studies. These aims were addressed through two research questions: "*What evidence from longitudinal trajectory studies in the general population supports the existence of late-onset ADHD*?" The second question examined was: "*What is currently known about the associated factors of late-onset ADHD based on these studies*?". By clarifying the characteristics and factorts of late-onset ADHD symptom trajectories, we will attempt to contribute to the ongoing conceptual debate on the emergence of ADHD in adolescence and young adulthood.

Methods

Protocol and registration

This scoping review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) guidelines.[20] The review did not involve stakeholder consultation and was not registered in a protocol repository.

Eligibility criteria

The inclusion criteria were designed to identify studies investigating ADHD symptom trajectories in the general population. These criteria prioritized longitudinal designs utilizing trajectory modeling and including analyses of factors associated with these diverse symptom patterns. In practice, all longitudinal trajectory studies identified in the general population fulfilled this criterion; therefore, no study was excluded for not examining associated factors. Importantly, studies were not required to identify a late-onset or delayed-onset subgroup to be included; rather, inclusion was based on modeling ADHD symptom trajectories, regardless of the specific trajectory patterns observed. See Table 1 for a detailed description of the eligibility criteria.

Information sources

A comprehensive literature search was conducted across four electronic databases: PsycINFO, PubMed, Web of Science, and Scopus, from database inception up to May 2025. The following Boolean search strategy was applied: ("ADHD" OR "attention deficit hyperactivity disorder") AND ("late-onset" OR "adult-onset" OR "emerging in adulthood" OR "adolescent-onset") AND ("trajectory" OR "trajectories" OR "longitudinal analysis" OR "growth mixture modeling" OR "latent class growth analysis" OR "developmental course").

Data source selection process

Two reviewers (S.B. and C.M.) independently screened all records at each stage of the review process. Of the 1132 records retrieved, 182 duplicates were removed. A total of 950 titles and abstracts were screened, and 32 full-text articles were assessed for eligibility based on predefined inclusion criteria (Table 1). Ultimately, 12 studies were retained for inclusion in the final synthesis. This process is illustrated in Fig. 1, in accordance with PRISMA-ScR guidelines.

Data charting process

Data were extracted independently by S.B and C.M using a standardized charting form. This form captured key study characteristics, including cohort name, sample size, country, age at assessment, ADHD assessment method, number and type of identified trajectories, and the associated factors examined. The form was piloted on a subset of studies

Table 1
Inclusion criteria for the selected studies.

Criterion	Details
Publication type	Peer-reviewed empirical articles; published in English or French; original studies (not reviews, editorials, theses or protocols without results).
Population	General population samples with participants assessed at least twice during childhood, adolescence, and/or early adulthood. Studies restricted to clinical or referred samples were excluded.
Phenotype	Assessment of ADHD symptoms (inattention and/or hyperactivity–impulsivity) or formal diagnosis based on DSM-5 criteria.
Methodology	Longitudinal designs employing developmental trajectory modeling (e.g., Latent Class Growth Analysis, Growth Mixture Modeling, Latent Class Analysis) or equivalent statistical approaches.
Study objectives	Examination of factors associated with symptom trajectories (e.g., cognitive, emotional, social, perinatal, or genetic), or analysis of subtypes including delayed or adolescent-onset patterns.
Publication period	No restriction on publication year. Articles published up to the final search date in May 2025 were eligible.

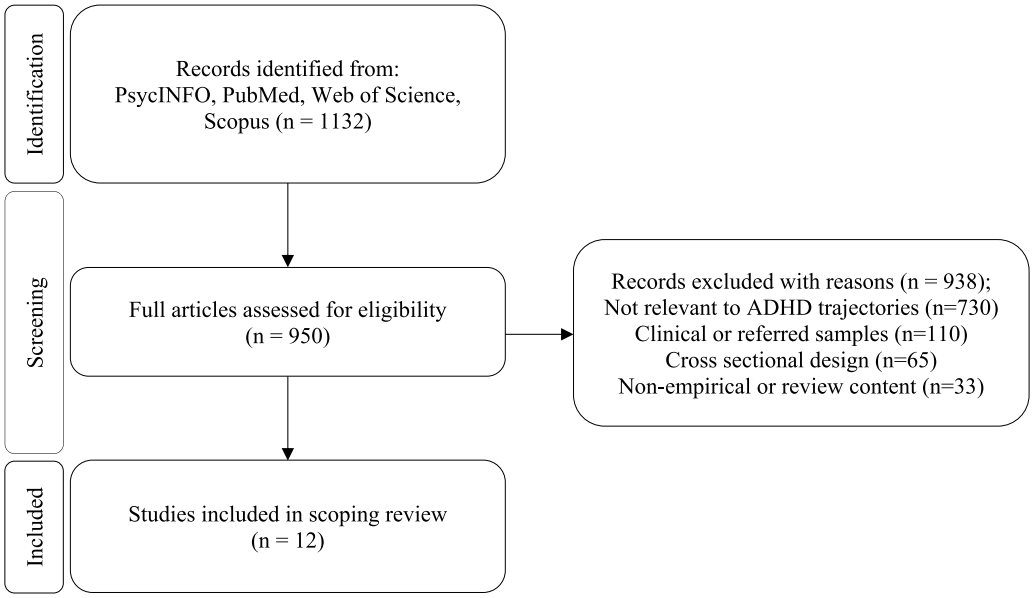

Fig. 1. PRISMA flow diagram showing the selection process of the included articles.

to ensure consistency and clarity.

Data synthesis

Data extracted from each included study were systematically recorded and organized to capture key study characteristics, identified ADHD symptom trajectories and associated factors. First, ADHD symptom trajectories were synthesized by grouping studies according to the same birth cohort. We independently identified the type and number of trajectories reported in each study. Prior to data extraction, they agreed on predefined definitions for the main trajectory patterns (e.g., persistent, remittent, late onset) to ensure a consistent classification framework. These trajectories were then classified according to these predefined patterns to facilitate comparisons between studies. Any discrepancies in classification were discussed until consensus was reached. Where available, prevalence rates by gender, ADHD symptom and informant type were reported. Secondly, associated factors were synthesized thematically according to conceptual domains. Terminological consistency was maintained by using the generic term "associated factors" to refer to variables reported in relation to ADHD trajectories, whether they were described as risk factors, predictors or markers in the original studies. This choice reflects the observational nature of the included studies and the general absence of causal inference.

Results

ADHD symptom trajectories

Cohorts – Table 2 summarizes the key characteristics of the twelve included studies, along with the developmental trajectories identified for ADHD symptoms. These studies were conducted across seven distinct population-based cohorts, distributed among six countries (Brazil, the United Kingdom, Switzerland, Germany, the Netherlands, and the United States). Sample sizes ranged from 1,571 to 11,316 participants, with follow-up periods extending from childhood (typically ages 3–10) through adolescence (approximately ages 11–17) and, in some cases, into early adulthood (up to age 25).

