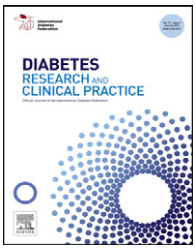
Contents available at Sciverse ScienceDirect

Diabetes Research and Clinical Practice

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

International Diabetes Federation



Review

Exercise training in high-risk ethnic populations with type 2 diabetes: A systematic review of clinical trials

William R. Sukala [a], Rachel Page [b], Birinder S. Cheema [c,*]

[a] School of Health and Human Sciences, Southern Cross University, Lismore, NSW, Australia
[b] Institute of Food, Nutrition & Human Health, Massey University, Wellington, New Zealand
[c] School of Biomedical and Health Sciences, University of Western Sydney, Campbelltown, Australia

ARTICLE INFO

Article history:
Received 9 October 2011
Received in revised form
27 January 2012
Accepted 2 February 2012
Published on line 3 March 2012

Keywords:
Aerobic training
Resistance training
Glycaemic control
Ethnicity
Race
Obesity

ABSTRACT

Background: To review clinical trials that have prescribed exercise training in high-risk, ethnic populations with type 2 diabetes mellitus (T2DM) and delineate areas for future research.

Method: A systematic review using computerized databases was performed.

Results: The systematic review located nine trials, including four uncontrolled trials, and five randomized controlled trials (RCTs) that included 521 participants. Cohorts studied included African, Indian, Polynesian, Hispanic, Arabian, and Chinese peoples and interventions included aerobic training, resistance training or a combination thereof. Several trials documented improvements in HbA1c, insulin action, body composition, blood lipids and systolic and diastolic blood pressure. In general, a longer duration and greater frequency of training resulted in greater adaptation. Studies demonstrating no effect were generally limited by an inadequate intervention. There was evidence of differential training responses between Caucasians and non-Caucasians in two studies drawing such comparisons.

Conclusions: Robust RCTs prescribing appropriate, targeted interventions and investigating relevant outcomes may be required to stimulate greater advocacy for exercise as a therapeutic adjunct for diabetes management in these populations. Investigations should be extended to other high-risk populations, particularly indigenous peoples who suffer an extreme burden of T2DM. Translation of research into clinical application should remain the overall objective.

Contents

1. Introduction . . . 207
2. Method . . . 208
2.1. Criteria for considering studies . . . 208
2.1.1. Study designs . . . 208
2.1.2. Participants . . . 208
2.1.3. Interventions . . . 208
2.1.4. Outcome measures . . . 208

* *Corresponding author at*: School of Biomedical and Health Sciences, University of Western Sydney, Locked Bag 1797, Penrith South DC, NSW 1797, Australia. Tel.: +61 2 4620 3795; mobile: +61 416 956 805; fax: +61 2 4620 3792.
E-mail address: B.Cheema@uws.edu.au (B.S. Cheema).

0168-8227/$ – see front matter
doi:10.1016/j.diabres.2012.02.001

2.2. Search method . . . 208
2.3. Assessment of research quality . . . 208
3. Results . . . 208
3.1. Studies retrieved and assessment of research quality . . . 208
3.2. Participants . . . 212
3.2.1. Sample size . . . 212
3.2.2. Ethnicity . . . 212
3.2.3. Gender . . . 212
3.2.4. Age . . . 212
3.2.5. Duration of diabetes . . . 212
3.2.6. Diabetes management . . . 212
3.2.7. Comorbidities and complications . . . 212
3.2.8. Participant attrition . . . 212
3.3. Exercise interventions . . . 212
3.3.1. Duration . . . 212
3.3.2. Frequency . . . 212
3.3.3. Modality . . . 212
3.3.4. Aerobic training intensity . . . 212
3.3.5. Aerobic training duration . . . 212
3.3.6. Resistance training intensity and volume . . . 212
3.3.7. Supervision . . . 213
3.3.8. Compliance . . . 213
3.3.9. Evidence of training effect . . . 213
3.4. Outcomes . . . 213
3.4.1. Adverse events . . . 213
3.4.2. Glycosylated hemoglobin (HbA1c) . . . 213
3.4.3. Insulin action . . . 213
3.4.4. Anthropometrics . . . 213
3.4.5. Blood lipids . . . 214
3.4.6. Haemodynamics . . . 214
4. Discussion . . . 214
References . . . 215

1. Introduction

The global prevalence of type 2 diabetes mellitus (T2DM) is projected to rise from an estimated 171 million cases in 2000 to 366 million cases in 2030 [1]. The statistics also clearly demonstrate that certain ethnic cohorts are more severely affected by this disease and related mortality. In fact, ethnicity itself is a well-established, non-modifiable risk factor for T2DM [2].

A prospective investigation of 78,419 apparently healthy women in the United States followed for a 20-year period noted that the relative risk of developing T2DM was 1.5 to 3-fold higher in those of Asian, Hispanic, and African descent versus those of Caucasian descent [3]. These findings have been corroborated by epidemiological investigation [4]. Indigenous populations throughout the world are particularly vulnerable to T2DM [5,6]. In New Zealand, for example, indigenous Polynesian peoples suffer nearly three times the prevalence of diabetes compared to the total population [7]. Such disparities have also been noted in Aboriginal Australian and Native American peoples [8–10].

Prescribed exercise, involving aerobic and resistance training modalities, has been shown to significantly reduce glycosylated hemoglobin (HbA_{1c}) and has therefore been recommended for the management and remission of T2DM [11–16]. Interestingly, however, the majority of trials used to formulate current exercise prescription guidelines have involved primarily Caucasian participants or the ethnicity of participants was not reported. This is notable given that certain cohorts are more severely affected by T2DM than others [17].

The mechanisms contributing to elevated risk of T2DM in certain ethnic groups remain to be fully elucidated; however, environmental, sociocultural, biological and genetic factors all play a role. The prevalence of T2DM has been shown to increase markedly in indigenous and ethnic populations subjected to westernized environments that depart from traditional food habits and lifestyle [17,18]. For example, Pima Indians living in urban areas in the United States have a diabetes prevalence of 54% and 37% for men and women, respectively, versus only 6% and 11% who maintain traditional lifestyle practices in Mexico [17]. Physiologic susceptibility also explains a greater burden of T2DM in certain cohorts. Markers such as fasting insulin, insulin sensitivity, pancreatic β-cell function, and glucoregulation have indeed been shown to differ by ethnicity, with non-Caucasian adults demonstrating greater susceptibility to T2DM and related non-communicable diseases [17,19–23]. Such disparities have also been noted in children [24,25].

