Can J Diabetes xxx (2026) 1–17

Contents lists available at ScienceDirect

Canadian Journal of Diabetes

journal homepage: www.canadianjournalofdiabetes.com

Review

Physiological Impacts of Type 1 Diabetes on Exercise Performance: A Systematic Review and Narrative Synthesis of the Literature

Shania N. Smee BSc [a,b]; Angela L. Spence BSc, FHEA, PhD [a]; Rhiannon Halse BSc, PhD [c]; Leo Ng BSc, PhD [a,d]; Vinutha B. Shetty MBBS, DCHDNB, MD, FRACP, PhD [b,e,f]; Elizabeth A. Davis MBBS, FRACP, PhD [b,e,f]; Raymond J. Davey BSc, PhD [a,*]

[a] Curtin School of Allied Health, Curtin University, Whadjuk Noongar Country, Perth, Western Australia, Australia
[b] Children's Diabetes Centre, The Kids Research Institute, Whadjuk Noongar Country, Perth, Western Australia, Australia
[c] Curtin School of Population Health, Curtin University, Whadjuk Noongar Country, Perth, Western Australia, Australia
[d] Department of Nursing and Allied Health, School of Health Science, Swinburne University of Technology, Wurundjeri Country, Melbourne, Victoria, Australia
[e] Department of Endocrinology and Diabetes, Perth Children's Hospital, Whadjuk Noongar Country, Perth, Western Australia, Australia
[f] Division of Paediatrics Within the Medical School, The University of Western Australia, Whadjuk Noongar Country, Perth, Western Australia, Australia

Key Messages

- Type 1 diabetes (T1D) influences both acute and adaptive exercise responses.
- This review summarizes the impact of T1D on exercise performance and identifies the underlying physiological mechanisms.
- There is weak evidence to suggest that aerobic exercise performance is reduced in individuals with T1D, perhaps mediated by glycemic management.

ARTICLE INFO

Article history:
Received 19 April 2026
Received in revised form
1 June 2026
Accepted 22 June 2026

Keywords:
athlete
exercise physiology
muscle strength
physical endurance
power
sport

ABSTRACT

Objective: Type 1 diabetes (T1D) may influence acute and adaptive responses to exercise training and performance. This systematic review aimed to summarize the evidence comparing exercise performance between individuals with and without T1D and to identify the physiological mechanisms underlying potential differences in exercise performance.
Methods: Seven electronic databases were searched for studies comparing exercise performance and underlying physiological mechanisms in individuals with and without T1D. Exercise performance was categorized as aerobic, anaerobic, or skill based, and physiological mechanisms were grouped across cardiorespiratory, neuromuscular, and metabolic domains.
Results: Of 6,563 studies, 28 met the inclusion criteria. Most (k=25) evaluated aerobic exercise performance with time-trial, time-to-exhaustion, and $\dot{V}O_{2peak}$ tests. Five evaluated anaerobic exercise performance with one-repetition maximum, maximum voluntary contraction, and supramaximal time-to-exhaustion tests. Of the aerobic studies, 88% reported lower aerobic performance in individuals with T1D compared with those without, potentially due to cardiovascular or circulatory mechanisms. Similarly, 60% of the anaerobic studies reported lower anaerobic performance in individuals with T1D compared with those without, potentially due to mitochondrial mechanisms and recovery. Certainty of evidence was assessed using the GRADE tool and was found to be very low.
Conclusion: Current evidence suggests that aerobic exercise performance may be reduced in individuals with T1D. However, the evidence is weak, and these differences seem to be marginal. Suboptimal glycemic management and longer T1D duration may influence physiological responses to exercise and reduce performance capacity. Further research is required to support these conclusions and to determine the impact of T1D on anaerobic and skill-based exercise performance.

* Address for correspondence: Raymond J. Davey BSc, PhD, Curtin School of Allied Health, Curtin University, Whadjuk Noogar Country, Bentley, Western Australia 6102, Australia.
Email address: ray.davey@curtin.edu.au

1499-2671/
https://doi.org/10.1016/j.jcjd.2026.06.007

Mots-clés:
athlète
physiologie de l'effort physique
force musculaire
endurance physique
puissance
sport

RÉSUMÉ

Contexte : Le diabète de type 1 (DT1) peut influencer les réponses aiguës et adaptatives à l'entraînement physique ainsi que la performance à l'exercice. Cette revue systématique visait à résumer les données probantes permettant de comparer les performances à l'exercice entre les personnes atteintes de DT1 et celles qui ne le sont pas, et d'identifier les mécanismes physiologiques sous-jacents aux différences potentielles en matière de performances à l'exercice.
Méthodologie : Sept bases de données électroniques ont été consultées afin de recenser les études comparant les performances physiques et les mécanismes physiologiques sous-jacents chez des personnes atteintes ou non de DT1. Les performances physiques ont été classées en trois catégories : aérobies, anaérobies ou basées sur l'habileté, tandis que les mécanismes physiologiques ont été regroupés selon les domaines cardiorespiratoire, neuromusculaire, métabolique et hormonal.
Résultats : Sur les 6 563 études, 28 répondaient aux critères d'inclusion. La plupart ($k = 25$) portaient sur les performances en exercice aérobie, évaluées à l'aide de tests chronométrés, de tests de temps jusqu'à l'épuisement et de tests de $\dot{V}O_2$pic. Cinq études ont évalué les performances en exercice anaérobie à l'aide d'un test de répétition maximale, d'un test de contraction volontaire maximale et d'un test supramaximal de temps jusqu'à l'épuisement. Parmi les études portant sur l'endurance aérobie, 88 % ont fait état d'une performance aérobie inférieure chez les personnes atteintes de DT1 par rapport à celles qui ne le sont pas, pouvant être due à des mécanismes cardiovasculaires, circulatoires ou métaboliques. De même, 60 % des études portant sur l'endurance anaérobie ont fait état d'une performance anaérobie inférieure chez les personnes atteintes de DT1 par rapport à celles qui ne le sont pas, ce qui pourrait s'expliquer par des mécanismes mitochondriaux. Le niveau de fiabilité des données probantes a été évalué à l'aide de l'outil GRADE et s'est révélé très faible.
Conclusion : Les données probantes actuelles suggèrent que les performances en exercice aérobie pourraient être réduites chez les personnes atteintes de DT1. Toutefois, ces données sont peu solides et ces différences semblent marginales. Une gestion glycémique sous-optimale et une durée plus longue du DT1 pourraient influencer les réponses physiologiques à l'effort et réduire la capacité de performance. Des recherches supplémentaires sont nécessaires pour étayer ces conclusions et déterminer l'impact du DT1 sur les performances en exercice anaérobie et dans les exercices fondés sur l'habileté.

Introduction

For athletes with type 1 diabetes (T1D), exercise performance requires tight blood glucose management alongside careful monitoring of training loads, nutritional intake, sleep and recovery practices, and overall well-being [1,2]. Developing individualized management strategies is challenging, requiring detailed planning and "trial-and-error" approaches to learn how different exercises, foods, and stressors affect blood glucose [3]. The steady rise in anecdotal resources and evidence-based recommendations, alongside advancements in the efficacy of available technologies and therapies, has enabled more athletes with T1D to participate in high-level sport and exercise [4]. It is therefore necessary to understand the unique physiology of athletes with T1D and how physiological differences may affect performance.

Current literature identifies several ways in which T1D may affect athletic performance, including the following: acute hyperglycemia or hypoglycemia through mechanisms such as altered substrate oxidation [5] and impaired cognitive processing [6]; suboptimal glycaemic management and/or increased duration of T1D through mechanisms such as reduced pulmonary diffusing capacity [7] and cardiac output [8]; and inherent physiological differences compared with similarly trained counterparts without T1D, such as decreased vasoreactivity [9] and mitochondrial oxidative capacity [10]. These differences in both acute responses and chronic adaptations to exercise suggest that performance may be capped or limited in individuals with T1D.

There are currently no studies that synthesize the impact of T1D on exercise performance or fully explore the physiological mechanisms that may be responsible for any differences observed. Furthermore, several studies report contradictory results, making the overall impact of T1D on exercise performance unclear. We therefore undertook a systematic review of the literature which aimed to determine whether differences exist in athletic performance between people with T1D and those without, who complete similar amounts of weekly physical activity. This review also evaluated the underlying physiological mechanisms that contribute to potential differences in aerobic, anaerobic, or skill-based performance.

Methods

This review was conducted following a protocol registered on the Open Science Framework (https://osf.io/xfjve/overview) and was reported following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines [11].

The review adopts a mixed etiologic and prognostic approach. Etiologic research explores the link between certain exposures and outcomes [12] and includes the component of this review that compares exercise performance between people with and without T1D, including the physiological mechanisms behind any differences. Prognostic research explores the link between prognostic factors and the potential outcomes of a condition [13] and includes the comparison of exercise performance between subgroups of people with T1D, including the physiological mechanisms underpinning any differences.

Study design, population, exposure, comparators, outcomes, and prognostic factors

The research question for this review was the following: "Does the presence of T1D affect exercise performance; if so, what physiological mechanisms are involved?" To address this question,

only primary studies published in peer-reviewed scholarly journals were included.

The population consisted of people aged 13 to 60 years with T1D and a comparison group of people without T1D with similar weekly physical activity. Both groups reported no other medical conditions, and the T1D group had experienced no complications related to their condition.

The primary outcome was the comparison of exercise performance between people with and without T1D. Exercise performance was categorized as either aerobic, anaerobic, or skill-based performance. We operationalized aerobic performance as the physiological capacity to execute physical tasks via energy production from predominantly aerobic metabolism; anaerobic performance as the physiological capacity to execute physical tasks via energy production from predominantly anaerobic metabolism; and skill-based performance as proficiency in performing physical tasks that replicate the technical, tactical, and/or skill demands of a sport. These can be assessed directly through performance outcomes or indirectly through physiological adaptations that transfer to exercise performance. Additional outcomes included the comparison of the physiological mechanisms reported alongside the results of exercise performance assessments. These were categorized according to physiological function (e.g. regional oxygen delivery, structural integrity of skeletal muscle). Prognostic factors were the characteristics of certain T1D subgroups (e.g. glycated hemoglobin [HbA1c], age at diagnosis, and T1D duration) that may affect the results of exercise performance assessments.

Search strategy for identification of studies

A structured electronic literature search was conducted across 7 databases (Medline, EMBASE, CINAHL Ultimate, SPORTDiscus with Full Text, Scopus, Web of Science, ProQuest) in April 2023, with an update in August 2024. The search strategy (Appendix A) used terms in the following 4 broad categories: (1) T1D, (2) physiological mechanisms, (3) exercise performance, and (4) age. The search was limited to human studies by using database-specific search phrases, and there was no date restriction. This strategy was developed by the investigators in consultation with an experienced university librarian.

The citations and abstracts of all articles identified in these searches were imported into EndNote 20 [14], and duplicates were removed (with https://sr-accelerator.com/#/deduplicator). They were then imported into *Research Screener*, a semi-automated machine learning tool that ranks abstracts by relevance based on user-input seed articles [5,9,15–20]. This assists with title and abstract screening by allowing investigators to identify all eligible studies after the first 50% of ranked abstracts have been screened [21]. Screening was performed independently by 2 members of the research team, with S.S. screening the first 50% of ranked abstracts and R.D. concurrently screening the first 20%, with any conflicts resolved by A.S. The remaining 50% of abstracts were not screened because they are unlikely to yield relevant studies, given that predictive modelling performance plateaus beyond this threshold [21].

Studies flagged as relevant by 2 researchers were sought from university library subscriptions or by contacting the corresponding author of the article. If the full text could not be acquired after 2 emails to the corresponding author and/or a full text request on ResearchGate, then the study was excluded. Full texts were assessed for eligibility by S.S., who applied the prespecified eligibility criteria; R.D. also screened the final list of studies. Once eligible studies were identified, their reference lists were manually searched forward and backward for additional eligible studies.

Data extraction and quality assessments

Data extraction was carried out independently by S.S., using a pre-prepared data extraction form adapted from the Cochrane Collaboration (Appendix B), with the following recorded from each paper: study characteristics, participant characteristics, exercise performance outcomes, physiological mechanisms, prognostic factors, and intervention (where relevant).

Risk of bias assessments were conducted to evaluate the methodological quality of each study using the Joanna Briggs Institute (JBI) critical appraisal tool for cross-sectional studies [22] or quasi-experimental studies [23], as appropriate. A risk of bias judgement was reported for each domain, and the overall risk of bias for each study was subjectively determined (low, moderate, or high). Two members of the research team (S.S. and R.H.) independently applied these tools to each study, with conflicts resolved through communication.

Certainty of evidence was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) tool [24]. Evidence was initially rated according to study design and then upgraded or downgraded depending on judgments of the evidence in each domain. One member of the research team (S.S.) independently applied the GRADE tool and determined the overall certainty of evidence for each exercise performance outcome (high, moderate, low, or very low).

Data synthesis and analysis

To summarize the effect of T1D on exercise performance, statistical mean or median outcome values were extracted for both T1D and control groups (at baseline and follow-up for intervention studies). Data were categorized as direct (e.g. time-trial, time-to-exhaustion) or indirect (e.g. $\dot{V}O_{2peak}$) assessments of aerobic performance; direct (e.g. Wingate test, one-repetition maximum for whole-body movement) or indirect (e.g. isometric strength, 40-m sprint) assessments of anaerobic performance; or direct (e.g. serving accuracy, agility test) or indirect (e.g. isolated joint range of motion, multiple object tracking task) assessments of skill performance. Statistical means and p values (vs controls without T1D) for physiological mechanisms related to these exercise performance assessments were categorized according to physiologic function.