ADHD assessment symptoms – The studies reviewed used a wide range of assessment tools to evaluate ADHD symptoms over time. The most frequently used instruments were the Strengths and Difficulties Questionnaire (SDQ), the Conners' Rating Scales, the Development and Well-Being Assessment (DAWBA) and the Adult ADHD Self-Report Scale (ASRS). These tests were generally administered on the basis of parent report, teacher report or self-report, depending on the age of the participant and the study design. Only one study applied the full DSM-5 diagnostic criteria at age 22, using clinician-administered interviews, resulting in a formal diagnosis.[13]

Trajectories – The number of trajectories identified per study ranged from three to six, reflecting considerable heterogeneity in symptom evolution patterns. Four main types of trajectories were consistently reported across studies: persistent/very stable trajectories, remission/decay trajectories, low-stability trajectories and late-onset/adolescent/increasing trajectories. Persistent/very stable trajectories are characterized by consistently high levels of symptoms since early childhood, and this pattern has been consistently observed in studies.[13,21,22] Trajectories of remission or decline reflect a decline in symptoms with age and are often interpreted as presentations limited to childhood or adolescence.[21,23] Unstable trajectories represent the most common pattern in cohorts, indicating minimal or absent symptoms at all time points.[15,17,24] Late-onset or adolescent-onset trajectories are defined by a marked increase in ADHD symptoms during adolescence or early adulthood, after a relatively symptom-free childhood period.[14,23,25]

Prevalence of late-onset trajectories – Among the twelve studies included in this scoping review, ten identified at least one symptom trajectory consistent with late-onset ADHD, with prevalence estimates ranging from 3 % to 17.5 % in the general population (in those cohorts), and were analyzed to examine associated factors. **It is important to note that not all studies explicitly used the term "late-onset" to describe these trajectories. Equivalent patterns were often labeled using alternative terms such as "increasing", "adolescent-onset", "late-emerging", or "ascending", all referring to the marked rise of ADHD symptoms during adolescence or early adulthood following a relatively symptom-free childhood period. This variability in prevalence estimates likely reflects differences in methodological approaches, symptom domain assessed, and the sex composition of the sample. For instance, Riglin et al. (2022) demonstrated marked discrepancies depending on the informant and instrument used.[5] Based on parent-reported Strengths and Difficulties Questionnaire (SDQ) data spanning childhood to early adulthood, a late-onset ADHD trajectory was identified in 8 % of participants, compared to 5 % who followed a child-onset persistent trajectory. Using the Development and Well-Being Assessment (DAWBA) symptom**

Table 2
Main characteristics of the studies.

Authors/ Year	Cohort Name	Country	Initial sample Size	Age at assessment	ADHD Assessment Method	Analysis	Number of trajectories	Type of trajectories
Breda et al. (2021)[13]	Pelotas Birth Cohort	Brazil	4676	11, 15, 18, and 22 years	SDQ Hyperactivity/Inattention subscale (parent and self-reported) at 11 and 15 years. Adult ADHD Self-Report Scale six-item screening tool at 18 and 22 years. DSM-5 ADHD criteria at 22 years.	LCMM	3	Stable (77.4 %), Descending (17.6 %), Ascending (4.9 %)
Van Lier et al. (2007)[15]	TRAILS	Netherlands	2076	4 to 18 years	CBCL (teacher)	GGMM	4	Near zero (males: 27 %; females: 27 %), Low (males: 40; females: 43 %), Moderate (males: 27 %; females: 26 %), High (males: 5 %; females: 4 %)
Döpfner et al. (2015)[24]	KiGGS	Germany	2593	7 to 19 years	ADHD Symptom Checklist from the DISYPS (parent)	LCGA	3	High (3.2 %), Moderate (14 %), Low (82.7 %)
Murray et al. (2020)[23]	Z-proso	Switzerland	1571	7, 8, 9, 10, 11, 12, 13 and 15 years	SDQ Hyperactivity/Inattention subscale (parent, teacher, self-reported according to age)	LCGA	4	Inattention trajectory: High stable (20 %), High decreasing (10 %), Low stable (63 %), Low increasing (7 %) Hyperactivity / impulsivity trajectory: High stable (8 %), High decreasing (13 %), Low stable (73 %), Low increasing (5 %)
Murray et al. (2020)[26]	Z-proso	Switzerland	1571	7, 8, 9, 10, 11, 12, 13, 15 and 17 years	SDQ Hyperactivity/Inattention subscale (parent, teacher, self-reported according to age)	GMM	4 OR 5	Inattention trajectory: Unaffected (63 %), Remitting (10 %), Persistent (20 %), Late-onset (8 %) Hyperactivity / impulsivity trajectory: Unaffected (73 %), Remitting (13 %), Persistent (8 %), Late-onset (5 %) Inattention trajectory with gender as a covariate: Unaffected (49 %), Remitting (10 %), Persistent (3 %), Mild (25 %), Late-onset (3 %) Hyperactivity / impulsivity trajectory with gender as a covariate: Unaffected (70 %), Remitting (15 %), Persistent (5 %), Late-onset (10 %)
Murray et al. (2022)[14]	MCS	UK	11316	3, 5, 7, 11, and 14 years	SDQ Hyperactivity/Inattention subscale (parent)	LCGA	6	Pre-school onset persistent (6.4 %), Subclinical remitting (12.4 %), Mildly affected (24.1 %), Pre-school onset partially remitting (14.1 %), Late childhood/ adolescent onset (7.6 %), Unaffected (34.9 %)
Carter et al. (2025)[16]	MCS	UK	10262	3, 5, 7, 11, 14 and 17 years	SDQ Hyperactivity/Inattention subscale (parent, self-reported according to age)	LCGA	6	Stable high (5.6 %), Subclinical remitting (14.4 %), Mildly affected (34.8 %), Adolescent onset (7.6 %), Unaffected (37.6 %)
Murray et al. (2022)[25]	UKHLS	UK	4866	10, 11, 12, 13, 14 and 15	SDQ Hyperactivity/Inattention subscale (self-reported)	LCGA	4	Mildly affected (males: 39.4 %; females: 48.3 %), High/ adolescent-increasing (males: NA; females: 17.1 %), Unaffected (males: 43.1 %; females: 34.7 %)
Durdurak et al. (2025)[21]	ALSPAC	UK	7811	8, 10, and 13 years	DAWBA (parent)	LCGA	4	Persistently high (9.1 %), Increasing (10.3 %), Remitting (14.5 %), Persistently low (66.1 %)
Riglin et al. (2016)[22]	ALSPAC	UK	9757	4 to 17 years	SDQ (parent)	LCGA	4	Low (82.6 %), Intermediate (7.7 %), Childhood-limited (5.8 %), Persistent (3.9 %)
Riglin et al. (2022)[5]	ALSPAC	UK	SDQ : 9764 DAWBA : 8132	4 to 25 years OR 7 to 25 years	Parent-reported/Self-reported SDQ at 4 to 25 years OR DAWBA (parent) at 7 to 25 years	GMM	5 OR 4	DAWBA: Low (81 %), Child-limited (7 %), Child/Adolescent-limited (6 %), Child-onset persistent (2 %), Late-onset (5 %) OR SDQ: Low (66 %), Child/ Adolescent-limited (21 %), Child-onset persistent (5 %), Late-onset (8 %)

(*continued on next page*)

Table 2 (*continued*)

Authors/ Year	Cohort Name	Country	Initial sample Size	Age at assessment	ADHD Assessment Method	Analysis	Number of trajectories	Type of trajectories
Shakeshaft et al. (2023)[17]	ALSPAC	UK	11316	4, 7, 8, 9, 12, 13, 17 and 25 years	SDQ (parent)	LCGA	3	Low-stable (87 %), Childhood/ Adolescent-declining (6 %), Late-emerging (6 %)

Note : ADHD: Attention-Deficit/Hyperactivity Disorder, **ALSPAC: Avon Longitudinal Study of Parents and Children, ASRS: Adult ADHD Self-Report Scale,** CBCL: Child Behavior Checklist, DAWBA: Development and Well-Being Assessment, DISYPS: Diagnostik-System für psychische Störungen im Kindes- und Jugendalter, GMM: Growth Mixture Modeling, GGMM: General Growth Mixture Modeling, KiGGS : Kinder- und Jugend gesundheit ssurvey, LCGA: Latent Class Growth Analysis, LCMM: Latent Class Mixed Models, MCS: Millennium Cohort Study, SDQ: Strengths and Difficulties Questionnaire, TRAILS: Tracking Adolescents' Individual Lives Survey, UKHLS**: UK Household Longitudinal Study, Z-proso: Zurich Project on the Social Development from Childhood to Adulthood.**
Percentages shown in the *Type of trajectories* column represent the proportion of participants within each identified trajectory, including late-onset or equivalent patterns (e.g., ascending, increasing, adolescent-onset, late-emerging). Two studies, Riglin et al. (2016; ALSPAC) and Döpfner et al. (2015; KiGGS/BELLA), did not identify any late-onset trajectory, and therefore no corresponding prevalence estimate was reported.