Given the extreme rates of T2DM and empirical evidence of differential mitigating risk factors such as environmental and biological mechanisms, there is a robust rationale for

investigating exercise responses in these ethnic diabetes populations independently and comparatively. Accordingly, a number of exercise intervention trials have recently been conducted in ethnic populations who suffer disproportionately from T2DM. Findings from these studies could yield useful insights for the development of efficacious and targeted exercise prescriptions to mitigate the burgeoning epidemic of T2DM globally. Therefore, our objectives were three-fold:

1. To systematically and critically evaluate clinical trials that have prescribed exercise training in high-risk, ethnic populations with T2DM;
2. To summarize the metabolic adaptations to exercise noted in these trials; and
3. To delineate areas for future empirical investigation and application.

2. Method

A systematic review of prospective, exercise intervention trials enrolling high-risk ethnic populations with T2DM was conducted. Due to the heterogeneity of interventions and cohorts, and a paucity of robust randomized controlled trials (RCTs), the pooling of effect sizes across studies in a meta-analysis was not considered appropriate at this stage. There were also clinically important findings to present from the relevant uncontrolled trials.

2.1. Criteria for considering studies

2.1.1. Study designs

RCTs and trials that did not involve a non-treatment or placebo control group were included. Abstracts and case reports were not considered.

2.1.2. Participants

Trials enrolling men and women (≥18 years) with a diagnosis of T2DM were included. Trials enrolling participants with pre-diabetes were excluded. Trials were included if the study population was noted as having a higher incidence and/or prevalence of T2DM versus Caucasian counterparts or, if these comparisons were not mentioned in the article, the study enrolled an ethnic group with a higher incidence and/or prevalence of T2DM versus Caucasians based on available data.

2.1.3. Interventions

Trials prescribing aerobic and/or resistance training of eight weeks duration or longer were included. While a minimum intervention period of 12 weeks is generally considered adequate to adapt HbA1c (due to the lifespan of the red blood cell) [26], trials prescribing 8–12 week interventions were included given their potential to shift other relevant metabolic outcomes (e.g. adiposity). Trials evaluating the efficacy of multi-disciplinary lifestyle interventions were excluded. Evidence of effective training intervention was evaluated via outcomes of physical fitness and functional tests, including changes in measures of functional capacity, cardiorespiratory fitness, and muscular strength.

2.1.4. Outcome measures

Trials evaluating HbA1c and additional clinically relevant outcomes, including: insulin action, anthropometrics (i.e. body mass index, body composition), blood lipids, and blood pressure, were included.

2.2. Search method

A literature review was conducted to include studies from 1966 to 2011, limited to the English language, using computerized databases, including Medline, CINAHL, SportDiscus, Embase, and Web of Science. The search combined key words related to T2DM (i.e. type 2 diabetes; diabetes; glycaemic control; glucose; insulin; insulin sensitivity; insulin resistance); exercise (i.e. exercise; physical activity; training; resistance training; weight training; strength training; aerobic training; muscle; endurance; fitness); and ethnicity (i.e. ethnicity; race; native; aboriginal; African; Hispanic; Latino; Pacific; Maori; Polynesian; Asian; Indian; Chinese). Reference lists of retrieved articles were screened for other additional relevant trials.

2.3. Assessment of research quality

Research quality was evaluated according to the Delphi List [27] for assessing the quality of RCTs, and the assessment was extended to uncontrolled trials as appropriate. An additional quality variable considered was supervision of exercise training sessions.

3. Results

3.1. Studies retrieved and assessment of research quality

The search resulted in 10 publications presenting the findings of nine trials, including four uncontrolled trials [28–31] and five RCTs [32–37]. A summary of the study quality assessment is presented in Table 1.

Of the nine studies retrieved, only two uncontrolled trials have compared exercise-induced adaptations between ethnic groups [28,29]. These trials compared Arabian versus Caucasian [28] and African versus Caucasian [29]. An other uncontrolled trial compared two exercise modalities in an all Polynesian cohort [30], while an additional uncontrolled trial involved a single treatment group design with repeated measures collected before and after training in an Indian cohort [31]. The main limitation of all uncontrolled trials [28–31] was the lack of an appropriate comparison group, including a non-exercise control group and/or a placebo group.

Four RCTs compared one or more exercise groups to a control group [32–36] while one RCT compared exercise to placebo (sham exercise, i.e. relaxation training) [37]. Only one of four RCTs, published as two articles, met all Delphi List quality criteria [32,33]. Common deficiencies of other trials primarily occurred in the reporting of randomization concealment [34–36], blinded assessment of outcome measures [34–36], and the use of an intention-to-treat statistical analysis procedure [34–37]. Only one RCT, the largest trial reviewed, provided a power calculation of the primary

Table 1 – Summary of research quality.

Citation	Randomization performed?	Treatment allocation concealed?	Groups similar at baseline regarding important prognostic values?	Eligibility criteria specified?	Blinded outcome assessors?	Compliance to exercise reported?	Supervision of exercise sessions?	Dropouts reported?	Did the analysis include an intention-to-treat analysis?	Were point estimates and measures of variability presented for the primary outcome measures?	Were between groups statistics reported?
Uncontrolled trials											
Winnick et al. [29]	n/a	n/a	n/a	Yes	n/a	No	Not reported	No	No	n/a	n/a
Misra et al. [31]	n/a	n/a	n/a	Yes	n/a	Yes	Yes	No	No	n/a	n/a
Glans et al. [28]	n/a	n/a	n/a	Yes	n/a	Yes	Partial	Yes	No	n/a	n/a
Sukala et al. [30]	n/a	n/a	n/a	Yes	n/a	Yes	Yes	Yes	No	n/a	n/a
Randomized controlled trials											
Castaneda et al. [33] Brooks et al. [32]	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Sykes et al. [36]	Yes	Not reported	Yes	Yes	Not reported	Yes	Yes	Yes	No	Yes	Yes
Van Rooijen et al. [37]	Yes	Yes	Yes	Yes	Yes	Yes	Partial	Yes	No	Yes	Yes
Shenoy et al. [34]	Yes	Not reported	Not reported	Yes	Not reported	No	Yes	Yes	No	Yes	Yes
Sridhar et al. [35]	Yes	Not reported	Yes	Yes	Not reported	No	Yes	No	No	Yes	Yes

Table 2 – Outcomes of exercise intervention trials conducted in high-risk ethnic populations with T2DM.

