

Progressive resistance exercise improves glycaemic control in people with type 2 diabetes mellitus: a systematic review

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Question: Is progressive resistance exercise a safe and effective form of exercise to improve glycaemic control in people with type 2 diabetes? **Design:** Systematic review with meta-analysis of randomised controlled trials. **Participants:** People with type 2 diabetes mellitus. **Intervention:** Progressive resistance exercise. **Outcome measures:** The primary outcome was glycaemic control measured as percentage glycosylated haemoglobin (HbA1c). Secondary outcomes were body composition (lean body and fat free mass in kg), and muscle strength (% change in 1RM, dynamometry, change in maximum weight lifted). **Results:** The search yielded nine relevant trials that evaluated 372 people with type 2 diabetes. Compared to not exercising, progressive resistance exercise led to small and statistically significant absolute reductions in HbA1c of 0.3% (SMD -0.25, 95% CI -0.47 to -0.03). When compared to aerobic exercise there were no significant differences in HbA1c. Progressive resistance exercise resulted in large improvements in strength when compared to aerobic (SMD 1.44, 95% CI 0.83 to 2.05) or no exercise (SMD 0.95, 95% CI 0.58 to 1.31). There were no significant changes in body composition. **Conclusions:** Progressive resistance exercise increases strength and leads to small reductions in glycosylated haemoglobin that are likely to be clinically significant for people with type 2 diabetes. Progressive resistance exercise is a feasible option in the management of glycaemia for this population. [Irvine C, Taylor NF (2009) Progressive resistance exercise improves glycaemic control in people with type 2 diabetes mellitus: a systematic review. *Australian Journal of Physiotherapy* 55: 237–246]

Key words: Review, Resistance training, Exercise, Diabetes, Glycosylated hemoglobin A, Hyperglycemia, Randomised controlled trial

Introduction

Diabetes is a metabolic disorder resulting from defective insulin secretion, insulin action, or both. As a consequence, people with diabetes have chronically elevated plasma glucose levels and disturbances in metabolism that lead to complications such as retinopathy, neuropathy, nephropathy, and an increased risk of cardiovascular disease. Type 2 diabetes, formerly known as non-insulin dependent diabetes mellitus, is due to relative, rather than absolute insulin deficiency and is characterised by a long period of hyperglycaemia that may last for years before symptoms appear (Laakso 2008). Obesity and lack of physical activity are major risk factors for the development of type 2 diabetes (Laakso 2008). In 1999/2000, an estimated 840 000 Australians had type 2 diabetes with one undiagnosed case for every diagnosed case (AIHW 2008). Type 2 diabetes accounts for 83% of diagnosed diabetes and is responsible for about 5% of the total burden of disease in Australia (AIHW 2008).

Achieving and maintaining appropriate plasma glucose levels is vital to managing diabetes and this has traditionally been achieved using medication, dietary intervention and aerobic exercise (ADA 2008). The gold standard test to monitor long-term plasma glucose levels (over 2–3 months) is the monitoring of glycosylated haemoglobin (HbA1c). Diabetic interventions aim to achieve glycosylated haemoglobin levels of less than 7% in people with diabetes in order to reduce complications associated with diabetes (ADA 2008).

Muscle weakness, decreased muscle mass, and changes in skeletal muscle fibres are related to compromised glycaemic control in diabetes, possibly because of peripheral neuropathy and reduced vascular supply (Schuller and Linke 2008). Studies in animal models of diabetes have shown that resistance exercise can lead to increased muscle mass (Farrell et al 1999). Skeletal muscle is a large reservoir for glucose disposal in the body (Schuller and Linke 2008) and exercise is a powerful stimulant of glucose uptake partly through the action of the skeletal muscle glucose transporter protein (Schuller and Linke 2008). Therefore, resistance exercise with its direct effect on skeletal muscle may have a role in the management of patients with type 2 diabetes.

According to the American College of Sports Medicine (2009), progressive resistance exercise is carried out 2–3 times a week and consists of 1–3 sets of 8–12 repetitions, progressed from a load of 45–50% to 70–80% of 1RM, where 1RM is the amount of weight that can be lifted just once through available range. Three previous systematic reviews have investigated both aerobic and resistance exercise and have provided evidence that exercise in general improves glycaemic control in people with type 2 diabetes (Boulé et al 2001, Snowling and Hopkins 2006, Thomas et al 2006). The Boulé et al (2001) review included three trials of resistance exercise which were inconclusive, Snowling and Hopkins' (2006) review included seven trials which produced a small benefit, and the Thomas et al (2006) review included three trials which were not analysed separately. No systematic review has focused on progressive resistance exercise alone

compared to other interventions, and included an evaluation of the safety of the intervention. Furthermore, new trials of resistance exercise have been published since 2006.

Therefore, the specific research questions for this review were:

1. Does progressive resistance exercise improve glycaemic control in people with type 2 diabetes more than aerobic or no exercise?
2. Does it improve strength and body composition?
3. Is it safe?

