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Review

Resistance training improves metabolic health in type 2 diabetes: A systematic review

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ABSTRACT

This paper systematically reviews the effect of resistance training (RT) on glycemic control and insulin sensitivity in adults with type 2 diabetes.

Twenty studies were included, with the volume, frequency and intensity of RT varying markedly. Supervised RT improved glycemic control and insulin sensitivity, however, when supervision was removed compliance and glycemic control decreased. Evidence indicates the mechanisms behind the improvements to glucose tolerance require further elucidation.

Although research demonstrates apparent benefits of RT for individuals with diabetes, further research is required to elucidate the minimum effective dose by describing frequency, intensity and the duration of acute and chronic improvements.

Contents

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Abbreviations: RT, resistance training; AT, aerobic training; CT, aerobic plus resistance training (combined training); RCT, randomized controlled trial; 1RM, one repetition maximum; 3RM, three repetitions maximum; HbA1c, glycosylated haemoglobin; OGTT, oral glucose tolerance test; AUC, area under the curve; FBG, fasting blood glucose; HOMA, homeostasis model assessment; ACSM, American College of Sports Medicine; ADA, American Diabetes Association; WHO, World Health Organization; GLUT4, glucose transporter 4; kcal/wk, kilocalories per week; LBM, lean body mass; GDR, glucose disposal rate.

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1. Introduction

The world-wide incidence of type 2 diabetes continues to increase [1] however, despite exercise being promoted as a vital part of the treatment process, exercise prescription does not vary between prevention and treatment. For individuals with existing diabetes, specific benefits of exercise include increased insulin sensitivity, improved glycemic control [2,3], improved lipid profile and lower blood pressure [3]. Importantly, individuals with diabetes completing exercise training using various exercise modes for between 8 weeks and 12 months have experienced decreased HbA1c by clinically significant levels (0.6%), improved insulin sensitivity and reduced serum triglycerides [4].

The American College of Sports Medicine (ACSM) endorses exercise as a treatment method for people with type 2 diabetes and currently recommends expending a minimum cumulative total of 1000 kcal/wk of energy from aerobic activities [5]. The American Diabetes Association (ADA) has similar recommendations for at least 150 min per week of moderate intensity aerobic physical activity and/or 90 min per week of vigorous aerobic exercise [6]. Accordingly, aerobic exercise has been the major focus for exercise-training studies due to consistent findings of improved glucose control [7,8], however long-term compliance to these recommendations remains low [9] necessitating the investigation of an effective strategy to improve adherence rates.

More recently, resistance training has been the focus of increased research and is suggested to improve glycemic control and insulin sensitivity partially via similar mechanistic pathways to aerobic training [10], and partially through discrete pathways providing additive insulin signalling benefits. The focus on resistance training is in part due to a recognition that individuals with type 2 diabetes, who are also likely to be obese or suffering from other co-morbidities, are likely to struggle to achieve the volume and intensity of aerobic training that is required to be effective [10,11], and therefore compliance to resistance training may be higher. Both the ACSM and the ADA have now included resistance training in their exercise prescription guidelines for younger individuals with type 2 diabetes and for older individuals with type 2 diabetes free of contraindications. The recommendations are; one set of 10–15 repetitions for 8–10 exercises twice a week [5] and, progressing to three sets of 8–10 repetitions three times a week [6]. These recommendations have largely been based on information regarding healthy individuals and the few [12–15] randomized controlled trials of resistance training in individuals with type 2 diabetes completed at the time that they were published. However, it should be noted that significant improvements to insulin sensitivity in healthy individuals have been reported only when resistance training was performed three or more days a week [16] and the responses of individuals with diabetes may differ. It is therefore the purpose of this paper to systematically review the literature on the effects of resistance training on the diabetes markers of glycemic control and insulin sensitivity in individuals with type 2 diabetes.

2. Methods

2.1. Search strategy

Ovid MEDLINE (1950 to August week 3, 2008), Ovid MEDLINE In-Process (September 02, 2008), OLD MEDLINE (1950–1965), CINAHL (1982 to August week 5, 2008) and EMBASE (1980 to 2008 week 35) electronic databases were searched on September 03, 2008. First, three keyword and categorical searches were performed (i) 'diabetes', or 'diabetes mellitus', or 'type 2 diabetes mellitus'; (ii) 'weight lifting', or 'resistance training', or 'strength training', or 'weight training', or 'progressive resistance training', or 'circuit training'; (iii) 'glucose intolerance', or 'blood glucose', or 'glucose', or 'glucose metabolism disorders', or 'glucose tolerance test', or 'insulin', or 'insulin resistance', or 'diabetes complications', or 'haemoglobin A', or 'glycosylated haemoglobin A', or 'HbA1c'. Second, categories i–iii were combined using 'and', limited to humans and reported in the English language with duplicates removed. In addition, reference lists of all publications meeting the inclusion criteria were manually searched to identify any relevant studies not found through electronic searching.

2.2. Inclusion and exclusion criteria

Studies that met the following criteria were included in this review: (i) published in English (ii) cohorts were adults above the age of 18 years with type 2 diabetes, (iii) a form of resistance training was included as an isolated intervention arm, (iv) it was an intervention study, (v) one diabetes marker (HbA1c, fasting glucose or insulin, insulin sensitivity) or an insulin signalling outcome were reported. Non-trial studies, review or opinion/editorial papers were excluded along with studies that did not report diabetes or insulin signalling markers or studies that investigated only individuals without diabetes. Interventions that combined resistance training with another intervention (aerobic training or diet) or did not involve ongoing training were also excluded.

2.3. Statistical analyses

To avoid misrepresentation of the presented data, a meta-analysis has not been conducted due to the methodological differences in terms of frequency and intensity of training, along with the number and type of exercises completed. Clinical significance has been interpreted as a 0.6% improvement in HbA1c [4]. Effect sizes were not calculated as only four papers included in the review provided enough information to enable effect size to be calculated.

3. Results

3.1. Search results

Twenty-four papers from 20 studies met the criteria and are included in this review. Search results are shown in Fig. 1. One doctoral dissertation was excluded, but its related publication identified and also excluded [17]. A paper reporting insulin sensitivity data was excluded [18] as this data had been

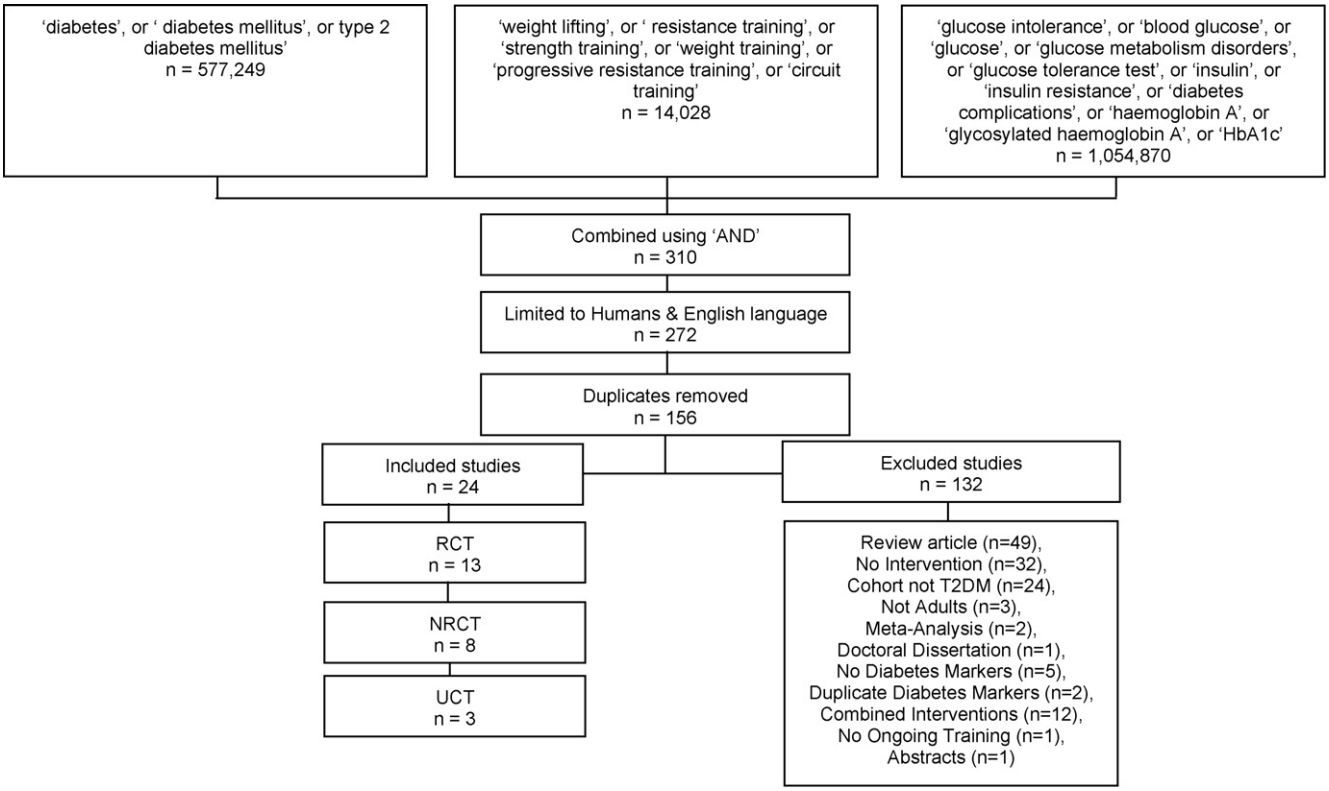

Fig. 1 – Sequence of searching and search results. RCT, randomized controlled trial; NRCT, non-randomized controlled trial; UCT, uncontrolled trial; combined interventions, aerobic and resistance training OR diet and resistance training.