Authors (year) Country	N	Study groups (*n*)	Ethnicity	Exercise intervention			Change in HbA1c and significant outcomes		
				Modality	Prescription	Duration	Variable	Change	*p* value
Uncontrolled trials									
Winnick et al. [29] (2008) USA	59	African: Exercise 1 (*n* = 12) Exercise 2 (*n* = 24) Caucasian: Exercise 1 (*n* = 8) Exercise 2 (*n* = 15)	African/Caucasian	RT AER	RT 10 reps, sets and intensity not defined AER Treadmill 30–40 min ~1000 kcal/wk 3×/week	8 weeks	Africans in RT: HOMA-IR (% change) Body mass index (% change)	 −19.2 −2.6	 ≤0.05* ≤0.05*
Misra et al. [31] (2008) India	30	Exercise (*n* = 30)	Indian	RT	6 machine-based exercises, 2 sets × 10 reps, Intensity not clearly described, 3×/week	12 weeks	HbA_{1c} (%) Insulin sensitivity (K_{ITT}) Total cholesterol (mmol/L) Triglycerides (mmol/L) VLDL cholesterol (mmol/L) Waist circumference (cm) Hip circumference (cm) Mid-thigh circumference (cm) Mid-arm circumference (cm) Central skinfolds (mm) Peripheral skinfolds (mm)	−0.5 +0.9 −0.4 −0.4 −0.4 −1.6 −1.8 −1.6 −1.2 −5.2 −4.6	≤0.001‡ ≤0.001‡ ≤0.003‡ ≤0.001‡ ≤0.003‡ ≤0.001‡ ≤0.001‡ ≤0.001‡ ≤0.001‡ ≤0.001‡ ≤0.001‡
Glans et al. [28] (2009) Sweden	32	Arabian (*n* = 18) Caucasian (*n* = 14)	Arabian/Caucasian	COMBO	Weeks 1–12 RT + AER, 6 weight stations plus 6 min cycling, 3×/week, Weeks 13–24 Cycling, Walking, 60–70% VO_{2max}, 45 min, 3×/week	24 weeks	Arabian: HbA_{1c} (%) Caucasian: Significant outcomes: HbA_{1c} Insulin sensitivity (*M* value)	 −0.1 −0.6 +0.9	 NS ≤0.05‡ ≤0.005‡
Sukala et al. [30] (2011) New Zealand	18	Exercise 1 (*n* = 9) Exercise 2 (*n* = 9)	Polynesian	RT AER	RT 8 machine-based exercises, 3 sets × 6–8 reps, 85% 1RM, 3×/week AER Cycle ergometer 40–60 min 3×/wk	16 weeks	RT group: HbA1c (% change) AER group: HbA1c (% change) Systolic blood pressure (mmHg) Diastolic blood pressure (mmHg) Triglycerides (mmol/L)	 −0.1 −0.0 −16.2 −4.7 +0.3	 0.86 0.60 ≤0.01‡,* ≤0.05‡ <0.01‡,*

Randomized controlled trials									
Castaneda et al. [33] (2002) Brooks et al. [32] (2007) United States	62	Exercise (n = 31) Control (n = 31)	Hispanic	RT	5 machine-based exercises, 3 sets × 8 reps, 80% of 1RM, 3×/week	16 weeks	HbA_{1c} (%) HOMA-IR Trunk fat mass (kg) Whole body fat-free mass (kg) Systolic blood pressure (mmHg)	−1.1 −1.8 −0.7 +1.2 −9.7	≤0.01[#] ≤0.05[#] ≤0.01[#] ≤0.04[#] ≤0.05[#]
Shenoy et al. [34] (2009) India	30	Exercise 1 (n = 10) Exercise 2 (n = 10) Control (n = 10)	Indian	RT AER	RT 7 machine-based exercises, 3 sets × 10 reps, 60–100% 1RM, 2×/week AER Walking Intensity not stated, 3×/wk	16 weeks	RT group: HbA_{1c} (%) Systolic blood pressure (mmHg) Diastolic blood pressure (mmHg) AER group: HbA_{1c}(%)	 −1.9 −9.0 −8.0 −1.3	 ≤0.01[#] ≤0.01[#] ≤0.01[#] ≤0.001
Sridhar et al. [35] (2010) India	105	Exercise (n = 55) Control (n = 50)	Indian	AER	Cycle Ergometer or Treadmill, ≤45 min, 60% HR_{max}, 5×/wk	52 weeks	Significant outcomes in Ex group: HbA_{1c} (%) Systolic blood pressure (mmHg) Diastolic blood pressure (mmHg)	 −2.2 −8.7 −5.8	 ≤0.001[‡] ≤0.01[‡] ≤0.01[‡]
Sykes et al. [36] (2004) Hong Kong	36	Exercise (n = 24) Control (n = 12)	Chinese	AER	Treadmill, cycle, step, arm ergometer, 50–70% HRR, 45 min, 1×/wk	12 weeks	HbA_{1c} (%) Body mass (kg) Body mass index (kg/m^2) HDL cholesterol (mmol/L)	−0.4 −0.4 −0.2 +0.1	NS ≤0.045[‡] ≤0.043[‡] ≤0.002[‡]
Van Rooijen et al. [37] (2004) South Africa	149	Exercise (n = 75) Control (n = 74)	African	AER	Walking, Borg RPE scale = 12–14, 45 min, 5–7×/week	12 weeks	HbA_{1c} (%)	−0.4	0.052

AER, aerobic training; COMBO, aerobic training plus resistance training; HbA_{1c}, hemoglobin A_{1c}; HDL, high density lipoprotein; HOMA-β, homeostasis model assessment-beta cell; HOMA-IR, homeostasis model assessment-insulin resistance; HRR, heart rate reserve; K_{ITT}, disappearance of glucose per unit time insulin tolerance test; LDL, low density lipoprotein; RT, progressive resistance training; VLDL, very low density lipoprotein.