Method

Identification and selection of studies

Relevant randomised trials were identified using a predefined search strategy (see Appendix 1 on the eAddenda for full search strategy) to search three databases: CINAHL, MEDLINE, and EMBASE. These electronic databases were searched from the earliest date available until July 2008. Manual searches of reference lists were conducted to ensure all relevant studies were captured. Two reviewers (CI and NT) independently applied the inclusion and exclusion criteria (Box 1) to the titles and abstracts of all the studies retrieved and any that clearly did not fulfil the criteria were eliminated. Where it was not clear, the full text papers of the studies were obtained for review. The trials had to be randomised trials comparing progressive resistance exercise to a suitable control group (eg, aerobic exercise, flexibility training, or sedentary) of participants with type 2 diabetes mellitus. Trials that included combined or mixed training (where progressive resistance exercise was completed in combination with aerobic exercise) were excluded in order to focus on the effects of progressive resistance exercise alone. Trials should have described the diagnostic criteria for type 2 diabetes. Acceptable definitions include the World Health Organisation and the American Diabetes Association's criteria of fasting plasma glucose ≥ 7.0 mmol/l (WHO/IDF 2006). Only trials of greater than eight weeks' duration were included since periods shorter than this are unsuitable to show changes in glycosylated haemoglobin. Trials were excluded if the participants had other conditions such as type 1 diabetes mellitus, gestational diabetes, or impaired glucose tolerance, or if the intervention was another form of exercise or mixed training. Where results were duplicated in more than one paper the original paper was chosen for inclusion.

Assessment of characteristics of studies

Quality: All trials were critically appraised for methodological quality using the PEDro scale (www.pedro.org.au) by two authors independently (CI and NT). Trials were not excluded on the basis of quality; however, quality was taken into account when interpreting results.

Participants: Age, BMI, and baseline % HbA1c levels were recorded in order to compare the similarity of participants between trials.

Intervention: The target muscles, intensity, and duration of intervention were recorded. The control groups were categorised as non-exercise or aerobic training. For the purposes of this review, flexibility training was categorised as non-exercise since it has negligible energy expenditure and is therefore unlikely to have an effect on glycaemic control.

Box 1. Inclusion criteria.

<p>Design</p> <ul style="list-style-type: none"> • Randomised trials • Full peer-reviewed papers <p>Participants</p> <ul style="list-style-type: none"> • Type 2 diabetes mellitus (as defined by WHO 2006, ADA 2008) • Human <p>Intervention</p> <ul style="list-style-type: none"> • Progressive resistance exercise (as defined by ACSM 2009) • Exercise > 8 weeks duration <p>Outcome measures</p> <ul style="list-style-type: none"> • Glycaemic control as measured by glycosylated haemoglobin (HbA1c) <p>Comparisons</p> <ul style="list-style-type: none"> • Progressive resistance exercise versus no exercise • Progressive resistance exercise versus aerobic exercise

WHO = World Health Organisation, ADA = American Diabetes Association, ACSM = American College of Sports Medicine

Outcome measures: The primary outcome was glycaemic control measured as percentage glycosylated haemoglobin (HbA1c). Secondary outcomes were body composition (lean body mass and fat free mass), strength (% change in 1RM, dynamometry), and adverse events. Where results for upper and lower limb muscles were presented, the lower limb results were used, since the lower limbs would be expected to provide a larger reservoir for glucose disposal than the upper limbs and so be more relevant for people with type 2 diabetes. Where both sides were measured, the left side was chosen.

Data analysis: Data were extracted and entered onto a customised form to record baseline characteristics, details of intervention and control groups, adverse events, and primary and secondary outcomes. Where sufficient information was obtained, standardised mean differences (effect sizes) were calculated for the outcomes based on post intervention means and the pooled estimate of post exercise standard deviations, using Hedges *g*. Meta-analyses were performed using a random effects model for primary and secondary outcomes using inverse variance methods (RevMan 2008). Statistical heterogeneity was assessed using I^2 statistic with values of less than 25% assigned as representing low levels of heterogeneity (Higgins et al 2003). The strength of the standardised mean difference was determined descriptively according to Cohen et al (1962), where 0.2 is considered small, 0.5 moderate, and 0.8 large. Where standard errors were provided they were converted to standard deviations. Authors were contacted for further information where required.

To calculate the between group percentage difference in HbA1c a meta-analysis on mean differences was performed using a random effects model and inverse variance methods (RevMan 2008). Analysis was performed separately for results of trials that compared progressive resistance exercise with a non-exercising control and those that compared progressive resistance exercise with aerobic exercise.

