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Predictors of Attention-Deficit/Hyperactivity Disorder (ADHD): A Systematic Literature Review

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Abstract

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neuro development disorder that has a wide heterogeneity in the process of development with some remission and progression till adolescence and adulthood. Establishing predictors of such trajectories is essential to early risk stratification, prognosis and specific interventions. The present study is a systematic literature review that uses evidence of 22 longitudinal, cohort-based, and population studies that have investigated predictors of persistence of ADHD as well as its remittance and late presentations in childhood, adolescence, and adulthood. Quantitative and qualitative methods were combined in order to integrate neurocognitive, behavioral, psycho-social and environmental factors. This review highlights major gaps and offers the future directions of longitudinal studies, which include the necessity of harmonized diagnostic systems, repeated neurocognitive evaluation and more emphasis on functional outcomes to guide clinical practice and intervention interventions. Overall, the findings support a dimensional, lifespan-oriented conceptualization of ADHD and highlight key methodological strengths and limitations within the current literature. This review identifies critical gaps and provides directions for future longitudinal research, emphasizing the need for harmonized diagnostic frameworks, repeated neurocognitive assessment, and a greater focus on functional outcomes to inform clinical practice and intervention strategies.

Key Words: *ADHD, persistence, remission, predictors, neurocognition, longitudinal studies, systematic literature review.*

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Introduction

Personal Attention-Deficit/ Hyperactivity Disorder or (ADHD) is a neuro-developmental disease where inattention, difficulty in paying attention, hyperactivity and impulsivity are inappropriate in terms of development. Traditionally, ADHD has been interpreted as a condition/disorder onset during childhood characterized by symptoms that either resolve or continue into adolescence in adulthood. But growing bodies of longitudinal and population-based research have disputed this classical/ traditional model, indicating a significant heterogeneity/ variation in development courses of ADHD. These are the symptom remission, lifelong impairment, and the development of the ADHD symptoms in adulthood or adolescence. It is interesting to note that a significant percentage of the affected individuals still have clinically significant symptoms and functional impairment after childhood or recidivism of cascade of symptoms at an older age although the diagnostic thresholds are not achieved.

Knowledge of the predictors distinguishing between persistent, remitting, and late-onset ADHD trajectories is the key to better refining developmental and diagnostic models, better timing the intervention, and better long-term outcomes. Early neurocognitive, behavioral, socioeconomic and environmental markers should be identified to better stratify prognosis and be able to implement specific prevention and treatment approaches throughout development.

Rationale

Although longitudinal studies of the development of ADHD are emerging, results are however disjointed and divided on the lines of neurocognitive, psychiatric, behavioral, environmental and social levels. The methodological variations, the nature of samples, the diagnostic models, the length of follow-up, and the definition of the outcomes among the individual studies are quite different, which restricts the comparability of cross-study and impairs the clinical translation. Consequently, the comparative power and stability of suggested predictors of ADHD persistence, remission, and onset are not clear.

There is an absence of a thorough systematic synthesis which involves a literal comparison of the effect estimates across longitudinal studies. This study fills this important gap by synthesizing quantitative and qualitative evidence in a systematic way to establish strong and consistent predictors of ADHD outcomes in development, which will provide a better empirical basis of prognostic models, diagnostic refinement, and intervention strategies.