Results

Literature search results

The study selection process is found in Figure 1. Database searches identified 10,220 records, and after duplicate removal, 6,563 were screened at the title/abstract level using *Research Screener*. This identified 388 full texts requiring assessment for eligibility, of which 27 studies were found. One additional study was identified by forward/backward search, resulting in a total of 28 included studies in this review.

Study characteristics

Characteristics of the studies included in this review are outlined in Table 1. All studies were published in English-language journals between 1963 and 2022. The studies are heterogeneous with respect to the outcomes assessed, study design, study quality, sample size, and participant characteristics. The studies evaluated various aspects of exercise performance, with some including more than one. Aerobic performance was evaluated in 25 studies

(89%); anaerobic performance was evaluated in 5 studies (18%); and no eligible studies on skill performance were identified.

Aerobic performance

Of the studies assessing aerobic performance, 19 were cross-sectional and 6 were of quasi-experimental study design. A summary of the risk of bias assessments for these studies is provided in Appendix C. Of the cross-sectional studies, the risk of bias was classified as low in 10 studies, moderate in 8 studies, and high in 1 study. For the quasi-experimental studies, the risk of bias was classified as moderate in 5 studies and high in 1 study. Participant characteristics for the aerobic performance studies are recorded in Table 2.

Aerobic exercise performance was assessed directly through time-trials (k=1) and time-to-exhaustion (k=2) tests, but more often, indirectly through maximal oxygen consumption ($\dot{V}O_{2peak}$) tests (k=23). The time-trial and time-to-exhaustion studies were all cross-sectional. One study assessed time-trial performance using a half-marathon distance, where the mean time was 6.31% (+7.0 min) higher in the T1D group than in the control (CON) group [25]. Two protocols were used to assess time-to-exhaustion in separate studies [26,27]. One study exercised participants at 85% peak power until exhaustion; mean time-to-exhaustion was 11.47% (−61.0 s) lower in the T1D group than in the CON group [27]. The other study exercised participants at 55% to 60% $\dot{V}O_{2peak}$ until exhaustion; mean time-to-exhaustion was 43.59% (−51.0 min) lower in the T1D group than in the CON group [26].

Peak oxygen consumption was assessed in studies using both cross-sectional and quasi-experimental study designs. Mean $\dot{V}O_{2peak}$ was lower in the T1D group than in the CON group in 86.96% (k=20/23) of cross-sectional and quasi-experimental (pre-intervention value) studies combined. In these studies, mean $\dot{V}O_2$peak ranged from 0.80 to 9.50 mL/kg/min lower in the T1D group compared with the CON group. The remaining 3 studies revealed either no difference or a marginally higher mean $\dot{V}O_{2peak}$ (range: 0.70 to 3.30 mL/kg/min) in the T1D group. Mean improvement in $\dot{V}O_{2peak}$ after exercise training was lower in the T1D group than in the CON group in 80.00% (k=4/5) of quasi-experimental studies.

Anaerobic performance

Of the 5 studies assessing anaerobic performance, 2 used a cross-sectional study design and 3 used a quasi-experimental study design. A summary of the risk of bias assessments for these studies is provided in Appendix C. In both cross-sectional studies, the risk of bias was classified as moderate; of the quasi-experimental studies, 2 were classified as having a moderate risk of bias and 1 as having a high risk of bias. Participant characteristics for the anaerobic performance studies are also recorded in Table 2. Anaerobic exercise performance was assessed directly through a one-repetition maximum for whole-body movements

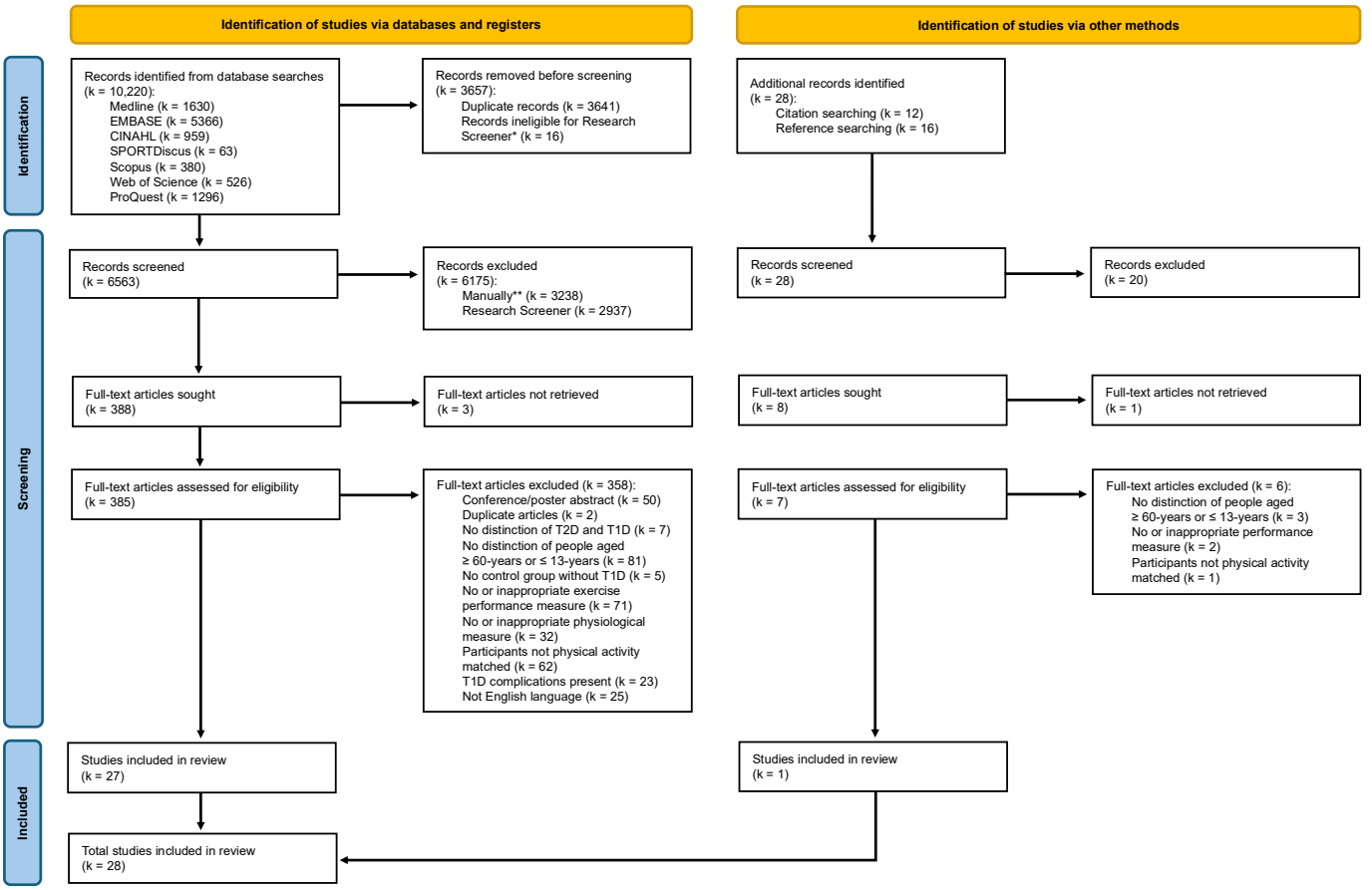

Figure 1. PRISMA diagram outlining the identification of eligible studies. *Titles and abstracts of these records were manually screened, and none met the eligibility criteria for inclusion in this systematic review. **Reasons for manual exclusion: only T2D; animal study; secondary study; no performance measure; no control without T1D; no physiological measure; only people aged ≥60 years or ≤13 years. *PRISMA*, Preferred Reporting Items for Systematic Reviews and Meta-Analyses; T1D, type 1 diabetes; T2D, type 2 diabetes.

Table 1
Characteristics of studies included in this review

Reference	Country	Study design	Risk of bias	Group	n	Sex	Age (year)	T1D dur. (year)	HbA1c (%)	Athletic calibre	Exercise test/s	T1D difference	Primary proposed mechanism
Adolfsson et al 2012 [49]	Sweden	Cross-sectional	Moderate	T1D CON	12 12	6F, 6M 6F, 6M	16 [14–20] 17 [15–19]	6.3 [0.7–15.6] n/a	7.9 [6.6–9.6] -	Tier 1 Tier 1	$\dot{V}O_{2peak}$– ergocycle	↓ (49.8 vs 50.7 mL/kg/min)	*Hormonal regulation* No difference in glucagon, GH, cortisol, adrenaline, noradrenaline ($p>0.05$)
Baldi et al 2010 [47]	United States	Cross-sectional	Low	T1D CON	12 10	3F, 9M 2F, 8M	40 ± 8 33 ± 8	- n/a	7.3 ± 0.8 5.2 ± 0.5	Tier 2 Tier 2	$\dot{V}O_{2peak}$– ergocycle	↓ (42 vs 45 mL/kg/min)	*Central oxygen delivery* No difference in HR, SV, Q ($p>0.05$)
Costil et al 1979 [58]	United States	Quasi-experimental	Moderate	T1D CON	12 13	0F, 12M 0F, 13M	21 ± 1.4 23 ± 1.1	0.4–11 n/a	- -	Tier 0 Tier 0	$\dot{V}O_{2peak}$– treadmill	PRE: ↑ (49.2 vs 48.5 mL/kg/min) POST: ↓ (54.6 vs 54.8 mL/kg/min)	*Oxygen metabolism* No difference in muscle enzyme activity, muscle C-palmityl CoA oxidation (pre- and post-training; $p>0.05$).
Dial et al 2021 [15] (female)	Canada	Cross-sectional	Moderate	T1D CON	10 7	10F, 0M 7F, 0M	26 ± 2.0 25 ± 2.0	10 ± 2.0 n/a	7.9 ± 0.5 5.2 ± 0.1	Unclas Unclas	MVC– max knee extension	↑ (192 vs 187 N)	*Structural integrity of skeletal muscle* No difference in myofibre feret diameter, % MYH-7, % grouped fibres ($g < 0.2$) *Repair, growth, remodelling of skeletal muscle* No difference in SC content *Regional oxygen delivery* Higher capillary density ($g\geq0.2$), capillary-to-fibre ratio ($g\geq0.5$).
Dial et al 2021 [15] (male)	Canada	Cross-sectional	Moderate	T1D CON	6 6	0F, 6M 0F, 6M	27 ± 2.0 26 ± 2.0	16 ± 3 n/a	7.1 ± 0.4 5.4 ± 0.2	Unclas Unclas	MVC– max knee extension	↑ (290 vs 288 N)	*Structural integrity of skeletal muscle* No difference in myofibre feret diameter, myofibre group size (rest; $g<0.2$) *Repair, growth, remodelling of skeletal muscle* No difference SC content

(*continued on next page*)

Table 1 (*continued*)

Reference	Country	Study design	Risk of bias	Group	n	Sex	Age (year)	T1D dur. (year)	HbA1c (%)	Athletic calibre	Exercise test/s	T1D difference	Primary proposed mechanism
Dial et al 2021 [28]	Canada	Cross-sectional	Moderate	T1D CON	9 9	5F, 4M 5F, 4M	23 ± 2.0 22 ± 1.4	9.7 ± 2.4 n/a	7.4 ± 0.7 5.4 ± 0.4	Tier 2 Tier 2	MVC– max knee extension	Male: ↑ (3.87 vs 3.50 Nm/kg) Female: ↑ (2.72 vs 2.66 Nm/kg)	*Structural integrity of skeletal muscle* Higher myofibres with sarcolemmal damage (g≥0.8), ECM content ($p<0.05$) *Repair, growth, remodelling of skeletal muscle* Lower SC content ($p<0.05$) and delayed SC proliferation; higher serum creatine kinase (g≥0.2). Dysregulated gene networks involving RNA translation and mitochondrial respiration
Gusso et al 2012 [8]	New Zealand	Cross-sectional	Moderate	T1D CON	53 22	26F, 27M 12F, 10M	16 ± 0.2 17 ± 0.2	6.0 ± 4.0 n/a	8.7 ± 0.2 5.3 ± 0.4	Unclas Unclas	$\dot{V}O_{2peak}$– ergocycle	↓ (44.7 vs 48.5 mL/kg FFM/min)	*Central oxygen delivery* No difference HR, LV mass ($p>0.05$).
Gusso et al 2017 [48]	New Zealand	Quasi-experimental	Moderate	T1D CON	38 22	18F, 20M 12F, 10M	16 ± 1.3 17 ± 1.5	5.4 ± 1.3 n/a	PRE: 8.8 [8.4-9.3] POST: 8.5 [8.1-8.9] PRE: 5.2 [4.4-5.9] POST: 5.1 [4.5-5.8]	Unclas Unclas	$\dot{V}O_{2peak}$ – ergocycle	PRE: ↓ (45.0 vs 48.5 mL/kg FFM/min) POST: ↓ (49.9 vs 53.6 mL/kg FFM/min)	*Central oxygen delivery* No difference HR, O_2 pulse, LV mass (pre- and post-training; $p>0.05$).
Harmer et al 2006 [29]	Australia	Quasi-experimental	High	T1D CON	8 7	3F, 5M 3F, 4M	25 ± 4 24 ± 5	7.1 ± 4 n/a	8.6 ± 0.8 5.3 ± 0.3	Unclas Unclas	TTE - 130% $\dot{V}O_{2peak}$ $\dot{V}O_{2peak}$– ergocycle	PRE: ↑ (78 vs 62 s) POST: not recorded PRE: (3.30 vs 3.17 L/min) POST: (3.38 vs 3.43 L/min)	*Contractile function of skeletal muscle* No difference Na^{2+}-K^{+}-ATPase content, rise in plasma [K^{+}] (pre- or post-training; $p>0.05$)
Harmer et al 2014 [30]	Australia	Quasi-experimental	Moderate	T1D CON	8 8	3F, 5M 3F, 5M	25 ± 4 23 ± 5	7.1 ± 4 n/a	8.6 ± 0.8 5.3 ± 0.3	Unclas Unclas	TTE - 130% $\dot{V}O_{2peak}$	PRE: ↑ (95 vs 74 s) POST: not recorded	*Contractile function of skeletal muscle* Lower [iCa^{2+}] (pre- and post-training; $p<0.05$); higher Ca^{2+}-ATPase activity, Ca^{2+} uptake (pre- and post-training; $p<0.05$).

