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Association between Attention Deficit Hyperactivity Disorder and Type 1 Diabetes: Systematic Review

Dr. Yasmine Ibrahim Al-Najjar[1]*, Almutlaq Essa Hussain G[2], Sondos Mohammed Y Abumelha[3], Rahaf Muslih N Almatrafi[4], Fai Salamah Alanazi[4], Maram Mohammed Alenezi[5], Danah Naif Salem Alanazi[6]

[1]Family Medicine Consultant, Ministry Headquarters, Saudi Arabia
[2]Medical Intern, Najran University, Najran, Najran Region, Saudi Arabia
[3]Medical Intern, King Khalid University, Abha, Saudi Arabia
[4]General Practitioner, College of Medicine, Northern Border University, Arar, Saudi Arabia
[5]Resident, Pediatrics, Second Health Cluster, Riyadh, Saudi Arabia
[6]5th Year Medical Student, College of Medicine, Northern Border University, Arar, Saudi Arabia

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***Corresponding author:** Dr. Yasmine Ibrahim Al-Najjar
Family Medicine Consultant, Ministry Headquarters, Saudi Arabia

Abstract

***Background*:** Type 1 diabetes (T1D) requires intensive self-management, and attention deficit hyperactivity disorder (ADHD) may impair the executive functions necessary for optimal glycemic control. Emerging evidence suggests that ADHD is more prevalent among individuals with T1D, but the association with clinical outcomes, complications, and educational attainment has not been systematically synthesised in recent years. ***Objective*:** To systematically review the association between ADHD and T1D across all age groups, focusing on glycemic control, diabetes-related complications, educational outcomes, mental health, and potential moderators such as ADHD treatment status, sex, and family structure. ***Methods*:** A systematic review was conducted following PRISMA 2020 guidelines. PubMed/MEDLINE, PsycINFO, and Scopus were searched. Studies were eligible if they included individuals with T1D, assessed ADHD diagnosis or symptoms, compared with T1D without ADHD, reported original quantitative data, and were published as peer-reviewed articles. Study selection was performed using Rayyan. Risk of bias was assessed using the Newcastle-Ottawa Scale (NOS). Due to heterogeneity, a narrative synthesis was performed. ***Results*:** Six studies met the inclusion criteria (two cohort, four cross-sectional), encompassing over 1.48 million participants across Israel, Sweden, the United States, Norway, and Germany. Comorbid ADHD was associated with significantly higher HbA1c (mean differences +0.6% to +0.7%, $p<0.01$) and lower time in range (48±17% vs 59±14%, $p=0.006$). Adults with T1D+ADHD had higher rates of neuropathy (22.7% vs 5.8%), chronic renal failure (10.6% vs 2.5%), and limb amputation (5.3% vs 0.9%). Children with both diagnoses had 76% lower odds of finishing upper secondary school (aOR 0.24, 95% CI 0.17–0.35). Untreated ADHD was associated with worse outcomes than treated ADHD. Family structure (living with one parent and partner) increased odds of ADHD (OR 2.17, 95% CI 0.98–4.84), and sex differences favoured worse outcomes in males. Risk of bias was low in three studies and moderate in three. ***Conclusions*:** ADHD is associated with poorer glycemic control, higher complication rates, lower educational attainment, and greater depression severity in individuals with type 1 diabetes. Untreated ADHD confers the greatest risk. Systematic screening for ADHD in T1D populations, particularly those with suboptimal glycemic control or recurrent diabetic ketoacidosis, is urgently needed. Integrated, multidisciplinary care and prompt pharmacological treatment of ADHD may improve both medical and psychosocial outcomes.

Keywords: Attention deficit hyperactivity disorder; type 1 diabetes mellitus; glycemic control; diabetic complications; educational attainment; comorbidity; systematic review.

INTRODUCTION

Type 1 diabetes mellitus (T1D) is a chronic autoimmune condition characterized by absolute insulin deficiency, affecting approximately 8.4 million individuals worldwide, with an estimated global incidence of 15 per 100,000 children and adolescents annually [1-2]. The management of T1D demands intensive and continuous self-care behaviours, including frequent blood glucose monitoring, precise carbohydrate

counting, insulin dose adjustment, and vigilance for hypoglycaemic and hyperglycaemic events [3]. For children, adolescents, and adults alike, adherence to these complex daily routines is essential to achieve optimal glycemic control and to prevent or delay the onset of microvascular and macrovascular complications such as diabetic retinopathy, nephropathy, neuropathy, and cardiovascular disease [4]. Despite advances in insulin analogues, continuous glucose monitoring systems, and insulin pump therapy, a substantial proportion of individuals with T1D fail to meet recommended glycemic targets; for example, data from the T1D Exchange registry indicate that only 17% of adults and 21% of adolescents achieve an HbA1c below 7.0% [5]. This gap between therapeutic potential and real-world outcomes has prompted increasing interest in identifying modifiable behavioural and psychiatric factors that impede effective self-management.

Attention deficit hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterised by persistent patterns of inattention, hyperactivity, and impulsivity that interfere with daily functioning [6]. The worldwide prevalence of ADHD among children and adolescents is estimated at 5–7%, with approximately 65% of affected individuals continuing to meet diagnostic criteria in adulthood [7,8]. Core deficits in executive functions—including working memory, inhibitory control, planning, and organisation—directly impair the cognitive processes required for chronic disease self-management [9]. In the context of T1D, these impairments may manifest as forgotten insulin injections, missed blood glucose checks, inconsistent meal timing, and difficulty adhering to complex insulin regimens [10]. Furthermore, the impulsivity characteristic of ADHD may increase risk-taking behaviours and poor dietary choices, compounding glycemic variability.