[*] Significant change over time versus comparison group(s).

[‡] Significant versus baseline values within group.

[#] Significant versus control group only.

outcome a priori [37]. The RCTs generally conducted appropriate statistical analyses (i.e. group × time interaction) [32–37], and reported complete [32–36] or partial supervision [37] of exercise intervention(s).

3.2. Participants

Cohorts, interventions and clinical outcomes are presented in Table 2.

3.2.1. Sample size

Five hundred and twenty-one ($n = 521$) participants were included in the final data analyses in the nine trials reviewed. The sample sizes ranged from 18 [30] to 149 participants [37]. Five of nine trials (56%) included fewer than 36 participants [28,30,31,34,36], while only two RCTs (22%) included over 100 participants [35,37].

3.2.2. Ethnicity

Indian [31,34,35], African [29,37], Chinese [36], Latino (Hispanic) [32,33], Arabian [28], and Polynesian [30] cohorts were included in the nine trials reviewed. As mentioned, an ethnically distinct comparison group (i.e. Caucasian) was included in only two trials [28,29].

3.2.3. Gender

Three hundred and twenty-eight women and 134 men were included across eight trials that specified gender [28,30–37]. The largest trial was comprised exclusively of women [37]. One trial did not specify gender [29]. However, subsequent contact with the authors confirmed the inclusion of 35 women and 24 men [29].

3.2.4. Age

Age was expressed as mean ± SD in 8 trials [28–36], and ranged from 41 ± 8 years [31] to 66 ± 2 years [32,33]. One trial provided the mean age but no standard deviation [37]. In studies that mentioned an age range [29–31,34,37], the youngest and oldest participants enrolled were 24 years [31] and 70 years [34], respectively.

3.2.5. Duration of diabetes

Six trials [28,30,32–36] reported the time from diagnosis of T2DM to study enrolment, which ranged from 3 ± 3 years [30] to 11 ± 1 years [32,33]. Three trials listed a minimum duration of T2DM as an entry criterion (>6 months to >3 years) [32–34,37].

3.2.6. Diabetes management

Approximately 76% of participants were prescribed oral hypoglycaemic agents and 27% were receiving insulin injections across six trials that detailed pharmacotherapy [28,30,32–35,37]. Five trials reported on the number of participants receiving non-pharmacological therapy [28,30,32–34,37]. Across these trials, approximately 5% of participants were managing their diabetes with diet only [28,30,32–34,37].

3.2.7. Comorbidities and complications

The prevalence of hypertension and ischaemic heart disease ranged from 23.8% to 62.4% [28,32,33,35,37] and 6% to 59.7% [28,32,33], respectively, in trials reporting these data. In other trials, the average baseline clinical data or pharmacological information gave an indication of underlying hypertension [29,30,34] and hyperlipidaemia [30,31,36]. Only one trial provided a breakdown of active diabetes-related complications (i.e. neuropathy, retinopathy, micro- and macroalbuminuria) [28], while most investigations excluded such patients [29–31,34,36,37].

3.2.8. Participant attrition

Five trials reported the number of participants unavailable for follow-up testing [28,30,32–34,37]. Of the participants enrolled in these trials ($n = 299$), only 8% ($n = 25$) were lost to follow-up. Only four trials specified reasons for dropout, including: no reason given, time constraints, pregnancy, bone fracture, medical reason unrelated to the study, and death [30,32–34,37].

3.3. Exercise interventions

3.3.1. Duration

Exercise training interventions ranged from 8 weeks [29] to 52 weeks [35], with the majority of trials prescribing 12–24 weeks of exercise (Table 2) [28,30–34,36,37]. Only one trial prescribed greater than 24 weeks of training (i.e. 52 weeks) [35].

3.3.2. Frequency

Frequency of training ranged from one [36] to seven sessions per week [37], with the majority of trials prescribing three to five sessions per week [28–35].

3.3.3. Modality

An aerobic training group was employed in six trials [29,30,34–37] and consisted of walking, leg and arm cycle ergometry, and stepping exercise. A resistance training group was employed in five trials [29–34]. These trials prescribed 5–8 machine-based exercises targeting the upper and lower body. One trial prescribed a combination of aerobic and resistance training for the first 12 weeks of training [28]. The aerobic training was performed on a cycle while the resistance training involved 6 machine-based exercises [28]. The following 12 weeks of training for this cohort involved aerobic exercise only (i.e. cycling and walking) [28].

3.3.4. Aerobic training intensity

Aerobic training intensity was defined in four trials according to percentage of maximal oxygen consumption [28], percentage of heart rate reserve [30,36], or percentage of maximal heart rate [37]. These prescriptions ranged from 50% to 85% of maximum intensity. Two trials did not specify aerobic training intensity [29,34].

3.3.5. Aerobic training duration

Aerobic exercise sessions ranged from 30 to 60 min [28–30,34–37] with three trials explicitly mentioning warm-up and cool-down components included within [28] or in addition to [35,36] the total exercise time.

3.3.6. Resistance training intensity and volume

Resistance training intensity was defined in four studies, generally at moderate-to-high-intensity, two to three sets at

6–10 repetitions per set [30–34]. Intensity was not clearly defined in two trials [28,29]. Six studies reported the number of resistance exercises performed, which ranged from four to eight [28–34]. Glans et al. [28] mentioned that each resistance exercise was performed for a 3 min duration, suggesting that the exercises were low intensity, hence, likely more cardiovascular in nature.