Heyman et al 2020 [17]	France	Cross-sectional	Low	T1D CON	16 16	4F, 12M 4F, 12M	29 ± 6.8 28 ± 6.6	8.5 ± 5.2 n/a	8.3 ± 1.5 5.2 ± 0.2	Tier 1 Tier 1	$\dot{V}O_{2peak}$—ergocycle	↓ (34.9 vs 40.7 mL/kg/min)	*Regional oxygen delivery; oxygen extraction; oxygen metabolism* Lower exercise-induced increase in muscle blood volume ($p<0.05$); lower O_2 extraction ($p<0.05$); lower mitochondrial complex IV capacity relative to complex I capacity ($p<0.05$)
Hyryla et al 2022 [31]	Finland	Cross-sectional	Moderate	T1D CON	13 13	0F, 13M 0F, 13M	34 ± 6.6 37 ± 8.6	12 ± 7 n/a	7.5 ± 0.8 -	Tier 1 Tier 1	$\dot{V}O_{2peak}$—ergocycle	↓ (35.6 vs 39.8 mL/kg/min)	*Oxygen intake* No difference in inspiratory values ($p>0.05$).
Item et al 2010 [27]	Switzerland	Cross-sectional	Low	T1D CON	29 9	29F, 0M 9F, 0M	24 ± 4.2 27 ± 5.2	13.1 ± 6.6 n/a	7.6 ± 0.4 5.3 ± 0.2	Tier 0 Tier 0	$\dot{V}O_{2peak}$ - ergocycle TTE - 85% $\dot{V}O_{2peak}$	↑ (2.21 vs 2.17 L/min) ↓ (471 vs 532 s)	*Systemic and regional oxygen delivery* No difference in Q ($p>0.05$); no difference in capillary-to-fibre ratio ($p>0.05$) *Oxygen metabolism* No difference oxidative enzyme activity
Jakober et al 1983[71]	United States	Cross-sectional	High	T1D CON	6 7	0F, 6M 0F, 7M	21 ± 1.1 23 ± 0.7	5.2 n/a	- -	Tier 0 Tier 0	$\dot{V}O_{2peak}$—ergocycle	↓ (39.0 vs 48.3 mL/kg/min)	*Hormonal regulation* Lower adrenaline, noradrenaline; higher GH, ACTH, cortisol *Oxygen metabolism* Higher free glycerol, FFA, lactate, pyruvate
Jlali et al 2023 [32]	France	Cross-sectional	Low	T1D CON	17 17	6F, 11M 6F, 11M	28 ± 7.2 28 ± 6.3	9.1 ± 5.5 n/a	8.0 ± 1.3 5.2 ± 0.2	Tier 1 Tier 1	$\dot{V}O_{2peak}$—ergocycle	↓ (34.7 vs 37.9 mL/kg/min)	*Oxygen intake* Lower TV ($p<0.05$); no difference DLCO ($p>0.05$).
Koponen et al 2013 [36]	Finland	Cross-sectional	Low	T1D CON	12 23	0F, 12M 0F, 23M	33 ± 7 33 ± 7	4-27 n/a	7.7 ± 0.8 -	Tier 1 Tier 1	$\dot{V}O_{2peak}$—ergocycle	↓ 21% (35.4 vs 44.9 mL/kg/min)	*Oxygen-carrying capacity* Lower tHb-mass, BV, EV, PV ($p<0.05$); no difference [Hb] ($p>0.05$);
Larsson et al 1964 [46]	Sweden	Quasi-experimental	Moderate	T1D CON	6 6	0F, 6M 0F, 6M	16 [15-19] 17 [15-19]	7.3 [4-12] n/a	- -	Unclas Unclas	$\dot{V}O_{2peak}$—ergocycle	PRE: ↓(2.33 vs 2.55 L/min) $POST_1$: ↓(2.58 vs 3.00 L/min) $POST_2$: ↓(3.08 vs 3.33 L/min)	*Systemic oxygen delivery* Lower heart volume (pre- and post- training); no difference HR (pre- and post-training)

(*continued on next page*)

Table 1 (*continued*)

Reference	Country	Study design	Risk of bias	Group	n	Sex	Age (year)	T1D dur. (year)	HbA1c (%)	Athletic calibre	Exercise test/s	T1D difference	Primary proposed mechanism
Lespagnol et al 2021 [9]	France	Cross-sectional	Low	T1D CON	22 21	7F, 15M 7F, 14M	27 ± 7.0 27 ± 5.8	8.7 ± 5.4 n/a	8.0 ± 1.4 5.2 ± 0.3	Tier 1 Tier 1	$\dot{V}O_{2peak}$– ergocycle	↓ (35.6 vs 39.6 mL/kg/min)	*Regional oxygen delivery* Lower exercise-induced increase in muscle blood volume, plasma L-arginine, plasma urate ($p<0.05$)
Minnock et al 2022 [18]	Ireland	Quasi-experimental	Moderate	T1D CON	10 10	6F, 4M 6F, 4M	32 ± 3.7 28 ± 5.9	17.8 ± 9.2 n/a	7.9 ± 3.4 -	Tier 0 Tier 0	1RM – goblet squat $\dot{V}O_{2peak}$– ergocycle	PRE: ↓ (42.6 vs 49.3 kg) POST: ↓ (47.9 vs 58.3 kg) PRE: ↓ (31.6 vs 32.76 mL/kg/min) POST: ↓ (35.0 vs 41.7 mL/kg/min)	*Repair, growth, remodelling of skeletal muscle* Alteration in muscle markers of mitochondrial functions, inflammation, ageing and growth/atrophy (pre- and post-training; $p<0.05$)
Mourot et al 2020 [25]	Italy	Cross-sectional	Moderate	T1D CON	9 9	1F, 8M 1F, 8M	39 ± 11.1 42 ± 5.8	- n/a	- -	Tier 2 Tier 2	Time-trial – half marathon	↑ (111.0 vs 104.0 min)	*Systemic oxygen delivery* No difference in SV, Q ($p>0.05$) *Autonomic nervous system recovery* No difference in BRS, HRV ($p>0.05$)
Peltonen et al 2012 [37]	Finland	Cross-sectional	Moderate	T1D CON	10 10	0F, 10M 0F, 10M	33 ± 7 32 ± 7	11 ± 6 n/a	7.7 ± 0.7 -	Tier 0 Tier 0	$\dot{V}O_{2peak}$– ergocycle	↓ (35.4 vs 43.8 mL/kg/min)	*Regional oxygen delivery* Lower exercise-induced increase in muscle blood volume at a given submaximal work rate, but not peak exercise ($p<0.05$)
Ramires et al 1993 [26]	Brazil	Cross-sectional	Moderate	T1D CON	15 16	0F, 15M 0F, 16M	21 ± 0.9 23 ± 0.6	3-5 n/a	< 12.0 -	Tier 0 Tier 0	$\dot{V}O_{2peak}$ – ergocycle TTE 55-60% $\dot{V}O_{2peak}$	↓ (40 vs 41 mL/kg/min) ↓ (66 vs 117 min)	*Oxygen metabolism* Higher lactate, FFA ($p<0.05$) *Hormonal regulation* Higher glucagon ($p<0.05$); no difference in cortisol, GH ($p>0.05$)

Rissanen et al 2015 [33]	Finland	Cross-sectional	Moderate	T1D CON	7 10	0F, 7M 0F, 10M	35 ± 6.0 34 ± 7.0	15 ± 9 n/a	7.4 ± 0.9 5.3 ± 0.2	Tier 1 Tier 1	$\dot{V}O_{2peak}$—ergocycle	↓ (47 vs 56 mL/kg/min)	*Systemic oxygen delivery* Lower FFM-adjusted SV and Q ($p<0.05$) *Regional oxygen delivery* Lower exercise-induced increase in muscle blood volume ($p<0.05$)
Rissanen et al 2018 [34]	Finland	Quasi-experimental	Moderate	T1D CON	8 8	0F, 8M 0F, 8M	33 ± 6.3 38 ± 7.1	10.5 ± 6.8 n/a	7.3 ± 0.9 5.3 ± 0.2	Tier 1 Tier 1	$\dot{V}O_{2peak}$—ergocycle	PRE: O (38 vs 38 mL/kg/min) POST: O (41 vs 41 mL/kg/min)	*Systemic oxygen delivery* No difference O_2 pulse, HR (pre- and post-training; $p<0.05$) *Regional oxygen delivery* No difference in exercise-induced increase in muscle blood volume (pre- or post-training; $p>0.05$)
Tagougui et al 2015 [41] (low HbA1c)	France	Cross-sectional	Low	T1D CON	11 11	0F, 11M 0F, 11M	27 ± 6.1 26 ± 5.6	4.5 ± 3.6 n/a	6.6 ± 0.7 5.5 ± 0.2	Unclas Unclas	$\dot{V}O_{2peak}$—ergocycle	↓ (40.9 vs 43.0 mL/kg/min)	*Systemic oxygen delivery* No difference O_2 pulse ($p>0.05$) *Regional oxygen delivery* No difference exercise-induced increase in muscle blood volume ($p>0.05$) *Oxygen-carrying capacity* No difference in erythrocyte 2,3-DPG ($p>0.05$)
Tagougui et al 2015 [41] (high HbA1c)	France	Cross-sectional	Low	T1D CON	12 12	5F, 7M 5F, 7M	26 ± 7.3 26 ± 5.0	10.9 ± 3.4 n/a	9.1 ± 0.7 5.3 ± 0.2	Unclas Unclas	$\dot{V}O_{2peak}$—ergocycle	↓ (34.6 vs 41.2 mL/kg/min)	*Systemic oxygen delivery* No difference O_2 pulse ($p>0.05$). *Regional oxygen delivery* Lower exercise-induced increase in muscle blood volume ($p<0.05$) *Oxygen-carrying capacity* No difference erythrocyte 2,3-DPG, arterial O_2 content ($p>0.05$)

(*continued on next page*)

Table 1 (*continued*)

Reference	Country	Study design	Risk of bias	Group	n	Sex	Age (year)	T1D dur. (year)	HbA1c (%)	Athletic calibre	Exercise test/s	T1D difference	Primary proposed mechanism
Tagougui et al 2015 [50] (low HbA1c)	France	Cross-sectional	Low	T1D CON	8 8	1F, 7M 1F, 7M	30 ± 6.8 30 ± 4.5	4.3 ± 3.5 n/a	6.8 ± 0.7 5.3 ± 0.2	Unclas Unclas	$\dot{V}O_{2peak}$– ergocycle	↓ (39.6 vs 41.7 mL/kg/min)	*Oxygen-carrying capacity* Higher arterial O_2 content ($p<0.05$)
Tagougui et al 2015 [50] (high HbA1c)	France	Cross-sectional	Low	T1D CON	10 10	4F, 6M 4F, 6M	26 ± 7.8 26 ± 5.9	10.7 ± 3.7 n/a	9.0 ± 0.7 5.3 ± 0.3	Unclas Unclas	$\dot{V}O_{2peak}$– ergocycle	↓ (34.6 vs 40.3 mL/kg/min)	*Oxygen-carrying capacity* No difference arterial O_2 content ($p>0.05$).
Van Ryckeghem et al 2021 [20]	Belgium	Cross-sectional	Low	T1D CON	19 19	6F, 13M 5F, 14M	15 ± 1.9 14 ± 1.3	5.8 ± 3.0 n/a	7.4 ± 0.9 5.3 ± 0.3	Tier 1 Tier 1	$\dot{V}O_{2peak}$– ergocycle	↑ (43.8 vs 40.5 mL/kg/min)	*Systemic oxygen delivery* No difference cardiac dimensions and structure, parameters of diastolic function and systolic function, O_2 pulse ($p>0.05$)
Wheatley et al 2011 [19]	United States	Cross-sectional	Moderate	T1D CON	12 18	3F, 9M 5F, 13M	40 ± 8 34 ± 10	- n/a	7.3 ± 0.8 5.2 ± 0.4	Tier 2 Tier 2	$\dot{V}O_{2peak}$– ergocycle	↓ (41.7 vs 42.5 mL/kg/min) Low HbA1c (6.6%, n=6) 44.1 mL/kg/min; high HbA1c (8.0%, n=6) 39.3 mL/kg/min	*Oxygen intake* Lower DLCO/Q and DM/Q ($p<0.05$); *Systemic oxygen delivery* No difference Q ($p>0.05$).
Wilson et al 2017 [35]	New Zealand	Cross-sectional	Low	T1D CON	23 17	11F, 12M 8F, 9M	32 ± 13 32 ± 12	15.2 ± 13.6 n/a	8.4 ± 3.7 -	Tier 1 Tier 1	$\dot{V}O_{2peak}$– ergocycle	↓ (32 vs 37 mL/kg/min)	*Autonomic nervous system recovery* Lower HRV metrics, HRR ($p<0.05$) in T1D (peak exercise; $p<0.05$) *Hormonal regulation* No difference adrenaline or noradrenaline ($p>0.05$).