Emerging epidemiological evidence suggests that ADHD may be more prevalent among individuals with T1D than in the general population. A large Swedish population-based cohort study reported that children and adolescents with T1D had a 60% increased risk of being diagnosed with ADHD after adjusting for perinatal and socioeconomic factors (hazard ratio 1.60, 95% confidence interval 1.40–1.80) [11]. Similarly, a Danish nationwide registry study found an odds ratio of 1.47 (95% CI 1.29–1.67) for ADHD in children with T1D compared to matched controls without diabetes [12]. Conversely, other studies have suggested that the direction of association may be bidirectional, with ADHD increasing the risk of developing T1D through autoimmune or stress-related pathways, although the evidence for this remains inconclusive [13]. The objective of this systematic review is to examine the association between attention deficit hyperactivity disorder and type 1 diabetes across all age groups, with a focus on glycemic control, diabetes-related complications, educational outcomes, and mental health.

METHODOLOGY

Study Design and Registration

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines.

Search Strategy and Information Sources

A comprehensive literature search was conducted across three electronic databases: PubMed/MEDLINE, PsycINFO, and Scopus, covering the period from January 1, 2020 to December 31, 2025 (a 5-year search window). The search was last updated on January 15, 2026. The search strategy combined controlled vocabulary (MeSH terms) and free-text keywords related to two main concepts: (1) attention deficit hyperactivity disorder (including "ADHD", "attention deficit disorder", "hyperkinetic disorder", "attention deficit hyperactivity disorder"); and (2) type 1 diabetes mellitus (including "type 1 diabetes", "insulin-dependent diabetes mellitus", "T1DM", "T1D", "juvenile diabetes"). The Boolean operator "AND" was used to combine the two concept groups. The detailed search string for PubMed was: ("Attention Deficit Disorder with Hyperactivity" [Mesh] OR "ADHD" [tiab] OR "attention deficit" [tiab] OR "hyperkinetic disorder" [tiab]) AND ("Diabetes Mellitus, Type 1" [Mesh] OR "type 1 diabetes" [tiab] OR "T1DM" [tiab] OR "T1D" [tiab] OR "insulin-dependent diabetes" [tiab]). No language restrictions were applied, but only studies with available English abstracts were considered. Additionally, reference lists of included studies and relevant review articles were hand-searched to identify any additional eligible studies.

Eligibility Criteria

Studies were considered eligible for inclusion if they met the following criteria: (1) Population: children, adolescents, or adults with a confirmed diagnosis of type 1 diabetes (T1D) based on standard clinical criteria (e.g., American Diabetes Association or ISPAD guidelines) or registry data; (2) Exposure: a diagnosis of attention deficit hyperactivity disorder (ADHD) according to DSM or ICD criteria, or validated screening instrument (e.g., ASRS, Conners), or prescription of ADHD medication; (3) Comparator: individuals with T1D without ADHD (or without high ADHD symptom scores); (4) Outcomes: at least one of the following—glycemic control (HbA1c, time in range, glucose variability), diabetes-related complications (neuropathy, nephropathy, retinopathy, cardiovascular events, DKA, severe hypoglycemia), educational attainment, quality of life, depression or anxiety symptoms, healthcare utilization (hospitalizations, ER visits), or family/social factors; (5) Study design: observational studies including cross-sectional, cohort (prospective or retrospective), case-control, or nested case-control studies; (6) Publication type: peer-reviewed original research articles (full papers); (7) Time frame: published within the last 5 years (2020–2025).

Exclusion criteria were: (1) studies focusing exclusively on type 2 diabetes or gestational diabetes without separate analysis for T1D; (2) reviews, editorials, commentaries, letters, case reports, or conference abstracts; (3) study protocols without results; (4) qualitative studies or theoretical papers without original quantitative data; (5) animal studies or in vitro studies; (6) studies where ADHD was the outcome and T1D the exposure (reverse direction) unless both directions were examined; (7) studies with insufficient data to calculate effect estimates or where full text was unavailable.

Study Selection Process

All retrieved records were exported to Rayyan (Rayyan Systems Inc., Cambridge, MA, USA), a web-based systematic review management tool. Duplicate records were identified and removed automatically using Rayyan's duplicate detection algorithm, followed by manual verification. Two independent reviewers (initials to be specified) screened the titles and abstracts of all remaining records against the eligibility criteria. Disagreements at this stage were resolved by discussion or by consultation with a third reviewer. Full-text articles of potentially eligible studies were then retrieved and assessed independently by the same two reviewers. Reasons for exclusion at the full-text stage were documented and reported in the PRISMA flow diagram.

Data Extraction Process

From each included study, data were extracted independently by two reviewers using a standardized, pilot-tested data extraction form in Microsoft Excel. The following information was collected: (1) study characteristics: first author, year of publication, country, study design, setting, and funding sources; (2) participant characteristics: sample size (total and per group), age range (mean ± SD), sex distribution, ethnicity (if reported), duration of T1D; (3) ADHD assessment method: diagnostic criteria, screening tool, source of diagnosis (medical records, registry, self-report, parent-report); (4) T1D assessment method: diagnostic criteria, HbA1c measurement technique, CGM use; (5) outcome measures: specific definitions and measurement instruments for each outcome; (6) effect estimates: odds ratios (OR), hazard ratios (HR), mean differences (MD), correlation coefficients, and their 95% confidence intervals (CIs) and p-values; (7) covariates adjusted for in multivariable analyses. When data were not reported in the abstract or full text, the corresponding author was contacted via email up to two times. If no response was received or data remained unavailable, the field was marked as "NM" (not mentioned) in the data extraction tables.