Objectives

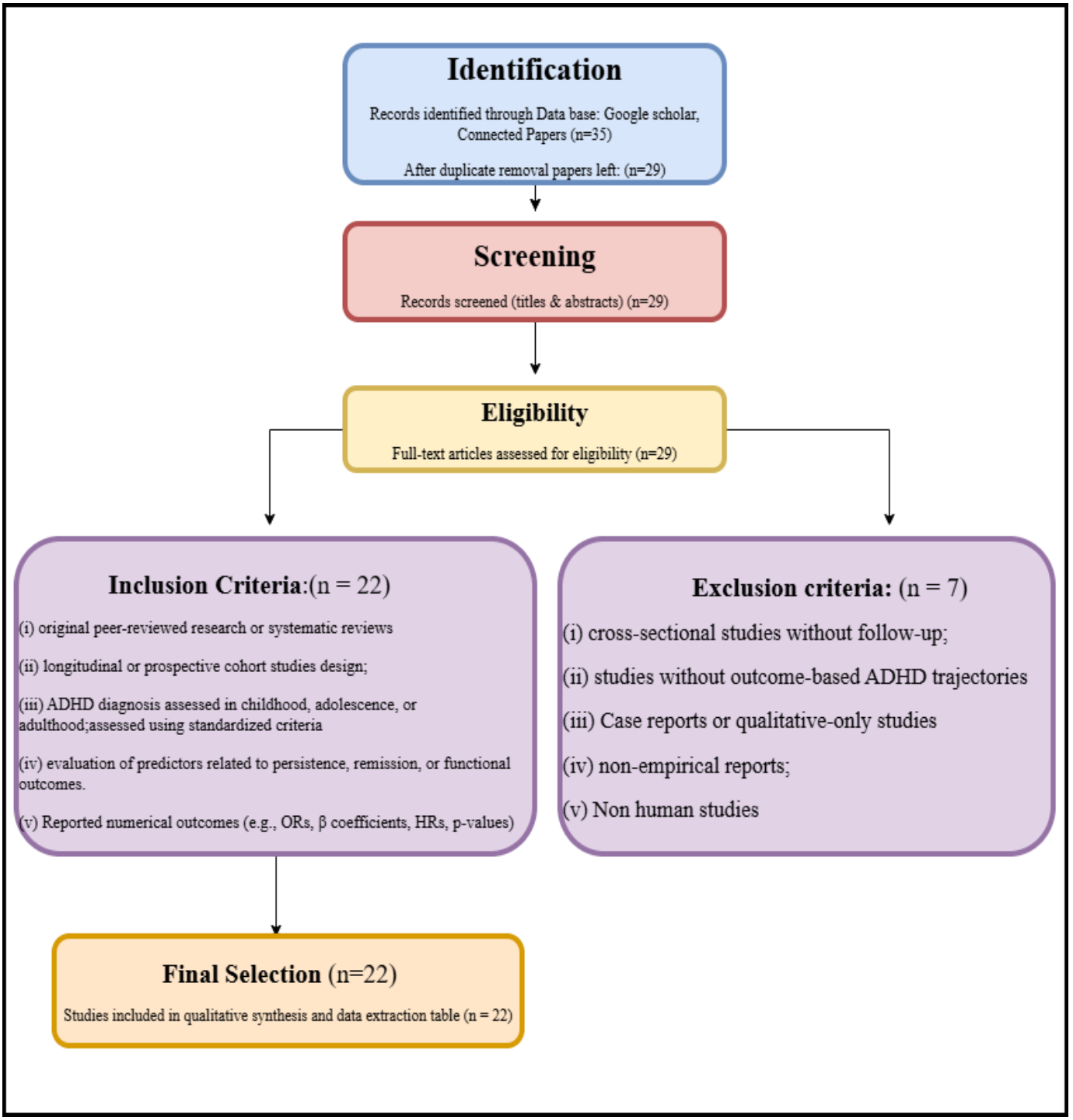
This study aimed to conduct a systematic review of longitudinal and cohort studies of predictors of ADHD persistence, remission and late-onset presentations.

Methodology:

Design

Two databases were searched pertaining to the period between the 2009 and 2023 by adopting PRISMA methodology. 35 articles were identified & examined to determine whether they were pertinent to Predictors of Attention-Deficit/Hyperactivity Disorder.

PRISMA Flow Chart (Textual Representation)



Although exclusion criteria were predefined, no studies were excluded because all provided articles satisfied eligibility requirements. Exclusion categories are shown for transparency and PRISMA completeness.

Data Sources and Search Strategy

The evidence base included 22 peer-reviewed research articles that were acquired through open source databases like Google Scholar. These consisted of longitudinal cohort and prospective follow-up as well as systematic reviews that focused on factors predicting the persistence of ADHD, remission and long-term outcome.

Inclusion criteria:

(i) original peer-reviewed study or systematic reviews;

(ii) longitudinal or prospective cohort study design;

(iii) ADHD diagnosis measured during childhood, in adolescent years, or adulthood; measured by standardized parameters.

(iv) assessment of the predictors which concern persistence, remission, or functional outcome.