1RM, one repetition maximum; *ATCH*, anticorticotrophic hormone; *BRS*, baroreflex sensitivity; *BV*, blood volume; *CON*, control; *DLCO*, diffusing capacity of the lungs for carbon monoxide; *DM*, membrane diffusing capacity; *ECM*, extracellular matrix; *EV*, erythrocyte volume; *F*, female; *FFA*, free fatty acids; *FFM*, fat free mass; *GH*, growth hormone; *[Hb]*, haemoglobin concentration; *HbA1c*, glycated hemoglobin; *HR*, heart rate; *HRV*, heart rate variability; *LVM*, left ventricular mass; *M*, male; *MVC*, maximal voluntary contraction; *O_2 pulse*, oxygen pulse; *PV*, plasma volume; *Q*, cardiac output; *SC*, satellite cell; *SV*, stroke volume; *T1D*, type 1 diabetes; *tHb mass*, haemoglobin mass; *TTE*, time to exhaustion; *TV*, tidal volume.

(k=1), or indirectly through a one-repetition maximum for isolated joint movements (k=1), maximal voluntary isometric contraction (k=2), and time-to-exhaustion at supra-maximal intensity (k=2).

One-repetition maximum (1RM) for whole body movements (goblet squat) and isolated joint movements (bicep curl, leg extension, seated cable row, squat press, triceps extension) was measured in 1 quasi-experimental study [18]. Pre-intervention, mean one-repetition maximum for whole body movements was 13.59% (−6.70 kg) lower in the T1D group than in the CON group, whereas mean one-repetition maximum for combined isolated joint movements was 0.66% (+1.20 kg) higher in the T1D group. After exercise training, the T1D group had a lower mean improvement in one-repetition maximum for whole body movements (−3.70 kg) and combined isolated joint movements (−3.48 kg) compared with the CON group.

Both maximal voluntary isometric contraction (MVC) studies were cross-sectional and evaluated either peak force or peak torque during maximal knee extension. Mean peak force was 1.45% (3.50 N) higher in the T1D group compared with the CON group [15]; mean peak torque was 6.53% (0.22 Nm) higher in the T1D group [28]. Two studies exercised participants at a power output equivalent to 130% $\dot{V}O_{2peak}$ until exhaustion; mean time-to-exhaustion was 22.11% (21 s) and 20.51% (16 s) higher in the T1D group compared with the CON group in each study, respectively [29,30].

GRADE assessment

Using the GRADE tool, all outcomes were initially rated as low certainty due to the observational nature of the studies included in this review. No outcomes met the criteria for upgrading. Evidence evaluating time-trial, submaximal time-to-exhaustion, full-body and isolated-joint 1RM, maximal voluntary isometric contraction, and supramaximal time-to-exhaustion was downgraded to very low certainty, primarily due to concerns with inconsistency and imprecision. Evidence evaluating $\dot{V}O_2$peak remained of low certainty. The five categories used to downgrade the certainty of evidence are detailed in Appendix D.

Discussion

This review explores differences in athletic performance between people with and without T1D and the underlying physiological mechanisms that may explain these differences. Studies have predominantly revealed lower aerobic exercise performance in people with T1D, which may be attributed to the pulmonary, cardiovascular, circulatory, neuromuscular, and/or metabolic effects of T1D. However, these differences were marginal and were primarily determined through $\dot{V}O_{2peak}$ assessments, which measure aerobic capacity but do not directly measure exercise performance. Limited research into anaerobic exercise performance has revealed mixed results, with some differences potentially attributable to the neuromuscular effects of T1D. No studies assessing skill-based performance were included.

Proposed mechanisms underlying differences in aerobic exercise performance

Ventilatory capacity and pulmonary diffusing capacity: Most studies found no significant difference between T1D and CON in inspiratory values at peak exercise [31] or minute ventilation at peak exercise [17,31-35]. However, Koponen [36] and Peltonen [37] found a significantly lower minute ventilation in T1D compared with CON. This is potentially explained by the lower tidal volume [31,32,35] but similar respiratory rates [17,31,32] observed in T1D. This could be a result of faster respiratory muscle fatigue and could cause the large differences in $\dot{V}O_{2peak}$ found in these 2 studies.

Pulmonary diffusing capacity increases progressively with exercise intensity, and athletes with a higher $\dot{V}O_{2peak}$ have been found to achieve higher diffusion capacities during intense exercise [38]. Several studies [17,19,32] found no significant difference in lung diffusing capacity for carbon monoxide (DLCO; a measure of pulmonary diffusion capacity), or its components, alveolar-capillary membrane conductance (D_M) and pulmonary capillary blood volume (V_C), between T1D and CON at peak exercise. However, Wheatley [19] found that DLCO was lower in the T1D group when corrected for cardiac output at peak exercise. They suggest that this limitation becomes increasingly functionally significant as erythrocyte transit time through the pulmonary capillaries is shortened (i.e. at higher exercise intensities). This is particularly relevant for endurance athletes, a population in which rapid pulmonary blood flow during vigorous exercise may already prevent complete aeration of pulmonary capillaries due to a higher capacity of the cardiovascular system in relation to the pulmonary system [39]. Ljubic [40] suggests that reduced D_M and V_C are a result of changes in lung connective tissue (particularly collagen and elastin) and microangiopathy of the pulmonary vasculature from prolonged hyperglycemia.

Oxygen-carrying capacity: An increase in the oxygen-carrying capacity of blood results in an increase in $\dot{V}O_{2peak}$ [39]. Most studies reported no significant difference in hematocrit [9,29,36], hemoglobin concentration [9,29,33,34,36,41], or erythrocyte 2-3DPG concentration [17,41] between T1D and CON at rest and at peak exercise. However, hemoglobin mass may better predict blood oxygen-carrying capacity and $\dot{V}O_{2peak}$ than hemoglobin concentration [42], and several studies have found lower erythrocyte volume [36] and hemoglobin mass [9,36,41] in T1D compared with CON. Low hemoglobin mass may suggest a shorter erythrocyte lifespan [43] or blunted erythropoietin production [44], which results from damage to the erythrocyte or peritubular fibroblasts of the renal cortex, respectively, due to prolonged hyperglycemia.

Systemic oxygen delivery: Larger left ventricular dimensions are associated with greater training intensity and a higher $\dot{V}O_{2peak}$ [45]. Studies evaluating left ventricular mass [8,20,46,48] reveal no difference between T1D and CON. Van Ryckegham [20] also found no differences in cardiac dimensions, including left ventricular wall thickness and left ventricular or atrial diameters. Notably, the participants in this study completed low-moderate levels of habitual weekly physical activity, so it is unclear whether the impact of T1D on exercise-induced cardiac remodelling is mediated by exercise exposure.

Variations in maximal cardiac output (driven by maximal stroke volume) are principally responsible for differences in $\dot{V}O_{2peak}$ between sedentary and trained individuals [39]. Four studies [19,25,27,47] found no difference in cardiac output at peak exercise. This is accompanied by most studies reporting no difference in heart rate [8,17,20,25,26,31,32,33,34,35,36,37,41,47,48,49,50] stroke volume [25,47] or O_2 pulse (a surrogate measure of stroke volume) [17,32,41,48,50,] at peak exercise. In the study by Rissanen [34], there was also a similar increase in O_2 pulse in T1D and CON after 1 year of combined endurance and resistance training.

However, Rissanen [33] revealed a lower peak cardiac output in T1D compared with CON. This can be attributed to a lower peak stroke volume, as Koponen [36] also recorded a lower O_2 pulse in T1D compared with CON. Reduced peak stroke volume could be a result of lower preload due to reduced blood volume [33,36] or

Table 2
Participant characteristics of studies included in this review

Characteristic	Aerobic performance ($k = 25$)		Anaerobic performance ($k = 5$)	
	T1D	CON	T1D	CON
Total participants (n)	410	356	51	47
Participants per study (n)	12 [9–16]	11 [9–17]	9 [8 – 10]	8 [7–9]
Age (years)	27.3 [21.1–33.0]	27.1 [23.4–32.5]	25.5 [25.0 – 26.8]	24.5 [23.3–25.8]
Sex (F%)	25 [0–41]	25 [0–42]	47 [38 – 59]	49 [39–59]
Physical activity (h/week)	3.3 [1.9–4.0]	3.5 [1.7–4.6]	9.4 [5.7 – 13.1]	9.3 [5.7–13.0]
Athletic calibre				
Tier 0 (%)	6 (24)	6 (24)	1 (20)	1 (20)
Tier 1 (%)	10 (40)	10 (40)	0 (0)	0 (0)
Tier 2 (%)	3 (12)	3 (12)	1 (20)	1 (20)
Unclassified (%)	6 (24)	6 (24)	3 (60)	3 (60)
HbA1c (%)	7.8 [7.4–8.4]	n/a	7.9 [7.5–8.4]	n/a
T1D duration (year)	8.7 [6.0–11.0]	n/a	9.7 [7.1–10.0]	n/a

CON, control; *HbA1c*, glycated hemoglobin; *n/a*, not available; *T1D*, type 1 diabetes.

higher afterload due to greater systemic vascular resistance [33]. Hypovolemia in people with T1D is often a result of osmotic diuresis from hyperglycemia [51]. Higher systemic vascular resistance may be linked to impairments in endothelial function and vascular smooth muscle (e.g. lower distensibility, reactivity, and release of vasodilatory substances), which are present even in individuals with T1D who do not have clinically relevant vascular complications [52]. These impairments are more pronounced in individuals with T1D who have a higher HbA1c, likely due to the impact of oxidative stress from prolonged hyperglycemia on blood vessel integrity [52].

Regional oxygen delivery: Endurance-trained muscles have greater hyperemic responses to exercise than untrained muscles, potentially contributing to a higher $\dot{V}O_{2peak}$ in these individuals [53]. In the study by Lespagnol [9], regional blood volume (of the vastus lateralis) rose across the exercise test, with marked increases occurring at 70% $\dot{V}O_{2peak}$ in T1D and 50% $\dot{V}O_{2peak}$ in CON. This difference is potentially responsible for the lower change in regional blood volume found in T1D at peak exercise [9,17,41]. For a subgroup of participants with low HbA1c value [34,41], the difference between T1D and CON groups was not significant. Further, Rissanen [33] found that although local blood flow (through the vastus lateralis) was similar between T1D and CON during submaximal exercise, it was lower in T1D during maximal exercise. These changes in the microvascular perfusion of skeletal muscle could be an early result of endothelial dysfunction or altered capillary recruitment from chronic hyperglycemia and/or long-term T1D duration [54]. This indicates a potential limitation in the delivery of oxygen to active muscles despite similar capillary density [15] and capillary-to-fibre ratio [15,27] in T1D and CON.

Oxygen extraction: A curvilinear relationship exists between local muscle oxygen extraction and $\dot{V}O_{2peak}$, with a plateau reached in endurance-trained athletes [55]. The arteriovenous oxygen difference was not significantly different between T1D and CON [27,33]. Changes in tissue deoxygenation (a proxy for local muscle oxygen extraction) were also assessed in several studies, with mixed results. Three studies [33,34,37] found tissue deoxygenation (in the vastus lateralis) to be higher in T1D compared with CON at both submaximal and maximal intensities. This enhanced deoxygenation, alongside diminished oxygen delivery (e.g. lower cardiac output, impaired microvascular perfusion), may suggest that people with T1D rely more heavily on oxygen extraction to maintain oxygen uptake at a given work rate. Consequently, they might reach maximal oxygen extraction and $\dot{V}O_{2peak}$ earlier than CON. Furthermore,

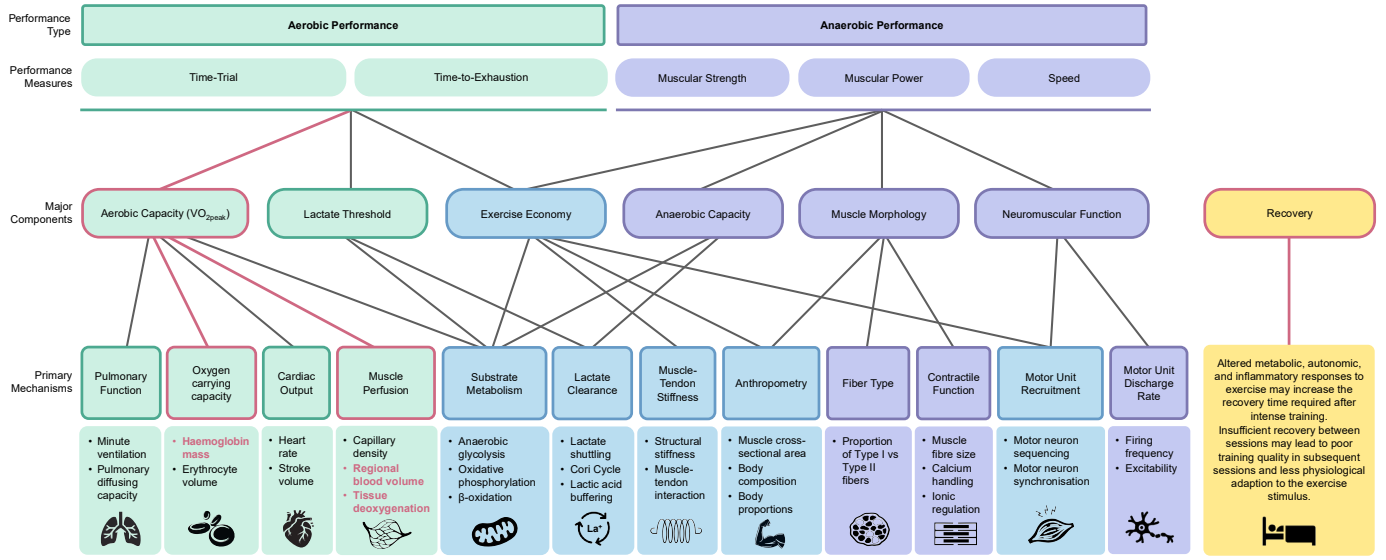

Figure 2. This diagram depicts the primary physiological mechanisms underpinning aerobic and anaerobic performance (as discussed in this review). The red lines and text indicate the key ways in which T1D *may* affect this framework. *T1D*, type 1 diabetes.