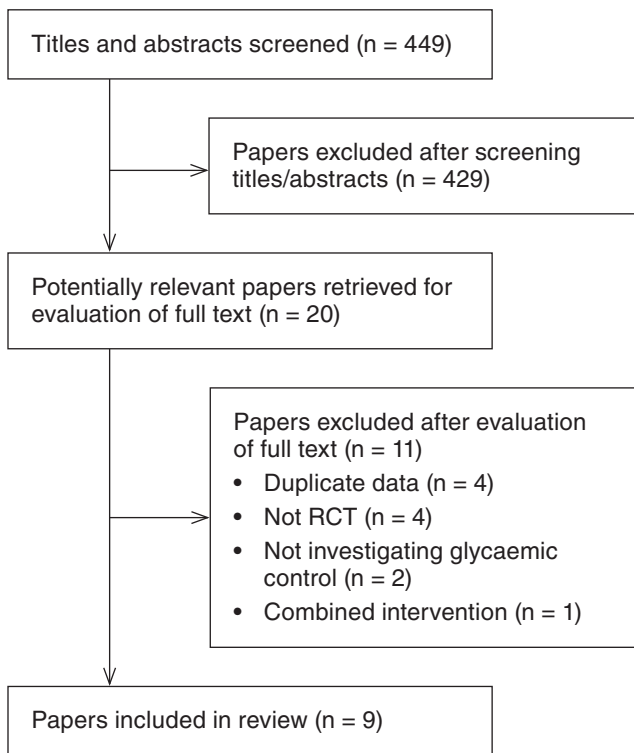


Figure 1. Flow of studies through the review.

Results

Flow of studies through the review

The search yielded a total of 449 studies. A manual search of reference lists did not yield any more trials. From the titles and abstracts, 429 studies clearly did not fulfil the criteria or existed in more than one database and were eliminated. Full text copies of 20 studies were obtained for further examination. Eleven of these were excluded because: duplicate data existed in four articles (Brooks et al 2007, Daly et al 2005, Dunstan et al 2005, Gordon et al 2006), four were not randomised trials (De Fayter et al 2007, Ibanez et al 2005, Ishii et al 1998, Lomangino et al 2005), two did not investigate glycaemic control (Brandon et al 2003, Castaneda et al 2006), and one involved combined exercise (Cuff et al 2003). Consensus was reached to yield a total of nine relevant trials (Figure 1).

Characteristics of included studies

Quality: The methodological quality of the studies sourced was low to moderate with a mean PEDro score of 5.0 out of 10 (SD 1.6) (Table 1). Quality scores ranged from 3 (Cauza et al 2005b) to 8 (Sigal et al 2007). No trials blinded participants or therapists. Since blinding of participants and therapists is not feasible with an intervention of progressive resistance exercise the highest expected score was 8. Only one trial had blinded assessors (Sigal et al 2007) and this was the only trial with concealed allocation of participants. Six out of nine trials had 85% retention rates and all trials reported point measures and measures of variability.

Participants: The review included 372 participants, of whom 192 completed progressive resistance exercise. Participants were males (66%) and females with a weighted mean average age of 58.4 years ranging between 46.5 (Baldi and Snowling 2003) and 67.6 years (Dunstan et al 2002). Most participants were overweight or obese with a

Table 1. Quality (PEDro scores) of included studies.

Study	Random allocation	Concealed allocation	Groups similar at baseline	Participant blinding	Therapist blinding	Assessor blinding	< 15% dropouts	Intention-to-treat analysis	Between-group difference reported	Point estimate and variability reported	Total (0 to 10)
Baldi and Snowling (2003)	Y	N	Y	N	N	N	Y	Y	N	Y	5
Baum et al (2007)	Y	N	Y	N	N	N	N	N	Y	Y	4
Castaneda et al (2002)	Y	N	Y	N	N	Y	Y	Y	Y	Y	7
Cauza et al (2005a)	N	N	Y	N	N	N	Y	Y	Y	Y	5
Cauza et al (2005b)	N	N	N	N	N	N	Y	N	Y	Y	3
Dunstan et al (1998)	Y	N	Y	N	N	N	N	N	Y	Y	4
Dunstan et al (2002)	Y	N	Y	N	N	N	N	N	Y	Y	4
Honkola et al (1997)	N	N	Y	N	N	N	Y	Y	Y	Y	5
Sigal et al (2007)	Y	Y	Y	N	N	Y	Y	Y	Y	Y	8

Table 2. Summary of included studies (n = 9).