Table 1 – Study quality.

Reference		Design					Subjects			Intervention			Compliance			Outcome Measures				
	Publication Date	Study Design	Randomization	Concealment	Assessor Blinding	Participant Blinding	Eligibility Criteria	Analysis between groups at baseline	Treatment vs Control similar at baseline	Details of RT exercise prescription	Intensity of RT	Adverse Events Reported	Loss to Follow-up	Attendance	Treatment of missing data specified	Statistical Analysis Specified	Primary &Secondary Outcomes Identified	Sample Size (determined by power calculations	Control &Treatment Method &Analysis the same	CV's Provided
Resistance Training																				
Winnick[28]	2008	RCT	Y	N	N	N	Y	Y	Y	Y	N	N	N	N	N	Y	Y	N	Y	N
Baum [21]	2007	RCT	Y	N	N	N	Y	N	?	Y	Y	N	N	N	N	Y	N	N	Y	N
Brooks [22]	2007	RCT	Y	N	Y	N	Y	Y	Y	Y	Y	N	N	Y	Y	Y	Y	N	Y	Y
Sigal [27]	2007	RCT	Y	Y	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	Y	N
Castaneda [29]	2006	RCT	Y	N	Y	N	N	Y	Y	Y	Y	N	N	N	N	Y	Y	N	Y	Y
Dunstan [25]	2006	RCT	Y	N	N	N	Y	Y	Y	Y	N	Y	Y	Y	Y	Y	Y	N	Y	N
Gordon [26]	2006	RCT	Y	N	N	N	Y	Y	Y	Y	Y	N	Y	Y	N	Y	Y	N	Y	Y
Cauza [24]	2005	RCT	Y	N	N	N	Y	Y	Y	Y	Y	Y	Y	N	N	Y	N	N	Y	N
Cauza [23]	2005	RCT	Y	N	N	N	Y	Y	N	Y	Y	Y	N	N	N	Y	Y	N	Y	N
Dunstan [20]	2005	RCT	Y	N	N	N	N	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	N	Y	Y
Baldi [12]	2003	RCT	Y	N	N	N	N	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	N	Y	Y
Castaneda [13]	2002	RCT	Y	N	Y	N	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	Y
Dunstan [15]	1998	RCT	Y	N	N	N	Y	Y	Y	Y	Y	N	Y	N	Y	Y	Y	N	Y	Y
Ibanez [35]	2008	NRCT	N	N	N	N	Y	Y	N	Y	Y	N	N	Y	N	Y	Y	N	Y	Y
Colberg [31]	2006	NRCT	N	N	N	N	Y	Y	N	Y	Y	N	N	N	N	Y	Y	N	Y	N
Wojtaszewski [34]	2005	NRCT	N	N	N	N	N	Y	N	Y	Y	N	N	N	Y	N	N	N	Y	N
Fenicchia [32]	2004	NRCT	N	N	N	N	Y	Y	N	Y	Y	N	N	N	N	Y	Y	N	Y	N
Holten [19]	2004	NRCT	N	N	N	N	N	Y	N	Y	Y	N	N	N	N	Y	Y	N	Y	N
Ishii [30]	1998	NRCT	N	N	N	N	N	Y	Y	Y	Y	N	N	N	N	Y	Y	N	Y	N
Honkola [36]	1997	NRCT	N	N	N	N	N	N	?	Y	Y	Y	Y	Y	Y	Y	Y	N	N	N
Smutok [37]	1994	NRCT	N	N	N	N	Y	Y	Y	Y	Y	N	N	N	N	Y	Y	N	Y	N
Misra [40]	2008	UCT	N	N/A	N	N	Y	N/A	N/A	Y	Y	N	N	Y	N	Y	Y	N	N/A	N
Ibanez [39]	2005	UCT	N	N/A	N	N	Y	N/A	N/A	Y	Y	Y	Y	Y	N	Y	Y	N	N/A	Y
Eriksson [38]	1997	UCT	N	N/A	N	N	N	N/A	N/A	Y	Y	Y	N	N	N	Y	Y	N	N/A	N

RCT, Randomized controlled trial; NRCT, non-randomized controlled trial; UCT, uncontrolled trial; ?, not specified; Y, yes; N, no; N/A, not applicable; Adverse events reported, refers to whether the authors reported on adverse events, not that adverse events occurred; CV's provided, coefficient of variation of the measure reported within the methodology section, indicating reliability of the measure.

published previously [19]. A paper reporting phase one of a study [14] was excluded due to having a weight loss diet added to the resistance training, however a paper describing phase two [20] was included as dietary modification was ceased at the completion of phase one.

3.2. *Study design/quality assessment*

Most (10/13) of the papers based on randomized controlled trials (RCT) [13,15,21–28] reported eligibility criteria (Table 1); as did just more than half (6/11) of the trials that had no randomization.

Assessors were reported to be blinded in only three papers [13,27,29]. In all studies, previous medical intervention was maintained with changes to drug regimes occurring only where medically required. With one exception [30], all studies were completed using an out-patient design with participants under free-living conditions.

3.3. *Baseline characteristics*

Generally, there were no differences between intervention and control groups except where studies were intentionally designed to compare different cohorts [19,31–35] (Table 1). Baseline characteristics differed in one RCT [23] with the RT group having higher fasting blood glucose levels and lower body mass index and fat mass than untrained controls. Two studies did not report any analysis between groups at baseline [21,36], although there appears to be some differences in the data presented in these [21,36].

3.4. *Statistical analysis and power calculations*

The purpose of the research was outlined in all studies except two [21,24], however one paper [34] did not report the purpose despite a previous paper [19] from the same study reporting this information. Additionally, few papers (6/21) reported how missing data were treated (Table 1). With the exception of one study [36], the intervention and control groups were subjected to the same research methodology and analysis. Only one study [27] reported determining sample size by a priori power calculation.

4. Resistance training for type 2 diabetes

Within the included studies, RT was almost always completed using machines, including pin-loaded machines

Table 2 – Exercise intervention characteristics.

Author (year) country	N = Sample M/F Age y	Control/Comparison condition	Exercise mode and intervention	F: Frequency I: Intensity D: Duration	Strength test	Resistance training equipment	Duration	Supervision
Winnick et al. (2008) USA [28]	*n* = 59 Whites = 23 RT = 8 AT = 15 African = 36 RT = 12 AT = 24 M = ?/F = ? 25–60 y	White subjects Aerobic: 30–40 min walking on motorized treadmill, 3 week^{-1} for first 4 weeks expending ~600 kcal/wk, then 5 week^{-1} expending ~1000 kcal/wk	Progressive resistance training: 8 exercises: not specified, modified after 4 weeks in accordance with performance outcomes	F: NR I: NR D: NR	10RM All exercises	Machine weights	8 weeks	NR
Baum et al. (2007) Germany [21]	*n* = 40 RT = 13 Flex = 13 Vib = 14 M = 24/F = 16 62.9 ± 7.3 y	Flexibility: 8 exercises, 15 min Vibration: 8 exercises, 20 min	Resistance training 8 whole-body exercises: leg extension, seated leg flexion, leg press, seated calf raise, lat pulley, horizontal chest press, butterfly and rowing Wk 1–6 70% 1RM Wk 7–9 increase to 2 sets × 12 reps, Wk 10–12, 3 sets × 10 reps, 80% 1RM	F: 3 week^{-1} I: 1 set × 12 reps D: 45 min total	1RM Max isometric torque (quads)	Machine weights	12 weeks	All sessions: unspecified personnel
Brooks et al. (2007) USA [22]	*n* = 62 RT = 31 C = 31 M = 40/F = 22 66 ± 15.7 y	Standard type 2 DM care	Resistance training and standard care 5 whole-body exercises: upper back, chest press, leg press, knee extension and flexion Wk 1–8 60–80% 1RM Wk 10–14 70–80% 1RM	F: 3 week^{-1} I: 3 sets × 8 reps High intensity D: 45 min total, 5 min warm-up and cool down	1RM - Upper and lower body	Pneumatic Machine weights	16 weeks	NR
Sigal et al. (2007) Canada [27]	*n* = 251 RT = 64 AT = 60 CT = 64 C = 63 M = 160/F = 91 54.7 ± 7.5 y	Aerobic: 3 week^{-1} 45 min @ 75% HRmax Combined: Aerobic and resistance 3 week^{-1} Control: No exercise intervention	Resistance training 7 whole-body exercises: Abdominal crunches, seated row, biceps curls, bench press, leg press, shoulder press and leg extension. Progressing from 1 set of 15 reps @ 15RM to 3 sets of 8 reps @ 8RM	F: 3 week^{-1} I: 2–3 sets × 7–9 reps D: NR 2–3 min between sets	8RM	Machine weights	26 weeks	Weekly first 4 weeks, then fortnightly: Unspecified personnel