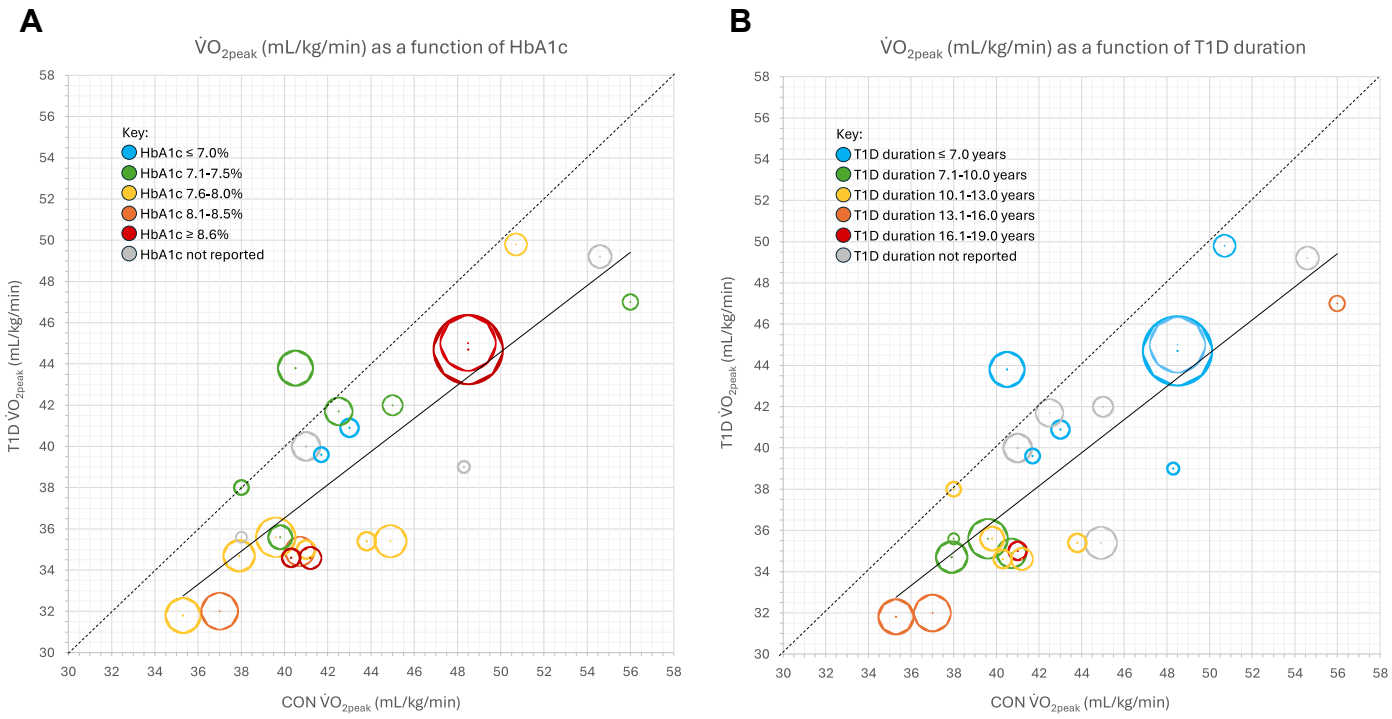

Figure 3. $\dot{V}O_{2peak}$ reported in T1D vs CON in included studies, with bubble size based on total number of participants. The dotted line indicates equal achievement of $\dot{V}O_{2peak}$ by T1D and CON participants. CON, control; HbA1c, glycated hemoglobin; T1D, type 1 diabetes.

the effect of this compensatory mechanism diminishes with endurance training as the maximal limits of leg muscle oxygen extraction are approached [55].

Three different studies [9,17,41] found tissue deoxygenation (in the vastus lateralis) to be significantly lower in T1D than in CON at both submaximal and maximal intensities (except in a subgroup with low HbA1c, where this difference was not significant [41]). This lower muscle oxygen extraction may be explained by either a reduced capacity to use oxygen (e.g. mitochondrial dysfunction; discussed subsequently) or a reduced capacity for oxyhemoglobin dissociation. An 8% HbA1c level (which is often found in people with T1D) reduces the kinetics of hemoglobin oxygen release by 10% compared with a 4% HbA1c level [56]. This mechanism is the most likely cause of increased oxygen affinity for hemoglobin, because other factors with the potential to cause this effect (blood pH, erythrocyte 2,3-DPG, $PaCO_2$) are comparable between T1D and CON.

Rissanen [34] also revealed that endurance training increased maximal tissue deoxygenation in the CON group but not in the T1D group. They proposed that unaltered oxygen extraction in the T1D group is a result of unchanged enzymatic oxidative capacity in muscle mitochondria or unchanged HbA1c. This difference, however, did not prevent an overall improvement in exercise performance compared with CON [34].

Oxygen metabolism: Mitochondrial oxidative capacity is a product of mitochondrial density, mitochondrial efficiency, and mitochondrial respiration. Neither Heyman [17] nor Item [27] found a significant difference in mitochondrial oxidative capacity (of the vastus lateralis or gastrocnemius muscle fibres, respectively) between T1D and CON. Mitochondrial density is comparable between those with and without T1D [17], whereas mitochondrial efficiency might be lower [57]. Overall mitochondrial respiratory capacity did not differ between TID and CON in the studies included in this review [17], but alterations in individual complex-supported mitochondrial respiration may exist. Heyman [17] suggests that complex IV respiratory capacity (relative to complex I capacity) is impaired in people with T1D, with oxygen consumption being 29% lower compared with CON. No differences were found between T1D and CON in the other complexes [17,27,58]. Long-standing diabetes and a high HbA1c may accelerate mitochondrial degeneration in people with T1D. Prolonged hyperglycemia has been found to increase the production of reactive oxygen species, causing a concomitant change in mitochondrial morphology [59] and damage to specific complex subunits [60]. It is unclear whether this reduces $\dot{V}O_{2peak}$ in people with T1D, given that the mitochondria's ability to consume oxygen far exceeds the cardiorespiratory system's ability to supply it during maximal whole-body exercise [39]. However, prolonged submaximal exercise may be impaired in T1D if mitochondrial enzymes cause lower fat oxidation and higher lactate production than in CON.

In vivo mitochondrial dysfunction may be related to defects in oxygen or substrate delivery. In their examination of physiological responses to a time-to-exhaustion assessment, Ramires [26] suggests that higher plasma lactate concentrations at exhaustion in the T1D group could indicate a lower rate of glucose oxidation. Similarly, a lower respiratory exchange ratio and free fatty acid levels at exhaustion in the T1D group suggest that they relied more on fats as an energy source [26]. Under fasted conditions, people with T1D have a higher absolute fat oxidation and lower absolute carbohydrate oxidation at submaximal exercise intensities (45% to 80% $\dot{V}O_{2peak}$), compared with CON [5,61]. Lower carbohydrate oxidation at exhaustion may impact performance in high-intensity aerobic activities.

Summary: The differences in physiological mechanisms underpinning aerobic performance were modest and supported by only a few studies. Lower hemoglobin mass, regional blood volume, and

local muscle oxygen extraction may be potential mechanisms of impairment. This is depicted in Figure 2.

Proposed mechanisms underlying differences in anaerobic exercise performance

Structural integrity of skeletal muscle: Decreases in both the quantity and quality of skeletal muscle tissue can lead to reduced skeletal muscle strength [62]. The studies evaluating MVC in this review reveal similar performance results in TID and CON [15,28]. Though no differences were found in fibre size and fibre type, several other physiological differences were identified. Dial [28] reported higher laminin content and incidence of dystrophin disruption in T1D at rest and postexercise. These findings suggest lower skeletal muscle integrity, which may reduce force production and increase the susceptibility of muscle tissue to damage [62]. The mechanism behind these changes in skeletal muscle integrity is unclear.

Contractile function of skeletal muscle: Ionic regulation is crucial for muscle excitation and contraction, and therefore for muscle function. Harmer [29] revealed that during intense exercise, muscle Na^+-K^+-ATPase content and the rise in plasma [K^+] did not differ between T1D and CON, despite a higher supramaximal time-to-exhaustion in T1D. After 7 weeks of sprint training, plasma [K^+] regulation improved similarly in both groups. The decrease in extracellular [K^+] accumulation helps to maintain muscle membrane excitability, delaying peripheral fatigue and improving tolerance of intense exercise. This was linked to an increase in Na^+-K^+-ATPase content for CON, but not for T1D, suggesting different contributions of mechanisms controlling [K^+] regulation.

Harmer [30] revealed that during intense exercise, Ca^{2+}-ATPase activity and Ca^{2+} uptake are higher in T1D than in CON, reflecting their higher supramaximal time-to-exhaustion. Increased Ca^{2+}-ATPase activity promotes Ca^{2+} reuptake into the sarcoplasmic reticulum during intense exercise, improving Ca^{2+} availability for repeated contractions. This may delay fatigue and support high-intensity exercise performance. Interestingly, 7 weeks of sprint training had no impact on Ca^{2+}-ATPase activity or Ca^{2+} uptake in either T1D or CON.

Repair, growth, and remodelling of skeletal muscle: After damaging exercise, the proliferation of satellite cells into myoblasts aids muscle growth and repair. Two studies found that people with T1D had lower satellite cell content and a delay in satellite cell proliferation compared with CON [15,28], perhaps due to persistent activation in Notch signalling from chronic hyperglycemia [63]. This delayed reparative response impacts future training quality and performance. Minnock [18] found that although 1RM was similar between T1D and CON before exercise intervention, a larger improvement was observed in CON after 12 weeks of combined resistance and aerobic training. They suggest that T1D might affect exercise-induced training adaptations by decreasing mitochondria-related gene expression and increasing inflammation-related gene expression. Optimal adaptation to exercise training occurs when exercise intensity is matched with adequate recovery [2]. Hence, athletes with T1D may require greater recovery periods after anaerobic training sessions.

Summary: The differences in physiological mechanisms underpinning anaerobic performance were slight and often only reported in single studies. Interestingly, the main differences seem to involve the recovery period after exercise, with greater inflammatory activity potentially preventing optimal muscle growth and repair. This is depicted in Figure 2.

Effects of acute and long-term glycemic management on exercise performance

Glycemia: Low blood glucose levels may affect exercise performance. Time spent at < 3.9 mmol/L has been found to decrease endurance capacity, increase perceived exertion [64], and impair sports skill performance [6]. For this reason, Riddell et al [65] recommend that time spent at <3.9 mmol/L be limited to <5% for optimal performance. High blood glucose levels may have a lesser impact on exercise performance. A meta-analysis by McGuire et al [66] determined that blood glucose levels between 10.0 and 17.0 mmol/L did not affect $\dot{V}O_{2peak}$. Similarly, a blood glucose level between 14.0 and 17.0 mmol/L did not affect performance in vertical jump, triplicate 6-second sprint cycling, grip strength, balance [67], or sports skill assessments [6]. However, it is important to note that people with T1D anecdotally report worsened exercise quality with hyperglycemia.

Most studies included in this review only evaluated data where participants had not experienced hypoglycemia before or during exercise, because antecedent hypoglycemia diminishes glucoregulatory responses to subsequent exercise [68]. Although this removes confounding factors, it reduces generalizability for people with T1D.