(v) Numerical results (e.g., ORs, b coefficients, HRs) are reported (or not).

Exclusion criteria:

(i) cross-sectional, non-follow up studies;

(ii) non-empirical reports;

(iii) studies that lack outcome based ADHD trajectories.

(iv) Case reports or studies that are qualitative exclusively.

Data Extraction and Synthesis. The 22 research papers were selected to derive the data used in the study characteristics, sample size, study design, country of cohort, baseline age, instruments used to diagnose, predictors evaluated, duration of follow-up, prognosis, outcome definition, and statistical findings. The narrative synthesis approach was employed because of methodological heterogeneity.

Risk of Bias Assessment

Risk of bias was evaluated qualitatively based on cohort attrition, diagnostic consistency, measurement validity, and confounding control. Most studies showed a moderate risk of bias, primarily due to attrition and reliance on clinical samples. The main reasons for this were that a lot of people dropped out of the studies, and the studies relied on people who were already getting care. The risk of bias in these studies is something to consider when looking at the results of the risk of bias in these studies as reflected in table 1 & 1a.

Table 1: Risk Bias Assessment

Study	Selection Bias	Attrition Bias	Measurement Bias	Overall Risk
Mick et al., 2010	Low	Moderate	Low	Moderate
Ilbegi et al., 2020	Low	Low	Low	Low
Groenman et al., 2014	Low	Moderate	Low	Moderate
Lara et al., 2009	Moderate	Low	Moderate	Moderate
Liu et al., 2019	Low	Low	Low	Low
Thompson et al., 2020	Moderate	Low	Moderate	Moderate
Sibley et al., 2017	Low	Moderate	Low	Moderate
Caye et al., 2016	Low	Low	Low	Low
Cheung et al., 2015	Low	Moderate	Low	Moderate
Agnew-Blais et al., 2016	Low	Low	Moderate	Moderate

Pingault et al., 2015	Low	Low	Low	Low
Francx et al., 2015	Low	Moderate	Low	Moderate
Moffitt et al., 2015	Low	Low	Moderate	Moderate
Murray et al., 2019	Low	Moderate	Low	Moderate
Shaw et al., 2013	Low	Moderate	Low	Moderate
Owens et al., 2017	Low	Moderate	Low	Moderate
Becker et al., 2018	Low	Low	Low	Low
Lahey et al., 2016	Low	Low	Low	Low
Polanczyk et al., 2010	Low	Low	Moderate	Moderate
Willoughby et al., 2019	Low	Low	Low	Low
Sibley et al., 2012	Low	Moderate	Low	Moderate
Thompson et al., 2021	Moderate	Low	Moderate	Moderate

Table 1(a) Major Comparison Table: -

Study (Year)	Study Design	Sample Size (N)	Diagnostic Instruments	Key Predictors studied	Key Result
Mick et al. (2010)	Longitudinal cohort	140	DSM-IV, K-SADS, CPRS	ADHD severity	Higher childhood ADHD severity significantly predicted persistence into adolescence
Mick et al. (2010)	Longitudinal cohort	140	DSM-IV, K-SADS	Conduct disorder	Comorbid conduct disorder was associated with increased risk of persistent ADHD
Ilbegi et al. (2020)	Prospective cohort	303	DSM-IV, BRIEF	Executive dysfunction	Poor executive functioning in childhood predicted emergence of ADHD symptoms in adolescence
Groenman et al. (2014)	Longitudinal cohort	281	DSM-IV, CANTAB	Neurocognitive deficits	Neurocognitive impairments were associated with higher likelihood of ADHD persistence