Table 2 (*Continued*)

Author (year) country	N = Sample M/F Age y	Control/Comparison condition	Exercise mode and intervention	F: Frequency I: Intensity D: Duration	Strength test	Resistance training equipment	Duration	Supervision
Castaneda et al. (2006) Germany [29]	*n* = 18 RT = 13 C = 5 M = 6/F = 12 66 ± 8 y	Standard type 2 DM care	Resistance training 5 whole-body progressive exercises: 2 upper body, 3 lower body exercises 60–65% 1RM, increasing to 75–80% 1RM by week 4	F: 3 week^{-1} I: 3 sets × 8 reps Moderate-high intensity D: 45 min total, 5 min warm-up and cool down	1RM - 2 upper and 3 lower body exercises	Pneumatic Machine weights	16 weeks	All sessions: unspecified personnel
Dunstan et al. (2006) Australia [25]	*n* = 60 Int = 28 C = 29 M = 33/F = 27 60.5 ± 8.2 y	Home-based resistance training: given 1 dumbbell and weight plates. Monthly telephone call	Community gym-based resistance training 8 whole-body exercises: similar to program undertaken in a supervised setting previously	F: 2 week^{-1} I: 3 sets × 8 reps Increase weight when able to perform 3 sets × 8 reps D: NR	1RM -Bench press - Leg extension	Machine and free weights	12 months	Yes, YMCA staff
Gordon et al. (2006) USA [26]	*n* = 30 RT = 15 C = 15 M = 15/F = 15 67 ± 11 y	Standard type 2 DM care: no exercise, fortnightly telephone interview	Resistance training and standard care 5 whole-body progressive exercises: knee extension, chest press, leg curl, upper back and leg press 60–65% 1RM, increasing to 75–80% 1RM by week 4	F: 3 week^{-1} I: 3 sets × 8 reps D: 45 min total, 1–2 min rest between sets, 5 min warm-up	1RM	Pneumatic Machine weights	16 weeks	Yes: unspecified personnel
Cauza et al. (2005) Austria [24]	*n* = 43 RT = 22 AT = 17 M = 22/F = 21 56 ± 6.6 y	Aerobic training: cycle 3 week^{-1}, 15 min progressing 5 min per week to 90 min @ 60% VO_{2max}	Resistance training 10 min warm-up moderate cycling Minimal weight wk 1 and 2 to teach technique Progressive resistance from wk 3 10 whole-body exercises: bench press, chest cross, shoulder press, pull downs, biceps curls, triceps extensions, situps, leg press, calf raises, leg extensions, increasing to 4, 5 and 6 sets/wk	F: 3 week^{-1} I: 3 sets/wk × 10–15 reps i.e. 1 set × 10–15 reps each session Weight increase when able to complete 15 reps D: NR	1RM - bench press - rowing - leg press All seated	Machine and free weights	4 months	All sessions: Professional instructor, Physician

Cauza et al. (2005) Austria [23]	$n = 15$ RT = 8 AT = 7 M = 4/F = 11 55 ± 7.8 y	Aerobic training: cycle 3 week^{-1}, 15 min progressing 5 min per week to 90 min @ 60% VO_{2max}	Resistance training 10 min warm-up moderate cycling Minimal weight wk 1 and 2 to teach technique Progressive resistance from wk 3 10 whole-body exercises: bench press, chest cross, shoulder press, pull downs, biceps curls, triceps extensions, sit-ups, leg press, calf raises, leg extensions, increasing to 4, 5 and 6 sets	F: 3 week^{-1} I: 3 sets × 10–15 reps Weight increase when able to complete 15 reps D: NR	1RM - seated bench press	Machine and free weights	4 months	All sessions: Professional instructor, Physician
Dunstan et al. (2005) Australia [20]	$n = 36$ RT = 14 C = 12 M = 21/F = 15 60–80 y	Home-based flexibility training, 3 week^{-1}, telephoned fortnightly	Home-based resistance training 9 whole-body exercises: lying dumbbell flies, seated single-leg extension, dumbbell shoulder press, dumbbell bent-over row, standing leg curl, dumbbell biceps curls, dumbbell triceps kickback, abdominal curls. 60–80% 1RM Additional weights provided to facilitate progression	F: 3 week^{-1} I: 3 sets × 8–10 reps D: NR	1RM	Free weights	6 months	No, telephone monitoring weekly first 4 weeks, then fortnightly
Baldi and Snowling (2003) New Zealand [12]	$n = 18$ RT = 9 Con = 9 M = 18/F = 0 47.9 y	No exercise completed	Resistance training 10 whole-body progressive exercises: not specified 1 set × 12 reps in wk 1 then 2 sets × 12 reps. Resistance progressed by 5% when able to successfully complete the program	F: 3 week^{-1} I: 2 sets × 12 reps Max weight for 10 reps for upper body and 15 reps for lower body exercises Moderate intensity D: NR, 60 s rest between sets	Max isokinetic torque - Leg and arm flexion	NR	10 weeks	All sessions: Unspecified personnel
Castaneda et al. (2002) USA [13]	$n = 62$ RT = 31 C = 31 M = 22/F = 40 66 ± 11.8 y	Standard type 2 DM care: Telephone call fortnightly	Resistance training 5 whole-body progressive exercises: chest press, leg press, upper back, knee extension and flexion. Wk 1–8 = 60–80% 1RM Wk 10–14 = 70–80% 1RM Wk 9 and 15 = 10% decrease	F: 3 week^{-1} I: 3 sets × 8 reps High intensity D: 45 min total, 5 min warm-up and cool down	1RM - 2 upper and 3 lower body exercises	Pneumatic Machine weights	16 weeks	All sessions: Unspecified personnel

Table 2 (*Continued*)

Author (year) country	*N* = Sample M/F Age y	Control/Comparison condition	Exercise mode and intervention	F: Frequency I: Intensity D: Duration	Strength test	Resistance training equipment	Duration	Supervision
Dunstan et al. (1998) Australia [15]	*n* = 27 RT = 15 C = 12 M = 17/F = 10 50 ± 10.4 y	Control: no exercise intervention with medical review fortnightly	Progressive circuit resistance training 10 whole-body exercises: leg extension, bench press, leg curl, biceps curls, behind neck pull down, calf raise, overhead press, seated rowing, triceps extension and abdominal curls. Wk 1–2 = 2 sets Wk 3–8 = 3 sets	F: 3 week^{-1} I: 2–3 sets × 10–15 reps D: 60 min total, 30 s per exercise, 30 s active rest including warm-up and cool down	1RM - all intervention exercises performed	Machine and free weights	8 weeks	All sessions: Instructor, Physician

RT, resistance training; Flex, flexibility training; Vib, vibration training; 1RM, 1 repetition maximum; mins, minutes; Max, maximum; C, control; standard type 2 DM care, routine medical care for type 2 diabetes mellitus; NR, not reported; AT, aerobic training; CT, combined aerobic and resistance training; Int, intervention; HRmax, maximum heart rate; wk, week; secs, seconds; VO_{2max}, maximal oxygen uptake.

(20/24 papers), with some studies incorporating the use of free-weights [15,23–25,30,32,37] (Table 2). Two studies varied the delivery of RT using circuit-type training [15,36]. A whole-body training protocol, mostly progressive in nature where the weight lifted, or sets and repetitions completed increased at varying stages was favoured by most researchers (19/20 studies; Table 2). However, one study [19,34] used only three exercises and focused solely on the lower limbs.