3.3.7. *Supervision*

Six trials involved direct supervision of exercise sessions by research staff [28,30–33,35,36]. Van Rooijen et al. [37] prescribed unsupervised walking plus supervised fortnightly sessions to educate and reinforce home-based exercise guidelines. Two studies were unclear regarding the level of supervision provided [29,34].

3.3.8. *Compliance*

Four trials reported participant compliance as percentage of exercise sessions attended [28,30,32,33,36]. Attendance ranged 67–91% across these trials [28,30,32,33,36].

3.3.9. *Evidence of training effect*

Two trials reported exercise-induced improvements in functional capacity evaluated via 6-min walk test [36,37]. Ventilatory threshold was shown to improve in Swedish but not Arabian participants secondary to 24 weeks of exercise intervention, however other parameters of graded exercise test performance (i.e. VO_{2max} and maximum workload) remained unchanged in either group [28,36]. Measures of muscular strength were evaluated and improved in two trials secondary to resistance training [32,34] and aerobic training [34]. Three studies did not measure physical fitness or functional adaptation to training [29,31,35].

3.4. *Outcomes*

3.4.1. *Adverse events*

Four trials provided information on adverse events related to exercise participation [30,32–34]. Castaneda et al. [32,33] reported five hypoglycaemic events and three episodes of chest pain. Sukala et al. [30] reported one episode of uncomplicated syncope. All events were managed and resolved conservatively, without complication, and none resulted in participant drop-out. Van Rooijen et al. [37] and Shenoy et al. [34] attributed no adverse events to exercise training.

3.4.2. *Glycosylated hemoglobin (HbA1c)*

Only one trial of the nine trials reviewed did not evaluate HbA1c as an outcome measure [29]. Of the remaining trials, statistically significant reductions in HbA1c (−0.4% to −2.2%) were noted in trials secondary to resistance training [31–34], and aerobic training [34,35,37]. The largest improvements in HbA1c were noted in Latino and Indian participants prescribed resistance training or aerobic training of greater than 16 weeks duration [32–35]. Glans et al. [28] noted that 12 weeks of combined aerobic plus resistance training followed by 12 weeks of aerobic training only significantly reduced HbA1c in their Caucasian participants but not in their Arabian participants; the difference between ethnic groups was significant ($p < 0.05$).

Van Rooijen et al. [37] noted an improvement in HbA1c over time in their African participants prescribed 12 weeks of home-based aerobic training. However, this treatment effect did not achieve statistical significance versus sham relaxation training ($p = 0.052$). HbA1c remained unchanged in trials prescribing 12 and 16 weeks of aerobic training in Chinese [36] and Polynesian [30] participants, respectively. The latter trial [30] also noted no change in HbA1c secondary to 16 weeks of resistance training.

3.4.3. *Insulin action*

Measures of insulin sensitivity were quantified by various methods in six trials [28–33,36]. Winnick et al. [29] documented significantly reduced insulin resistance, measured via homeostasis model assessment (HOMA-IR) in African–American versus Caucasian participants prescribed eight weeks of resistance training ($p < 0.05$). The African participants performing resistance training also showed a trend toward improved pancreatic β-cell function (HOMA-β) versus the Caucasian participants ($p < 0.10$) [29]. By contrast, eight weeks of aerobic training did not improve measures of insulin resistance or pancreatic β-cell function within or between these ethnic groups secondary to eight weeks of aerobic training.

Brooks et al. [32] reported a reduction in HOMA-IR, from 7.1 ± 5.7 to 5.3 ± 5.5, in their Latino participants randomized to 16 weeks of resistance training versus control ($p < 0.05$). However, these findings were not supported by other trials that prescribed aerobic training [30,36] or resistance training [30] in other ethnic cohorts.

Misra et al. [31] measured insulin sensitivity by using the short insulin tolerance test (K_{ITT}) and reported an increase in this marker secondary to 12 weeks of resistance training in their participants. Glans et al. [28] determined insulin sensitivity from glucose infusion rates during the last 60 min of the euglycaemic insulin clamp (*M*-value) expressed as glucose uptake per kilogram of body weight. Within groups analyses revealed that the Caucasian participants significantly improved this marker at 6-months whereas the Arabian participants did not, while between group comparisons did not achieve statistical significance [28].

3.4.4. *Anthropometrics*

At baseline, body mass index (BMI) ranged from 24.1 to 45.0 kg/m^2 across all trials. In one trial, which included visceral adiposity was an entry criterion, the mean BMI indicated Class III obesity (43.8 ± 9.5 kg/m^2) [30]. Winnick et al. [29] reported a significant reduction of BMI in African participants versus Caucasian participants engaging in eight weeks of resistance training ($p < 0.05$). However, no changes in BMI were noted secondary to aerobic training in either ethnic group [29]. Waist-to-hip ratio and body fat percentage did not change in either African or Caucasian participants secondary to aerobic or resistance training. Sykes et al. [36] noted a significant reduction in BMI in an Chinese cohort performing 12 weeks of aerobic training. The change in BMI was concomitant with a significant reduction of body mass ($p = 0.045$), but no change in body fat percentage, waist circumference, hip circumference or waist-to-hip ratio [36].

Two uncontrolled trials [28,30] reported no significant change in BMI or body composition outcomes (i.e. fat-free

mass, waist circumference or body fat percentage) secondary to chronic aerobic [30], resistance [30] or combined exercise training [28] in obese Polynesian [30], Middle-Eastern and Caucasian participants with T2DM [28]. In a separate uncontrolled trial, Misra et al. [31] reported no statistically significant changes in BMI, waist-to-hip ratio, or whole body or regional fat and fat-free mass in their Indian cohort secondary to 12 weeks of resistance training. However, interestingly, the authors did report statistically significant reductions in several circumference and skinfold measures. Van Rooijen et al. [37] noted no significant difference in BMI between 12 weeks of aerobic exercise and sham relaxation training in a cohort of African participants ($p = 0.28$).

Castaneda et al. [33] reported a significant decrease in visceral fat ($p = 0.01$), an increase in whole body fat-free mass ($p = 0.04$), and a trend toward increased trunk fat-free mass ($p = 0.08$), determined via dual energy X-ray absorptiometry (DEXA), after 16 weeks of resistance training versus control in older (aged $\geq$55 years) Latino participants. The authors also noted a 2.2 cm reduction in waist circumference in the exercising participants; however, this finding did not achieve statistical significance ($p = 0.07$) [33].