scores from the same cohort, the prevalence of late-onset trajectories was estimated at 5 %, whereas only 2 % were classified as child-onset persistent.[5] **In addition,** a critical factor in the identification of late-onset trajectories was the duration and timing of longitudinal follow-up. Studies that extended data collection through late adolescence or early adulthood (≥18 years) consistently detected these trajectories.[5,13,17]. In contrast, Döpfner et al. (2015) and **Riglin et al. (2016)** whose assessments ended before age 15, did not report any trajectories consistent with late-onset ADHD.[22,24]

Sex and symptom-specific trajectories of ADHD – In parallel, the Zurich Project on the Social Development from Childhood to Adulthood studies applied growth mixture modeling to assess inattention and hyperactivity/impulsivity dimensions separately.[23,26] Both dimensions yielded a low-increasing trajectory broadly corresponding to late-onset ADHD, with higher prevalence observed for inattention (8 %) than for hyperactivity/impulsivity (5 %). Furthermore, gender-stratified models revealed differences in class membership: although males were generally overrepresented in symptomatic trajectories, the proportion of females was relatively greater in the late-onset group—particularly in the inattention domain. A similar pattern emerged in Murray et al (2020) using data from the UK Household Longitudinal Study, who identified a trajectory of ADHD appearing in adolescence only in girls (17.1 %), while boys were more likely to have high or stable early symptom profiles.[26] In the Dutch cohort of the Tracking Adolescents' Individual Lives Survey, Van Lier et al. (2007) also identified an increasing trajectory of symptoms, with symptom levels rising throughout adolescence, consistent with a late-onset profile, and showing similar prevalence in boys (5 %) and girls (4 %).[15]

Associated factors of late-onset ADHD

Among the twelve studies included in this scoping review, ten identified at least one symptom trajectory consistent with late-onset ADHD and were analyzed to examine associated factors. These factors spanned multiple domains, including genetic susceptibility, early life risk factors, cognitive functioning and executive resources, psychosocial maladjustment and psychiatric vulnerabilities, family context and socioeconomic environment, and sex differences. Table 3 provides an overview of the specific correlates examined across studies and summarizes the heterogeneity of findings related to each domain.

Among these same studies, ten examined factors potentially associated with late-onset ADHD in greater detail. For each domain, the number of studies assessing and reporting significant associations is summarized in Table 4. The most consistently replicated associations concerned prematurity, female sex, internalizing symptoms, and psychosocial maladjustment during adolescence, whereas findings for genetic susceptibility and socioeconomic background were more heterogeneous.

Genetic susceptibility – Results concerning genetic susceptibility are mixed. Individuals following a late-onset trajectory do not systematically present lower polygenic risk scores (PRS) for ADHD.[5] When late-onset ADHD was based on parent-reported symptoms, PRS levels were similar to those of individuals with persistent childhood ADHD, whereas self-reported late-onset symptoms in adulthood were associated with lower PRS. Other results indicated higher genomic propensity scores (GPS) for ADHD and schizophrenia, and lower GPS for executive function in the late-onset group.[17]

Early life risk factors – Preterm birth was consistently associated with late-onset ADHD (Murray et al., 2022; Shakeshaft et al., 2023, Riglin et al., 2022). No significant association was found for low birth weight across studies.[5,13,14,17]

Cognitive functioning and executive resources – In the Pelotas cohort, individuals with late-onset ADHD (ascending trajectory) showed higher intellectual quotient scores compared with those with a stable/ persistent ADHD trajectory.[13] However, others found the opposite: those following a late-emergence trajectory had lower intellection functionning than individuals in the low-stability group.[17] Self-reported late-onset ADHD was also associated with better performance on sustained attention and response inhibition tasks than persistent ADHD, while no significant differences were observed for the parent-reported group.[5]

Psychosocial maladjustment and psychiatric vulnerabilities – Compared with persistent ADHD trajectories, late-onset ADHD was less often associated with early externalizing disorders such as oppositional defiant disorder and conduct disorder.[14] During adolescence, however, individuals in the late-onset group showed more emotional difficulties, including symptoms of anxiety and depression[14], as well as more general indicators of poor mental health, such as psychological distress and decreased well-being.[16] Substance use was also high in this group, with an increased risk of alcohol use but not cannabis[16], while other studies have reported higher rates of both alcohol and cannabis use.[17]

Symptom-based analyses further revealed specific behavioral patterns associated with late-onset ADHD. Both late-onset inattention and hyperactivity/impulsivity trajectories were linked to elevated externalizing behaviors in adolescence, including proactive and reactive aggression, violent ideation, delinquent behavior and cigarette smoking, compared with unaffected peers.[26] However, compared with persistent inattention trajectories, individuals in the late-onset group showed fewer differences, with the exception of lower levels of reactive aggression. Similarly, late-onset hyperactivity/impulsivity trajectories were associated with increased externalizing problems compared to unaffected adolescents, but with lower levels of anxiety and reactive aggression than persistent profiles.[26] One study also reported an association between inattention symptoms and hypomanic traits during adolescence.[21]

Psychosocial difficulties also emerged more broadly in late-onset ADHD, including experiences of peer victimization[14,17], low self-esteem[16], and engagement in delinquent behavior.[14] Finally, no significant associations were found between late-onset ADHD and a range of psychiatric disorders in early adulthood, including major depressive

Table 3
Associated factors with late-onset ADHD.

Authors/Year	Studied associated factors	Associated factor with Late-Onset ADHD
Breda et al. (2021)[13]	Prenatal tobacco exposure; Newborn gender; Birth weight; Ethnicity from the mothers in maternity; General health; Income; Substance use; Years of schooling; IQ at 18 years of age; Major depressive disorder, bipolar disorder, social anxiety disorder, generalized anxiety disorder, posttraumatic, stress disorder and antisocial personality disorder	Female Gender; Higher IQ
Van Lier et al. (2007)[15]	Conduct disorder; Oppositional defiant disorder	Females: High oppositional defiant disorder trajectory and high ADHD trajectory. Males: High oppositional defiant trajectory
Murray et. (2020)[23]	Gender; Anxiety; Reactive aggression; Sensation seeking; Risk taking	Inattention symptoms: female gender, higher sensation seeking compared to the low stable group; lower anxiety, lower reactive aggression, and lower risk taking compared to the high stable group Hyperactivity/impulsivity symptoms: male gender compare to the low stable group; lower anxiety and lower reactive aggression compared to the high stable group
Murray et al. (2020)[26]	Internalizing symptoms; Prosociality; Aggression; Violent ideations; Substance use; Delinquency	Inattention and hyperactivity/impulsivity symptoms: higher level of proactive and reactive aggression, violent ideations, delinquency and substance use compared to the unaffected group Inattention symptoms: no significant differences between late-onset vs. persistent group Hyperactivity/impulsivity symptoms: lower level of reactive aggression compared to the persistent group
Murray et al. (2022)[14]	Prematurity; Low birth weight; Maternal educational level; Early child temperament; Early child cognitive ability; Conduct problems; Peer problems and emotional problems	Prematurity; Lower maternal education; Higher conduct and peer problems at age 3
Carter et al. (2025)[16]	Peer victimization; Substance use (alcohol consumption, cannabis use); Mental health (anxiety and depression symptoms; well-being; self-esteem); Delinquency	Higher peer victimization, alcohol use; increased anxiety and depression symptoms; lower well-being and self-esteem; higher delinquency. Compared to subclinical remitting, mildly affected, and unaffected groups, the adolescent-onset trajectory was associated with significantly higher levels of peer victimization, psychological distress, and delinquency, as well as lower self-esteem and wellbeing at age 17. Alcohol use was lower than in the unaffected group.
Murray et al. (2022)[25]	Conduct problem and internalizing problem symptoms	Females: Frequent co-occurrence with internalizing and moderate conduct problems. Males: Frequent co-occurrence with severe conduct problems and less pronounced internalizing problems
Durdurak et al. (2025)[21]	Bipolar spectrum disorder	ADHD total score and inattentive symptoms were associated with hypomanic symptoms
Riglin et al. (2022)[5]	**ADHD polygenic risk scores**; Preterm birth; Low birth weight; Sustained attention; Response inhibition; Verbal and Reading ability; Family income; Maternal education; Depression genetic risk; Maternal depression	Parent-Reported Late-Onset ADHD: higher levels of childhood verbal and reading ability, family income and maternal education compared to child-onset persistent Self-Reported Late-Onset ADHD: lower PRS; higher performance on sustained attention and response inhibition tasks; higher levels of childhood verbal and reading ability, family income and maternal education compared to child-onset persistent
Shakeshaft et al. (2023)[17]	Sex; Preterm birth; Low birth weight; Family income; Emotional problems; Conduct problems; Anxiety disorder; Mental Well-being; Depressive disorder; Childhood IQ; Childhood epileptic seizures; peer problems; Education, employment or training; Alcohol abuse; Cannabis abuse; genetic liability	Females; Lower preterm birth; lower family income; Emotional and conduct problems at all ages; Childhood epileptic seizures; low IQ; peers' problems at all ages; Higher rates of substance abuse; higher ADHD genetic liability; higher schizophrenia genetic liability; lower executive function genetic liability compared to the low-stable group