Study	Participants		Intervention		Outcome measures
	Experimental	Control	Experimental	Control	
Baldi and Snowling (2003)	n = 9 Age (yr) = 47 (SD 6) BMI (kg/m ²) = 34.3 (SD 9.6) HbA1c (%) = 8.9 (SD 3.6)	Type: PRE Muscles: major muscle groups for UL and LL Intensity: 2 x 12RM UL or 2 x 15RM LL x 3/wk x 10 wk	Type: no intervention	Diabetes: HbA1c (%) Strength: peak force (Nm) Body composition: FFM Adverse events: recorded Follow up: 0, 10 wk	
Baum et al (2007)	n = 13 Age (yr) = 63 (SD 7) BMI (kg/m ²) = not reported HbA1c (%) = 6.8 (SD 0.17)	Type: PRE Muscles: hip E, knee F, knee E, ankle PF, shoulder horizontal F/E, shoulder adduction, shoulder E Intensity: Wk 1-6: 1 x 12 70%1RM x 3/wk Wk 7-9: 2 x 12 70%1RM x 3/wk Wk 10-12: 3 x 10 80%1RM x 3/wk	Type: no intervention (flexibility)	Diabetes: HbA1c (%) Strength: max relative isometric torque (Nm/kg) Body composition: not measured Adverse events: recorded Follow up: 0, 12 wk	
Castaneda et al (2002)	n = 31 Age (yr) = 66 (SD 11) BMI (kg/m ²) = 30.9 (SD 6.1) HbA1c (%) = 8.7 (SD 1.7)	Type: PRE Muscles: hip E, knee E, knee F, shoulder horizontal F, upper back Intensity: Wk 1-8: 3 x 8 60-80% baseline 1RM x 3/wk Wk 10-14: 3 x 8 70-80% midstudy 1RM x 3/wk	Type: no intervention	Diabetes: HbA1c (%) Strength: sum 1RM (kg) Body composition: lean tissue mass Adverse events: recorded Follow up: 0, 16 wk	
Cauza et al (2005a)	n = 8 Age (yr) = 55 (SD 5) BMI (kg/m ²) = 29.9 (SD 2.3) HbA1c (%) = 7.5 (SD 1.4)	Type: PRE Muscles: shoulder horizontal F, shoulder F/abduction, hip E, knee F, knee E, shoulder adduction/E, elbow F, elbow E, ankle PF Intensity: 1-2 x 10-15RM x 3/wk x 16 wk	Type: aerobic exercise	Diabetes: HbA1c (%) Strength: bench press (kg) Body composition: LBM Adverse events: recorded Follow up: 0, 16 wk	
Cauza et al (2005b)	n = 22 Age (yr) = 56 (SD 5) BMI (kg/m ²) = 31.1 (SD 4.2) HbA1c (%) = 8.3 (SD 8.0)	Type: PRE Muscles: shoulder horizontal F, shoulder F/abduction, hip E, knee F, knee E, shoulder adduction/E, elbow F, elbow E, ankle PF Intensity: 1-2 x 10-15RM x 3/wk x 16 wk Reps: 10-15	Type: aerobic exercise	Diabetes: HbA1c (%), glucose profiles Strength: leg press (kg) Body composition: LBM Adverse events: recorded Follow up: 0, 16 wk	

Study	Participants	Intervention		Outcome measures
		Experimental	Control	
Dunstan et al (1998)	n = 11 Age (yr) = 50 (SD 7) BMI (kg/m ²) = 28.3 (SD 2.7) HbA1c (%) = 8.2 (SD 1.7)	Type: PRE Muscles: abdominals, hip E, knee E, knee F, shoulder horizontal F, elbow E, elbow F, shoulder F/abduction, shoulder adduction/E, ankle PF, shoulder E/scapula retraction Intensity: 2–3 x 10–15 50–55% 1RM x 3/wk x 8 wk	Type: no intervention	Diabetes: HbA1c (%) Strength: leg press change from baseline (% change) Body composition: not measured Adverse events: recorded Follow up: 0, 8 wk
Dunstan et al (2002)	n = 16 Age (yr) = 68 (SD 5) BMI (kg/m ²) = 31.5 (SD 3.7) HbA1c (%) = 8.1 (SD 1.0)	Type: PRE Muscles: abdominals, hip E, knee E, knee F, shoulder horizontal F, elbow E, shoulder F, shoulder abduction, shoulder adduction/E Intensity: Wk 1–2: 3 x 8–10 50–60% 1RM Wk 3–24: 3 x 8–10 75–85% current 1RM	Type: no intervention (stretching)	Diabetes: HbA1c (%) Strength: leg press change from baseline (% change) Body composition: LBM Adverse events: recorded Follow up: 0, 12, 24 wk
Honkola et al (1997)	n = 18 Age (yr) = 62 (SD 9) BMI (kg/m ²) = 30.2 (SD 5.1) HbA1c (%) = 7.5 (SD 1.3)	Type: PRE Muscles: knee F, knee E, shoulder, trunk F, trunk E Intensity: 2 x 12–15 Borg moderate intensity x 2/wk x 20 wk	Type: no intervention	Diabetes: HbA1c (%) Strength: not measured Body composition: not measured Adverse events: not recorded Follow up: 0, 20 wk
Sigal et al (2007)	n = 64 Age (yr) = 55 (SD 8) BMI (kg/m ²) = 34.1 (SD 9.6) HbA1c (%) = 7.5 (SD 1.5)	Type: PRE Muscles: exercises on weight machines Intensity: 3 x 8–12 7–9RM x 3/wk x 26 wk	Type: no intervention	Diabetes: HbA1c (%) Strength: not measured Body composition: LBM Adverse events: recorded Follow up: 0, 12, 24 wk
Sigal et al (2007)	n = 64 Age (yr) = 55 (SD 8) BMI (kg/m ²) = 34.1 (SD 9.6) HbA1c (%) = 7.5 (SD 1.5)	Type: PRE Muscles: exercises on weight machines Intensity: 3 x 8–12 7–9RM x 3/wk x 26 wk	Type: aerobic exercise	Diabetes: HbA1c (%) Strength: not measured Body composition: LBM Adverse events: recorded Follow up: 0, 12, 24 wk