3.4.5. *Blood lipids*

Four studies evaluated changes in blood lipid profiles [30–33,36]. Misra et al. [31] determined that 12-weeks of resistance training induced statistically significant reductions in total cholesterol and triglycerides and very low density lipoprotein (VLDL) cholesterol, while low-density lipoprotein (LDL) and high-density lipoprotein (HDL) cholesterol remained unchanged. Sykes et al. [36] noted a significant increase in HDL cholesterol, but no change in triglycerides, LDL, or total cholesterol secondary to once weekly aerobic training in a Chinese cohort. Castaneda et al. [32,33] reported a trend toward reduced triglycerides ($p = 0.08$) secondary to 16 weeks of resistance training in their Latino cohort, but noted no change in total cholesterol, LDL, or HDL cholesterol. Sukala et al. [30] reported that total, HDL, and LDL cholesterol remained unchanged in Polynesian participants performing 16 weeks of resistance or aerobic training. There was a significant increase in mean triglyceride levels in the aerobic training group ($p = 0.004$), which may have been due to unreported changes in diet, medication, or a possible regression to the mean.

3.4.6. *Haemodynamics*

Resistance training of 16 weeks proved to be beneficial in reducing systolic and/or diastolic blood pressure in Latino and Indian participants [32–34]. Two trials also showed a statistically significant reduction in these parameters in Polynesian [30] and Indian participants [35] following chronic aerobic training. Other studies showed no significant change in these measures following aerobic training [34,37] or did not consider measuring systolic or diastolic blood pressure.

4. Discussion

The disparities in T2DM prevalence and prognosis in high-risk ethnic groups are likely the result of the complex interaction between environmental, sociocultural, genetic, and physiological factors. The present systematic review suggests that exercise training can significantly improve chronic glucoregulation (HbA1c) in some ethnic populations [31–35]. The improvement of HbA1c has been concomitant with improvements in insulin action [32,33], body composition [31–33], blood lipid profile [31], and haemodynamic parameters [34,35]. These adaptations have been shown to occur without life-threatening adverse events, including myocardial infarction, cerebrovascular accident or death. Accordingly, the trials reviewed [31–35] support current position statements advocating the use of aerobic and/or resistance training to mitigate T2DM [11], and provide impetus for further research and application in high-risk ethnic populations.

The largest reductions in HbA1c (-1.1 to -2.2%) were noted secondary to aerobic and resistance training prescribed for a minimum of 16 weeks [33–35]. The practical significance of reduced HbA1c have been noted in the United Kingdom Prospective Diabetes Study [38], in which each 1% reduction in HbA_{1c} resulted in a 14% reduced risk of myocardial infarction, a 37% reduced risk of microvascular complications, and a 21% reduced risk of disease-specific mortality. Physiologically, the reduction in HbA1c is likely mediated by exercise-induced augmentation of whole body composition, particularly the reduction of intramyocellular triglyceride [39,40], which can enhance intracellular, insulin-dependent mechanisms of glucose uptake [41].

The lack of improvement of HbA1c and several body adiposity measures in a Chinese cohort studied by Sykes et al. [36] was likely due to an insufficient stimulus. The researchers prescribed only one training session per week for 12 weeks whereas the majority of trials prescribed a minimum of three sessions per week and a longer training duration. In another trial, Van Rooijen et al. [37] noted a trend toward reduced HbA1c ($p = 0.052$) and no change in BMI or systolic and diastolic blood pressure in African women randomized to perform 45 min of daily unsupervised walking for 12 weeks. Exercise logbooks completed by participants revealed poor compliance with exercise recommendations and may therefore have resulted in a less than optimal training and adaptation.

Sukala et al. [30] found no significant change in HbA1c secondary to aerobic or resistance training in a Polynesian cohort suffering from T2DM and morbid obesity (BMI = 43.8 ± 9.5 kg/m^2). Further, insulin sensitivity, blood lipids and adiposity measures were not favourably altered in either training group. The lack of adaptation could have been due to less than optimal attendance and the level of obesity in the cohort, which may have been so extreme as to override the potential benefits of 16 weeks of exercise training. Sukala et al. [30] suggested that a greater frequency and duration of training may have reduced fat mass and/or increased fat-free mass sufficiently to improve HbA1c. Future investigations should prescribe at least three exercise sessions per week for 16 weeks or longer, dependent on initial level of adiposity, provide adequate supervision, and report detailed compliance data in order to identify contributing factors which may explain enhanced or decreased efficacy of specific exercise interventions.

Sukala et al. [30] also postulated that Polynesian people may be able to tolerate and respond more favourably to higher doses of exercise versus other ethnic cohorts, given that their genotype expression has been influenced by extreme levels of

physical activity for daily living, food procurement, dance and rituals, and inter-island travel [42]. RCTs stratifying by ethnicity, and contrasting various exercise interventions, are required to investigate this hypothesis [30]. Sigal et al. [43] have shown that multimodal exercise interventions are more effective than isolated modalities, and such interventions may be more suitable to adapt HbA1c and other clinical outcomes in an obese Polynesian cohort.

To date, only two trials have compared the exercise training response between different ethnic cohorts [28,29]. Glans et al. [28] reported a statistically significant 0.6% reduction in HbA_{1c} and a 41% increase in insulin sensitivity in their Caucasian participants, but no significant change in their Arabian participants. The lack of adaptation in the Arabian group may be attributed to the fact that these participants did not progress their training intensities (i.e. workloads and heart rate) as rapidly as the Caucasian participants [28]. The authors speculated that cultural differences may potentially explain these findings. Sociocultural norms have been identified as a barrier to exercise participation in Arabian women, for example [44]. Ali et al. [44] suggest that culturally sensitive exercise facilities could potentially be used to facilitate exercise adoption, progression, and physiological adaptation. Such considerations may enhance future investigations with this and other ethnic cohorts that have cultural taboos surrounding exercise participation.