Note: ADHD: Attention-Deficit/Hyperactivity Disorder**; PRS: polygenic;**

disorder, bipolar disorder, social and generalized anxiety disorders, posttraumatic stress disorder, and antisocial personality disorder.[13]

Family context and socioeconomic environment – Associations between late-onset ADHD and socioeconomic background were inconsistent. One study found a link between lower family income and late-onset ADHD trajectories[17], while another reported higher family income and maternal education in those with late-onset ADHD, irrespective of whether symptoms were self-reported or parent-reported.[5]

Sex differences – Across studies, the proportion of females within late-onset ADHD trajectories ranged from approximately 50 % to 70 %, compared with 30 % to 50 % in persistent or early-onset groups.[15,23,24,25,26,17] Females were over-represented in late-onset ADHD trajectories, with profiles more frequently marked by internalizing symptoms, particularly anxiety and depression during adolescence.[13,14,17] In contrast, boys with late-onset ADHD were more likely to exhibit externalizing behaviors, including conduct problems and aggression.[14] Co-occurring trajectories of oppositional defiant disorder were also identified among both males and females with late-onset ADHD, indicating that oppositional behaviors can accompany late-emerging ADHD regardless of sex.[15]

Overlapping cohorts and complementary analyses across longitudinal studies

Several studies included in this review were based on overlapping longitudinal cohorts but analyzed different developmental periods, outcomes, or methodological questions. In the Avon Longitudinal Study of Parents and Children, several studies have examined ADHD symptom trajectories across development using different methodological and conceptual lenses. **Riglin et al. (2016)** focused on genetic correlates of ADHD symptom persistence, whereas **Riglin et al. (2022)** investigated

Table 4
Frequency and significance of factors associated with late-onset ADHD across included studies.

Domain of associated factors	Studies assessing	Studies assessing	Summary of significance across studies
Genetic susceptibility	2	Riglin et al. (2022)[5]; Shakeshaft et al. (2023)[17]	All of 2 studies reported significant associations between late-onset ADHD and polygenic liability, although findings were heterogeneous: Riglin et al. (2022)[5] observed comparable or lower ADHD polygenic risk scores depending on informant source, whereas Shakeshaft et al. (2023) found higher polygenic scores for ADHD and schizophrenia and lower scores for executive functioning[17]
Perinatal & early life factors	3	Murray et al. (2022)[14]; Riglin et al., (2022); Shakeshaft et al., (2023)[17]	All of 3 studies reported significant perinatal or early-life associations
Cognitive functioning / Executive resources	2	Breda et al. (2021)[13]; Riglin et al., (2022)	All of 2 studies reported significant associations (higher IQ or cognitive performance)
Psychiatric & psychosocial vulnerabilities	6	Van Lier et al. (2007)[15]; Murray et al. (2020)[23]; Murray et al. (2022)[25]; Carter et al. (2025)[16]; Durdurak et al. (2025)[21]	All of 6 studies identified significant associations with internalizing/ externalizing or psychosocial outcomes
Family context / Socioeconomic environment	3	Murray et al. (2022)[14]; Riglin et al. (2022)[5]; Shakeshaft et al. (2023)[17]	All of 3 studies reported significant associations between late-onset ADHD and socioeconomic indicators, but in opposite directions: Murray et al. (2022) found that the late-onset trajectory was associated with lower maternal education[14]; Riglin et al. (2022) observed higher family income and higher maternal education in individuals with late-onset ADHD compared with those with persistent childhood-onset ADHD[5]; Shakeshaft et al. (2023) reported lower family income in the late-emerging ADHD group compared with the low-stable group[17]
Sex differences	5	Van Lier et al. (2007)[15]; Murray et al. (2020)[23]; Murray et al. (2022)[26]; Shakeshaft et al. (2023)[17]; Carter et al. (2025)[16]	All of 5 studies found sex effects; female over-representation in late-onset trajectories

Note: ADHD: Attention-Deficit/Hyperactivity Disorder.

the nature and validity of late-onset ADHD up to age 25 using multiple informants and diagnostic instruments.[5,22] **Shakeshaft et al. (2023)** modelled the joint developmental trajectories of ADHD and social communication traits from childhood to early adulthood, identifying distinct subgroups with divergent patterns of emergence, decline, and psychosocial and genetic correlates.[17] Finally, **Durdurak et al. (2025)** explored the associations between ADHD trajectories and hypomanic symptoms in young adults, extending the developmental window into emerging adulthood.[21] Similarly, in the UK Millennium Cohort Study[14,16] and in the Zurich Project on the Social Development from Childhood to Adulthood[23,26] successive analyses examined predictors and later outcomes of identified developmental subtypes. These complementary investigations contribute to a richer understanding of ADHD symptom trajectories across development, but their shared sample bases may introduce partial dependency and should thus be interpreted as sequential analyses within the same population rather than independent replications. Importantly, across these three longitudinal cohorts, as participants age and successive analyses extend into late adolescence or young adulthood, the identification of a late-onset trajectory remains remarkably consistent. Despite differences in measurement instruments, informants, and modeling approaches, each cohort reproduces the presence of a subgroup showing emergent ADHD symptoms during adolescence or early adulthood, supporting the robustness of this latent pattern as cohorts mature.

Discussion

This literature review synthesized twelve longitudinal studies that used trajectory modeling in population-based cohorts to examine the evolution of ADHD symptoms and factors associated with late-onset trajectories. Across these studies, four main patterns emerged consistently: unstable, remittent, persistent and late-onset trajectories. Although late-onset patterns were generally less frequent than the other trajectories, they were identified in the vast majority of cohorts. The reported prevalence of these trajectories varies considerably from study to study, reflecting differences in sample characteristics, modeling strategies, sources of information and symptom definitions. A broad range of factors were identified in relation to late-onset trajectories, spanning from early indicators, such as cognitive performance, sex, family socioeconomic background, and polygenic scores, to later emerging characteristics, including psychopathological symptoms and psychosocial maladjustment during adolescence or early adulthood. To deepen our understanding of these findings, the remainder of the discussion is organized into four parts. First, we examine the clinical validity and developmental interpretation of late-onset ADHD trajectories, including their symptom profiles and timing of emergence. Second, we review key methodological influences that may account for variability in their identification. Third, we synthesize the main associated factors reported across studies, spanning genetic, cognitive, psychosocial, and contextual domains. Finally, we reflect on the nosological implications of these findings and discuss future research directions.