SD = standard deviation, BMI = body mass index, HbA1c = glycosylated haemoglobin, PRE = progressive resistance exercise, RM = repetition maximum, E = extension, F = flexion, PF = plantarflexion, FFM = fat free mass, LBM = lean body mass

weighted mean body mass index of 32.0 kg/m². Duration of diabetes ranged from a mean of 4.8 (Dunstan et al 1998) to 9.0 years (Cauza et al 2005a) with a weighted average duration of 7.2 years. Participants had a weighted average baseline HbA1c of 7.9% (SD 1.4) representing a slightly elevated level of HbA1c (Table 2). Cauza et al (2005a and b) were contacted and confirmed that the two trials involved different participants.

Intervention: All interventions occurred on three non-consecutive week days with the exception of one in which exercise occurred twice per week (Honkola et al 1997). Exercise was supervised in research centres or community settings utilising free weights or weight machines. Supervision was provided by physiotherapists, research assistants, nurses, or personal trainers. Interventions ran for a weighted average of 19.8 weeks, ranging from 8 weeks (Dunstan et al 1998) to 26 weeks (Sigal et al 2007). Sessions typically lasted 45–50 minutes and consisted of 2–3 sets (with the exception of Cauza et al 2005a and b where 1–2 sets were completed) of 8–15 repetitions of 5–10 exercises. All trials increased the intensity of training over the duration of the intervention. Different methods were used to progress training. Three trials based progression on 1RM tested at baseline and mid-study (Castaneda et al 2002, Dunstan et al 1998, Dunstan et al 2002), others progressed by increasing sets (Baldi and Snowling 2003, Baum et al 2007), or increasing resistance systematically when targets were reached (Cauza et al 2005a and b, Sigal et al 2007), and in one trial the method of progression was not detailed (Honkola et al 1997). The weighted average rate of adherence to progressive resistance exercise was 87% of scheduled sessions.

Outcome measures: All measurements of strength were of maximum voluntary force production by dynamometry or maximum weight moved (1RM). Three trials measured quadriceps strength, one measured the bench press, one measured both bench press and leg press, one measured strength of the upper and lower limb exercises, and one presented results as the sum of scores. The remaining two trials did not assess strength (Honkola et al 1997, Sigal et al 2007). Lean body mass was measured by dual x-ray absorbiometry (Castaneda et al 2002, Dunstan et al 2002) or calculated from skin fold measurements (Cauza et al 2005a and b). Fat-free mass was calculated by densitometry through hydrostatic weighing (Baldi and Snowling 2003) and bioelectrical impedance analysis (Sigal et al 2007). Baum et al (2007) was contacted and confirmed that measures of dispersion of HbA1c values were represented as standard deviations and not standard errors.

Effect of progressive resistance exercise

Glycosylated haemoglobin: When compared with non-exercising controls in seven trials, progressive resistance exercise lowered HbA1c by a small but significant amount (SMD -0.25, 95% CI -0.47 to -0.03) (Figure 2a, see also Figure 3a on the eAddenda for detailed forest plot). The absolute reduction of HbA1c was 0.3% (95% CI 0.0 to 0.6, I² 0%) with Baum et al (2007) excluded, and 0.1% (95% CI -0.1 to 0.30, I² 8%) with Baum et al (2007) included, with only 26 participants accounting for 62% of the weight (due to the very small standard deviations).

When compared with aerobic exercise in three trials, progressive resistance exercise had no significant effect on HbA1c (SMD -0.04, 95% CI -0.38 to 0.30, I² 13%) (Figure