In another study comparing exercise modality by ethnicity, Winnick et al. [29] reported a significant reduction in BMI and HOMA-IR scores and a trend toward improved β-cell function in African participants receiving eight weeks of resistance training compared to Caucasian subjects receiving identical training volumes. Africans exhibit higher levels of insulin resistance than their Caucasian counterparts [20]. Therefore, the preferential reduction in insulin resistance (HOMA-IR) and improved insulin secretory capacity (β-cell function) in Africans are important findings [29]. These findings suggest that similar exercise modalities may exert differential physiological effects in different ethnic groups and warrant further investigation with larger-scale intervention trials.

The statistically significant reductions in systolic [33,34] and diastolic blood pressure [34] in Latino [33], Indian [34,35] and Polynesian [30] participants after exercise training are also clinically important findings. Patients with T2DM have a two- to four-fold increased risk of adverse cardiovascular events [45]. Hence, any regimen that can reduce hypertension in this patient population is desirable. A 2 mmHg reduction of systolic or diastolic blood pressure translates to a reduction in risk of coronary artery disease and stoke by 6–17% in the general population [46]. Four trials reviewed evaluated changes in blood lipid profiles and the findings were equivocal [30–33,36]. Confounding variables such as use of statins and diet may have impacted on the outcomes and hence need to be evaluated more rigorously in future trials. Further, baseline values may have been within normal limits, due to medication usage, hence limiting the adaptability of these outcome measures. Sub-group analyses in future large-scale investigations should be conducted to evaluate the effect of exercise training on underlying dyslipidaemia.

Although the available literature generally supports the prescription of exercise training in high-risk ethnic cohorts with T2DM, many methodological limitations exist within this body of literature and many research questions remain to be investigated. Effectively designed RCTs prescribing appropriate, targeted interventions and investigating a broad spectrum of relevant outcome measures may be required to stimulate greater advocacy for exercise as an essential therapeutic adjunct for diabetes management in these populations. It is also essential that the exercise stimulus is evaluated with appropriate physical fitness and functional testing. These investigations should be extended to other high-risk populations, particularly indigenous peoples who suffer an extreme burden of T2DM [6]. The translation of research investigations into clinical application should remain the overall objective.

Only five trials to date have involved randomization of participants to exercising and non-exercising group [32,33]. Three of these trials were of larger scale ($n = 62–149$) and therefore may have been adequately powered to test the state hypotheses. The RCTs by Castaneda et al. [32,33] and Van Rooijen et al. [37] were methodologically robust according to the current standards of reporting [27] and provide guidance for the development of future trials. Findings of the small-scale RCTs [34,36], and uncontrolled trials [28–31] should be considered for the development of larger scale, robustly designed RCTs in various ethnic populations that should be studied independently and comparatively.

Although this systematic review is limited by the small number of published exercise trials in high-risk ethnic groups with T2DM, it provides the necessary starting point to facilitate discussions and prompt additional research in this area. A greater understanding of the physiological impact of exercise on glycaemic control and associated clinical outcomes may be useful in establishing targeted evidence-based exercise prescription guidelines which may lead to enhanced diabetes management strategies and reduced morbidity and mortality in high risk ethnic groups with T2DM.

Conflict of interest

There are no conflicts of interest.

REFERENCES

[1] Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care 2004;27:1047–53.
[2] Paulweber B, Valensi P, Lindstrom J, Lalic N, Greaves C, McKee M, et al. A European evidence-based guideline for the prevention of type 2 diabetes. Horm Metab Res 2010;42:S3–6.
[3] Shai I, Jiang R, Manson J, Stampfer M, Willett W, Colditz G, et al. Ethnicity, obesity, and risk of type 2 diabetes in women. Diabetes Care 2006;29:1585–90.
[4] Mokdad A, Bowman B, Ford E, Vinicor F, Marks J, Koplan J. The continuing epidemics of obesity and diabetes in the United States. J Am Med Assoc 2001;286:1195–200.
[5] Zimmet P. Globalization, coca-colonization and the chronic disease epidemic: can the doomsday scenario be averted. J Intern Med 2000;247:301–10.
[6] Anderson I, Crengle S, Kamaka M, Chen T, Palafox N, Jackson-Pulver L. Indigenous health in Australia, New Zealand, and the Pacific. Lancet 2006;367:1775–85.