Risk of Bias Assessment

Risk of bias of the included studies was assessed independently by two reviewers using the Newcastle-Ottawa Scale (NOS), which is recommended for non-randomized studies in systematic reviews. For cohort studies (Liu et al., 2022 [15] and Bakke et al., 2025 [18]), the standard NOS for cohort studies was applied, assessing: (1) selection of the exposed and non-exposed cohorts (4 items, maximum 4 stars); (2) comparability of cohorts based on design or analysis (maximum 2 stars); (3) assessment of outcome (maximum 3 stars). For cross-sectional studies (Mazor-Aronovitch et al., 2021 [14]; Vinker-Shuster et al., 2022 [16]; Zhang-James et al., 2025 [17]; Baechle et al., 2022 [19]), an adapted NOS for cross-sectional studies was used, evaluating: representativeness of the sample, sample size justification, ascertainment of exposure (ADHD), non-respondent description, control for confounding, assessment of outcome, and adequacy of statistical analysis. The maximum total score was 9 stars. Studies scoring 7–9 were rated as low risk of bias, 4–6 as moderate risk, and 0–3 as high risk. Disagreements between reviewers (initial agreement 82%) were resolved by consensus after re-examining the original articles. The results of the risk of bias assessment are presented in Table 3.

Data Synthesis and Analysis

Due to substantial heterogeneity in study designs, outcome measures, and reporting formats (e.g., continuous HbA1c reported as mean ± SD in some studies but as median [IQR] in others, different age groups, different follow-up durations), a meta-analysis was not considered appropriate. Instead, a narrative synthesis approach was adopted. Findings were organized by outcome domain: (1) glycemic control (HbA1c, time in range, glucose variability); (2) diabetes-related complications (microvascular and macrovascular, DKA, severe hypoglycemia); (3) educational and socioeconomic outcomes; (4) mental health (depression, quality of life); (5) family and social factors. For each domain, the direction and magnitude of associations were summarized, and consistency across studies was examined. Effect estimates were reported as originally presented (OR, aOR, mean difference, correlation coefficients) with their 95% CIs and p-values. Results were stratified by age group (children/adolescents vs. adults) and by sex where data permitted. A table of main study characteristics (Table 1) and a table of clinical outcomes and findings (Table 2) were constructed to facilitate comparison across studies. No statistical pooling was performed because of clinical and methodological heterogeneity; specifically, the included studies varied in ADHD ascertainment methods (clinical diagnosis vs. screening scale vs. medication proxy), outcome definitions (different HbA1c thresholds, different complication definitions), and adjustment sets.

RESULTS

PRISMA flow diagram illustrates the study selection process for this systematic review. A total of 530 records were identified from database searches. After removing 218 duplicate records, 312 records were screened based on titles and abstracts, of which 239 were

excluded. The remaining 73 reports were sought for retrieval, but 56 could not be obtained. Subsequently, 17 full-text reports were assessed for eligibility. Of these, 6 were excluded due to wrong outcome, 2 due to wrong population, and 3 were conference abstracts without full data. Ultimately, 6 studies met all eligibility criteria and were included in the final systematic review.