4.1. *Frequency*

Resistance training protocols were commonly performed on three non-consecutive days/wk (Table 2), although one non-randomized study [30] admitted their participants to hospital to complete low intensity RT 5 days/wk. Four studies [25,36,38,39] performed RT on 2 days/wk, with one study [25] prescribing 2 days/wk to maintain benefits achieved from previously training 2 days/wk.

4.2. *Intensity*

The intensity of each RT protocol varied considerably, with some studies giving precise information about initial intensities and progression points, while other studies provided vague details of increasing the weight (by an unspecified amount) when participants were able to complete a certain number of sets and repetitions (Table 2). Two studies [35,39] specifically reported completing power exercises using low weight and high velocity movement, in addition to their normal RT program. Two studies prescribed the training weight as percentage 1RM but measured strength using 3RM [19,34] or KIN-COM [30], while another study [33] prescribed RT based on percentage 10RM after conducting 1RM testing. The precise intensities reported for each RCT are shown in Table 2.

4.3. *Duration*

The duration of all studies varied from 4–6 weeks to 12 months of training. One study [32] reported the acute effects of RT, before also reporting 6-week follow-up data. An additional study reported data at 6 weeks [19,34], while another study reported a duration of 4–6 weeks [30]. Six studies had durations of 6–16 weeks [12,15,28,31,35,40], and another study examined changes over 6 months [27]. Additionally, one paper reported a 6-month follow-up period [20] after a 6-month RT and weight loss intervention [14]. One study reported a 2-month supervised introductory phase, followed by 12 months of home-based maintenance [25].

4.4. *Compliance*

When the interventions were completed at a specific exercise venue, eight papers reported compliance levels of ≥85% with most of the training completed under supervision (Table 2). When direct supervision was removed during maintenance programs at home or at a leisure-centre, adherence dropped to 67–72% [20,25] and 68% [25], respectively.

4.5. *Adverse events*

Although information regarding adverse events was not reported in 6 of 13 papers reporting RCT's [15,21,22,26,28,29] and only 3 of 11 papers describing non-RCT's reported information on adverse events [36,38,39] (Table 1), the interventions seemed to be well tolerated in these clinical populations with co-morbidities. Cases of hypoglycemia were reported in five RT studies, during training [25], immediately following training [13], during the night after RT [23], or at unspecified times, with medication decreased to counteract this outcome [27,38]. Hypoglycemic events were also reported with combined training (CT), AT and in the control group, with medication adjusted for this [27]. Additionally, hypoglycemia occurred frequently in one individual both before and after AT [23], while seven hypoglycemic events were reported in a control group [13]. In only one case, was hypoglycemia severe enough to warrant medical attention [25]. One study reported musculoskeletal conditions requiring the program to be modified [27], episodes of chest pain were reported twice [13,27] and one study reported a case of hypotension [38].

5. Glycemic control

5.1. *Glycosylated haemoglobin*

Glycosylated haemoglobin (HbA1c) is considered the optimal way of measuring long-term (120 days) glycemic control [41], with HbA1c values of <7.0% accepted as representing good glucose control [41]. Nine RCT's reported HbA1c data (Table 3), with two studies [13,22,24,26] reporting HbA1c reduced by 1.0–1.2%, from above 8.0% prior to 16 weeks of moderate-high intensity training. Baldi and Snowling [12] showed an improvement over the intervention period which approached significance ($P = 0.057$) after 10 weeks of RT with HbA1c levels reducing from 8.9% to 8.4%. Maintenance programs completed at home [20,25], or at a community-gym [25] reported glycemic control returned towards baseline after 6 months or became worse after 12 months, which is likely to be a result of decreased compliance to the prescribed training. Interestingly, RT appears to be as effective as AT at improving HbA1c when compared to control groups [27] and more effective when compared to AT [24]. This finding requires further validation though as the RT group appeared to spend a larger volume of time training than the AT group. Sigal et al. [27] however, concluded that CT was superior at improving glycemic control to either RT or AT on their own.

Seven non-RCT's reported HbA1c data with only the trials reporting different subjects (diabetes vs. non-diabetes) indicating significant differences. Two [38,40] reported an improvement over time, although another [30] reported a 2% improvement in HbA1c which was not significant.

The greatest improvements to glycemic control occurred when HbA1c was poor (>8.0%) at baseline however, based on current literature [4], clinically relevant improvements of 0.6% were generally seen with moderate-high intensity RT or where the duration of training lasted 10 weeks or longer. The exception to this was 4–6 weeks of low intensity RT 5 days/wk resulting in a 2.0% improvement of HbA1c [30], although this study was not randomized and participants were remarkably light and had a low body mass index, reducing the generalizability of this study.

5.2. *Fasting blood glucose*

Fasting blood glucose (FBG) is less frequently used as a measure of glycemic control but can be a substitute when HbA1c is not measured, for instance when the intervention duration is less than that required for a change in glycemic control to be fully reflected in HbA1c (<3 months). Seven RCT's reported FBG levels (Table 3), with only one [24] reporting a significant change when compared to the comparison group (AT). This was quite a large improvement (3.2 mmol/L) and included some subjects taking insulin, where no other study included subjects taking insulin. This study however was not identically matched in terms of volume, with the RT group completing up to six sets of 10–15 repetitions per week for 10 exercises (estimated to be 120 min of exercise per week plus 120 min of rest/recovery during the sessions) and the AT group completing up to 90 min per week. Again, only two [39,40] of six [19,31,32,38–40] non-RCT's reporting FBG indicated an improvement over time.

6. Insulin sensitivity

6.1. *Euglycemic–hyperinsulinemic clamp*

Although considered the gold-standard for determining insulin sensitivity levels [42], only two studies used the euglycemic–hyperinsulinemic clamp [19,30]. Holten et al. [19] reported that despite individuals with diabetes having significantly lower glucose disposal rates (GDRs) and therefore greater insulin resistance than controls, leg glucose clearance rates increased during the second stage of the euglycemic–hyperinsulinemic clamp, showing that improvements are achievable with RT despite being less sensitive to insulin. Ishii et al. [30] also used an euglycemic–hyperinsulinemic clamp, reporting a 48% ($P < 0.05$) increase in insulin sensitivity with RT and no change in sedentary individuals with diabetes acting as controls.

Comparing these studies is difficult due to one [19] reporting GDR at varying levels of insulin infusion, and another [30] reporting final GDR. However, it is likely that RT for 4–6 weeks will result in increased insulin sensitivity.

6.2. *Oral glucose tolerance test*

Insulin sensitivity using area under the curve (AUC) equations for glucose and insulin levels during an oral glucose tolerance test (OGTT) has been validated against the euglycemic–hyperinsulinemic clamp [42] with lower glucose values indicating better glucose tolerance and lower insulin values indicating increased insulin sensitivity. The OGTT was used in two RCT's [15,21] (Table 3), with results indicating an improvement in insulin sensitivity when compared to sedentary controls [15] but not when compared to vibration or flexibility training [21], although the method of performing this analysis varied from other studies as blood was drawn

Table 3 – Metabolic outcomes.