[7] Ministry of Health. A portrait of health: key results of the 2006/2007 New Zealand Health Survey. Wellington; 2008.
[8] Burrows N, Geiss L, Engelgau M, Acton K. Prevalence of diabetes among Native Americans and Alaska Natives. Diabetes Care 2000;23:1786–90.
[9] Harjo T, Perez A, Lopez V, Wong N. Prevalence of diabetes and cardiovascular risk factors among California adults compared to other ethnicities. Metab Syndr Relat Disord 2011;9:49–54.
[10] The health and welfare of Australia's Aboriginal and Torres Strait Islander peoples 2005. Canberra: Australian Bureau of Statistics and AIHW; 2005.
[11] Albright A, Franz M, Hornsby G, Kriska A, Marrero D, Ullrich I, et al. Exercise and type 2 diabetes. Med Sci Sports Exerc 2000;32:1345–60.
[12] American Diabetes Association. Physical activity/exercise and diabetes. Diabetes Care 2004;27:S58–62.
[13] Warburton D, Nicol C, Bredin S. Health benefits of physical activity: the evidence. Can Med Assoc J 2006;174:801–9.
[14] Eves N, Plotnikoff R. Resistance training and type 2 diabetes. Diabetes Care 2006;29:1933–41.
[15] Snowling N, Hopkins W. Effects of different modes of exercise training on glucose control and risk factors for complications in type 2 diabetic patients. Diabetes Care 2006;29:2518–27.
[16] Thomas D, Elliott E, Naughton G. Exercise for type 2 diabetes mellitus. Cochrane Database Syst Rev 2006;19:3. Art. No.: CD002968.
[17] Abate N, Chandalia M. The impact of ethnicity on type 2 diabetes. J Diabetes Complications 2003;17:39–58.
[18] Raschke V, Cheema B. Colonisation, the new world order and the eradication of traditional food habits in East Africa. Public Health Nutr 2008;11:662–74.
[19] Osei K, Schuster D. Effects of race and ethnicity on insulin sensitivity, blood pressure, and heart rate in three ethnic populations: comparative studies in African–Americans, African immigrants (Ghanaians), and white Americans using ambulatory blood pressure monitoring. Am J Hypertens 1996;9:1157–64.
[20] Osei K, Schuster D, Owusu S, Amoah A. Race and ethnicity determine serum insulin and c-peptide concentrations and hepatic insulin extraction: comparative studies of three populations in West African ancestry and White Americans. Metabolism 1997;46:53–8.
[21] Glans F, Elgzyri T, Shaat N, Lindholm E, Apelqvist J, Groop L. Immigrants from the Middle-East have a different form of type 2 diabetes compared with Swedish patients. Diabet Med 2008;25:303–7.
[22] Misra A, Vikram N. Insulin resistance syndrome (metabolic syndrome) and obesity in Asian Indians: evidence and implications. Nutrition 2004;20:482–91.
[23] UK Prospective Diabetes Study Group. UK Prospective Diabetes Study X. Differences between Asian, Afro-Caribbean and white Caucasian type 2 diabetic patients at diagnosis of diabetes. Diabet Med 1994;11:670–7.
[24] Raschke V, Elmadfa I, Bermingham M, Steinbeck K. Low density lipoprotein subclasses in Asian and Caucasian adolescent boys. Asia Pac J Clin Nutr 2006;15:496–501.
[25] Whincup P, Gilg J, Papacosta O, Seymour C, Miller G, Alberti K, et al. Early evidence of ethnic differences in cardiovascular risk: cross sectional comparison of British South Asian and white children. Br Med J 2002;324:1–6.
[26] Reynolds T, Smellie W, Twomey P. Glycated haemoglobin (HbA1c) monitoring. Br Med J 2006;333:586–8.
[27] Verhagen A, de Vet H, de Bie R, Kessels A, Boers M, Bouter L, et al. The Delphi list: a criteria list for quality assessment of randomized clinical trials for conducting systematic reviews developed by Delphi consensus. J Clin Epidemiol 1998;51:1235–41.
[28] Glans F, Eriksson K, Segerstrom A, Thorsson O, Wolmer P, Groop L. Evaluation of the effects of exercise on insulin sensitivity in Arabian and Swedish women with type 2 diabetes. Diabetes Res Clin Pract 2009;85:69–74.
[29] Winnick JGT, Gaillard T, Schuster D. Resistance training differentially affects weight loss and glucose metabolism of White and African American patients with type 2 diabetes mellitus. Ethn Dis 2008;18:152–6.
[30] Sukala W, Page R, Rowlands D, Krebs J, Lys I, Leikis M, et al. South Pacific Islanders resist type 2 diabetes: comparison of aerobic and resistance training. Eur J Appl Physiol 2008;112:317–25.
[31] Misra A, Alappan N, Vikram N, Goel K, Gupta N, Mittal K, et al. Effect of supervised progressive resistance exercise training protocol on insulin sensitivity, glycemia, lipids, and body composition in Asian Indians with type 2 diabetes. Diabetes Care 2008;31:1282–7.
[32] Brooks N, Layne J, Gordon P, Roubenoff R, Nelson M, Castaneda-Sceppa C. Strength training improves muscle quality and insulin sensitivity in Hispanic older adults with type 2 diabetes. Int J Med Sci 2007;4:19–27.
[33] Castaneda C, Layne J, Muñoz-Orians L, Gordon P, Walsmith J, Foldvari M, et al. A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. Diabetes Care 2002;25: 2335–41.
[34] Shenoy S, Arora E, Jaspal S. Effects of progressive resistance training and aerobic exercise on type 2 diabetes in Indian population. Int J Diabetes Metab 2009;17:27–30.
[35] Sridhar B, Haleagrahara N, Ramesh B, Kulur A, Avabratha S, Adhikary P. Increase in the heart rate variability with deep breathing in diabetic patients after 12-month exercise training. Tohoku J Exp Med 2010;220:107–13.
[36] Sykes K, Yeung T, Ko G. A 12-week prospective randomized controlled trial to investigate the effects of aerobic training on type 2 diabetes patients. Am J Recreation Ther 2004;3:36–42.
[37] Van Rooijen A, Rheeder P, Eales C, Becker P. Effect of exercise versus relaxation on haemoglobin A1c in black females with type 2 diabetes mellitus. Q J Med 2004;97:343–51.
[38] Stratton I, Adler A, Neil H, Matthews D, Manley S, Cull C, et al. Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. Br Med J 2000;321:405–12.
[39] Stannard S, Holdaway M, Sachinwalla T, Cunningham C. Insulin sensitivity and intramyocellular lipid concentrations in young Maori men. Diabet Med 2007;24:1205–12.
[40] Stannard S, Johnson N. Energy well spent fighting the diabetes epidemic. Diabetes Metab Res Rev 2006;22:11–9.
[41] Rice B, Janssen I, Hudson R, Ross R. Effects of aerobic or resistance exercise and/or diet on glucose tolerance and plasma insulin levels in obese men. Diabetes Care 1999;22:684–91.
[42] Price W. Nutrition and physical degeneration. La Mesa, CA: Price-Pottenger Nutrition Foundation; 1939.
[43] Sigal R, Kenny G, Boulé N, Wells G, Prud'homme D, Fortier M, et al. Effects of aerobic training, resistance training, or both on glycemic control in type 2 diabetes. Ann Intern Med 2007;147:357–69.
[44] Ali H, Baynouna L, Bernsen R. Barriers and facilitators of weight management: perspectives of Arab women at risk for type 2 diabetes. Health Soc Care Community 2010;18:219–28.
[45] Kannel W, McGee D. Diabetes and cardiovascular disease: the Framingham Study. J Am Med Assoc 1979;241:2035–8.
[46] Pescatello L, Franklin B, Fagard R, Farquhar W, Kelley G, Ray C, et al. American College of Sports Medicine position stand. Exercise and hypertension. Med Sci Sports Exerc 2004;36:533–53.