

Obesity Management

Resistance training for obese, type 2 diabetic adults: a review of the evidence

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Summary

In both developed and developing countries, increased prevalence of obesity has been strongly associated with increased incidence of type 2 diabetes mellitus (T2DM) in the adult population. Previous research has emphasized the importance of physical activity in the prevention and management of obesity and T2DM, and generic exercise guidelines originally developed for the wider population have been adapted for these specific populations. However, the guidelines traditionally focus on aerobic training without due consideration to other exercise modalities. Recent reviews on resistance training in the T2DM population have not compared this modality with others including aerobic training, or considered the implications of resistance training for individuals suffering from both obesity and T2DM. In short, the optimal mix of exercise modalities in the prescription of exercise has not been identified for its benefits to the metabolic, body composition and muscular health markers common in obesity and T2DM. Similarly, the underlying physical, social and psychological barriers to adopting and maintaining exercise, with the potential to undermine the efficacy of exercise interventions, have not been addressed in earlier reviews. Because it is well established that aerobic exercise has profound effects on obesity and T2DM risk, the purpose of this review was to address the importance of resistance training to obese adults with T2DM.

Keywords: Aerobic exercise, body weight, resistance training, type 2 diabetes mellitus.

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Introduction

The global epidemic of obesity (1) has contributed to a concomitant increase in the prevalence of type 2 diabetes mellitus (T2DM) (2,3). Both conditions are escalating at a frightening rate, are prevalent in adults and youth and are not restricted to the developed world (2–4). The increasing prevalence of obesity and the associated dysfunctional state of adipose tissue are the major contributors to the incidence of T2DM (5). Obesity and the cluster of conditions known as the metabolic syndrome (i.e. dysglycaemia, dyslipidemia, hypertension and a procoagulant state) (6)

are the precursors of T2DM and cardiovascular disease (7). Given the close interplay between obesity and T2DM in many individuals, the term ‘diabesity’ has been used to portray this relationship (8). Because of its growing prevalence, greater attention is required regarding how best to treat diabesity. Lifestyle modification, specifically changes in diet, physical activity (PA) and exercise is considered the cornerstone of both obesity and T2DM management (9). PA substantially reduces the risk of T2DM, particularly in individuals with an increased baseline risk such as the obese (10). PA guidelines for the adult population have evolved over the last decade, as seen in Fig. 1. The 1995