Author (year) country	Group	Time of follow-up	Type of change	HbA1c (%)	Glucose (mmol/L)	Insulin (pmol/L)	Insulin sensitivity method	Insulin sensitivity
Winnick et al. (2008) USA [28]	Whites RT AT African RT AT	NR	Pre:Post Δ Time effect Group × time	7.9 ± 2.0:NR 7.8 ± 1.2:NR 6.5 ± 1.0:NR 7.6 ± 1.5:NR NR NR			HOMA IR	6.8 ± 4.8:NR +13.2% 10.6 ± 8.5:NR −3.68% 5.8 ± 2.4:NR −19.15% 8.6 ± 7.4:NR +3.79% NR $P < 0.05$ RT African v Whites $P > 0.05$ AT African v Whites
Baum et al. (2007) Germany [21]	RT Vib Flex	72–96 h	Pre:Post Δ Time effect Group × time	6.8% ± 0.17:NR +0.2 ± 0.15 Δ 7.3% ± 0.66:NR −0.3 ± 0.22 Δ 6.7% ± 0.26:NR +0.34 ± 0.26 Δ NR NR	6.99 ± 1.28: 6.66 ± 1.22 7.38 ± 3.16: 6.77 ± 1.94 6.66 ± 1.39: 6.38 ± 1.22 NR NR		OGTT – ear lobe Glucose only	NR:NR −5.6% Δ NR:NR −6.3% Δ NR:NR 0.00% Δ $P < 0.05$ RT and Vib NR
Brooks et al. (2007) USA [22]	RT Con	72 h	Pre:Post Δ Time effect Group × time	8.7 ± 10.0: 7.6 ± 8.4 −1.0 ± 1.1 Δ 7.8 ± 8.9: 8.3 ± 7.2 +0.4 ± 1.7 Δ NR $P < 0.001$	8.8 ± 2.8:7.9 ± 2.2 −0.9 ± 2.8 Δ 9.9 ± 3.9:9.5 ± 3.3 −0.3 ± 4.5 Δ NR $P = 0.92$	116 (124):105 (70)* −16 (69)* Δ 115 (131):133 (126)* +6 (86)* Δ NR $P = 0.27$	HOMA-IR	7.1 (5.7):5.3 (5.5)* −0.7 (3.6)* Δ 6.7 (9.0):6.4 (6.8)* +0.8 (3.8)* Δ NR $P = 0.05$
Sigal et al. (2007) Canada [27]	RT AT CT Con	Minimum 48 h	Pre:Post Time effect Group × time	7.5 ± 1.5: 7.2 ± 1.5 7.4 ± 1.5: 7.0 ± 1.5 7.5 ± 1.5: 6.6 ± 1.6 7.4 ± 1.4: 7.5 ± 1.5 $P = 0.018$ RT $P = 0.002$ AT $P < 0.001$ CT $P = 0.57$ Con $P = 0.038$ RT v Con $P = 0.007$ AT v Con $P = 0.001$ CT v RT $P = 0.014$ CT v AT				

Dunstan et al. (2006) Australia [25]	Centre Home	48 h	Pre:Post Δ Time effect Group × time	7.8 ± 0.9:NR +0.1 ± 1.0 Δ 7.5 ± 0.5:NR +0.2 ± 1.2 Δ $P < 0.05$ both grps NS	9.0 ± 2.0:NR −0.3 ± 1.8 Δ 8.4 ± 1.9:NR −0.2 ± 2.2 Δ NS NS	143.7 ± 66.1:NR −21 ± 47.6 Δ 126.6 ± 55.1:NR −8.5 ± 32.8 Δ $P < 0.05$ centre NS	HOMA	46.9 ± 26.1:NR +9.4 ± 16.4 Δ 50.7 ± 24.6:NR +2.4 ± 12.4 Δ $P < 0.05$ centre NS
Gordon et al. (2006) USA [26]	RT Con	72 h	Pre:Post Time effect Group × time	8.7 ± 1.9:7.7 ± 1.6 8.0 ± 1.6:8.3 ± 1.6 NR $P < 0.01$		173 ± 108:132 ± 54 157 ± 101:168 ± 139 NR $P < 0.05$	HOMA-IR	8.5 (7.2):5.3 (6.3)* 6.7 (7.8):7.1 (7.4)* NR $P = 0.08$
Cauza et al. (2005) Austria [24]	RT AT		Pre:Post Δ Time effect Group × time	8.3 ± 8.0:7.1 ± 1.7 −1.2 Δ 7.7 ± 1.2:7.4 ± 1.2 −0.3Δ $P = 0.001$ RT, NS AT $P = 0.009$	11.32 ± 7.62:8.16 ± 3.77 −3.2 Δ 8.88 ± 2.06:8.83 ± 2.31 −0.05 Δ $P < 0.001$ RT, NS AT $P = 0.002$	130.9 ± 84.0:118.4 ± 85.4 −12.5 Δ 105.1 ± 77.5:125.6 ± 96.1 +20.46 Δ NS both grps $P = 0.04$	HOMA-IR	9.1 ± 7.0:7.2 ± 5.6 −2.0 Δ 6.8 ± 5.8:8.4 ± 7.8 +1.5 Δ $P = 0.04$ RT, NS AT $P = 0.009$
Cauza et al. (2005) Austria [23]	RT AT		Pre:Post Time effect Group × time	7.5 ± 1.4:7.0 ± 2.1 8.0 ± 3.8:7.6 ± 4.8 NS both groups NR				
Dunstan et al. (2005) Australia [20]	RT Con	48 h	Pre:post Δ Time effect Group × time	Returned towards baseline Returned towards baseline $P < 0.05$ NR	NR:NR +0.3 ± 2.2 Δ NR:NR −0.5 ± 2.1 Δ NS both grps NS	NR:NR −0.1 ± 46.8 Δ NR:NR −19.3 ± 50.1 Δ $P < 0.05$ Con, NS RT NS	HOMA-IR	NR:NR +0.04 ± 5.5 Δ NR:NR +5.4 ± 6.5 Δ $P < 0.05$ Con, NS RT NS
Baldi and Snowling (2003) New Zealand [12]	RT Con	36–48 h	Pre:Post Time effect Group × time	8.9 ± 3.6:8.4 ± 1.8 8.5 ± 2.4:8.4 ± 1.8 $P = 0.057$ RT, 0.64 Con NR	12.0 ± 2.7:11.4 ± 2.4 11.1 ± 3.3:11.0 ± 3.0 $P < 0.05$ RT NR	268.1 ± 35.4:146.5 ± 28.5 191.7 ± 63.9:214.6 ± 52.1 $P < 0.05$ RT NR	Insulin sensitivity index 0.120	20.3 ± 3.9:22.6 ± 3.9 22.2 ± 11.4:19.9 ± 5.1 NS NR
Castaneda et al. (2002) USA [13]	PRT Con	48 h	Pre:Post Δ Time effect Group × time	8.7 ± 1.7:7.6 ± 1.1 −12.6 ± 11.1% Δ 8.4 ± 1.7:8.3 ± 2.8 +1.2 ± 5.6% Δ NR $P = 0.01$	8.8 ± 2.8:7.9 ± 2.2 9.7 ± 3.9:8.9 ± 3.9 NR $P = 0.34$			
Dunstan et al. (1998) Australia [15]	CRT Con	48 h	Pre:Post Δ Time effect Group × time	8.2 ± 1.9:8.0 ± 1.9 8.1 ± 2.1:8.3 ± 2.4 NS both grps NS	9.6 ± 3.5:9.4 ± 3.1 9.9 ± 4.2:9.8 ± 4.5 NS both grps NS	64.3 ± 49.1:63.1 ± 48.8 82.6 ± 36.4:93.8 ± 43.7 NS both grps NS	OGTT - Glucose AUC - Insulin AUC	−22 ± 240Δ −2183 ± 6053Δ +191 ± 291Δ +3947 ± 5352Δ NR $P < 0.05$ glucose and insulin

RT, resistance training; Flex, flexibility training; Vib, vibration training; Con, control; NR, not reported; AT, aerobic training; NS, not significant; PRT, progressive resistance training; CRT, circuit resistance training; CT, combined aerobic and resistance training; *, values are median (interquartile range). Castaneda [29] did not report any metabolic variables.

from the ear lobe, rather than the commonly used antecubital vein and only glucose was measured, not insulin as well. Two non-RCT's [32,37] completed OGTT's with AUC for glucose and insulin improving over time with both RT and AT [37], although Fenicchia et al. [32] showed no change after 6 weeks of RT despite reporting an improvement 12–24 h after the first RT session, however, the time of completing the OGTT post training was later. The time utilized for each OGTT trial varied considerably between 24 and 72–96 h post-training (Table 3). This may be a factor in whether studies reported improvements or not as it is still unclear precisely how long insulin sensitivity remains increased following RT, and therefore acute rather than chronic training effects could have been reported. The training regimes may also have contributed to the varied results as different protocols at different intensities were employed by each study.

6.3. *Homeostasis model assessment*

The homeostasis model assessment (HOMA) is a mathematical model of determining insulin resistance from fasting glucose and insulin concentrations which has been validated against the euglycemic–hyperinsulinemic clamp [42]. This was the most common method of determining insulin resistance and estimating insulin sensitivity, possibly because of its ease and speed of completion as it requires only a fasting blood sample, with six papers describing five RCT's using this method [20,22,24–26,28] (Table 3). HOMA was originally developed in 1985 and updated in 1996 to estimate insulin sensitivity [43] although it is unclear whether any of the studies using HOMA modelling utilized the updated version.

A reduction in insulin resistance after 4 months of RT ($P = 0.04$) was reported in a study with 22 participants [24], while 12 months of centre-based maintenance following a 2-month introductory period saw insulin sensitivity improve ($P < 0.05$) [25]. Comparing RT with the control group significantly improved ($P < 0.05$) [22] and tended to improve ($P = 0.08$) [26] insulin resistance, while RT compared with AT also showed a trend towards ($P = 0.09$) improvement of insulin resistance [24]. Winnick et al. [28] reported a significant improvement in insulin resistance for African Americans completing RT when compared to Whites completing RT. However, there was no difference between ethnicity when AT was completed.