Identifying late-onset ADHD in population cohorts: insights from longitudinal trajectories

Clinical validity and developmental interpretation – Across the twelve longitudinal population-based studies included, ten trajectories characterized by ADHD symptom emergence during adolescence or early adulthood were identified. These late-onset patterns were typically derived using person-centered statistical approaches, such as latent class or growth mixture modeling, allowing for the identification of distinct developmental profiles over time. Although less prevalent than persistent childhood-onset trajectories, they were consistently observed across diverse cohorts[5,14,16], with prevalence estimates ranging from 3 % to 17.5 %, depending on sample characteristics, age range, and measurement strategy. These findings align with calls to reconceptualize ADHD

as a lifespan-oriented condition rather than a strictly early-onset neurodevelopmental disorder.[27,28] However, as these prevalence rates derive from general population cohorts, many individuals classified within late-onset trajectories may display subthreshold or context-dependent ADHD-like symptoms rather than clinically impairing syndromes. It is therefore essential to distinguish symptom trajectories identified in population-based models from clinically diagnosed late-onset ADHD. Symptom domain specificity may also shape these trajectories. When inattention and hyperactivity-impulsivity are modeled separately, late-onset patterns tend to emerge predominantly in the inattentive domain, while hyperactivity-impulsivity usually follows earlier, more stable developmental courses.[23] This likely reflects the subtler and more internal nature of inattention, which often goes unnoticed in childhood, especially when externalizing behaviors are absent, whereas the more overt features of hyperactivity are typically flagged earlier by caregivers or educators. These observations support dimensional models and underscore the differing developmental sensitivity of ADHD symptom domains.[29,30] However, the consistency of these findings must be interpreted in light of key methodological features, which may shape the likelihood of detecting late-onset trajectories across studies.

Methodological and Conceptual Artifacts – The identification of late-onset ADHD trajectories remains highly sensitive to methodological factors. Informant source is crucial: studies using adolescent self-reports tend to report higher prevalence, possibly due to increased awareness of internalized symptoms, but also risk over-identification.[2,23,31] Symptom measurement tools also vary in sensitivity. Brief scales like the ASRS or SDQ may miss subtle or context-dependent patterns that structured interviews, such as the DAWBA, are more likely to detect, though the latter may be inflated by comorbid distress.[27] In addition, the use of different instruments across cohorts (e.g., SDQ, DAWBA, ASRS) complicates cross-study comparisons and may partly explain discrepancies in the prevalence and characterization of late-onset trajectories. Short screening tools often rely on limited symptom domains or rating contexts, whereas structured diagnostic interviews provide more comprehensive but potentially less comparable data. These methodological differences can influence prevalence estimates, the identification of trajectory classes, and the apparent overlap with internalizing or externalizing comorbidities, thereby limiting cross-cohort comparability. To improve the robustness and developmental sensitivity of future research, several refinements are warranted. The systematic use of multi-informant assessments across all waves of data collection would help disentangle true developmental change from informant-related variance, a critical issue in the identification of late-emerging trajectories. In parallel, incorporating explicit functional impairment criteria would allow researchers to distinguish subthreshold symptom fluctuations from clinically significant presentations. Sensitivity analyses comparing alternative symptom thresholds or cut-points could further clarify the extent to which late-onset profiles depend on measurement definitions rather than genuine developmental differences. Finally, the development and validation of instruments specifically designed to capture late developmental manifestations of ADHD (e.g., cognitive fatigability, mind-wandering, procrastination, time management deficits, or emotional instability and stress intolerance) remain essential. These dimensions often characterize adult-onset presentations that differ from classical childhood forms. The availability of developmentally sensitive tools would substantially strengthen the ecological validity and comparability of future longitudinal studies. Detection also depends on follow-up timing. Late-onset profiles are more likely to emerge in studies extending into early adulthood[5,17] than those ending in mid-adolescence.[24] Similarly, modeling choices influence findings: latent class and growth mixture models with flexible parameters tend to reveal late-onset groups more frequently than constrained models with limited timepoints.[13,14,16] These variations highlight the importance of developmentally sensitive designs and caution against overgeneralizing across heterogeneous methodologies.[32] Taken together, these methodological factors may partly explain inconsistencies in prevalence and profile characteristics, and caution against overgeneralizing findings across studies without careful consideration of design features.

Associated factors of late-onset ADHD: evidence from longitudinal trajectories

Genetic susceptibility – Findings on genetic susceptibility in late-onset ADHD trajectories reveal a complex and somewhat inconsistent pattern. Riglin et al (2022) reported that individuals classified as having late-onset ADHD on the basis of parent-reported symptoms had similar ADHD PRS to those with persistent childhood-onset ADHD, suggesting comparable genetic liability.[5] In contrast, self-reported late-onset cases in adulthood had lower ADHD polygenic risk scores, raising the possibility that some cases reflect non-specific distress, over-reporting or context-dependent symptom expression rather than a genetic condition. Adding further complexity, Shakeshaft et al. (2023) found higher polygenic scores for both ADHD and schizophrenia, along with lower scores for executive functioning, in late-onset trajectories.[17] Together, these findings point to potential etiological heterogeneity, with some individuals exhibiting genuine neurodevelopmental vulnerability and others reflecting environmentally reactive or transdiagnostic profiles.[33] Clarifying these pathways will require genetically informed longitudinal studies using multi-informant data and refined phenotyping strategies.

Early life risk factors – Prematurity has been consistently linked to neurodevelopmental vulnerabilities, including increased risk for ADHD [34,35]. In the Avon Longitudinal Study of Parents and Children cohort, both Riglin et al. (2022) and Shakeshaft et al. (2023) identified a higher prevalence of preterm birth among individuals with late-onset ADHD trajectories.[5,17] Riglin et al. found elevated rates of prematurity in late-onset compared to persistent and low-symptom groups, while Shakeshaft et al. reported similar associations in both late-emerging and childhood-declining profiles.[5,17] These findings suggest that prematurity may act as a latent risk factor, contributing to symptom emergence only when increasing developmental demands exceed compensatory resources. However, given the modest effect sizes and limited number of studies addressing this factor directly, further research is needed to clarify its role in shaping ADHD symptom trajectories across development.

Cognitive functioning and executive resources – Cognitive profiles of individuals in late-onset ADHD trajectories appear to differ meaningfully from those observed in early-onset or persistent cases. Two cohorts have reported that individuals with adolescent-onset ADHD, particularly within inattentive trajectories, exhibit higher childhood intellectual quotient, better early inhibitory control, and stronger language or reading skills.[5,16] These preserved cognitive abilities may serve as compensatory mechanisms, delaying the onset of functional impairment by temporarily buffering the expression of core ADHD symptoms. Such mechanisms align with broader compensatory models proposed in neurodevelopmental conditions such as autism spectrum disorder, where cognitive strengths can mask underlying deficits until external demands intensify.[7] This interpretation resonates with the threshold-exceedance model, which suggests that symptoms remain latent until environmental demands exceed individual regulatory capacities.[36] It also aligns with the life-transition model, which emphasizes how major developmental shifts, such as entering post-secondary education or losing parental scaffolding, can unmask previously compensated vulnerabilities.[37]

Psychosocial maladjustment and psychiatric vulnerabilities – Late-onset ADHD trajectories appear to be associated with a distinct pattern of psychosocial vulnerabilities, particularly regarding internalizing symptoms, substance use, and peer-related difficulties. Unlike early-onset persistent profiles, individuals with late-emerging ADHD symptoms often show fewer early conduct problems, possibly reflecting preserved behavioral regulation in childhood, supportive early environments, or alternative expressions of dysregulation such as anxiety or

inhibition.[16,21,23] However, during adolescence, these individuals frequently experience academic failure, low self-esteem, and social withdrawal, which in turn elevate the risk of depressive and anxious symptoms.[21,23] Substance use also emerges as a prominent associated factor. Three cohorts report higher rates of alcohol use in late-onset or adolescent-increasing trajectories, likely reflecting attempts at emotional or cognitive self-medication, increased impulsivity in social contexts, and affiliations with deviant peer groups.[16,21,23] Importantly, these patterns are often compounded by comorbid oppositional or conduct disorders, which are robust predictors of substance. Peer victimization is also more frequent in these profiles, potentially resulting from poor early social skills or later-emerging social disruptions.[16,21] Taken together, these findings raise the hypothesis that late-onset ADHD may not simply reflect a delayed expression of earlier vulnerability but could involve qualitatively distinct developmental pathways shaped by specific emotional, behavioral, and contextual dynamics.