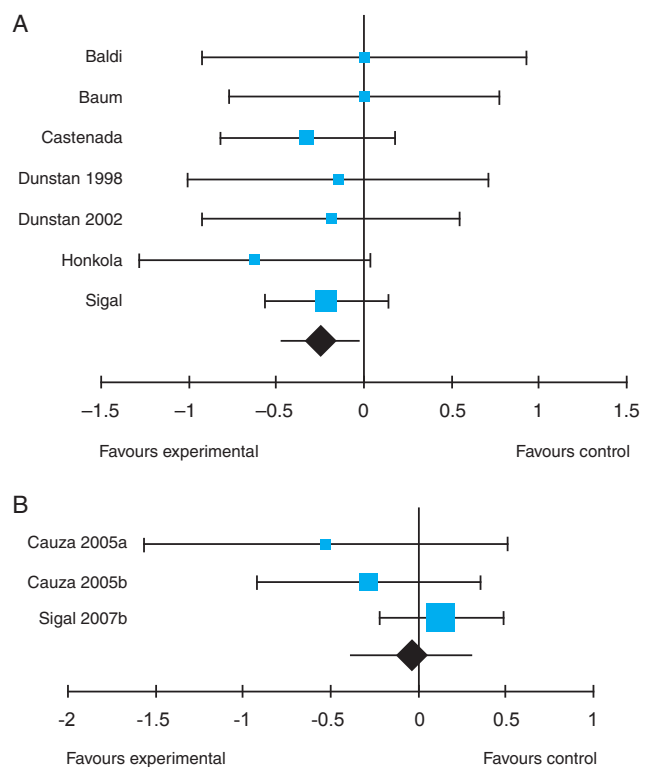


Figure 2. SMD (95% CI) of effect of progressive resistance exercise on HbA1c compared with a) no exercise by pooling data from 7 studies (n = 321), and b) aerobic exercise by pooling data from 3 studies (n = 178).

2b, see also Figure 3b on the eAddenda for detailed forest plot).

Strength: One trial that tested strength did not present post intervention strength data so was not included in the analyses (Dunstan et al 1998). When compared with non-exercising controls in four trials, progressive resistance exercise resulted in a large and significant increase in muscle strength (SMD 0.95, 95% CI 0.58 to 1.31, I² 34%) (Figure 4a, see also Figure 5a on the eAddenda for detailed forest plot). This was equivalent to a weighted average increase in muscle strength of 35%.

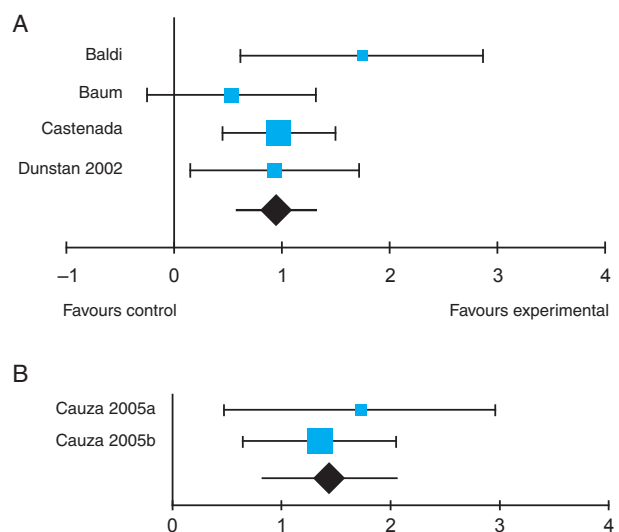


Figure 4. SMD (95% CI) of effect of progressive resistance exercise on muscle strength compared with a) no exercise by pooling data from 4 studies (n = 135), and b) aerobic exercise by pooling data from 2 studies (n = 44).

When compared with aerobic exercise in two trials, progressive resistance exercise resulted in a very large and significant increase in muscle strength (SMD 1.44, 95% CI 0.83 to 2.05, I^2 0%) (Figure 4b, see also Figure 5b on the eAddenda for detailed forest plot).

Body composition: When compared with non-exercising controls in 4 trials, progressive resistance exercise had no significant effect on body composition (SMD 0.13, 95% CI -0.14 to 0.40, I^2 5%) (Figure 6a, see also Figure 7a on the eAddenda for detailed forest plot).

When compared with aerobic exercise in 3 trials, progressive resistance exercise had no significant effect on body composition (SMD -0.08, 95% CI -0.37 to 0.22, I^2 0%) (Figure 6b, see also Figure 7b on the eAddenda for detailed forest plot.)

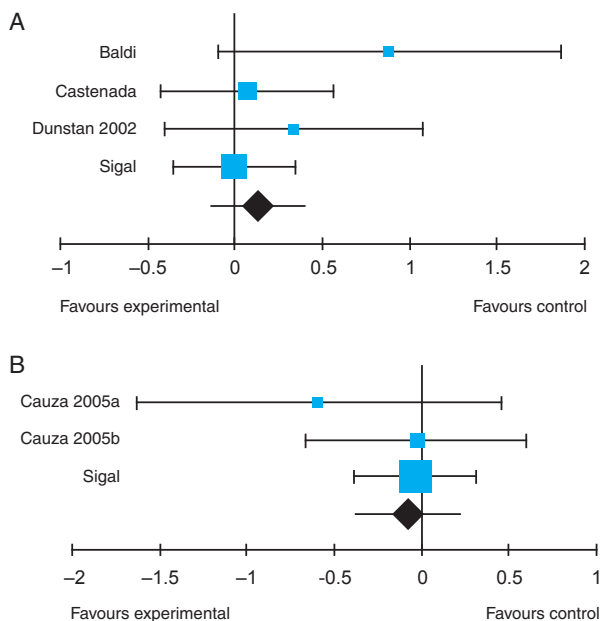


Figure 6. SMD (95% CI) of effect of progressive resistance exercise on body composition compared with a) no exercise by pooling data from 4 studies ($n = 236$), and b) aerobic exercise by pooling data from 3 studies ($n = 178$).