Insulin resistance improved by 3.2 when calculated using HOMA 72 h after the final session [26], which is supported by a 9.4% improvement in insulin sensitivity when measured 48 h after the final RT session [25]. Additionally one non-RCT [31] reported HOMA, stating no change in insulin resistance 48–72 h following the final RT session. The limited number of studies and the variation in HOMA limit the ability to make conclusions. However, insulin sensitivity seemed to at least tend to improve compared to a comparison group [15,22,24,26], though how long this improvement remains is unclear.

6.4. *Insulin sensitivity index*

The insulin sensitivity index is another validated mathematical model for determining insulin sensitivity [42], but was used by only one RCT [12] and one uncontrolled trial [39] with each using a different model. Contrasting results were reported, with Baldi and Snowling [12] finding no evidence of change in either RT (10 weeks) or control groups, while Ibanez et al. [39] observed a 46% improvement in insulin sensitivity ($P < 0.001$) after 16 weeks of RT. This difference could be time related as the improvement was measured 24 h after the final session [39] compared to 36–48 h when no improvement was seen [12], or this could be related to intensity or duration of training.

6.5. *Short insulin tolerance test*

One non-RCT [40] used the short insulin tolerance test to measure insulin sensitivity. This test was completed 72–96 h after the final training session of a 12-week program completed with free weights in a physiotherapy clinic, and reported a significant improvement in insulin sensitivity.

7. Insulin signalling

Only two studies reported data on glucose transport and insulin signalling in individuals with diabetes [19,29,34] with one of these being a RCT [29]. Improved glucose disposal, as measured by incorporation into muscle glycogen, support findings using the euglycemic–hyperinsulinemic clamp [19,29]. Changes in the glucose transporter-4 (GLUT4) are less clear with an earlier study [19] reporting a 40% increase ($P < 0.05$) in GLUT4 density compared to a more recent study [29] reporting no evidence of change in GLUT4 gene or protein expression. This could be due to population differences (males vs. females) or the different training protocols (whole-body vs. lower-limb).

Eight weeks of moderate-high intensity RT resulted in increased protein content of the insulin receptor, protein kinase-B, and glycogen synthase (GS) to similar levels in individuals with diabetes and healthy control subjects. However, no training effect was observed for protein content of insulin receptor substrate-1, the p85 subunit of phosphatidylinositol(PI)-3-kinase, or percent GS activity [19]. Moderate-intensity RT resulted in similar changes to various AMP-activated protein kinase (AMPK) subunit isoforms (α1: +16%, β2: +14%, γ1: +29%, γ3: −48%) in patients with diabetes and healthy controls [34], while muscle glycogen levels significantly increased with RT [19,29], when compared to controls ($P = 0.04$) [29].

8. Muscle strength

Ten papers from seven RCT's reported muscle strength data (Table 4), with all but one study [25] reporting improvements of at least 50% after completing RT. The study [25] reporting a decrease ($P < 0.05$) in strength after RT, reported small losses after 12 months of a home or leisure-centre based maintenance program following on from a 2-month supervised intervention period, although only lower body strength in the home-based group decreased below baseline, and was likely to be due to not being able to maintain the appropriate intensity. In most cases [13,20,22,29] these changes were significant when compared to sedentary controls, but not when

Table 4 – Body composition markers.

Author (year) country	Group	Type of change	Mass (kg)	BMI (kg m^{-2})	Waist circumference (cm)	Muscle strength (kg) unless specified	% Fat method	% Fat	Fat mass (kg) unless specified	LBM (kg)
Winnick et al. (2008) USA [28]	Whites RT AT African RT AT	Pre:Post Δ Time effect Group × time	98.1 ± 20.1:NR 99.1 ± 23.6:NR 109.5 ± 39.5:NR 99.5 ± 17.2:NR NR NR	35.1 ± 5.7:NR +2.6% 36.5 ± 6.6:NR −1.18% 33.6 ± 5.9:NR −2.6% 34.2 ± 5.9:NR −0.7% NR $P < 0.05$ RT African v Whites			DXA	40.2 ± 12.5:NR +1.38% 38.6 ± 9.3:NR −0.22% 38.5 ± 11.4:NR −0.85% 38.3 ± 9.8:NR −0.40% NR NS		
Baum et al. (2007) Germany [21]	RT Vib Flex	Pre:Post Δ Time effect Group × time	86.5 ± 14.7:NR −1.30 ± 2.36 Δ 83.3 ± 13.4:NR −0.86 ± 1.77 Δ 88.6 ± 24.1:NR −1.68 ± 4.57 Δ NS NR			NR:NR (Nm kg^{-1}) +14% Δ (left leg) NR:NR NR Δ NR:NR NR Δ NR NR				
Brooks (2007) USA [22]	RT Con	Pre:Post Δ Time effect Group × time		30.9 ± 6.13:NR NR Δ 31.1 ± 5.57:NR NR Δ NR	99.7 ± 12.81:NR NR Δ 100.1 ± 14.48:NR NR Δ NR NR	66 ± 22:90 ± 33^ +24 ± 11 Δ 338 ± 150:568 ± 189∨ +173 ± 106 Δ 62 ± 22:58 ± 22^ −4 ± 11 Δ 300 ± 156:285 ± 150∨ −19 ± 39 Δ NR $P < 0.001$	DXA		35.0 ± 12.25:NR NR Δ 33.7 ± 13.36:NR NR Δ NR NR	44.3 ± 9.47:45.5 ± 10.58 +1.1 ± 1.67 Δ 44.9 ± 10.58:44.8 ± 9.47 +0.4 ± 1.11 Δ NR $P = 0.04$
Sigal et al. (2007) Canada [27]	RT AT CT Con	Pre:Post Time effect Group × time	99.1 ± 30.4:98.0 ± 30.4 103.5 ± 31.0:100.9 ± 30.2 101.9 ± 30.4:99.3 ± 30.4 101.3 ± 28.6:101.0 ± 27.8 NR $P = 0.008$ AT v Con	34.1 ± 9.6:33.7 ± 9.6 35.6 ± 10.1:34.8 ± 10.1 35.0 ± 9.6:34.2 ± 9.6 35.0 ± 9.5:34.9 ± 8.7 NR $P = 0.009$ AT v Con	110 ± 24:107 ± 24 113 ± 23:110 ± 23 112 ± 24:108 ± 24 112 ± 24:111 ± 24 NR $P = 0.03$ AT v Con		Bioelectrical impedance	35.9 ± 9.6:35.0 ± 9.6 37.0 ± 9.3:36.3 ± 9.3 36.0 ± 9.6:35.0 ± 9.6 36.6 ± 8.7:36.9 ± 9.5 NR NS	36.5 ± 19.2:35.2 ± 19.2 39.2 ± 19.4:37.6 ± 19.4 37.6 ± 19.2:35.7 ± 19.2 38.0 ± 17.5:38.2 ± 17.5 NR $P = 0.44$ AT v Con	62.3 ± 13.6:62.5 ± 13.6 64.0 ± 13.9:63.0 ± 13.9 63.9 ± 13.6:63.2 ± 13.6 63.0 ± 12.7:62.5 ± 12.7 NR NS
Castaneda et al. (2006) Germany [29]	RT Con	Pre:Post Δ Time effect Group × time		32.1 ± 6.8:NR NR Δ 33.4 ± 6.3:NR NR Δ NR NR		NR:NR +43 ± 29% Δ* NR:NR +19 ± 31% Δ* NR $P = 0.01$				

Table 4 (*Continued*)