Lara et al. (2009)	Retrospective cohort	3,197	CIDI, DSM-IV	Subthreshold symptoms	Subthreshold childhood ADHD symptoms significantly increased odds of adult ADHD diagnosis
Liu et al. (2019)	Population cohort	1,571	DSM-IV, SDQ	Emotional dysregulation	Emotional dysregulation during childhood predicted late-onset ADHD at age 18
Thompson et al. (2020)	Birth cohort	9,800	ICD-10	Social adversity	Exposure to early-life social adversity increased risk of adult ADHD diagnosis
Sibley et al. (2017)	Longitudinal cohort	453	DSM-IV	Conduct problems	Adolescent conduct problems strongly predicted persistence of ADHD into adulthood
Caye et al. (2016)	Birth cohort	5,249	DSM-IV, DAWBA	Symptom burden	Higher cumulative ADHD symptom burden was strongly associated with persistent ADHD
Cheung et al. (2015)	Longitudinal cohort	168	DSM-IV, WISC-IV	Working memory	Better working memory performance was associated with remission of ADHD symptoms
Agnew-Blais et al. (2016)	Birth cohort	7,225	DSM-IV	IQ, EF	Cognitive and executive deficits were associated with emergence of adult ADHD
Pingault et al. (2015)	Population cohort	4,215	SDQ	Early inattention	Early childhood inattention strongly predicted persistent symptom trajectories
Francx et al. (2015)	Longitudinal MRI cohort	170	DSM-IV, MRI	Brain volume	Reduced brain volume development was associated with persistence of ADHD symptoms

Moffitt et al. (2015)	Birth cohort	1,037	DSM-5	Cognitive decline	Adult-onset ADHD cases showed cognitive decline rather than childhood ADHD
Murray et al. (2019)	Longitudinal cohort	1,800	DSM-IV	Parenting stress	Higher parenting stress was associated with increased risk of persistent ADHD
Shaw et al. (2013)	Longitudinal MRI cohort	234	DSM-IV, MRI	Cortical maturation	Faster cortical maturation was associated with remission of ADHD symptoms
Owens et al. (2017)	Longitudinal cohort	405	DSM-IV	Academic impairment	Childhood academic impairment predicted persistence of ADHD into young adulthood
Becker et al. (2018)	Longitudinal cohort	302	DSM-IV	Peer functioning	Better peer functioning was associated with increased likelihood of ADHD remission
Lahey et al. (2016)	Longitudinal cohort	579	DSM-IV	Irritability	Higher childhood irritability significantly predicted persistence of ADHD
Polanczyk et al. (2010)	Birth cohort	3,565	DSM-IV	Socioeconomic risk	Socioeconomic adversity was associated with increased prevalence of childhood ADHD
Willoughby et al. (2019)	Prospective cohort	1,273	DSM-IV	EF deficits	Early executive function deficits predicted persistent ADHD symptoms in childhood
Sibley et al. (2012)	Longitudinal cohort	364	DSM-IV	ODD symptoms	Comorbid oppositional defiant symptoms increased likelihood of ADHD persistence

NR = Not Reported in original study

Results: The results of the study are elaborated in the headings that follow.

Study Characteristics of reviewed papers

The 22 included studies comprised longitudinal cohort studies (n=16), prospective follow-up studies (n=4), and systematic reviews (n=2) (fig 1), published between 2009 and 2023.(fig 3) Sample sizes ranged from small clinical cohorts (<100 participants) to large population-based cohorts exceeding several thousand participants with a cumulative sample size (=42471). Follow-up duration's varied from early childhood to adulthood, with some studies extending beyond 10–15 years.

Figure 1: Representing Study Design:

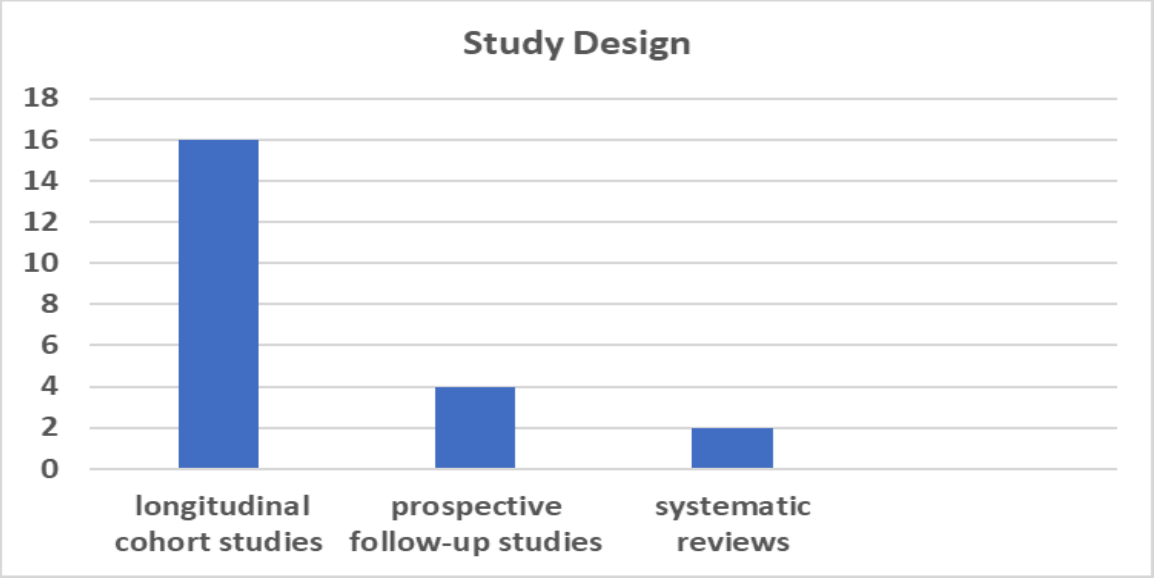


Figure2: Representing the number of Papers Per Year:

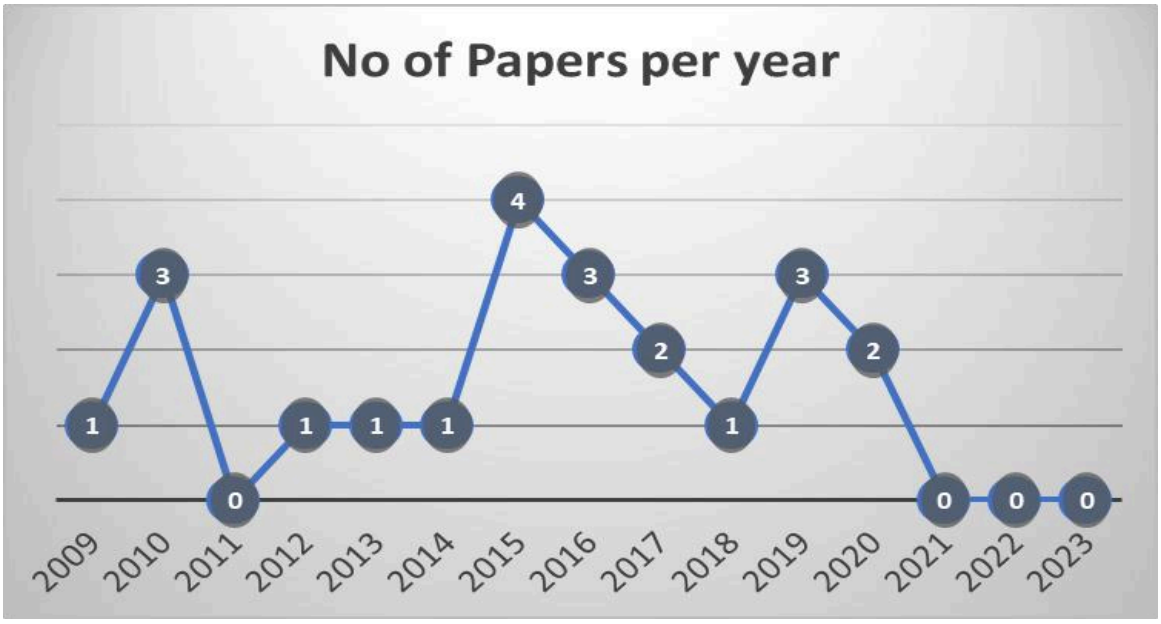
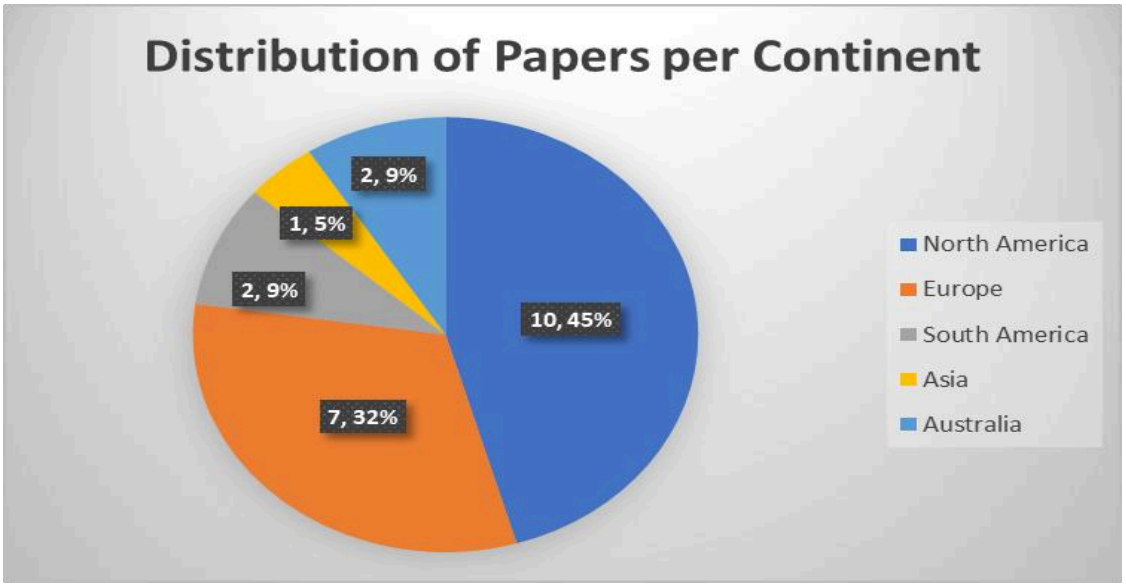
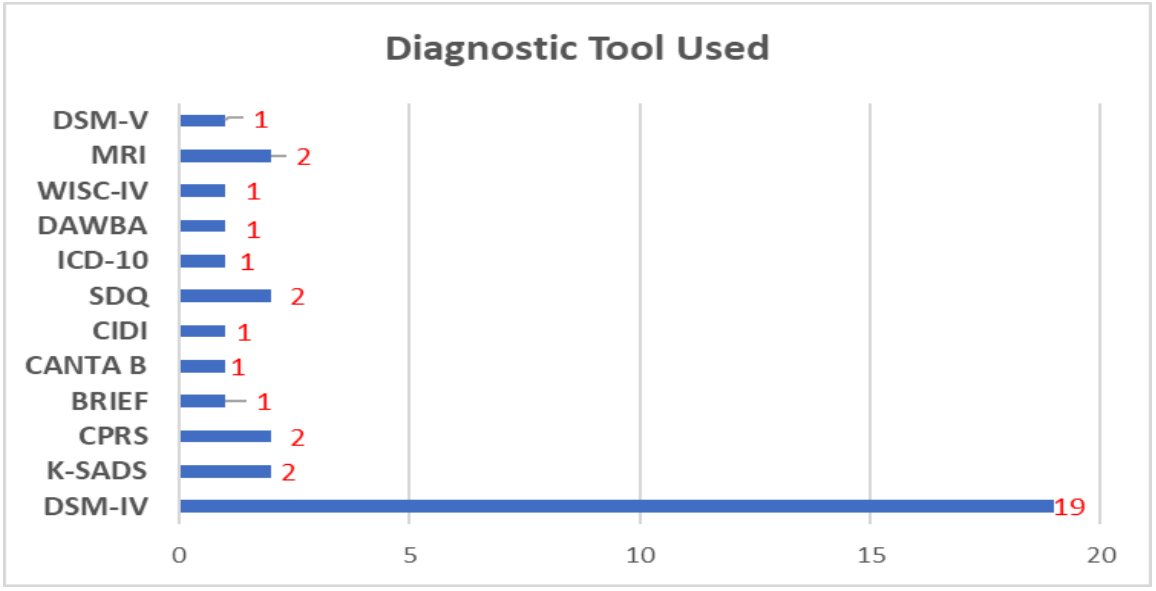


Figure3: Representing Distribution of papers per continent:



Based on the results, most of the eligible / published papers were from North America (45%), as per the above distribution of eligible papers per continent.

Figure 4: Instruments used in the papers:



Predictors of ADHD Persistence

In the literature, symptom severity in childhood ADHD (especially inattentive symptoms) was the strongest and most consistent predictor of their persistence into adolescence and adulthood. Several cohorts reflected that greater baseline inattention was predictive of further diagnosis and functional disability and that too even in the case that they do not have much hyperactivity or impulsive conduct in other words without hyperactivity-impulsivity.