Family context and socioeconomic environment – Children from low-income families or with lower maternal education levels are more likely to experience stressful environments, including household conflict, instability, and limited educational stimulation.[38] They are also less likely to be identified or supported early, even when presenting high intellectual potential. This combination of adversity and lack of intervention may delay the recognition of ADHD symptoms. As academic and social demands increase during adolescence, previously masked difficulties may become more pronounced, leading to later and more visible symptom expression. Supporting this view, Shakeshaft et al. (2023) identified lower family income as a factor associated with late-onset ADHD trajectories.[17] However, Riglin et al. (2022) found higher levels of family income and maternal education in late-onset cases, regardless of informant.[5] These contrasting findings suggest that late-onset ADHD may arise through multiple developmental pathways, either due to under-identification in disadvantaged contexts, or context-dependent decompensation in individuals previously functioning well. Importantly, such group-level associations reflect statistical trends and do not imply a single, unified profile. Each trajectory likely comprises heterogeneous subgroups with distinct contextual, cognitive, and developmental features.

Sex differences – Three cohorts included in this review reported a higher proportion of females in late-onset ADHD trajectories compared to persistent or early-onset profiles.[5,23,24] This overrepresentation may reflect longstanding sex-based disparities in the detection of ADHD.[39] Girls are more likely to present with inattentive symptoms, which are less disruptive and more likely to be overlooked in educational or family contexts. They may also develop compensatory strategies that mask their difficulties[37,40], and face gendered expectations that discourage help-seeking[41]. Additionally, diagnostic criteria historically based on male samples may lack sensitivity for female presentations[42], while internalizing comorbidities, more prevalent in females, can further obscure ADHD symptoms[43]. Together, these factors may delay identification and contribute to the apparent female overrepresentation in late-onset ADHD groups.

Taken together, the synthesis of associated factors suggests that late-onset ADHD trajectories are most consistently associated with internalizing and psychosocial vulnerabilities emerging during adolescence, as well as with female overrepresentation and a history of prematurity. However, findings regarding genetic susceptibility and socioeconomic background remain heterogeneous across studies, underlining the need for more standardized methodologies and longitudinal replications to clarify the mechanisms underlying these developmental pathways.

Reconsidering the nosological status of late-onset ADHD

From a developmental standpoint, the emergence of ADHD symptoms after childhood may reflect increasing cognitive, emotional, or environmental demands that exceed earlier compensatory capacities. This aligns with both the threshold-exceedance model, in which latent vulnerabilities become symptomatic under heightened pressure, and the life-transition model, which emphasizes how developmental shifts, such as entering higher education or assuming greater autonomy, can destabilize previously adaptive functioning. The findings of this review challenge the notion of a single explanatory pathway for late-onset ADHD. Some trajectories likely represent subthreshold neurodevelopmental difficulties that remained compensated in childhood, while others may reflect emerging psychiatric comorbidities, environmental stressors, or measurement artifacts. This heterogeneity complicates categorical classification and questions the strict age-of-onset threshold defined by current nosological systems. Although ADHD is classified as a childhood-onset neurodevelopmental disorder [1], longitudinal data consistently show that clinically impairing symptom constellations can appear later in development, sometimes without clear childhood antecedents. These patterns support a more dimensional and developmentally sensitive framework that considers individual variability in symptom emergence, functional impairment, and contextual influences. Clarifying whether late-onset profiles reflect a true ADHD variant, a distinct clinical entity, or a phenocopy remains a key challenge for future research and diagnostic refinement.

Strengths and limitations of the review

This scoping literature review provides a comprehensive synthesis of longitudinal studies on the trajectory of late-onset ADHD symptoms in general population cohorts. A strength is the inclusion of large-scale studies using person-centered approaches (e.g., LCGA, GMM), which provide a nuanced understanding of symptom emergence beyond diagnostic thresholds. The study also identifies points of convergence and methodological variability between cohorts, laying the foundations for future systematic studies. A number of limitations need to be highlighted. Firstly, grey literature and unpublished data were not taken into account, which could limit coverage. Secondly, although the analysis focused on general population samples, some of the included studies reported attrition or sampling bias that could impact on the representation of late-emerging profiles. Although this is not a limitation of the review itself, it affects the robustness of the evidence base. Third, no formal appraisal of study quality was conducted, consistent with scoping review methodology. Another limitation concerns the partial overlap between some included studies. In fact, several included studies were derived from the same longitudinal cohorts, most notably Avon Longitudinal Study of Parents and Children,[5,17,21,22] Millennium Cohort Study,[14,16] and Zurich Project on the Social Development from Childhood to Adulthood.[23,26] Although these studies applied distinct analytic strategies, follow-up periods, informant sources, or symptom measures, they rely on overlapping samples, which may reduce the effective diversity of the evidence base. Consequently, some of the associations summarized in Tables 3 and 4 represent partial analytical replications within the same cohort rather than independent findings across distinct populations. This overlap could therefore introduce some degree of dependency or overrepresentation of specific population characteristics and potentially inflate the apparent consistency of late-onset trajectories. Finally, heterogeneity in terminology and class definitions may limit comparability across studies. Despite these limitations, the review offers an integrative overview of a complex and understudied developmental phenomenon.

Future research directions

While the existing longitudinal trajectory studies offer valuable insights into ADHD's developmental patterns, they exhibit several key limitations in capturing late-onset presentations comprehensively.

First, none of the twelve studies included validated measures of insomnia or excessive daytime sleepiness, despite increasing evidence that sleep-wake disturbances contribute to ADHD expression. Excessive daytime sleepiness, in particular, has been proposed as a core

endophenotype of adult-onset ADHD, with nearly half of affected individuals reporting clinically significant sleepiness and over 20 % meeting criteria for hypersomnolence disorder.[44,45] Conversely, patients with central hypersomnolence disorders frequently report persistent ADHD symptoms, especially in the inattentive domain.[46] These bidirectional associations suggest shared mechanisms involving arousal regulation and executive control. Moreover, adolescent insomnia has been linked to a higher risk of developing ADHD symptoms,[47] and poor sleep quality in early adolescence predicts greater symptom severity later on.[48] Shared impairments in metacognitive and behavioral regulation may mediate these associations.[49] The absence of sleep-wake assessments in trajectory studies may obscure critical developmental pathways, lead to misclassification, and limit understanding of causal mechanisms.

Second, none tracked ADHD-related treatments or educational interventions across development including pharmacotherapy, behavioral therapies, or academic accommodations. These supports can significantly influence symptom expression, compensation, or masking of difficulties.[40] Without this context, distinguishing true emergence from treated, suppressed, or contextualized symptom trajectories is challenging.

Third, despite their focus on psychiatric comorbidities, studies overlooked neurological antecedents including concussions and traumatic brain injuries (TBIs) which may produce ADHD-like symptoms. This omission is striking given that recent data show children and adolescents with ADHD have approximately double the lifetime prevalence of concussion (10.6 % vs. 5.6 %), and large-scale cohort studies confirm elevated TBI risk, with hazard ratios around 1.6.[50] Concussions and mild TBIs significantly increase the risk of emergent attention problems, with ADHD diagnoses appearing post-injury in 5–53 % of cases depending on severity[51] Accounting for these neurological factors is essential to rule out acquired or mimicking pathways.