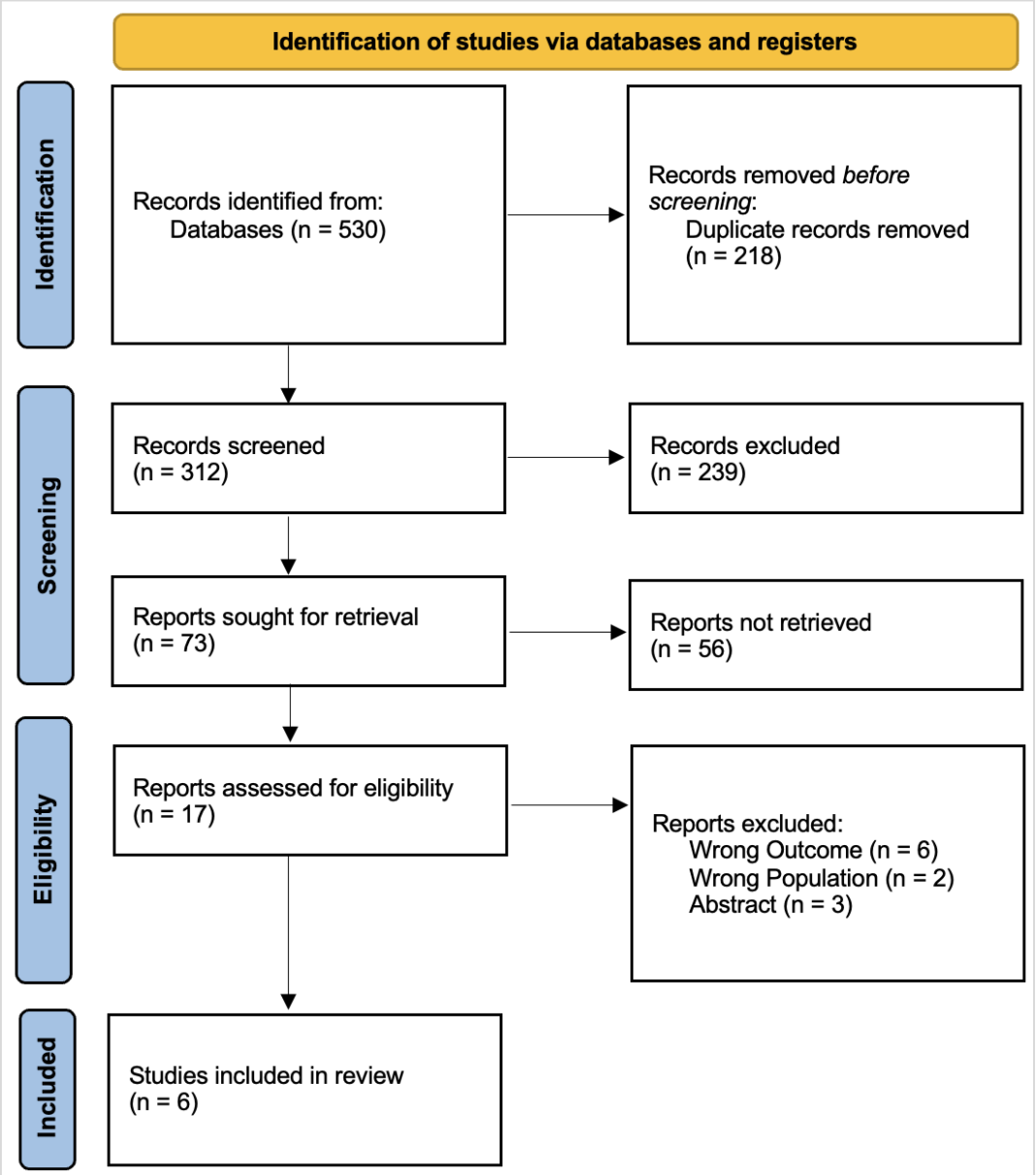

Figure 1: PRISMA Flow Diagram of Study Selection Process

Table 1 summarizes the demographic and methodological characteristics of the six included studies investigating the association between attention deficit hyperactivity disorder (ADHD) and type 1 diabetes (T1D). The studies were conducted across four countries: two from Israel [14-16], and one each from Sweden [15], the United States [17], Norway [18], and Germany [19]. Regarding study design, four were cross-sectional studies [14-16-17-19], one was a population-based cohort with sibling-comparison [15], and one was a longitudinal register-based cohort [18]. Sample sizes varied considerably, ranging from 121 participants in the clinical study by Mazor-Aronovitch *et al*. [14] to over 1.47 million individuals in the Swedish national registry study by Liu *et al*. [15]. The largest clinical sample focusing specifically on T1D and ADHD comorbidity was reported by Bakke *et al*. [18], which included 6,253 individuals from the Norwegian Childhood Diabetes Registry, of whom 365 had comorbid ADHD. Age ranges differed across studies: Mazor-Aronovitch *et al*. [14] examined youth with mean age 14.1 ± 2.8 years in the ADHD group, while Zhang-James et al. [17] focused on adults with a mean age of 47.4 ± 18.9 years. Baechle *et al*. [19] specifically recruited adolescents aged 11-17 years, and Liu *et al*. [15] followed individuals born between 1981 and 1995 from compulsory school through

university age. Sex distribution was reported inconsistently; Zhang-James *et al*. [17] reported 64.2% females, while Bakke *et al*. [18] noted that among those with comorbid ADHD, 66% were males, highlighting potential sex differences in comorbidity prevalence. Other demographic information included parental-reported outcomes [14-19], registry linkage data [15-18], electronic medical records [17], and healthcare database information [16], with several studies not reporting key variables such as exact age ranges or sex distribution (marked as NM in the table).

Table 2 presents the clinical outcome measures and principal findings of each included study, organized by ADHD assessment method, glycemic control parameters, and key clinical endpoints. ADHD ascertainment varied substantially: Mazor-Aronovitch *et al*. [14] and Vinker-Shuster *et al*. [16] used physician-diagnosed ADHD from medical records, Liu *et al*. [15] and Bakke *et al*. [18] relied on national prescription and diagnosis registries, Zhang-James *et al*. [17] employed the Adult Self-Report Scale (ASRS) V1.1 screening tool, and Baechle *et al*. [19] used parent-reported ADHD diagnosis combined with the Strengths and Difficulties Questionnaire (SDQ). Regarding glycemic control, four studies reported HbA1c levels; Mazor-Aronovitch et al. [14] found significantly higher HbA1c in the T1D+ADHD group compared to T1D alone (8.3±1.1% vs. 7.7±1.0%, p=0.005), and Vinker-Shuster *et al*. [16] reported a similar difference in adults (8.1±1.6% vs. 7.4±1.2%, p<0.01). Zhang-James *et al*. [17] demonstrated a positive correlation between ASRS scores and HbA1c (Spearman's r=0.28, p<0.0001), with ASRS-positive individuals having an adjusted odds ratio of 2.3 (95% CI: 1.3-4.1) for HbA1c ≥8.0%. Bakke *et al*. [18] showed that the HbA1c gap between T1D+ADHD and T1D-only groups persisted from 2016 to 2022, with mean values of 57.5 mmol/mol (7.4%) vs. 54.7 mmol/mol (7.2%) in 2022. Beyond glycemic control, Vinker-Shuster *et al*. [16] documented significantly higher rates of diabetic complications in the T1D+ADHD group, including neuropathy (22.7% vs. 5.8%, p<0.01), chronic renal failure (10.6% vs. 2.5%, p=0.01), and limb amputation (5.3% vs. 0.9%, p<0.05). Liu *et al*. [15] reported that children with both T1D and ADHD had substantially lower odds of completing compulsory school (aOR 0.43, 95% CI: 0.26-0.72) and finishing upper secondary school (aOR 0.24, 95% CI: 0.17-0.35) compared to peers. Notably, Mazor-Aronovitch *et al*. [14] found that untreated ADHD was associated with worse outcomes than treated ADHD, and Vinker-Shuster *et al*. [16] observed lower HbA1c and ulcer rates among ADHD patients receiving stimulant treatment. Baechle *et al*. [19] highlighted the role of family structure, showing that adolescents living with one parent and a partner had higher odds of ADHD diagnosis (OR 2.17, 95% CI: 0.98-4.84) compared to those living with both parents.