HbA1c and T1D duration: Figure 3 provides a visual comparison of $\dot{V}O_{2peak}$ between T1D and CON and indicates how HbA1c (Figure 3A) and T1D duration (Figure 3B) may modify this result. Nine studies evaluated the impact of HbA1c and/or T1D duration on aerobic capacity ($\dot{V}O_{2peak}$). Several found an inverse relationship between $\dot{V}O_{2peak}$ and HbA1c [8,17,32] and between $\dot{V}O_{2peak}$ and T1D duration [17], whereas one found a significant difference in $\dot{V}O_{2peak}$ between low-HbA1c and high-HbA1c subgroups [47]. A meta-analysis by Eckstein [69] revealed a weak but significant correlation between $\dot{V}O_{2peak}$ and HbA1c ($r=-0.19$, $p<0.001$). Meta-regression revealed that for each 1% increase in HbA1c, relative $\dot{V}O_{2peak}$ was lower by 1.46 mL/kg/min ($p<0.001$).

The studies included in this review also demonstrated an inverse correlation between HbA1c and lung diffusion capacity [19], stroke volume [33,47] cardiac output [27,33], peak leg muscle blood flow [33], leg muscle oxygen extraction [17], and mitochondrial capacity [27]. Heyman et al [17] identified an inverse correlation between T1D duration and complex IV relative capacity. These differences may be due to reduced oxygen-carrying capacity and long-term oxidative damage, which suggests that careful glycemic management is important for athletic performance in people with T1D.

Study Limitations

We have identified the following limitations to this review pertaining to the methodological heterogeneity of studies, the scope of performance outcomes, and population representation. First, owing to the substantial heterogeneity across study designs, populations, and outcomes, meta-analysis is precluded in the current review, and findings should be interpreted accordingly. Second, the predominant use of $\dot{V}O_{2peak}$ as the primary outcome measure in several studies reflects aerobic capacity rather than exercise performance, because although a higher $\dot{V}O_{2peak}$ is essential for meeting energy requirements, factors such as $\%\dot{V}O_{2peak}$ at lactate threshold, velocity at $\dot{V}O_{2peak}$, and running economy all also play a vital role in performance. Similarly, the anaerobic assessments used (i.e. 1RM, MVC, and supramaximal time-to-exhaustion) have variable predictive validity for real-world performance contexts. Last, most included studies examined sedentary or recreationally active male participants, emphasizing a male bias in the current literature. The underrepresentation of female participants (32.3%)

and the absence of studies evaluating athletes at or above a "highly trained" calibre (tier 3 and higher) mean that findings should not be extrapolated to female athletes or higher performance populations, where physiological differences may meaningfully alter the relationships observed. Furthermore, the tight glycemic management applied across most studies, while strengthening internal validity, limits the generalizability of findings to everyday athletic contexts where glycemic variability is often unavoidable.

Future Directions

Future research should prioritize direct performance assessments over surrogate measures of aerobic capacity, evaluate anaerobic and skill performance, and include female and higher-calibre athletic populations with T1D, where evidence remains sparse. This will allow more definitive conclusions about real-world athletic success to be drawn.

The underrepresentation of female participants in this review is compounded by their majority involvement (67.5%) in mixed-sex cohort studies. Research involving female-only cohorts or male- vs female-cohort analyses will help establish the impact of T1D on female athletes' responses to exercise and exercise training. This is particularly important for physiological mechanisms where sexual dimorphism has previously been reported. Furthermore, future research should endeavour to define the menstrual status of its participants, as outlined by Smith et al [70].

Exercise testing protocols in athletes with T1D should not assess performance alone, but also glycemia and glycemic management strategies. Intra-exercise glycemic variability may alter physiological responses to exercise, confounding the interpretation of performance outcomes. Furthermore, strategies to minimize the risk of hypoglycemia (e.g. defensive carbohydrate consumption, insulin dose reductions, insulin suspension) may contribute to between-study heterogeneity.

Standardizing and reporting glycemic management strategies during exercise testing (e.g. glycemic target ranges, insulin regimen, insulin-on-board, carbohydrate intake, antecedent exercise and hypoglycemia) may help distinguish the physiological effects of T1D from those related to acute glycemia. A glycemic target range of 4.0 to 10.0 mmol/L is often recommended to reduce the risk of hypoglycemia and minimize the risk of glycosuria and associated fluid loss. Future studies may benefit from narrower pre-exercise and intra-exercise glycemic targets (4.0 to 7.0 mmol/L, provided individuals are asymptomatic for hypoglycemia) to minimize confounding. However, studies that reflect real-world glycemic variability will also be relevant to understanding the practical implications for athletes with T1D.

Determining the impact of specific exercise types and/or training blocks on the recovery status of athletes with T1D may help coaches build more individualized training programs that maximize the adaptive capacity of these athletes. Monitoring perceived exertion, glucose trends, and recovery markers can guide whether recovery windows should be extended. Similarly, potential alterations in cardiovascular function may influence the physiological responses of athletes with T1D to prescribed training intensities. This means that physiological thresholds could occur at different relative exercise intensities, and training zones based on fixed percentages of maximal heart rate, power output, or $\dot{V}O_{2peak}$ may not provide equivalent training stimuli for athletes with T1D. Finally, potential limitations in neuromuscular function may support the prioritization of relative strength over absolute strength in athletic training. This facilitates performance in many sporting tasks which rely on strength-to-weight ratio rather than absolute strength alone.

Conclusion

This review supports the understanding of physiology in T1D that may affect exercise performance and adaptive responses to training. Although differences appear to exist, namely in mechanisms related to aerobic capacity, there is currently an absence of evidence to indicate that T1D is meaningfully limiting to exercise performance. These findings support the aspirations of athletes with T1D to participate at a high level, including the physical activity goals of all individuals with T1D.

The inverse relationship between HbA1c and several performance-relevant physiological parameters suggests that optimizing glycemic management may be an important adjunct to training in athletes with T1D. The clinical and practical complexity of achieving this during heavy training periods warrants careful, individualized management in consultation with relevant health care professionals. Many athletes with T1D have risen to this challenge, achieving elite and world-class levels of performance. Advances in the resources available to athletes with T1D (e.g. peer mentorship, online communities, evidence-based guidelines), and the efficacy of technologies and therapies (e.g. continuous glucose monitoring, insulin pumps, insulin pharmacokinetics) have also helped facilitate this athletic success in recent years.

Acknowledgments

The authors greatly appreciate their university librarian Vanessa Varis, whose knowledge and insight helped shape the search strategy used in this review.

Author Disclosures

Conflicts of interest: None.
Financial support: None.

Author Contributions

All authors were involved in study conception and design. S.S. completed all data collection. S.S., A.S., R.H. and R.D. analysed the data; while S.S., A.S. and R.D. interpreted the data. S.S. drafted the manuscript. Revision and editing were finalised by S.S., A.S., R.D., L.N. and V.S. All authors approved the final version of the paper.

References

[1] Riddell MC, Gallen IW, Smart CE, Taplin CE, Adolfsson P, Lumb AN, et al. Exercise management in type 1 diabetes: A consensus statement. Lancet Diabetes Endocrinol 2017;5:377–90.
[2] Yang P, Xu R, Le Y. Factors influencing sports performance: A multi-dimensional analysis of coaching quality, athlete well-being, training intensity, and nutrition with self-efficacy mediation and cultural values moderation. Heliyon 2024;10:e36646.
[3] Dizon S, Malcolm J, Rowan M, Keely EJ. Patient perspectives on managing type 1 diabetes during high-performance exercise: What resources do they want? Diabetes Spectr 2019;32:36–45.
[4] Yardley JE. The athlete with type 1 diabetes: Transition from case reports to general therapy recommendations. Open Access J Sports Med 2019;10:199–207.
[5] Dumke CL, Keck NA, McArthur MC, Corcoran MH. Patients with type 1 diabetes oxidize fat at a greater rate than age- and sex-matched controls. Phys Sportsmed 2013;41:78–85.
[6] Kelly D, Hamilton JK, Riddell MC. Blood glucose levels and performance in a sports cAMP for adolescents with type 1 diabetes mellitus: A field study. Int J Pediatr 2010;2010:216167.
[7] Lee MJ, Coast JR, Hempleman SC, Baldi JC. Type 1 diabetes duration decreases pulmonary diffusing capacity during exercise. Respiration 2016;91:164–70.
[8] Gusso S, Pinto TE, Baldi JC, Robinson E, Cutfield WS, Hofman PL. Diastolic function is reduced in adolescents with type 1 diabetes in response to exercise. Diabetes Care 2012;35:2089–94.
[9] Lespagnol E, Tagougui S, Fernandez BO, Zerimech F, Matran R, Maboudou P, et al. Circulating biomarkers of nitric oxide bioactivity and impaired muscle