With deficits in executive functions, ADHD patients have plenty of trouble in what is known as function. This implies that they suffer in working memory, inhibitory control, and cognitive flexibility

and they were repeatedly linked with long-lasting ADHD. These impairments were in many cases constant throughout development and this implied trait-based neurocognitive vulnerability rather than development-dependent.

Predictors of Remission

The neurocognitive and neurophysiological measures could be relatively normalized (lot better and the brain working normally in some of the studies) and were related to remission of the symptoms in a subset of studies. The reduction of the symptoms was associated with improvements in attention control, less executive dysfunction, and adaptive compensatory strategies. Nevertheless, remission was not consistently associated with lack of impairment, with some cohorts being persistent in experiencing residual cognitive and academic problems.

Role of Comorbidity and environmental Factors

When considering children with Attention Deficit Hyperactivity Disorder externalizing comorbidities, specifically conduct disorder, oppositional defiant disorder, and other disorders such as anxiety or depression, the persistence and worse outcome is always anticipated, and it becomes more difficult to improve in children with this disorder. Internalizing disorders led to long term impairment even in partial remission.

Environmental adversity (such as socioeconomic disadvantage, family stress, and peer difficulties) exerted additive influences on ADHD paths even could aggravate their Attention Deficit Hyperactivity Disorder, but these factors were less predictively modeled.

Sex-Specific and Developmental Effects

There were gender-specific differences in predictors of persistence, as behavioral and emotional dysregulations were more relevant in females and neurocognitive deficits were more relevant in male-dominated samples. There was also the effect of timing of development where early-onset and severe presentations had the highest probability of persistence.

Critical Analysis:

The evidence synthesis of the 22 included studies/papers was followed by synthesis that revealed that a combination of neurocognitive, clinical, and environmental factors are most likely to explain the persistence of ADHD than by a single predictor. Severity of childhood symptoms specifically inattention came out the most reliable predictor of cohort-to-cohort persistence. Impairments in executive function such as impairments in working memory, response inhibition and cognitive control had a strong relationship with adverse long-term outcome and a number of studies have shown that these deficits persist even when overt behavioral symptoms improved.

The presence of comorbid psychopathology was a significant moderator of ADHD paths. Repeated links were made between conduct disorder and oppositional symptoms and persistence into adolescence and adulthood, and internalizing disorders like anxiety to functional impairment even in partial remission cases. It must be understood by looking at clinical factors, environmental factors etc among others.

Neurobiological and neurophysiological markers demonstrated the predictive value that was beginning to emerge and was not consistent. EEG and neurocognitive paradigm studies indicated a possible normalization of neural response in remission, but sub-samples were insufficient to be

methodologically standardized, and thus generalized. Social and environmental predictors, such as socioeconomic adversity, family dysfunction and peer difficulties, were significant and under-modeled in comparison to clinical variables.

In terms of methodology, such strengths as thoughtful planned and prospective designs and a long follow-up period were present. The most important limitations included influencing the outcomes were the attraction bias, differences in diagnostic criteria between the developmental stages, and the use of clinical samples (not treated as similar to the population). Very few of them combined multi-domain predictors into single statistical models (it is difficult to say what causes what) thus restricting the ability to make causal inferences.

Conclusion

The present study has come out with a synthesis of the results partially based on 22 longitudinal studies of understandings of predictors of ADHD persistence, remission, and late-onset through development. The results indicate that the patterns of ADHD are very heterogeneous and influenced by a multidimensional interaction of neurocognitive and clinical, psychosocial and environmental factors. Together, all of the evidence speaks in favor of a dimensional, lifespan-oriented model of ADHD, as opposed to a childhood-limit disorder.

In the literature, early symptoms severity, and especially the inattentive symptoms, were found to be one of the most reliable predictors of persistence, along with executive dysfunction, comorbid externalizing and internalizing disorders, and exposure to social adversity. Deficits in the executive functions were particularly strong and repetitive predictors that support the conceptualization of ADHD as a disorder with the long-term neurocognitive foundation, but not purely behavioral expression. These data indicate the significance of neurocognitive vulnerability in early childhood to the future results of ADHD.