Safety: The presence or absence of adverse events was recorded in eight of the nine trials. Seven of these stated there were no exercise-related injuries or serious adverse events. The eighth trial, Cauza et al (2005a), reported there was no difference between the frequency of hyperglycaemic episodes in the progressive resistance exercise group compared with the aerobic exercise group. The most commonly reported problem was delayed onset muscle soreness.

Discussion

The results of this systematic review provide evidence from nine randomised trials involving 372 participants with type 2 diabetes that progressive resistance exercise improves glycaemic control by lowering glycosylated haemoglobin. In addition progressive resistance exercise leads to large increases in muscle strength for people with type 2 diabetes.

A 1% decrease in glycosylated haemoglobin is associated with a 37% decrease in the risk for microvascular complications and a 21% decrease in the risk of death associated with diabetes (Stratton et al 2000). The United Kingdom Prospective Diabetes Study concluded that any reduction in glycosylated haemoglobin is clinically significant as it is likely to reduce the risk of diabetic complications (Stratton et al 2000). One way of interpreting the results of this review is that participants who completed progressive resistance exercise made a 55% improvement towards the target value of glycosylated haemoglobin of 7.0% (where their glycosylated haemoglobin reduced by 0.5%, from 7.9% to 7.4%, during the intervention). It has long been recommended that the three cornerstones of diabetic therapy are medication, diet, and exercise (ADA 2008). Oral hypoglycaemic medications can reduce glycosylated haemoglobin by 0.5% to 2% (Willett et al 2004). These medications can be costly and are commonly associated with gastrointestinal side-effects (flatulence, nausea, diarrhoea, and abdominal pain) and weight gain (Willett et al 2004). Their efficacy also declines over time by 0.2% to 0.3% each year (Horan et al 2006). Dietary management including low GI, individualised meal plans and medical nutrition therapy can reduce glycosylated haemoglobin by 0.43% to 1% in people with established diabetes and is more effective in newly diagnosed people (Horan et al 2006). Therefore, compared with no exercise control groups, the overall absolute reduction of glycosylated haemoglobin of 0.3% found in this systematic review is small, suggesting that progressive resistance exercise should not be a stand-alone treatment for people with type 2 diabetes. However, the positive effect indicates that progressive resistance exercise may be part of a management plan, in combination with medication and an appropriate diet, to produce clinically significant reductions in complications associated with type 2 diabetes.

Snowling and Hopkins (2006) in their review reported a 0.5% decrease in glycosylated haemoglobin following progressive resistance exercise. The slight observed difference in results may be due to poorer quality trials in that review, the inclusion of trials of less than eight weeks duration and the smaller number of trials. The current review has also included a large trial of good quality which should be less subject to bias (Sigal et al 2007). Thomas et al (2006) reported a 0.6% decrease in glycosylated haemoglobin following exercise. Trials with combined training (both aerobic and resistance) were included in the Thomas review and may have an additive effect on results. The nine trials in the current review included trials of variable study quality ranging from a PEDro score of three to eight. The two trials that rated highest in trial quality (Castenada et al 2002, Sigal et al 2007) reported results similar to the meta-analysis. For the comparison of progressive resistance exercise with a no exercise control group, Castenada et al (2002) and Sigal et al (2007) reported standardised mean differences in glycosylated haemoglobin of 0.32 and 0.22, respectively, compared with the value of 0.25 from the meta-analysis in the current review. This suggests that the meta-analysis was not over-estimated due to influence of trials of lesser quality that would have been more subject to bias. Castenada et al (2002) and Sigal et al (2007) were also the two trials with the highest sample sizes, with well-described high intensity progressive resistance exercise interventions lasting from 16 to 26 weeks, suggesting that they also rated highly in the quality of the intervention as well as in quality of the study methods (Herbert and Bo 2005).

The results of the current systematic review show that progressive resistance exercise is significantly better than not exercising in reducing glycosylated haemoglobin to improve glycaemic control but it is not significantly better than aerobic exercise in achieving these improvements. This trend has been demonstrated before in a summary of systematic reviews on exercise by Taylor et al (2007). The authors found that across a broad range of musculoskeletal, cardiorespiratory, and neurological conditions exercise was better than not exercising; however, there was a lack of evidence to suggest that one type of exercise was better than another. One interpretation is that the choice of exercise for people with type 2 diabetes may be decided upon according to co-morbidities, personal preference, available resources, and a need for variety whilst knowing that progressive resistance exercise is a viable alternative to aerobic exercise.

Due to the small number and the mild nature of adverse events reported in the reviewed trials, progressive resistance exercise appears to be a relatively safe form of exercise for people with type 2 diabetes. Pollock et al (1991) established that there were twice as many complications during jogging and walking exercises when compared to strength training in older adults. Low rates of adverse events were also noted in a systematic review of progressive resistance exercise in community-dwelling older adults (Dodd et al 2002). Importantly, the participants in our review were overweight and previously sedentary, making it noteworthy that there were so few adverse events. However, it should also be considered that as reporting of adverse events was inconsistent and, as serious adverse events are rare, the sample sizes may have been too small to have detected serious adverse effects.