vasoreactivity to exercise in adults with uncomplicated type 1 diabetes. Diabetologia 2021;64:325–38.
[10] Monaco CMF, Hughes MC, Ramos SV, Varah NE, Lamberz C, Rahman FA, et al. Altered mitochondrial bioenergetics and ultrastructure in the skeletal muscle of young adults with type 1 diabetes. Diabetologia 2018;61:1411–23.
[11] Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. BMJ 2021;372:n71.
[12] Ramspek CL, Steyerberg EW, Riley RD, Rosendaal FR, Dekkers OM, Dekker FW, et al. Prediction or causality? A scoping review of their conflation within current observational research. Eur J Epidemiol 2021;36:889–98.
[13] Munn Z, Stern C, Aromataris E, Lockwood C, Jordan Z. What kind of systematic review should I conduct? A proposed typology and guidance for systematic reviewers in the medical and health sciences. BMC Med Res Methodol 2018;18:5.
[14] The EndNote Team. EndNote. Philadelphia, PA: Clarivate, 2013.
[15] Dial AG, Monaco CMF, Grafham GK, Patel TP, Tarnopolsky MA, Hawke TJ. Impaired function and altered morphology in the skeletal muscles of adult men and women with type 1 diabetes. J Clin Endocrinol Metab 2021;106:2405–22.
[16] Eckstein ML, Farinha JB, McCarthy O, West DJ, Yardley JE, Bally L, et al. Differences in physiological responses to cardiopulmonary exercise testing in adults with and without type 1 diabetes: A pooled analysis. Diabetes Care 2021;44:240–7.
[17] Heyman E, Daussin F, Wieczorek V, Caiazzo R, Matran R, Berthon P, et al. Muscle oxygen supply and use in type 1 diabetes, from ambient air to the mitochondrial respiratory chain: Is there a limiting step? Diabetes Care 2020;43:209–18.
[18] Minnock D, Annibalini G, Valli G, Saltarelli R, Krause M, Barbieri E, et al. Altered muscle mitochondrial, inflammatory and trophic markers, and reduced exercise training adaptations in type 1 diabetes. J Physiol 2022;600:1405–18.
[19] Wheatley CM, Baldi JC, Cassuto NA, Foxx-Lupo WT, Snyder EM. Glycemic control influences lung membrane diffusion and oxygen saturation in exercise-trained subjects with type 1 diabetes: Alveolar-capillary membrane conductance in type 1 diabetes. Eur J Appl Physiol 2011;111:567–78.
[20] Van Ryckeghem L, Franssen WMA, Verbaanderd E, Indesteege J, De Vriendt F, Verwerft J, et al. Cardiac function is preserved in adolescents with well-controlled Type 1 diabetes and a normal physical fitness: A cross-sectional study. Can J Diabetes 2021;45:718–724.e1.
[21] Chai KEK, Lines RLJ, Gucciardi DF, Ng L. Research Screener: A machine learning tool to semi-automate abstract screening for systematic reviews. Syst Rev 2021;10:93.
[22] Moola S, Munn Z, Tufanaru C, Aromataris E, Sears K, Sfetcu R, et al. Chapter 7. Systematic reviews of etiology and risk. In: Aromataris E, Munn Z, editors. JBI manual for evidence synthesis. Adelaide, Australia: The Joanna Briggs Institute, 2020.
[23] Barker TH, Habibi N, Aromataris E, Stone JC, Leonardi-Bee J, Sears K, et al. The revised JBI critical appraisal tool for the assessment of risk of bias for quasi-experimental studies. JBI Evid Synth 2024;22:378–88.
[24] Atkins D, Best D, Briss PA, Eccles M, Falck-Ytter Y, Flottorp S, et al. Grading quality of evidence and strength of recommendations. BMJ 2004;328:1490.
[25] Mourot L, Fornasiero A, Rakobowchuk M, Skafidas S, Brighenti A, Stella F, et al. Similar cardiovascular and autonomic responses in trained type 1 diabetes mellitus and healthy participants in response to half marathon. Diabetes Res Clin Pract 2020;160:107995.
[26] Ramires PR, Forjaz CL, Silva ME, Diament J, Nicolau W, Liberman B, et al. Exercise tolerance is lower in type I diabetics compared with normal young men. Metabolism 1993;42:191–5.
[27] Item F, Heinzer-Schweizer S, Wyss M, Fontana P, Lehmann R, Henning A, et al. Mitochondrial capacity is affected by glycemic status in young untrained women with type 1 diabetes but is not impaired relative to healthy untrained women. Am J Physiol Regul Integr Comp Physiol 2011;301:R60–6.
[28] Dial AG, Grafham GK, Monaco CMF, Voth J, Brandt L, Tarnopolsky MA, et al. Alterations in skeletal muscle repair in young adults with type 1 diabetes mellitus. Am J Physiol Cell Physiol 2021;321:C876–83.
[29] Harmer AR, Ruell PA, McKenna MJ, Chisholm DJ, Hunter SK, Thom JM, et al. Effects of sprint training on extrarenal potassium regulation with intense exercise in type 1 diabetes. J Appl Physiol (1985) 2006;100:26–34.
[30] Harmer AR, Ruell PA, Hunter SK, McKenna MJ, Thom JM, Chisholm DJ, et al. Effects of type 1 diabetes, sprint training and sex on skeletal muscle sarcoplasmic reticulum Ca2+ uptake and Ca2+-ATPase activity. J Physiol 2014;592:523–35.
[31] Hyrylä VV, Rissanen AE, Peltonen JE, Koponen AS, Tikkanen HO, Tarvainen MP. Altered expiratory flow dynamics at peak exercise in adult men with well-controlled Type 1 diabetes. Front Physiol 2022;13:836814.
[32] Jlali I, Heyman E, Matran R, Marais G, Descatoire A, Rabasa-Lhoret R, et al. Respiratory function in uncomplicated type 1 diabetes: Blunted during exercise even though normal at rest. Diabet Med 2023;40:e15036.
[33] Rissanen APE, Tikkanen HO, Koponen AS, Aho JM, Peltonen JE. Central and peripheral cardiovascular impairments limit VO(2peak) in type 1 diabetes. Med Sci Sports Exerc 2015;47:223–30.
[34] Rissanen AE, Tikkanen HO, Koponen AS, Aho JM, Peltonen JE. One-year unsupervised individualized exercise training intervention enhances cardiorespiratory fitness but not muscle deoxygenation or glycemic control in adults with type 1 diabetes. Appl Physiol Nutr Metab 2018;43:387–96.
[35] Wilson LC, Peebles KC, Hoye NA, Manning P, Sheat C, Williams MJA, et al. Resting heart rate variability and exercise capacity in type 1 diabetes. Physiol Rep 2017;5:e13248.
[36] Koponen AS, Peltonen JE, Päivinen MK, Aho JM, Hägglund HJ, Uusitalo AL, et al. Low total haemoglobin mass, blood volume and aerobic capacity in men with type 1 diabetes. Eur J Appl Physiol 2013;113:1181–8.
[37] Peltonen JE, Koponen AS, Pullinen K, Hägglund H, Aho JM, Kyröläinen H, et al. Alveolar gas exchange and tissue deoxygenation during exercise in type 1 diabetes patients and healthy controls. Respir Physiol Neurobiol 2012;181:267–76.
[38] Tedjasaputra V, Bouwsema MM, Stickland MK. Effect of aerobic fitness on capillary blood volume and diffusing membrane capacity responses to exercise. J Physiol 2016;594:4359–70.
[39] Bassett Jr DR, Howley ET. Limiting factors for maximum oxygen uptake and determinants of endurance performance. Med Sci Sports Exerc 2000;32:70–84.
[40] Ljubić S, Metelko Z, Car N, Roglić G, Drazić Z. Reduction of diffusion capacity for carbon monoxide in diabetic patients. Chest 1998;114:1033–5.
[41] Tagougui S, Leclair E, Fontaine P, Matran R, Marais G, Aucouturier J, et al. Muscle oxygen supply impairment during exercise in poorly controlled type 1 diabetes. Med Sci Sports Exerc 2015;47:231–9.
[42] Schmidt WF, Doerfler C, Wachsmuth N, Voelzke C, Treff G, Thoma S, et al. Influence of body mass, body composition, and performance state on total hemoglobin mass: 2790 board #184 May 29 3:30 PM – 5:00 PM. Med Sci Sports Exerc 2009;41:461.
[43] Nigra AD, Monesterolo NE, Rivelli JF, Amaiden MR, Campetelli AN, Casale CH, et al. Alterations of hemorheological parameters and tubulin content in erythrocytes from diabetic subjects. Int J Biochem Cell Biol 2016;74:109–20.
[44] Lush DJ, King JA, Fray JC. Pathophysiology of low renin syndromes: Sites of renal renin secretory impairment and prorenin overexpression. Kidney Int 1993;43:983–99.
[45] Weberruß H, Baumgartner L, Mühlbauer F, Shehu N, Oberhoffer-Fritz R. Training intensity influences left ventricular dimensions in young competitive athletes. Front Cardiovasc Med 2022;9:961979.
[46] Larsson Y, Persson B, Sterky G, Thoren C. Functional adaptation to rigorous training and exercise in diabetic and nondiabetic adolescents. J Appl Physiol 1964;19:629–35.
[47] Baldi JC, Cassuto NA, Foxx-Lupo WT, Wheatley CM, Snyder EM. Glycemic status affects cardiopulmonary exercise response in athletes with type I diabetes. Med Sci Sports Exerc 2010;42:1454–9.
[48] Gusso S, Pinto T, Baldi JC, Derraik JGB, Cutfield WS, Hornung T, et al. Exercise training improves but does not normalize left ventricular systolic and diastolic function in adolescents with type 1 diabetes. Diabetes Care 2017;40:1264–72.
[49] Adolfsson P, Nilsson S, Albertsson-Wikland K, Lindblad B. Hormonal response during physical exercise of different intensities in adolescents with type 1 diabetes and healthy controls. Pediatr Diabetes 2012;13:587–96.
[50] Tagougui S, Fontaine P, Leclair E, Aucouturier J, Matran R, Oussaidene K, et al. Regional cerebral hemodynamic response to incremental exercise is blunted in poorly controlled patients with uncomplicated type 1 diabetes. Diabetes Care 2015;38:858–67.
[51] Umpierrez GE, Davis GM, ElSayed NA, Fadini GP, Galindo RJ, Hirsch IB, et al. Hyperglycaemic crises in adults with diabetes: A consensus report. Diabetologia 2024;67:1455–79.
[52] Lespagnol E, Dauchet L, Pawlak-Chaouch M, Balestra C, Berthoin S, Feelisch M, et al. Early endothelial dysfunction in type 1 diabetes is accompanied by an impairment of vascular smooth muscle function: A meta-analysis. Front Endocrinol (Lausanne) 2020;11:203.
[53] Calbet JA, Joyner MJ. Disparity in regional and systemic circulatory capacities: Do they affect the regulation of the circulation? Acta Physiol (Oxf) 2010;199:393–406.
[54] Yang DR, Wang MY, Zhang CL, Wang Y. Endothelial dysfunction in vascular complications of diabetes: A comprehensive review of mechanisms and implications. Front Endocrinol (Lausanne) 2024;15:1359255.
[55] Skattebo Ø, Martin-Rincon M, Rud B, Nielsen J, Hegg LHN, Kleive AV, et al. Determinants of maximal oxygen uptake in highly trained females and males: A mechanistic study of sex differences using advanced invasive methods. J Physiol; 2025.
[56] Marschner JP, Seidlitz T, Rietbrock N. Effect of 2,3-diphosphoglycerate on O2-dissociation kinetics of hemoglobin and glycosylated hemoglobin using the stopped flow technique and an improved in vitro method for hemoglobin glycosylation. Int J Clin Pharmacol Ther 1994;32:116–21.
[57] Goulding RP, Charlton BT, Breedveld EA, Huijts JY, Van Der Laan M, Strating AR, et al. Skeletal muscle mitochondrial health in type 1 diabetes: The role of exercise capacity and lifestyle factors. Diabetologia 2025;68:1823–35.
[58] Costill DL, Cleary P, Fink WJ, Foster C, Ivy JL, Witzmann F. Training adaptations in skeletal muscle of juvenile diabetics. Diabetes 1979;28:818–22.
[59] Yu T, Robotham JL, Yoon Y. Increased production of reactive oxygen species in hyperglycemic conditions requires dynamic change of mitochondrial morphology. Proc Natl Acad Sci U S A 2006;103:2653–8.
[60] Choksi KB, Boylston WH, Rabek JP, Widger WR, Papaconstantinou J. Oxidatively damaged proteins of heart mitochondrial electron transport complexes. Biochim Biophys Acta 2004;1688:95–101.

[61] Raguso CA, Coggan AR, Gastaldelli A, Sidossis LS, Bastyr EJ, Wolfe RR. Lipid and carbohydrate metabolism in IDDM during moderate and intense exercise. Diabetes 1995;44:1066–74.
[62] Shaikh S, Ahmad K, Lim JH, Ahmad SS, Lee EJ, Choi I. Skeletal muscle aging: Enhancing skeletal muscle integrity and function as a potential pharmacological approach. Pharmaceuticals (Basel) 2025;18:1407.
[63] D'Souza DM, Zhou S, Rebalka IA, MacDonald B, Moradi J, Krause MP, et al. Decreased satellite cell number and function in humans and mice with type 1 diabetes is the result of altered Notch signaling. Diabetes 2016;65:3053–61.
[64] Riddell MC, Bar-Or O, Ayub BV, Calvert RE, Heigenhauser GJ. Glucose ingestion matched with total carbohydrate utilization attenuates hypoglycemia during exercise in adolescents with IDDM. Int J Sport Nutr 1999;9:24–34.
[65] Riddell MC, Scott SN, Fournier PA, Colberg SR, Gallen IW, Moser O, et al. The competitive athlete with type 1 diabetes. Diabetologia 2020;63:1475–90.
[66] McGuire B, Dadah H, Oliver D. The effects of acute hyperglycaemia on sports and exercise performance in type 1 diabetes: A systematic review and meta-analysis. J Sci Med Sport 2024;27:78–85.
[67] Rothacker KM, Armstrong S, Smith GJ, Benjanuvatra N, Lay B, Adolfsson P, et al. Acute hyperglycaemia does not have a consistent adverse effect on exercise performance in recreationally active young people with type 1 diabetes: A randomised crossover in-clinic study. Diabetologia 2021;64:1737–48.
[68] Galassetti P, Tate D, Neill RA, Morrey S, Wasserman DH, Davis SN. Effect of antecedent hypoglycemia on counterregulatory responses to subsequent euglycemic exercise in type 1 diabetes. Diabetes 2003;52(7):1761–9.
[69] Eckstein ML, Aberer F, Dobler FJR, Aziz F, Heise T, Sourij H, et al. Association of HbA1c with VO2max in individuals with type 1 diabetes: A systematic review and meta-analysis. Metabolites 2022;12:1017.
[70] Smith ES, McKay AKA, Ackerman KE, Harris R, Elliott-Sale KJ, Stellingwerff T, et al. Methodology review: A protocol to audit the representation of female athletes in sports science and sports medicine research. Int J Sport Nutr Exerc Metab 2022;32:114–27.
[71] Jakober B, Schmülling RM, Eggstein M. Carbohydrate and lipid metabolism in type I diabetics during exhaustive exercise. International journal of sports medicine 1983;4(2):104–8.

Appendix A
Search strategy

	Concept 1: Type 1 diabetes	Concept 2: Physiological differences	Concept 3: Exercise performance	Concept 4: Age
Key words *	"type 1 diabet*" OR "type I diabet*" OR T1DM OR T1D OR IDDM OR "insulin dependent diabet*" OR "autoimmune diabet*" OR "juvenile onset diabet*" OR "sudden onset diabet*" OR "ketosis prone diabet*" OR "brittle diabet*"	cardi* or vascular* or "diastolic function" or "systolic function" or "ventricular function" or "atrial function" or "stroke volume" or circulatory or "endothelial function" or respirat* or pulmonary or "blood lactate" or muscul* or muscle* or "joint mobility" or "sweat rate" or "core temperature" or mitochondria* or glycogen or "substrate oxid*" or "carbohydrate oxid*" or "glucose oxid*" or "fat oxid*" or "lipid oxid*" or "lipid peroxid*" or "beta oxidation" or "ketone body oxid*" or "protein oxid*" or "fuel metabolism" or "energy metabolism" or "carbohydrate metabolism" or "glucose metabolism" or "fat metabolism" or "lipid metabolism" or "ketone body metabolism" or "protein metabolism" or "fuel utili*" or glycolysis or "Krebs cycle" or "citric acid cycle" or "oxidative phosphorylation" or "hormon* regulation" or "hormon* response" or "growth hormone" or catecholamine or cortisol or epinephrine or norepinephrine or adrenaline or noradrenaline	exercis* or sport* or athlet* or "time trial" or run* or swim* or cycl* or soccer or football or basketball or netball or volleyball or cricket or hockey or dance or endurance or fitness or "physical capacity" or "physical exertion" or "perceived exertion" or "time to exhaustion" or "aerobic capacity" or "oxygen consumption" or "oxygen uptake" or "aerobic power" or "anaerobic power" or "anaerobic capacity" or "anaerobic threshold" or "lactate threshold" or "power output" or Wingate or "physical fatig*" or "muscle fatig*" or "musc* strength" or "musc* power" or "repetition max*" or "maximal voluntary contraction" or "isometric muscle contraction" or "isokinetic muscle contraction" or agility or flexib*	teen* or adolescen* or youth* or "young person*" or "young people" or adult* or "middle age*" or master*
Medline †	Diabetes mellitus, type 1/	exp Endocrine System/ or exp Cardiovascular System/ or exp Atrial Function/ or exp Ventricular Function or exp Myocardial Contraction/ or exp Blood Volume/ or Erythrocyte Indices/ or exp Respiratory System/ or Pulmonary Diffusing Capacity/ or Pulmonary Gas Exchange/ or exp Musculoskeletal System/ or exp Muscle Contraction/ or exp Muscle Development/ or exp Range of Motion, Articular/ or exp Nervous System/ or exp Energy Metabolism/ or exp Carbohydrate Metabolism/ or exp Lipid Metabolism/ or Lipid Peroxidation/	exp Exercise/ or Muscle Fatigue/ or exp Muscle Strength/ or exp Physical Endurance/ or Physical Exertion/ or exp Physical Fitness/ or Oxygen Consumption/ or Psychomotor Performance/ or Exercise Test/ or exp Exercise Therapy/ or exp Sports/	Adolescent/ or Adult/ or Young Adult/ or Middle Aged/
EMBASE †	Insulin-dependent diabetes mellitus/	exp Endocrine Function/ or exp Cardiovascular Function/ or exp Respiratory Function/ or exp Musculoskeletal Function/ or exp Nervous System Function/ or exp Energy Metabolism/ or exp Carbohydrate Metabolism/ or exp Lipid Metabolism/ or exp Protein Metabolism/	exp Exercise/ or exp Physical Activity/ or exp Physical Performance/ or exp Physical Capacity/ or Oxygen Consumption/ or exp Muscle Strength/ or exp Muscle training/ or exp Training/ or exp Exercise Test/ or exp Psychomotor Performance/ or exp Athlete/ or exp Sport/	Adolescent/ or Adulthood/ or Adult/ or Young Adult/ or Middle Aged/
CINAHL †	MH "Diabetes mellitus, type 1"	MH "Endocrine System+" or "Cardiovascular System+" or "Respiratory System+" or "Cardiopulmonary Physiology+" or "Musculoskeletal System+" or "Musculoskeletal System Physiology+" or "Nervous System+" or "Nervous System Physiology+" or "Energy Metabolism+" or "Carbohydrate Metabolism+"	MH "Exercise+" or "Physical Activity" or "Physical Fitness+" or "Physical Performance" or "Sports+" or "Exertion+" or "Exercise Test+" or "Oxygen Consumption+" or "Athletes+"	MH Adolescence or Adult or "Young Adult" or "Middle Age"
SPORTDiscus †	SU "Type 1 diabetes"	SU "Endocrine system" (explode) or "Cardiovascular system" (explode) or "Cardiopulmonary system" (explode) or "Respiratory mechanics" or "Respiratory muscles" or "Musculoskeletal System" (explode) or "Nervous System" (explode) or "Energy Metabolism" (explode) or "Carbohydrate Metabolism" (explode) or "Lipid Metabolism" (explode) or "Ketone body Metabolism" (explode) or "Protein Metabolism" (explode)	SU "Exercise" (explode) or "Physical activity" (explode) or "Physical fitness" (explode) or "Cardiopulmonary fitness measurement" (explode) or "Anaerobic capacity" (explode) or "Oxygen Consumption" (explode) or "Physical Training & conditioning" (explode) or "Sports" (explode) or "Sports participation" or "Athletes" (explode) or "Athletic ability" (explode) or "Athletics" (explode)	SU "Teenagers" or "Young adults" or "Older people" (explode) or "Older athletes" (explode)