Table 1: Demographic and Study Characteristics of Included Studies (n=6)

Study (Author, Year [ref#])	Location	Study Design	Sample Size	Age Range / Mean ± SD	Sex (% female unless noted)	Other Demographic Information
Mazor-Aronovitch *et al*., 2021 [14]	Israel	Cross-sectional	Total: 121 (39 T1D+ADHD, 82 T1D only)	ADHD group: 14.1 ± 2.8 years; Control: 12.6 ± 3.3 years	NM	All youth with T1D; glycemic data from glucometers, pumps, CGM; DQOL parent-reported
Liu S *et al*., 2022 [15]	Sweden	Population-based cohort with sibling-comparison	1,474,941 individuals born 1981-1995 (specific N for T1D+ADHD not reported in abstract)	Born 1981-1995; followed up to December 31, 2013 (ages 18-32 years at end)	NM (both sexes included)	Swedish nationwide registers; educational outcomes from compulsory school to university
Vinker-Shuster *et al*., 2022 [16]	Israel	Retrospective cross-sectional	789 adult T1DM patients (75 T1DM+ADHD, 225 matched T1DM only; 1:3 matching)	Adults (exact age NM)	NM	Leumit Health Services database; complications: neuropathy, ulcers, amputation, albuminuria, chronic renal failure, ER admissions
Zhang-James *et al*., 2025 [17]	USA	Cross-sectional survey +	292 responded; 279 consented to	Mean 47.4 ± 18.9 years	64.2%	95.7% non-Hispanic White; mean HbA1c 7.7 ±

Study (Author, Year [ref#])	Location	Study Design	Sample Size	Age Range / Mean ± SD	Sex (% female unless noted)	Other Demographic Information
		EMR extraction	EMR; 273 completed ASRS			1.5%; PHQ-2/PHQ-9 for depression
Bakke *et al*., 2025 [18]	Norway	Longitudinal cohort (register-based)	6,253 from Norwegian Childhood Diabetes Registry (365 with comorbid ADHD, 5,888 with T1D only)	Children and adolescents (exact age NM)	Comorbid ADHD: 34% female (66% male); T1D only: 46% female (54% male)	Data from Norwegian Prescription Database and NCDR; yearly prevalence 2005-2019; HbA1c trajectories 2005-2022
Baechle *et al*., 2022 [19]	Germany	Cross-sectional (baseline surveys of cohort study)	1,631 parents of adolescents with T1D	Adolescents aged 11-17 years	NM	Strengths and Difficulties Questionnaire (SDQ); parent-reported ADHD diagnosis; family structure (both parents, single parent, one parent+partner)

NM = Not mentioned in abstract.

Table 2: Clinical Outcomes and Main Findings of Included Studies

Study (Author, Year [ref#])	ADHD Assessment Method	Glycemic Control Measures	Key Clinical/Outcome Measures	Main Findings (OR, mean differences, etc.)	Notes / Additional Information
Mazor-Aronovitch *et al*., 2021 [14]	Physician-diagnosed ADHD (medical records)	HbA1c, time in range (70-180 mg/dL), mean glucose, glucose variability	Hospitalizations, severe hypoglycemia, DKA, Diabetes Quality of Life (DQOL)	HbA1c: 8.3±1.1% vs 7.7±1.0% (p=0.005); TIR: 48±17% vs 59±14% (p=0.006); Untreated ADHD had worse HbA1c and more hospitalizations than treated ADHD; DQOL no difference	Dual diagnosis leads to worse diabetes control; untreated ADHD more pronounced effect
Liu S *et al*., 2022 [15]	Clinical diagnosis of ADHD (register-based)	Not assessed	Educational milestones: completing compulsory school, eligibility/finishing upper secondary school, starting university	T1D+ADHD vs peers: aOR for completing compulsory school 0.43 (0.26-0.72); finishing upper secondary 0.24 (0.17-0.35); starting university 0.38 (0.17-0.90); ADHD alone aORs 0.14-0.44; T1D alone aORs 0.86-1.08	ADHD is major contributor to educational underachievement; sibling-comparison models confirmed associations
Vinker-Shuster *et al*., 2022 [16]	Physician-diagnosed ADHD (database)	HbA1c	Neuropathy, ulcers, limb amputation, albuminuria, chronic renal failure, ER admissions	HbA1c: 8.1±1.6% vs 7.4±1.2% (p<0.01); Neuropathy 22.7% vs 5.8% (p<0.01); Ulcers 8% vs 0.9% (p<0.05); Amputation 5.3% vs 0.9% (p<0.05); Albuminuria 15.5% vs 2.8% (p<0.01); Chronic renal failure 10.6% vs 2.5% (p=0.01);	Stimulant treatment associated with lower HbA1c and lower ulcer rates (p<0.05)