Complying with aerobic exercise recommendations can be challenging. Aerobic exercise needs to be completed on most, if not all, days of the week to be effective. The current review provides evidence that progressive resistance exercise can be effective when performed only three times a week and that compliance rates are high with 87% of scheduled sessions attended. Even walking may be difficult or risky because of co morbidities such as arthritis, cardiovascular disease, peripheral vascular disease, neuropathy, and mobility impairments. Progressive resistance exercise may be preferred by some older people with complications of diabetes. These include foot ulceration, Charcot's joint, amputations without prosthesis, angina, claudication, and people at risk of falls. In all these situations resistance exercise is not only a viable alternative, but it may also be more feasible than aerobic exercise.

For those who can participate safely in aerobic exercise, another option to consider is whether a combined exercise program is more beneficial than doing progressive resistance exercise program alone. Sigal et al (2007) reported that glycosylated haemoglobin values reduced significantly more in the combined exercise training group than in the aerobic or resistance exercise alone groups. However, the review of Snowling and Hopkins (2006) did not detect any significant additional benefits of combined training.

The results show that participants got stronger during the intervention but did not increase their lean body mass or fat free mass suggesting that the muscles became more efficient at glucose disposal without changing their morphology through hypertrophy. It is possible that measurement error in the methods of measuring body composition may have

obscured any true effects. However, the densitometry, dual X-ray absorptiometry and bioelectrical impedance techniques used in four of the six trials have demonstrated high precision in the measurement of body composition (Woodrow 2009). The two trials that estimated body composition based on skin fold measures (Cauza et al 2005a and b) may have had a limited ability to detect change (Woodrow 2009), although these trials only compared progressive resistance exercise to aerobic training, and so did not affect the meta-analysis comparing the intervention with no intervention. This leads to the question of whether longer trials that could influence muscle morphology might have a larger effect on glycaemic control, or whether participants trained with sufficient intensity to provide a stimulus to produce changes in muscle morphology. Since the average program ran for nearly 20 weeks, it appears that program length was sufficient. Ensuring adherence to protocol intensity for progressive resistance exercise, and ensuring frequent progression of the training intensity (as participants are starting from a low baseline of strength) may have the potential to further benefit glucose control.

A strength of this review is that it follows the QUOROM checklist for high quality reporting of systematic reviews (Moher et al 1999). It includes all recent and relevant trials, and an evaluation of adverse events. The results are clinically applicable since the included participants are typical of the diabetic population; higher risk with obesity, occurs most commonly in people over 40 years of age, with higher prevalence rates for males (AIHW 2008). The interventions are replicable as they were held in common, low-cost settings with readily available equipment, and were supervised by physiotherapists or other appropriately trained professionals.

A limitation of this review is that its conclusions rely on the quality of the included trials, where only three scored higher than four on the quality assessment scale. Good quality trials are less likely to be subject to bias and give the most accurate estimate of the effect of the intervention. Another limitation was that relevant data were not reported, or were only represented diagrammatically in some of the trials. For example, measures of strength in Baum et al (2007) were estimated from a figure. There was also a lack of long-term follow-up in the trials. Another limitation is that the meta-analysis of mean difference in percentage glycosylated haemoglobin may have been biased by the inclusion of a trial with very small standard deviations (Baum et al 2007), leading to this relatively small trial contributing more than 60% weight to the overall estimate of 0.1% glycosylated haemoglobin. We decided to complete a sensitivity analysis excluding this trial which resulted in a value of 0.3% glycosylated haemoglobin. This value was interpreted as more likely to reflect the true mean difference, as it was similar to the value reported in the largest high quality trial included in the review (Sigal et al 2007).

In conclusion, this systematic review has demonstrated that progressive resistance exercise leads to small but statistically significant improvements in glycosylated haemoglobin and therefore glycaemic control. The results are likely to be clinically significant since any improvement in glycaemic control that can be achieved safely is considered important. Eight weeks with two-three sessions of 45 minutes duration of progressive resistance exercise is sufficient to produce improvements in glycaemic control. Future research should focus on making progressive resistance exercise more

achievable and cost-effective by assessing the minimum duration of the program, minimum frequency of sessions and minimum duration of each session required to produce the desired benefits. Research should also address the possible additive benefits of combined aerobic and resistance exercise on glycosylated haemoglobin. Long-term trials would also be beneficial to determine how best to maintain these improvements in glycaemic control. Progressive resistance exercise is a feasible option to include in the management of glycaemic control for people with type 2 diabetes mellitus. ■

eAddenda: Appendix 1 Search strategy, and Appendix 2 Figures 3, 5, and 7, available at AJP.physiotherapy.asn.au

Support: Eastern Health, Victoria.

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