* Keywords were searched in the title, abstract, and keywords.
† Subject headings are not required for the other databases (Scopus, Web of Science, ProQuest).

Database	Strategy to limit search to human studies
Medline	not (exp animals/ not humans.sh.)
EMBASE	not ((exp animal/ or exp invertebrate/ or nonhuman/ or animal experiment/ or animal tissue/ or animal model/ or exp plant/ or exp fungus/) not (exp human/ or human tissue/))
CINAHL	NOT (((MH "Animals+") OR (MH "Animal Studies") OR (TI "animal model*")) NOT (MH "human"))
SPORTDiscus	NOT (SU "Animal breeding" (explode) or "Animal nutrition" (explode) or "Gait in animals" (explode))
Scopus	AND NOT ((INDEXTERMS(animals OR animal)) AND NOT (INDEXTERMS(humans OR human)))
Web of Science	NOT (filter described in https://journals.sagepub.com/doi/suppl/10.1177/00236772211045485/suppl_file/sj-pdf-6-lan-10.1177_00236772211045485.pdf)
ProQuest	None*

* ProQuest is a multidisciplinary database with limited uniformity in indexing and keywords, so no rigorous strategy exists to limit searches to human studies.

Appendix B
Data extraction form

General information

Study ID	
Study title	
Reference (APA 7th)	
Journal impact factor	
Study aim	
Research theme	Performance ☐ Health ☐ Indirect associations with performance or health ☐
Study design	
Risk of bias assessment outcome	Risk of bias assessment tool
Notes	

Participant characteristics

Country/s		
Eligibility criteria		
Recruitment method		
Baseline sample (n =)	T1D (n =)	CON (n =)
Age (years, mean ± SD)		
Sex (female, n[%]; male, n[%])		
Menstrual status (n[%]; tier)		
Athletic calibre (median tier)		
Physical activity (min/wk, mean ± SD)		
HbA1c (%[mmol], mean ± SD)		
T1D duration (years, mean ± SD)		N/A
Age at diagnosis (years, mean ± SD)		N/A
Insulin delivery (MDI, n[%]; CSII, n[%])		N/A
Insulin units/day, mean ± SD)		N/A
Notes		

Intervention groups (one table per intervention)

Duration of intervention		
Frequency and duration of each session		
Description of intervention		
Provider (*e.g. no., profession, training*)		
Compliance	T1D (n =)	CON (n =)
Notes		

Outcomes (one table per outcome)

Exercise performance classification	Aerobic (direct) ☐	Aerobic (indirect) ☐	Anaerobic (direct) ☐	Anaerobic (indirect) ☐
Assessment type (units) Assessment protocol Is the assessment validated?	Yes ☐	No ☐	Unclear ☐	
Time points measured Results Main result (units, mean ± SD) Change from baseline Other *(e.g. odds ratio, p value)* Missing participants (n; reasons) Statistical methods Notes	T1D before (n =)	T1D after (n =)	CON before (n =)	CON after (n =)

Exercise performance classification	Aerobic (direct) ☐	Aerobic (indirect) ☐	Anaerobic (direct) ☐	Anaerobic (indirect) ☐
Assessment type (units) Assessment protocol Is the assessment validated?	Yes ☐	No ☐	Unclear ☐	
Time points measured Results Main result (units, mean ± SD) Other *(e.g. odds ratio, p value)* Statistical methods Notes	T1D (n =)		CON (n =) before	

Prognostic factors (one table per factor)

Prognostic factor classification	Cardiovascular ☐ Pulmonary ☐ Circulatory ☐ Thermoregulatory ☐ Neuromuscular ☐ Hormonal ☐ Metabolic ☐			
Prognostic factor Assessment type (units) Assessment protocol Is the assessment validated?	Yes ☐ No ☐ Unclear ☐			
Time points measured Results Main result (units, mean ± SD) Change from baseline Other *(e.g. odds ratio, p value)* Missing participants (n; reasons) Statistical methods Notes	T1D before (n =)	T1D after (n =)	CON before (n =)	CON after (n =)

OR

Prognostic factor classification		Cardiovascular ☐ Pulmonary ☐ Circulatory ☐ Thermoregulatory ☐ Neuromuscular ☐ Hormonal ☐ Metabolic ☐	
Prognostic factor Assessment type (units) Assessment protocol Is the assessment validated?		Yes ☐ No ☐ Unclear ☐	
Time points measured Results Main result (units, mean ± SD) Other *(e.g. odds ratio, p value)* Statistical methods Notes		T1D (n =)	CON (n =) before

Risk of Bias Assessment

Risk of bias assessment tool			
Domain	Risk of bias	Support for judgement	Location
	Low ☐ Moderate ☐ High ☐ Unclear ☐		
	Low ☐ Moderate ☐ High ☐ Unclear ☐		
	Low ☐ Moderate ☐ High ☐ Unclear ☐		
	Low ☐ Moderate ☐ High ☐ Unclear ☐		
	Low ☐ Moderate ☐ High ☐ Unclear ☐		
	Low ☐ Moderate ☐ High ☐ Unclear ☐		
	Low ☐ Moderate ☐ High ☐ Unclear ☐		
	Low ☐ Moderate ☐ High ☐ Unclear ☐		
Overall bias	Low ☐ Moderate ☐ High ☐ Unclear ☐		N/A
Notes			

Other

Key conclusions
Funding sources
Conflicts of interest
Other relevant studies
Notes

Appendix C
Risk of bias assessments

Table 2
Risk of bias summary table

Cross-sectional studies									
Study	Clear criteria for inclusion in sample?	Participants and setting described in detail?	Exposure measured in valid and reliable way?	Objective criteria used for measurement of condition?	Cofounding factors identified?	Strategies to deal with cofounding factors?	Outcomes measured in valid and reliable way?	Appropriate statistical analysis?	Overall risk of bias
Adolfsson et al 2012 [49]	Unclear	Unclear	Yes	Yes	Yes	Unclear	Yes	Yes	Moderate
Baldi et al 2010 [47]	Unclear	Unclear	Yes	Unclear	Yes	Yes	Yes	Unclear	Low
Dial et al 2021 [15]	Yes	Unclear	Yes	Unclear	Yes	Yes	Unclear	Yes	Moderate
Dial et al 2021 [28]	Unclear	Yes	Yes	Unclear	Yes	Yes	Unclear	Yes	Moderate
Gusso et al 2012 [8]	Unclear	Unclear	Yes	Yes	Unclear	Unclear	Yes	Unclear	Moderate
Heyman et al 2020 [17]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
Hyryla et al 2022 [31]	Yes	Yes	Yes	Unclear	Unclear	Yes	Yes	Yes	Moderate
Item et al 2010 [27]	Yes	Yes	Yes	Yes	Unclear	Unclear	Yes	Yes	Low
Jakober et al 1983 [71]	Unclear	Unclear	Yes	Yes	Unclear	Unclear	Yes	Unclear	High
Jlali et al 2023 [32]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
Koponen et al 2013 [36]	Yes	Yes	Yes	Yes	Unclear	Yes	Yes	Yes	Low
Lespagnol et al 2021 [9]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
Mourot et al 2020 [25]	Yes	Unclear	Yes	Unclear	Unclear	Unclear	Yes	Yes	Moderate
Peltonen et al 2012 [37]	Yes	Yes	Yes	Unclear	Unclear	Yes	Yes	Yes	Moderate
Ramires et al 1993 [26]	Yes	Unclear	Yes	Yes	Unclear	Unclear	Yes	Yes	Moderate
Rissanen et al 2015 [33]	Yes	Unclear	Yes	Yes	Yes	Unclear	Yes	Yes	Moderate
Tagougui et al 2015 [41]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
Tagougui et al 2015 [50]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low
Van Ryckegham et al 2021 [20]	Yes	Yes	Yes	Yes	Unclear	Yes	Yes	Yes	Low
Wheatley et al 2011 [19]	Unclear	Unclear	Yes	Unclear	Yes	Yes	Yes	Yes	Moderate
Wilson et al 2017 [35]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Low

Quasi-experimental studies										
Study	Clear cause' and 'effect'?	Control group?	Participants in comparisons similar?	Treatment/care, other than intervention?	Outcomes measured pre and post intervention?	Outcomes measured in same way?	Outcomes measured in reliable way?	Follow-up complete?	Appropriate statistical analysis?	Overall risk of bias
Costil et al 1979 [58]	Yes	Yes	Yes	Unclear	Yes	Yes	Yes	Unclear	Unclear	Moderate
Gusso et al 2017 [48]	Yes	Yes	Yes	Unclear	Yes	Yes	Yes	Unclear	Yes	Moderate
Harmer et al 2006 [29]	Yes	Yes	Yes	Unclear	No	Yes	Yes	Unclear	Yes	High
Harmer et al 2014 [30]	Yes	Yes	Yes	Unclear	Unclear	Yes	Yes	Unclear	Yes	Moderate
Larsson et al 1964 [46]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Unclear	Moderate
Minnock et al 2022 [18]	Yes	Yes	Yes	Unclear	Yes	Yes	Yes	Yes	Yes	Moderate
Rissanen et al 2018 [34]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Unclear	Yes	Moderate

Appendix D
GRADE Table

Table 4
GRADE: Certainty of evidence table for primary outcomes

Number of studies	Study design	Risk of bias	Inconsistency	Indirectness	Imprecision	Publication bias	Overall certainty
*Is there a difference in **time-trial performance** between people with and without T1D?*							VERY LOW
1	Cross-sectional	Not serious	Not serious*	Not serious	Very serious	Not serious*	
*Is there a difference in **submaximal time-to-exhaustion** between people with and without T1D?*							VERY LOW
2	Cross-sectional	Not serious	Serious	Not serious	Very serious	Not serious*	
Is there a difference in $\dot{V}O_{2peak}$ between people with and without T1D?							LOW
23	Cross-sectional and quasi-experimental	Not serious	Not serious	Not serious	Serious	Not serious	
*Is there a difference in **one-repetition maximum performance (full-body movement)** between people with and without T1D?*							VERY LOW
1	Quasi-experimental	Not serious	Not serious*	Not serious	Very serious	Not serious*	
*Is there a difference in **one-repetition maximum performance (isolated joint movement)** between people with and without T1D?*							VERY LOW
1	Quasi-experimental	Not serious	Not serious*	Not serious	Very serious	Not serious*	
*Is there a difference in **maximal voluntary isometric contraction** between people with and without T1D?*							VERY LOW
2	Cross-sectional	Not serious	Not serious*	Not serious	Very serious	Not serious*	
*Is there a difference in **supramaximal time-to-exhaustion** between people with and without T1D?*							VERY LOW
2	Quasi-experimental	Serious	Not serious*	Not serious	Very serious	Not serious*	

* Low number of studies makes this category difficult to evaluate accurately.