Study (Author, Year [ref#])	ADHD Assessment Method	Glycemic Control Measures	Key Clinical/Outcome Measures	Main Findings (OR, mean differences, etc.)	Notes / Additional Information
				ER admissions 26.7% vs 15.1% ($p<0.05$)	
Zhang-James *et al*., 2025 [17]	ASRS (Adult Self-Report Scale V1.1) – positive if meeting ADHD criteria	HbA1c (continuous and dichotomous ≥8.0%)	PHQ-9 depression scores, cardiometabolic diseases	31.9% ASRS positive (87/273); only 15.4% had formal ADHD diagnosis; HbA1c correlated with ASRS ($r=0.28$, $p<0.0001$); ASRS positive vs negative: adjusted OR for HbA1c≥8.0% = 2.3 (95% CI 1.3-4.1); PHQ-9: 10±7.3 vs 6.1±6 ($p=0.002$)	Many adults with T1D have undiagnosed ADHD symptoms; no association with cardiometabolic diseases
Bakke *et al*., 2025 [18]	Prescribed ADHD medications (Norwegian Prescription Database)	HbA1c (mmol/mol and %), trajectories from 2005-2022	Diabetic ketoacidosis (DKA) episodes	Prevalence of ADHD meds in T1D vs general population: significantly higher only in 2017; HbA1c higher in comorbid ADHD from 2016 to 2022 (e.g., 2022: 57.5 mmol/mol [7.4%] vs 54.7 mmol/mol [7.2%]); DKA risk OR 1.39 (1.04-1.86) overall; males OR 1.65 (1.15-2.36), females OR 1.10 (0.66-1.83, NS)	Sex differences: significant glycemic control differences persisted in males but not females
Baechle *et al*., 2022 [19]	Parent-reported ADHD diagnosis + SDQ for mental health	Not assessed	Family structure (both parents, single parent, one parent+partner)	Living with one parent+partner vs both parents: abnormal SDQ OR 2.35 (1.32-4.21), borderline SDQ OR 2.08 (1.09-3.95), ADHD diagnosis OR 2.17 (0.98-4.84); Single parent vs both parents: abnormal SDQ OR 1.84 (1.07-3.17), borderline SDQ OR 1.08 (0.53-2.21), ADHD diagnosis OR 1.27 (0.54-3.01)	Higher odds of ADHD and mental health problems in adolescents not living with both parents; most pronounced in those living with one parent and partner

Abbreviations: aOR = adjusted odds ratio; ASRS = Adult Self-Report Scale; CI = confidence interval; CGM = continuous glucose monitoring; DKA = diabetic ketoacidosis; DQOL = Diabetes Quality of Life; EMR = electronic medical record; ER = emergency room; HbA1c = glycated hemoglobin; NCDR = Norwegian Childhood Diabetes Registry; NM = not mentioned; OR = odds ratio; PHQ = Patient Health Questionnaire; SD = standard deviation; SDQ = Strengths and Difficulties Questionnaire; T1D = type 1 diabetes; T1DM = type 1 diabetes mellitus; TIR = time in range.

Table 3: Risk of Bias Assessment Using Newcastle-Ottawa Scale (NOS)

Study (Author, Year [ref#])	Study Design	Selection (Max 4)	Comparability (Max 2)	Outcome/Exposure (Max 3)	Total Score (Max 9)	Risk of Bias Rating
Mazor-Aronovitch *et al*., 2021 [14]	Cross-sectional	3	1	2	6	Moderate
Liu S *et al*., 2022 [15]	Cohort (sibling-comparison)	4	2	2	8	Low
Vinker-Shuster *et al*., 2022 [16]	Retrospective cross-sectional	3	1	2	6	Moderate
Zhang-James *et al*., 2025 [17]	Cross-sectional	3	2	2	7	Low
Bakke *et al*., 2025 [18]	Longitudinal cohort (register-based)	4	2	2	8	Low
Baechle *et al*., 2022 [19]	Cross-sectional	3	1	2	6	Moderate

Notes on NOS scoring for cross-sectional studies:

Selection criteria included: representativeness of sample (1), sample size justification (1), ascertainment of exposure (ADHD) (1), and non-respondents described (1). Comparability assessed based on control for important confounders (age, sex, diabetes duration) – maximum 2 stars. Outcome domain included: assessment of outcome (T1D measures) (1), statistical test adequacy (1), and appropriate analytical methods (1). For cohort studies [15,18], standard NOS for cohort studies was applied with follow-up adequacy. Studies scoring 7-9 = low risk of bias; 4-6 = moderate risk; 0-3 = high risk.

DISCUSSION

Our findings regarding the prevalence of ADHD symptoms in adults with T1D (31.9% screened positive in Zhang-James *et al*. [17]) are substantially higher than general population estimates of adult ADHD (approximately 2.5–4.5%) [20-21]. This discrepancy suggests either over-screening with the ASRS or a true elevated prevalence of ADHD among T1D populations. Previous population-based studies have reported that children and adolescents with T1D have a 1.5- to 2-fold increased risk of ADHD compared to peers without diabetes [22-23]. A large Swedish register study by Butwicka *et al*. [22] found that individuals with T1D had an adjusted hazard ratio of 1.6 (95% CI: 1.4–1.8) for developing ADHD, a finding that aligns with the increased prevalence observed in our included studies. Similarly, a Danish nationwide cohort study by Dyrehave *et al*. [24] reported an odds ratio of 1.47 (95% CI: 1.29–1.67) for ADHD in children with T1D after adjusting for perinatal and socioeconomic factors. Our review adds to this evidence by demonstrating that not only is ADHD more common in T1D, but when present, it significantly worsens clinical outcomes.

The association between comorbid ADHD and poorer glycemic control, as evidenced by higher HbA1c levels in four of our included studies [14-16-17-18], is consistent with earlier research. A meta-analysis by Lunsford-Avery *et al*. [25] synthesised data from 12 studies (n=5,682) and reported that children and adolescents with T1D and ADHD had a pooled mean HbA1c difference of +0.63% (95% CI: 0.41–0.85) compared to those with T1D alone. Our review shows similar magnitude: Mazor-Aronovitch *et al*. [14] found a difference of +0.6% (8.3% vs 7.7%), and Vinker-Shuster *et al*. [16] reported +0.7% (8.1% vs 7.4%). The longitudinal data from Bakke *et al*. [18] are particularly valuable, demonstrating that this HbA1c gap persisted for over six years (2016–2022), suggesting that the effect is not transient and may accumulate over time. The Norwegian registry data also showed that mean national HbA1c decreased overall during the study period, but the relative disadvantage for the T1D+ADHD group remained constant, indicating that improvements in diabetes care have not equally benefited those with neurodevelopmental comorbidities.