Author (year) country	Group	Type of change	Mass (kg)	BMI (kg m^{-2})	Waist circumference (cm)	Muscle strength (kg) unless specified	% Fat method	% Fat	Fat mass (kg) unless specified	LBM (kg)
Dunstan et al. (2006) Australia [25]	Cent Home	Pre:Post Δ Time effect Group × time	92.6 ± 17.1:NR −2.1 ± 3.4 Δ 91.2 ± 13.6:NR −2.2 ± 3.2 Δ $P < 0.05$ both grps NS	32.8 ± 4.8:NR NR Δ 32.4 ± 4.4:NR NR Δ NR NR	105.6 ± 11.7:NR −1.3 ± 5.3 Δ 107.4 ± 10.8:NR −2.0 ± 5.9 Δ NS NS	78.8 ± 43.9:NR^ −3.4 ± 17.8 Δ 29.9 ± 10.1:NR∨ −7.2 ± 10.5 Δ 78.3 ± 49.1:NR^ −3.7 ± 19.6 30.3 ± 12.0:NR∨ −0.3 ± 6.3 Δ $P < 0.05$ RT∨ $P < 0.05$ ∨	Bioelectrical impedance		37.6 ± 12.3:NR −0.8 ± 3.2 Δ 35.8 ± 10.0:NR −1.0 ± 3.2 Δ NS NS	55.0 ± 9.8:NR −1.3 ± 1.6 Δ 55.4 ± 10.5:NR −0.9 ± 2.2 Δ $P < 0.05$ both grps NS
Gordon et al. (2006) USA [26]	RT Con	Pre:Post Time effect Group × time	80 ± 19:NR 88 ± 15:NR NR NR	30.7 ± 6.2:31.3 ± 6.2 33.5 ± 6.2:33.4 ± 5.8 NR $P = 0.05$	100 ± 13.4:101 ± 10.8 108 ± 11.2:109 ± 12.4 NR $P = 0.43$					
Cauza et al. (2005) Austria [24]	RT AT	Pre:Post Δ Time effect Group × time	91.3 ± 13.6:90.2 ± 13.1 −1.1% Δ 96.7 ± 18.6:95.4 ± 18.6 −1.1% Δ NS NR	31.3 ± 4.2:30.9 ± 4.2 −1.1% Δ 33.9 ± 5.4:33.5 ± 5.4 −1.1% Δ NS NR		54.6 ± 16.0:68.6 ± 18.8^ +26% Δ 114 ± 36.6:168 ± 45.5∨ +48% Δ 43.9 ± 15.7:45.0 ± 16.1^ +2.5% Δ 93 ± 35.9:107 ± 42.1∨ +15% Δ $P < 0.001$ RT^ ∨, ET∨ NR	10 site skinfolds	44.5 ± 3.8:40.5 ± 5.2 −9.1% Δ 46.3 ± 3.3:44.5 ± 3.3 −3.4% Δ $P < 0.001$ both grps NR	39.6 ± 6.6:35.8 ± 8.0 −9.7% Δ 44.8 ± 9.5:42.5 ± 8.7 −5.3% Δ $P < 0.001$ both grps NR	49.4 ± 8.4:52.6 ± 8.0 +6.5% Δ 51.9 ± 10.3:52.9 ± 11.1 +2% Δ $P < 0.001$ RT NR
Cauza et al. (2005) Austria [23]	RT AT	Pre:Post Time effect Group × time		29.9 ± 2.3:29.9 ± 2.8 36.3 ± 12.4:36.3 ± 6.9 NS $P = 0.03$		47.4 ± 15.0:59.7 ± 18.4^ 31.8 ± 10.6:31.7 ± 10.6^ $P = 0.01$ RT, NS AT NR	NR		38.9 ± 6.5:33.5 ± 7.9 46.9 ± 10.6:44.4 ± 10.3 $P < 0.01$ both grps $P = 0.04$	46.3 ± 7.4:51.9 ± 9.1 56 ± 10.3:58.2 ± 11.1 $P < 0.01$ RT, $P = 0.03$ AT NR
Dunstan et al. (2005) Australia [20]	RT Con	Pre:Post Δ Time effect Group × time	88.7 ± 10.9:NR Unspecified increase 89.5 ± 12.1:NR Unspecified increase $P < 0.05$ RT NR		NR:NR −3.4 ± 4.7 Δ NR:NR −2.0 ± 4.3 Δ $P < 0.05$ RT NS	NR:NR +26.4 ± 22.8^ Δ +4.9 ± 6.4∨ Δ NR:NR −0.2 ± 19.1^ Δ −0.1 ± 5.4∨ Δ $P < 0.05$ RT^∨ $P < 0.05$^∨	DXA		33.1 ± 7.4:NR NR Δ 35.6 ± 6.8:NR NR Δ $P < 0.01$ both grps NR	51.8 ± 8.1:NR NR Δ 49.7 ± 9.5:NR NR Δ NS $P < 0.08$

Baldi and Snowling (2003) New Zealand [12]	RT	Pre:Post	112.3 ± 12.0:114.0 ± 12.3	34.3 ± 9.6:NR		NR/NR:NR/	Hydrostatic	32.4 ± 3.3:NR	38.1 ± 10.5:37.0 ± 10.5	74.3 ± 3.6:76.9 ± 3.3
	Con	Δ	110.3 ± 21.9:110.9 ± 22.2	36.4 ± 9.3:NR		NR(ext/flx)	weighing	30.7 ± 6.6:NR	37.7 ± 17.1:40.3 ± 18.9	72.6 ± 9.6:70.6 ± 9.0
		Time effect	$P < 0.05$ RT, NS Con	NR		+32.0/+3.2%^ Δ		NR	$P < 0.05$ Con	$P < 0.05$ RT, NS Con
		Group × time	NR	NR		+18.1%/+37.0%∨ Δ		NR	NR	NR
						NR/NR:NR/NR				
						NR/NR Δ				
						$P < 0.05$ RT^∨				
						NR				
Castaneda et al. (2002) USA [13]	PRT	Pre:Post	79.3 ± 17.8:79.5 ± 18.4		99.7 ± 12.8:97.5	389 ± 167:518	DXA		35.0 ± 12.3:34.0 ± 12.8	44.3 ± 9.5:45.5 ± 10.6
	Con	Δ	78.6 ± 17.3:79.4 ± 16.2		± 12.8	± 267*			33.7 ± 13.4:34.6 ± 12.3	44.9 ± 10.6:44.8 ± 9.5
		Time effect	NR		100 ± 14.5:102	+33 ± 7% Δ			NR	NR
		Group × time	$P = 0.89$		± 12.3	351 ± 173:299			$P = 0.26$	$P = 0.004$
					NR	± 167*				
					$P = 0.07$	−15 ± 3% Δ				
						NR				
						$P = 0.0001$				
Dunstan et al. (1998) Australia [15]	CRT	Pre:Post	83.6 ± 14.3:83.2 ± 14.3	28.3 ± 3.1:28.1 ± 3.1		NR:NR	7 site	NR	NR	NR
	Con	Δ	82.7 ± 12.8:83.7 ± 13.2	30.1 ± 3.8:30.4 ± 3.8		+15 ± 6%^ Δ	skinfolds			
		Time effect	NR	NR		+43 ± 12%∨ Δ				
		Group × time	$P < 0.05$	$P < 0.05$		NR:NR				
						NR^∨ Δ				
						$P < 0.05$^∨				
						NR				

RT, resistance training; Flex, flexibility training; Vib, vibration training; Con, control; Cent, centre-based training; AT, aerobic training; NS, not significant; PRT, progressive resistance training; CRT, circuit resistance training; ^, upper body; ∨, lower body; *, whole-body; ext, extension; flx, flexion; CT, combined aerobic and resistance training; NR, not reported.

compared to AT [23,24]. Seven non-RCT's [19,30–32,35,37,39] also reported muscle strength improved, with similar improvements in muscle strength observed in individuals with diabetes compared to those without diabetes [19,31]. One study also reported muscle power output improved over time [35]. Studies that reported greater improvements in muscular strength, utilized durations between 16 weeks [23,24,39] and 6 months [20] at moderate or moderate-high intensities. In contrast to other results, one study [32] reported highly significant ($P < 0.01$) increases in muscle strength after 6 weeks of moderate intensity RT. However, overall it appears that higher intensity RT is appropriate and more time efficient for muscle strength gains, although data evaluating lower intensity RT in patients with diabetes is limited (1/17 studies).

Improved glycemic control was observed in five [12,13,22,24,26] of the 10 papers from RCT's (3 studies) that reported significant improvements in strength, while four RCT's [15,21,22,24,26] that increased strength also improved insulin sensitivity, leaving two RCT's [12,20] that did not improve insulin sensitivity despite increasing strength. In non-RCT's, no studies that improved strength reported improved glycemic control, yet four studies [19,30,37,39] that improved strength reported improved insulin sensitivity and two of six studies [31,32] did not improve insulin sensitivity.

9. Body composition

9.1. *Lean body mass*

Lean body mass (LBM) was measured by dual energy X-ray absorptiometry (DXA) in four studies [13,20,22,30,40] including two RCT's [13,20,22], or estimated after accounting for fat mass in a further six studies [12,24,25,27,32,33,37] including four RCT's [12,24,25,27], with one study [23] not specifying the method used (Table 4). Results varied, with significant LBM increases of 3–6 kg with RT [12,23,24] and 2 kg with AT [23]. Two studies reported significant ($P < 0.05$; $P = 0.04$) [13,22] or a trend ($P < 0.08$) [20] towards improvements for LBM when RT was compared with the non-exercising control group.