Importantly, as current longitudinal cohorts continue to age, forthcoming data from adolescence into adulthood will be pivotal. These follow-ups promise to unlock fundamental insights into late-emerging symptom trajectories revealing how developmental timing, environmental context, and compensatory mechanisms shape ADHD expression across the lifespan.

Conclusion

This scoping review examined longitudinal evidence on late-onset ADHD trajectories in population-based cohorts. Consistent patterns of predominantly inattentive symptoms emerging in adolescence or adulthood were observed in ten out of the twelve included studies, supporting a developmental reconceptualization of ADHD beyond childhood. These findings call into question rigid age-of-onset criteria and underline the value of dimensional, lifespan approaches. A range of associated factors has been identified, including cognitive, psychiatric, genetic and sociodemographic variables. However, methodological heterogeneity and key omissions, such as sleep and wakefulness disorders, treatment history, contextual support and neurological history, limit the scope and comparability of current findings. Future research should incorporate these missing elements using ecological and developmentally sensitive models to better distinguish true late presentations from mimicry or compensatory pathways. Concretely, future longitudinal studies should systematically integrate the assessment of sleep and wakefulness disorders (e.g., insomnia, hypersomnolence), track treatment exposure and educational support (e.g., pharmacotherapy, academic accommodations), and account for neurological antecedents such as concussions or mild traumatic brain injuries. In addition, multi-informant designs spanning adolescence and early adulthood, combined with dimensional modeling and objective measures of executive and arousal regulation, will be crucial to capture the heterogeneity of late-onset profiles. Such insights will be essential to refine diagnostic criteria and support more nuanced therapeutic approaches.

Ethics approval and informed consent

This study did not involve the collection of original human data and is based entirely on previously published literature. Therefore, ethics approval and informed consent were not required.

Funding statement

This research received no specific funding from any public, commercial, or not-for-profit agency.

Data availability statement

This scoping review is based entirely on data extracted from previously published studies. No new data were generated or analyzed. All relevant data extracted from the included studies are summarized within the manuscript. No additional data are available.

Declaration of competing interest

The authors have no conflicts of interest to disclose.

References

1. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*. 5th ed. American psychiatric association; 2013.
2. Asherson P, Agnew-Blais J. Annual Research Review: Does late-onset attention-deficit/hyperactivity disorder exist? *Child Psychol Psychiatry*. 2019;60(4):333–352. https://doi.org/10.1111/jcpp.13020.
3. Faraone SV, Biederman J. Can Attention-Deficit/Hyperactivity Disorder Onset Occur in Adulthood? *JAMA Psychiatry*. 2016;73(7):655. https://doi.org/10.1001/jamapsychiatry.2016.0400.
4. Barkley RA, Fischer M, Smallish L, Fletcher K. The persistence of attention-deficit/hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *J Abnorm Psychol*. 2002;111(2):279–289. https://doi.org/10.1037/0021-843X.111.2.279.
5. Riglin L, Wootton RE, Livingston LA, et al. Late-onset" ADHD symptoms in young adulthood: Is this ADHD? *J Atten Disord*. 2022;26(10):1271–1282. https://doi.org/10.1177/10870547211066486.
6. Kosaka H, Fujioka T, Jung M. Symptoms in individuals with adult-onset ADHD are masked during childhood. *Eur Arch Psychiatry Clin Neurosci*. 2019;269(6):753–755. https://doi.org/10.1007/s00406-018-0893-3.
7. Livingston LA, Happé F. Conceptualising compensation in neurodevelopmental disorders: Reflections from autism spectrum disorder. *Neurosci Biobehav Rev*. 2017;80:729–742. https://doi.org/10.1016/j.neubiorev.2017.06.005.
8. Agnew-Blais JC, Polanczyk GV, Danese A, Wertz J, Moffitt TE, Arseneault L. Young adult mental health and functional outcomes among individuals with remitted, persistent and late-onset ADHD. *Br J Psychiatry*. 2018;213(3):526–534. https://doi.org/10.1192/bjp.2018.97.
9. Thapar A, Collishaw S, Pine DS, Thapar AK. Depression in adolescence. *Lancet*. 2012;379(9820):1056–1067. https://doi.org/10.1016/S0140-6736(11)60871-4.
10. Fischer AG, Bau CHD, Grevet EH, et al. The role of comorbid major depressive disorder in the clinical presentation of adult ADHD. *J Psychiatr Res*. 2007;41(12):991–996. https://doi.org/10.1016/j.jpsychires.2006.09.008.
11. Spencer T, Biederman J, Wilens T. Attention-deficit/hyperactivity disorder and comorbidity. *Pediatr Clin N Am*. 1999;46(5):915–927. https://doi.org/10.1016/S0031-3955(05)70163-2.
12. Sibley MH, Rohde LA, Swanson JM, et al. Late-Onset ADHD Reconsidered With Comprehensive Repeated Assessments Between Ages 10 and 25. *AJP*. 2018;175(2):140–149. https://doi.org/10.1176/appi.ajp.2017.17030298.
13. Breda V, Rohde LA, Menezes AMB, et al. The neurodevelopmental nature of attention-deficit hyperactivity disorder in adults. *Br J Psychiatry*. 2021;218(1):43–50. https://doi.org/10.1192/bjp.2020.200.
14. Murray AL, Hall HA, Speyer LG, et al. Developmental trajectories of ADHD symptoms in a large population-representative longitudinal study. *Psychol Med*. 2022;52(15):3590–3596. https://doi.org/10.1017/S0033291721000349.
15. Van Lier PAC, Der Ende JV, Koot HM, Verhulst FC. Which better predicts conduct problems? The relationship of trajectories of conduct problems with ODD and ADHD symptoms from childhood into adolescence. *Child Psychol Psychiatry*. 2007;48(6):601–608. https://doi.org/10.1111/j.1469-7610.2006.01724.x.
16. Carter L, Speyer L, Caye A, Rohde L, Murray AL. Late adolescent outcomes of different developmental trajectories of ADHD symptoms in a large longitudinal study. *Eur Child Adolesc Psychiatry*. 2025;34(2):709–719. https://doi.org/10.1007/s00787-024-02516-5.
17. Shakeshaft A, Heron J, Blakey R, et al. Co-development of attention deficit hyperactivity disorder and autistic trait trajectories from childhood to early adulthood. *Child Psychol Psychiatry*. 2023;64(11):1596–1607. https://doi.org/10.1111/jcpp.13851.