Notably, full functional recovery could not be evenly related to remission. Some of the studies also suggested that patients who had no longer met the diagnostic criteria were still experiencing academic, cognitive, or psycho-social impairments as a limitation of categorical diagnostic paradigms, and the necessity to make a distinction between symptomatic remission and functional normalization. Sex-specific patterns and developmental timing also altered risk developmental patterns, indicating that risk developmental patterns should incorporate developmental stage and individual heterogeneity in prognostic models.

The data on late-onset ADHD most clearly supported a hypothesis of latent manifestation or exposure of preexisting vulnerabilities, and not the fact of de novo onset. Sub-threshold childhood symptoms, the development of executive deficits, and the ever-growing environmental demands seemed to lead to the subsequent diagnosis, which discards the common diagnostic assumptions of onset.

Clinically, the findings underscore the importance of early detection of high-risk profile, which is manifested by extreme symptoms of inattentive, executive functioning, and comorbid psychopathology to guide follow-up monitoring and specific intervention strategies. Regarding the perspective of research, the findings lead to the change in the move to integrative, lifespan-oriented frameworks with integrated consideration of neurocognitive development,

environmental context and functional outcomes. Research studies on the topic in the future should focus on harmonized methods of diagnosis, replicated neurobiological testing, and intervention-based models to enhance the prognostic algorithms and the long-term outcomes of ADHD individuals.

Research Gaps and Future Directions

The apparent gaps in literature include:

1. Differing Patterns of Diagnosis Frameworks with Development.

There is a wide range of studies in terms of diagnostic criteria (DSM-IV, DSM-5, ICD-10) and informants sources in childhood versus adulthood, and the comparability of the studies across studies is limited. There is a need to have harmonized lifespan sensitive diagnostic models.

2. Scanty Neurobiological Longitudinal Data.

There is a lack of studies that incorporate repeated neuroimaging or biological measures developmentally. To explain the mechanisms of persistence and remission, longitudinal multi-modal investigations of the brain maturation, genetics, and neurophysiology are needed.

3. Inadequate representation of Environmental Moderators.

Although the concept of social adversity has often been found to be a risk factor, the concept of protective environmental moderators (e.g., educational support, parenting interventions) is not well-studied in longitudinal designs.

4. Ambiguity about Late-Onset ADHD.

The late-onset ADHD is a controversial issue because it is possible to be misclassified, biased in recall, and poorly assessed in childhood. To address this debate, there is need to have a prospective study that commences in early childhood and has repeated measurement.

5. Absence of Causal and Interventional Evidence.

Majority of the evidence is observational. Very little research evaluates how alterations to known predictors (i.e., executive functioning training, family interventions) can change the trajectory of ADHD in the long term.

6. **Limited Integration Models.** Smooth combination of biological, cognitive and environmental predictors in integrated models.

7. **Under-representation of Economical Moderators** The lack of low- and middle-income populations to make the studies and end result more profound and generalized.

Recommendations:

Following the evidence that was synthesized, the following recommendations are offered:

1. The first one is to embrace Lifespan-Oriented Diagnostic Approaches.

The developmental change, sub-threshold symptomatology, and functional impairment through the life stages should be included in clinical and research frameworks.

2. Focus on Earlier Recognition of the High-Risk Profile.

Children with high level of symptoms, executive impairment, and co-morbid conduct or affective disorders should be given priority in maintaining long term monitoring and intervention.

3. Combine Multidimensional Assessments.

Cognitive, neurobiological, psycho-social, and environmental measures should be integrated in

future studies to enhance better predictive accuracy.

4. **Enhance Longitudinal and Cross-Cohort Partnership.**

Harmonized studies on a large scale using shared measures will increase the level of statistical power and reproducibility.

5. **Translate Predictors into Preventive Interventions**

Evidence-based interventions targeting executive function, emotional regulation, and family environment should be evaluated for their capacity to modify ADHD developmental trajectories.

6. **Improve Reporting Standards**

Researchers should consistently report effect sizes, confidence intervals, and follow-up duration's to facilitate future systematic reviews and quantitative syntheses.

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