9.2. *Fat mass*

Fat mass was typically determined through mathematical equations after measuring body mass and using various techniques to estimate percentage fat. Significant decreases in fat mass of 1–4.5 kg with RT [20,23,24,32,39] and 2 kg with AT [23,24] occurred over the training duration (Table 4). One study [12] reported no changes in fat mass with RT, compared with a 3.5 kg increase ($P < 0.05$) in controls over 10 weeks. With the exception of one study [32], interventions with durations less than 10 weeks did not report fat mass (Table 4). The current evidence suggests that moderate or high intensity training of greater than 10 weeks tends to reduce fat mass in individuals with diabetes.

9.3. *Percentage body fat*

One non-RCT [30] reported a decrease in percentage body fat as measured by DXA. With one RCT [28] and one non-RCT [40]

reporting no change. Percentage body fat results were not reported in other studies despite utilizing DXA [13,20,22]. Two further studies [12,37] utilized hydrostatic weighing, reporting no evidence of change to percentage body fat (Table 4). Bioelectrical impedance was used in three studies [25,27,32], with changes only reported when AT was compared to controls ($P = 0.008$) [27] (Table 4). Four studies utilized the less sensitive measure of skin-fold measurements where decreases in body fat of up to 9.1% were reported (Table 4). One non-RCT [35] reported percentage body fat results, but not how it was measured.

9.4. *Body mass*

Typically there was no change in body mass with any exercise regimen, however, one study [12] reported a 2 kg increase ($P < 0.05$) after 10 weeks of moderate intensity RT (Table 4). After 6 months of home-based maintenance [20], body mass significantly increased ($P < 0.05$), although final levels remained lower than baseline.

9.5. *Girth measures*

Measures of waist circumference were not routinely completed (7/20 studies; Table 4) [13,20,22,25–27,32,38,40] with change occurring when comparing sedentary controls with RT [13], AT [27] and over time with RT [20,40]. Waist circumference was reported to remain decreased after 6 months of home-based RT maintenance [20].

10. Cardiac risk factors

10.1. *Lipid profile*

Blood lipids were reported in nine studies with general improvements in total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and triglycerides reported after RT ($P < 0.001$; $P < 0.01$) [24,36,40].

10.2. *Blood pressure*

Blood pressure was measured in 10 studies. Three studies reported beneficial changes in systolic blood pressure associated with all forms of training [13,21,24]. Improvements to diastolic blood pressure were less frequently observed, but still occurred over time with RT and AT [24].

11. Discussion

Individuals with diabetes are able to complete RT with minimal risk of negative health outcomes or injury, while improving overall glycemic control, insulin sensitivity and muscular strength. Overall, the quality of study design was good with 13 papers reporting on 10 RCT's, of which all but three were published since 2005. The major findings from these studies are that completing RT, and AT over extended durations will result in similar improvements to glycemic control [23,27]. However, RT could potentially provide greater benefits in terms of glycemic control than AT with researchers and practitioners intimating that RT, comprising short bouts with intermittent rest periods, is better tolerated than AT [10,44,45]. To further improve the quality of studies and knowledge in this area and to enable comprehensive comparison between studies in the future, consideration needs to be given to quantifiable and replicable exercise prescriptions, specifying how missing data is treated and determining sample sizes by power calculations.

A clinically relevant lowering of HbA1c, a key marker of improved long-term glycemic control, was reported in a number of RT studies while those reporting no effect were intervention studies with durations of 10 weeks or less. These changes appear to be of a similar or greater magnitude to aerobic training [23,24,27], however the effect of combining RT with AT remains unclear with only one study [27] making a direct comparison between combined training and isolated RT or AT interventions.

Interestingly, insulin sensitivity was only evaluated using the euglycemic–hyperinsulinemic clamp in non-RCT's [19,30]. These studies reported increased insulin sensitivity following RT, despite the time that the measure was performed varying from 16 to 48 h following the final exercise session and the intensity and frequency of the training varying markedly. Other, less precise measures of insulin sensitivity, generally indicated improvements at times ranging from 24 to 72–96 h [21,40] following the final RT session of a long-term training program. However, the effect of a single session of RT on insulin sensitivity in previously untrained subjects has not been investigated beyond 12–24 h after the session [32,33]. This raises questions about the training frequency that should be prescribed, which is currently based on improvements to HbA1c. Eight RCT's included in this systematic review [12,15,20–22,24–26,28] present HbA1c and insulin sensitivity data, with only one [12] indicating that insulin sensitivity did not improve when HbA1c improved. Furthermore, two studies [15,21] indicated that insulin sensitivity improved but was not reflected in HbA1c, which did not change. Additionally, insulin sensitivity improved after 12 months of gym-based maintenance despite glycemic control becoming worse [25]. Therefore, further investigations as to whether RT should be prescribed based on insulin sensitivity should be undertaken. If RT should be prescribed based on insulin sensitivity, RT may need to be prescribed everyday in this population, at least initially, as the length of time insulin sensitivity remains improved following a single RT session has not been adequately evaluated. After 12–16 weeks of training, improved insulin sensitivity appears to be maintained for 4–5 days [21,22,26], therefore glucose control may potentially be improved or maintained with one or two RT sessions each week. Both of these possibilities vary considerably from current RT recommendations of 3 days/wk [6]. It is possible that RT should be performed more regularly initially to improve insulin sensitivity and glycemic control, before it can be performed less frequently to maintain the benefits; however this is yet to be thoroughly examined.

The training environment and intensity of RT also need further investigation, as decreased compliance to the training protocol appears to be associated with a decline in insulin sensitivity as demonstrated by the lower adherence level and

increased insulin resistance reported with home-based training [20,25]. While high adherence to RT protocols resulted in significant muscle strength improvements, changes in body mass were generally not observed. In contrast, LBM increased and percentage body fat decreased, confirming that body composition is improved with RT. Therefore, RT may provide further benefits for individuals with diabetes attempting to lose weight as RT may counteract the loss of muscle mass typically associated with isolated hypocaloric diets [46]. However, changes to body composition are unlikely to account for any changes in insulin sensitivity, as this can be increased following a single exercise session [32,33]. Although changes to body composition may not improve insulin sensitivity, individuals with diabetes are at an increased risk of cardiac co-morbidities for which improved body composition would reduce this risk. Additionally, RT has the ability to improve muscle quality (defined as a functional measure of strength per unit volume of muscle) and change the characteristics of a muscle fibre [22,47], suggested to result in increased glucose transport. Although, limited data from individuals with diabetes suggest that muscle mass or body composition changes do not influence insulin sensitivity, local contraction-mediated responses from RT might [19], resulting in increased intracellular signalling [10] leading to increased membrane bound GLUT4 transporters and improved insulin sensitivity. Despite mechanisms for why RT improves insulin sensitivity not yet being fully elucidated, they are understood to have some common mechanisms to AT as well as some unique adaptations attributable to RT alone [7,37].

Although not reviewed in detail here, RT invokes many health benefits for individuals with diabetes in addition to improved glycemic control. These include improvements in bone strength, minimization of sarcopenic losses or muscle weakness associated with aging, improved balance and reduced falls risk [48]. The beneficial effects of RT on lowering cardiovascular risk (i.e. blood pressure and blood lipids) have been reviewed elsewhere [4]. Of the studies reviewed here, the impact of RT on lipid profiles is minimal in individuals who were normal or just above normal at baseline, but it is promising that beneficial blood pressure effects have been reported in hypertensive patients with type 2 diabetes [13,21,24]. Decreasing body mass by dieting (energy restriction) has detrimental effects on muscle mass and while AT is only able to maintain the integrity of muscle [49] it is suggested that RT is able to counteract these detrimental effects in a way that AT cannot by actually improving the amount and integrity of muscle mass [46].

Compelling evidence from both RCT's and non-RCT's is that RT is safe for individuals with diabetes who are likely to have complex co-morbidities, although it needs to be noted that all studies to date have excluded patients with contraindications to RT [50]. Resistance training is effective in improving glycemic control and increasing insulin sensitivity. Higher intensity and longer intervention duration of RT appear most beneficial, but this along with training frequency, are parameters that require further investigation. It is likely that individualized programs, taking into account an individual's current level of strength, severity of diabetes and also co-morbidities will optimise the adaptive response and enhance compliance. Determining the minimum effective dose of RT, or if appropriate in conjunction with AT, would possibly improve ongoing compliance, and therefore lead to improved health outcomes. Resistance training has been shown to not only be equivalent to AT in ameliorating diabetes and its associated complications; it may also be the exercise of choice for individuals with diabetes or pre-diabetes who find adherence to continuous moderate intensity aerobic training too physically challenging.

Conflict of interest

The authors declare that they have no conflict of interest.

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