18. Costello EJ, Maughan B. Annual Research Review: Optimal outcomes of child and adolescent mental illness. *Child Psychol Psychiatry*. 2015;56(3):324–341. https://doi.org/10.1111/jcpp.12371.
19. Stergiakouli E, Martin J, Hamshere ML, et al. Association between polygenic risk scores for attention-deficit hyperactivity disorder and educational and cognitive outcomes in the general population. *Int J Epidemiol*. 2016:dyw216. https://doi.org/10.1093/ije/dyw216. Published online September 30.
20. Tricco AC, Lillie E, Zarin W, et al. PRISMA Extension for Scoping Reviews (PRISMA-ScR): Checklist and Explanation. *Ann Intern Med*. 2018;169(7):467–473. https://doi.org/10.7326/M18-0850.
21. Durdurak BB, Morales-Muñoz I, Hosang GM, Marwaha S. ADHD symptom trajectories across childhood and early adolescence and risk for hypomanic symptoms in young adulthood. *Eur Psychiatr*. 2025;68(1):e37. https://doi.org/10.1192/j.eurpsy.2025.24.
22. Riglin L, Collishaw S, Thapar AK, et al. Association of Genetic Risk Variants With Attention-Deficit/Hyperactivity Disorder Trajectories in the General Population. *JAMA Psychiatry*. 2016;73(12):1285–1292. https://doi.org/10.1001/jamapsychiatry.2016.2817.
23. Murray AL, Booth T, Auyeung B, Eisner M, Ribeaud D, Obsuth I. Outcomes of ADHD Symptoms in Late Adolescence: Are Developmental Subtypes Important? *J Atten Disord*. 2020;24(1):113–125. https://doi.org/10.1177/1087054718790588.
24. The BELLA study group, Döpfner M, Hautmann C, Görtz-Dorten A, Klasen F. Ravens-Sieberer U. Long-term course of ADHD symptoms from childhood to early adulthood in a community sample. *Eur Child Adolesc Psychiatry*. 2015;24(6):665–673. https://doi.org/10.1007/s00787-014-0634-8.
25. Murray AL, Ushakova A, Speyer L, Brown R, Auyeung B, Zhu X. Sex/gender differences in individual and joint trajectories of common mental health symptoms in early to middle adolescence. *JCPP Adv*. 2022;2(1), e12057. https://doi.org/10.1002/jcv2.12057.
26. Murray AL, Eisner M, Obsuth I, Ribeaud D. Identifying Early Markers of "Late Onset" Attention Deficit and Hyperactivity/Impulsivity Symptoms. *J Atten Disord*. 2020;24(13):1796–1806. https://doi.org/10.1177/1087054717705202.
27. Faraone SV, Banaschewski T, Coghill D, et al. The World Federation of ADHD International Consensus Statement: 208 Evidence-based conclusions about the disorder. *Neurosci Biobehav Rev*. 2021;128:789–818. https://doi.org/10.1016/j.neubiorev.2021.01.022.
28. Thapar A, Cooper M. Attention deficit hyperactivity disorder. *Lancet*. 2016;387(10024):1240–1250. https://doi.org/10.1016/S0140-6736(15)00238-X.
29. Barkley RA. *Attention-Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment*. 4. Guilford Press; 2015.
30. Michelini G, Barch DM, Tian Y, Watson D, Klein DN, Kotov R. Delineating and validating higher-order dimensions of psychopathology in the Adolescent Brain Cognitive Development (ABCD) study. *Transl Psychiatry*. 2019;9(1):261. https://doi.org/10.1038/s41398-019-0593-4.
31. Sayal K, Prasad V, Daley D, Ford T, Coghill D. ADHD in children and young people: prevalence, care pathways, and service provision. *Lancet Psychiatry*. 2018;5(2):175–186. https://doi.org/10.1016/S2215-0366(17)30167-0.
32. Surman CBH, Goodman DW. Is ADHD a valid diagnosis in older adults? *ADHD Atten Def Hyp Disord*. 2017;9(3):161–168. https://doi.org/10.1007/s12402-017-0217-x.
33. ADHD Working Group of the Psychiatric Genomics Consortium (PGC). Early Lifecourse & Genetic Epidemiology (EAGLE) Consortium, 23andM Research Team, et al. Discovery of the first genome-wide significant risk loci for attention deficit/hyperactivity disorder. *Nat Genet*. 2019;51(1):63–75. https://doi.org/10.1038/s41588-018-0269-7.
34. Franz AP, Bolat GU, Bolat H, et al. Attention-Deficit/Hyperactivity Disorder and Very Preterm/Very Low Birth Weight: A Meta-analysis. *Pediatrics*. 2018;141(1), e20171645. https://doi.org/10.1542/peds.2017-1645.
35. Johnson S, Wolke D, Hennessy E, Marlow N. Educational Outcomes in Extremely Preterm Children: Neuropsychological Correlates and Predictors of Attainment. *Dev Neuropsychol*. 2011;36(1):74–95. https://doi.org/10.1080/87565641.2011.540541.
36. Turgay A, Goodman DW, Asherson P, et al. Lifespan Persistence of ADHD: The Life Transition Model and Its Application. *J Clin Psychiatry*. 2012;73(02):192–201. https://doi.org/10.4088/JCP.10m06628.
37. Young S, Adamo N, Ásgeirsdóttir BB, et al. Females with ADHD: An expert consensus statement taking a lifespan approach providing guidance for the identification and treatment of attention-deficit/hyperactivity disorder in girls and women. *BMC Psychiatry*. 2020;20(1):404. https://doi.org/10.1186/s12888-020-02707-9.
38. Bradley RH, Corwyn RF. Socioeconomic Status and Child Development. *Annu Rev Psychol*. 2002;53(1):371–399. https://doi.org/10.1146/annurev.psych.53.100901.135233.
39. Quinn PO, Madhoo M. A Review of Attention-Deficit/Hyperactivity Disorder in Women and Girls: Uncovering This Hidden Diagnosis. *Prim Care Companion CNS Disord*. 2014. https://doi.org/10.4088/PCC.13r01596. Published online May 15.
40. Hinshaw SP, Nguyen PT, O'Grady SM, Rosenthal EA. Annual Research Review: Attention-deficit/hyperactivity disorder in girls and women: underrepresentation, longitudinal processes, and key directions. *Child Psychol Psychiatry*. 2022;63(4):484–496. https://doi.org/10.1111/jcpp.13480.
41. Quinn PO. Treating adolescent girls and women with ADHD: Gender-Specific issues. *J Clin Psychol*. 2005;61(5):579–587. https://doi.org/10.1002/jclp.20121.
42. Nussbaum NL. ADHD and Female Specific Concerns: A Review of the Literature and Clinical Implications. *J Atten Disord*. 2012;16(2):87–100. https://doi.org/10.1177/1087054711416909.
43. Rucklidge JJ. Gender differences in ADHD: implications for psychosocial treatments. *Expert Rev Neurother*. 2008;8(4):643–655. https://doi.org/10.1586/14737175.8.4.643.
44. Becker SP, Kapadia DK, Fershtman CEM, Sciberras E. Evening circadian preference is associated with sleep problems and daytime sleepiness in adolescents with ADHD. *J Sleep Res*. 2020;29(1), e12936. https://doi.org/10.1111/jsr.12936.
45. Lopez R, Micoulaud-Franchi JA, Camodeca L, Gachet M, Jaussent I, Dauvilliers Y. Association of Inattention, Hyperactivity, and Hypersomnolence in Two Clinic-Based Adult Cohorts. *J Atten Disord*. 2020;24(4):555–564. https://doi.org/10.1177/1087054718775826.
46. Kim J, Lee GH, Sung SM, Jung DS, Pak K. Prevalence of attention deficit hyperactivity disorder symptoms in narcolepsy: a systematic review. *Sleep Med*. 2020;65:84–88. https://doi.org/10.1016/j.sleep.2019.07.022.
47. Liu CY, Asherson P, Viding E, Greven CU, Pingault JB. Early Predictors of De Novo and Subthreshold Late-Onset ADHD in a Child and Adolescent Cohort. *J Atten Disord*. 2021;25(9):1240–1250. https://doi.org/10.1177/1087054719892888.
48. Lecendreux M, Silverstein M, Konofal E, Cortese S, Faraone SV. A 9-Year Follow-Up of Attention-Deficit/Hyperactivity Disorder in a Population Sample. *J Clin Psychiatry*. 2019;80(3). https://doi.org/10.4088/JCP.18m12642.
49. Madiouni C, Broc G, Cindy L, Bayard S. Attention-Deficit/Hyperactivity Disorder, Insomnia, and Sleepiness Symptoms among a Community Adult Sample: The Mediating Effect of Executive Behavioral Regulation and Metacognition Abilities. *Arch Clin Neuropsychol*. 2022;37(5):916–928. https://doi.org/10.1093/arclin/acac006.
50. Maietta JE, Iverson GL, Cook NE. Lifetime history of concussion among children and adolescents with attention-deficit/hyperactivity disorder: examining differences stratified by age, medication status, and parent-reported severity. *Front Neurol*. 2025;15, 1487909. https://doi.org/10.3389/fneur.2024.1487909.
51. Stojanovski S, Scratch SE, Dunkley BT, Schachar R, Wheeler AL. A Systematic Scoping Review of New Attention Problems Following Traumatic Brain Injury in Children. *Front Neurol*. 2021;12, 751736. https://doi.org/10.3389/fneur.2021.751736.