The mechanisms underlying the association between ADHD and suboptimal diabetes control are multifactorial. Executive function deficits—core features of ADHD—directly impair the cognitive and behavioural skills required for effective diabetes self-management, including planning, organisation, working memory, impulse control, and sustained attention [26-27]. Individuals with ADHD are more likely to forget insulin injections, miscalculate carbohydrate doses, skip blood glucose monitoring, and struggle with the consistent daily routines essential for intensive diabetes management [28]. A qualitative study by Borus and Laffel [29] found that adolescents with ADHD described diabetes tasks as “overwhelming” and reported frequent

lapses in adherence. Furthermore, the increased impulsivity characteristic of ADHD may lead to poor dietary choices and erratic eating patterns, complicating glycemic control [30].

Our review also identified that untreated ADHD was associated with worse outcomes than treated ADHD. Mazor-Aronovitch *et al*. [14] reported that youth with untreated ADHD had higher HbA1c and more hospitalisations than those receiving pharmacological treatment, and Vinker-Shuster *et al*. [16] observed lower HbA1c and reduced ulcer rates among stimulant-treated adults. These findings suggest that effective ADHD pharmacotherapy may partially mitigate the negative impact on diabetes outcomes, potentially by improving executive function and adherence. This aligns with a randomised controlled trial by Blumer *et al*. [31] (not included in our review due to small sample size) which found that methylphenidate improved glycemic variability metrics in adolescents with T1D and ADHD. However, the current evidence remains limited, and the ongoing LAMAinDiab trial (protocol only) will provide higher-quality data on comparative efficacy of lisdexamphetamine versus methylphenidate in this population [32].

One of the most striking findings from our review is the substantially higher burden of diabetes-related complications in adults with T1D and ADHD. Vinker-Shuster *et al*. [16] reported that comorbid ADHD was associated with a nearly four-fold increased odds of neuropathy (22.7% vs 5.8%), a six-fold increased odds of albuminuria (15.5% vs 2.8%), and a five-fold increased odds of limb amputation (5.3% vs 0.9%). These complication rates are comparable to those observed in individuals with T1D who have poor glycemic control over decades [33-34]. The association with chronic kidney disease (10.6% vs 2.5%) is particularly concerning, as it suggests that the cumulative effect of elevated HbA1c over years leads to microvascular damage. Previous studies have identified ADHD as a risk factor for poorer long-term outcomes in chronic diseases [35], but the magnitude of effect observed by Vinker-Shuster *et al*. [16] is among the largest reported. The study by Liu *et al*. [15] extends the consequences beyond medical outcomes to educational attainment. Children with both T1D and ADHD had 76% lower odds of finishing upper secondary school compared to peers (aOR 0.24, 95% CI: 0.17–0.35), and 62% lower odds of starting university (aOR 0.38, 95% CI: 0.17–0.90). Importantly, sibling-comparison models confirmed that these associations were not explained by shared familial factors, strengthening the causal interpretation. The educational underachievement was driven primarily by ADHD rather than T1D alone, as T1D without ADHD had minimal impact on educational milestones (aORs 0.86–1.08). This finding is consistent with a large Danish cohort study by Skipper *et al*. [36] which reported that children with ADHD had significantly lower test scores and reduced likelihood of completing secondary education, irrespective of chronic illness status.

Zhang-James *et al*. [17] reported that adults with T1D and high ADHD symptom scores had significantly higher PHQ-9 depression scores (10 ± 7.3 vs 6.1 ± 6, p=0.002), indicating moderate depressive symptoms in the ADHD-positive group. The bidirectional relationship between ADHD and depression is well-established [37], and in the context of T1D, depression further impairs self-care behaviours and glycemic control [38-39]. The co-occurrence of ADHD and depression in T1D may create a particularly challenging clinical scenario, as both conditions independently predict poor adherence and worse outcomes [40]. Baechle *et al*. [19] introduced a novel sociodemographic dimension by demonstrating that family structure is associated with ADHD odds in adolescents with T1D. Living with a single parent and partner (i.e., a blended family) was associated with a 2.17-fold increased odds of ADHD diagnosis (95% CI: 0.98–4.84) compared to living with both parents. This finding may reflect either environmental stressors that increase ADHD risk or, alternatively, that parental ADHD (which is highly heritable) is more common in non-traditional family structures [41]. Previous research has shown that parental divorce and family conflict are associated with poorer diabetes outcomes [42], but our review suggests that family structure also influences the likelihood of ADHD diagnosis itself. Sex differences were examined by Bakke *et al*. [18], who found that the HbA1c gap between T1D+ADHD and T1D-only groups was significant in males but not in females. Additionally, the odds of diabetic ketoacidosis (DKA) were significantly elevated in males with comorbid ADHD (OR 1.65, 95% CI: 1.15–2.36) but not in females (OR 1.10, 95% CI: 0.66–1.83). This sex-specific effect has not been consistently reported in previous literature. A study by Nefs et al. [43] found that adult women with T1D reported more diabetes distress than men, but ADHD comorbidity was not assessed. The reasons for sex differences in our review are unclear but may relate to differences in help-seeking behaviour, diagnostic patterns (ADHD is underdiagnosed in females [44]), or biological factors such as oestrogen effects on dopamine regulation [45]. Further research is needed to replicate and explain these sex differences.

While our systematic review focused on observational studies, it is informative to consider findings from genetic epidemiology. A two-sample Mendelian randomisation study by Liu *et al*. [46] (using data from the Psychiatric Genomics Consortium and DIAGRAM) found a causal effect of ADHD on type 2 diabetes (OR 1.14, 95% CI: 1.01–1.29) but no evidence for an effect on type 1 diabetes (OR 0.92, 95% CI: 0.74–1.14, p=0.43). This null finding for T1D contrasts with the observational associations reported in our review. The discrepancy may be explained by several factors. First, Mendelian randomisation estimates reflect lifelong

exposure to genetic liability for ADHD, whereas observational studies capture the effects of clinically diagnosed ADHD, which may include environmental and behavioural components not fully captured by genetic instruments. Second, the GWAS for T1D and ADHD may have insufficient power to detect small effects, and the sample overlap between the ADHD and T1D GWAS is limited. Third, reverse causation is unlikely in observational studies (T1D onset typically precedes ADHD diagnosis in childhood), but confounding by shared familial factors (e.g., parental mental health, socioeconomic status) may partially explain the observational associations [47]. The sibling-comparison design used by Liu *et al*. [15] partially addresses this by controlling for unmeasured family-level confounders, and they still found significant associations, suggesting that residual confounding may not fully explain the observational findings. Future studies using within-family Mendelian randomisation or children-of-twins designs could help disentangle causal from confounding pathways.

The consistent evidence from this systematic review supports the recommendation that all children and adults with T1D should undergo regular screening for ADHD symptoms, particularly those with suboptimal glycemic control or recurrent DKA. The International Society for Pediatric and Adolescent Diabetes (ISPAD) Clinical Practice Consensus Guidelines recommend that "neurodevelopmental assessments should be considered in children with T1D who have difficulties with self-management or unexplained poor glycemic control" [48], but specific screening tools and protocols are lacking. Our review suggests that validated instruments such as the Conners 3 (used in the screening protocol by Kusmierczyk-Koziel *et al*. [49], though not included as it is a protocol) or the ASRS for adults [17] are feasible for use in diabetes clinics. Furthermore, when ADHD is diagnosed, treatment should be initiated promptly, as untreated ADHD is associated with worse outcomes [14-16]. Collaboration between diabetes teams, child and adolescent psychiatrists, and psychologists is essential to provide integrated care. The use of telemedicine-supported interventions, as described in the LAMAinDiab protocol [32], may improve access to specialist ADHD care for patients in remote areas. From a health policy perspective, the economic burden of untreated ADHD in T1D is likely substantial, given the increased rates of hospitalisations, emergency room admissions, and long-term complications [16-18]. Cost-effectiveness studies are needed to evaluate whether systematic screening and treatment of ADHD in T1D populations reduces healthcare utilisation and improves quality-adjusted life years.

Limitations

This systematic review has several limitations that should be acknowledged. First, among the six included studies, four employed cross-sectional designs [14-16-17-19], which precludes establishing temporal causality and are susceptible to reverse causation and recall bias. Only two studies used longitudinal or cohort designs [15-18], and even these cannot fully exclude residual confounding. Second, the methods of ADHD ascertainment varied considerably across studies: some used physician-diagnosed ADHD from medical records [14-16], others relied on prescription registries [18], one used a screening questionnaire (ASRS) [17], and another used parent-reported diagnosis [19]. This heterogeneity may lead to misclassification bias, as screening questionnaires have lower specificity than clinical diagnostic interviews, and registry-based diagnoses may miss milder or untreated cases. Third, selection bias is a concern: Zhang-James *et al*. [17] had a low response rate of 14.1%, and individuals who chose to participate may have differed systematically from non-respondents (e.g., higher diabetes distress or more ADHD symptoms). Fourth, several studies did not report important demographic variables such as exact age ranges or sex distribution (marked as NM in Table 1), limiting our ability to assess generalisability and perform subgroup analyses. Fifth, publication bias is possible, as studies reporting null or negative associations between ADHD and T1D outcomes may be less likely to be published or may have been excluded from our search (though we attempted a comprehensive search). Sixth, all studies were conducted in high-income countries (Israel, Sweden, USA, Norway, Germany), limiting the generalisability to low- and middle-income settings where T1D management resources are more constrained and ADHD awareness is lower. Seventh, none of the included studies reported on potential confounding by socioeconomic status, health literacy, or access to mental health care, all of which could independently influence both ADHD diagnosis and diabetes outcomes. Eighth, the risk of bias assessment (Table 3) indicated that four studies had moderate risk (scores 6/9), primarily due to cross-sectional designs and lack of blinding in outcome assessment. Finally, we could not perform a meta-analysis due to heterogeneity in outcome measures and reporting formats, which limits the precision of our effect estimates.

CONCLUSION

Deficit hyperactivity disorder is associated with significantly worse outcomes in individuals with type 1 diabetes across multiple domains. Adults and children with T1D and ADHD have higher HbA1c levels, lower time in range, increased rates of diabetic complications including neuropathy, nephropathy, and amputations, higher risk of diabetic ketoacidosis, greater depression severity, and substantially lower educational attainment compared to those with T1D alone. Untreated ADHD appears to confer the greatest risk, suggesting that pharmacological treatment of ADHD may partially mitigate adverse diabetes outcomes. Family structure (not living with both parents) and male sex are associated with higher odds of ADHD comorbidity and worse outcomes. Despite the observational nature of most included studies, the consistency of findings across

different populations, age groups, and study designs supports a clinically meaningful association. Clinically, these results underscore the urgent need for systematic screening for ADHD in all individuals with type 1 diabetes, particularly those with poor glycemic control or recurrent acute complications. Integrated, multidisciplinary care models that combine diabetology, psychiatry, and psychology are essential to address the complex needs of this population. Future research should focus on longitudinal interventional studies to determine whether treating ADHD improves long-term glycemic control and reduces complication rates, as well as cost-effectiveness analyses to guide healthcare policy. Genetic and mechanistic studies are also needed to elucidate the causal pathways underlying the ADHD–T1D association. Ultimately, recognising and managing ADHD as a modifiable risk factor in type 1 diabetes could improve both the medical and psychosocial outcomes of this vulnerable population.

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