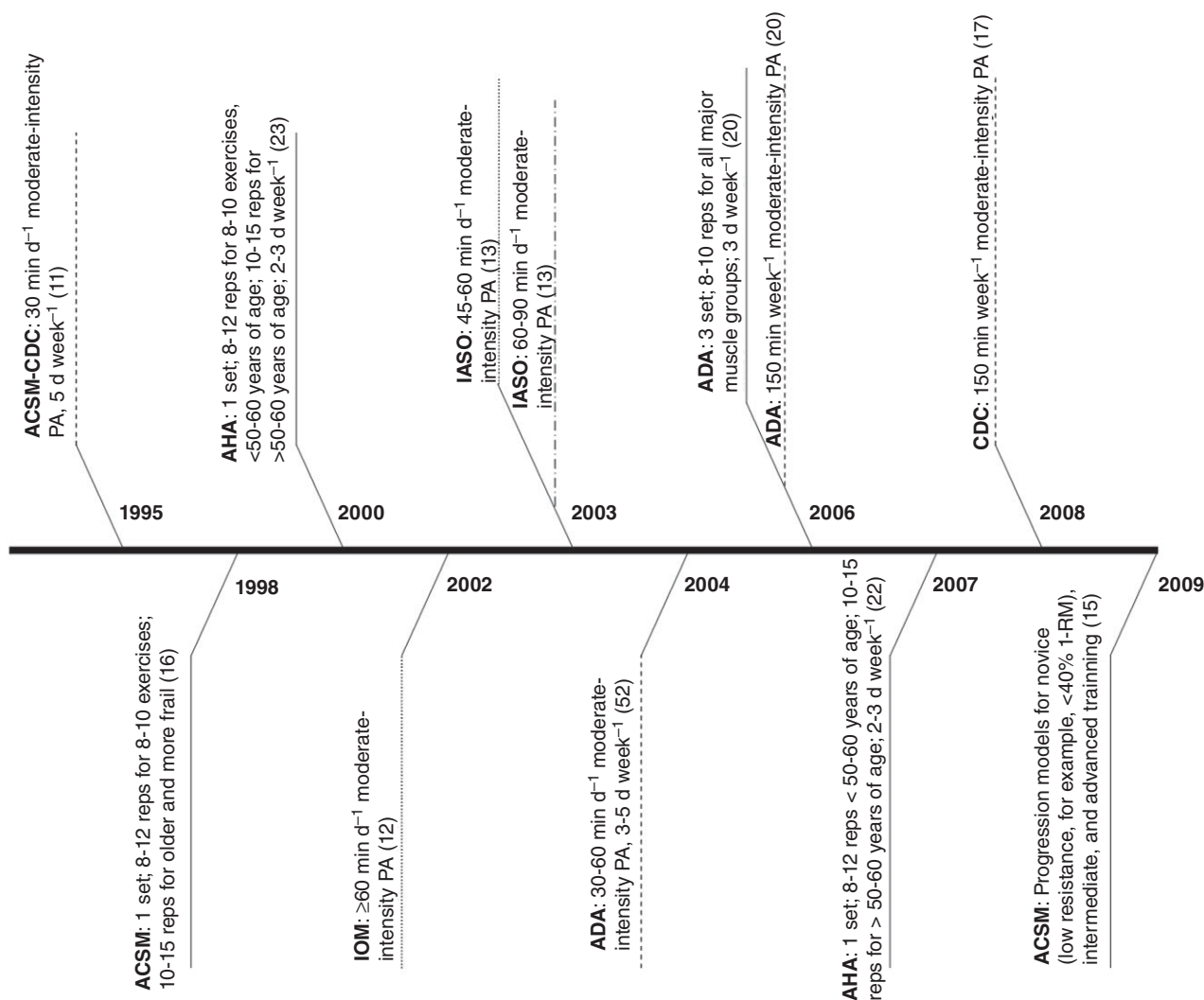


Figure 1 Chronology of consensus recommendations regarding PA/exercise for weight management and type 2 diabetes mellitus in adults. The lines indicate factors that were promoted by PA. Dashed lines indicate health improvements; dotted lines indicate prevention of weight gain. Dash-dot lines indicate maintenance of weight loss, and solid lines indicate resistance training. 1 RM, 1 repetition maximum; ACSM, American College of Sports Medicine; ADA, American Diabetes Association; AHA, American Heart Association; CDC, Centers for Disease Control and Prevention; IASO, International Association for the Study of Obesity; IOM, Institute of Medicine; PA, physical activity; reps, repetitions.

American College of Sports Medicine (ACSM) and the Centers for Disease Control and Prevention recommendations for PA and health included 30 min d⁻¹ (or more) of at least moderate-intensity PA on most days of the week and was largely framed with respect to cardiovascular benefits (11). With the dramatic increases in obesity rates in recent decades, and the recognition of the integral role of PA in weight management, studies and guidelines by the Institute of Medicine (12), International Association for the Study of Obesity (13), and the ACSM (14-16) consistently support the need for more than 150-250 min week⁻¹ of moderate-intensity PA to prevent weight gain and maintain weight loss. Additionally, the 1996 Surgeon General's Report (17) referenced the benefits of PA for

diabetes and obesity, indicating that regular PA lowers the risk of developing T2DM and may favourably affect body fat distribution. However, specific PA recommendations for adults with T2DM were not developed by the ACSM until 2000 (18). T2DM patients were recognized as having lower fitness levels than healthy individuals, and exercise intensity was set at a rating of perceived exertion of 10-12, or low- to moderate-intensity for 10-30 min d⁻¹ (3-4 d week⁻¹). The general guidelines suggested a minimum activity expenditure of 1000 kcal week⁻¹, with a frequency (3 non-consecutive days) necessary for the maintenance of blood glucose levels.

Traditionally, aerobic training (AT) has been promoted as the most effective mode of exercise for treating T2DM

with improvements in lipid profiles and insulin sensitivity (19). AT, especially high-intensity AT, is metabolically demanding on skeletal muscle, which is a primary target for insulin transport. Therefore, it is suggested that AT may be a useful tool in the prevention and treatment of T2DM (20). Mechanisms for the AT-induced improvement in insulin sensitivity and lipid profile are unknown, although a number of potential metabolic factors have been identified for affecting insulin signalling and insulin-responsive glucose transporter 4 up-regulation and transport. Some of these include decreased cytokines, decreased energy surplus in the skeletal muscle, up-regulation of uncoupling protein 3, more efficient fatty acid metabolism and increased skeletal muscle mitochondrial function (21). More recently, published guidelines have recognized the importance of strength or resistance training (RT) (22,23) and acknowledge the potential role for progressive RT to increase muscle mass, and in turn, 24-h energy expenditure (15). RT normally involves lifting weights (machines or free weights) typically at loads greater than 65% of the one repetition maximum (1 RM).

The 2007 (22) and 2009 (24) statements by the American Heart Association and ACSM underline the role of RT to enhance muscular strength and endurance, functional capacity and independence, and quality of life, while the recent ACSM statement highlights the importance of progression in RT with particular detail devoted to individuals with different levels of training (15). RT has also received considerable attention in recent research literature because of its ability to improve glycaemic control and enhance muscular strength and endurance in T2DM patients (25–27). Despite the level of interest in the field, current guide-

lines do not specifically address obesity and/or T2DM. Therefore, the purpose of this review was to focus on the potential and unique effect RT may have on T2DM and obesity (recognizing the existing barriers to exercise experienced by this population).

Obesity, body fat distribution and type 2 diabetes mellitus

Obesity is characterized by an excess accumulation of total body fat that influences metabolic processes and predisposes an individual to chronic non-communicable diseases. While total body fat is important, the distribution of excess fat in the abdominal or gluteal regions modifies the health risk profile. Abdominal obesity is particularly relevant and can be further subdivided into subcutaneous and intra-abdominal (visceral) fat regions. Visceral adiposity is 'traditionally' implicated in the aetiology of a number of metabolic complications and is expected to decrease the sensitivity of target tissues to insulin (28) (Fig. 2). There is some evidence that subcutaneous fat may also contribute to insulin resistance and the metabolic syndrome (28). Concomitant decreases in insulin resistance and abdominal fat, specifically in the subcutaneous compartment, would support the relationship between this anatomical compartment and insulin resistance (29,30).

The few studies that have specifically addressed the influence of RT on abdominal fat consistently show a decrease in visceral fat (31–34). Recent unpublished research has found that following a 15% (12 kg) diet-induced weight loss, individuals who participated in RT lost 37% of their visceral fat and increased insulin sensitivity 49%. The results

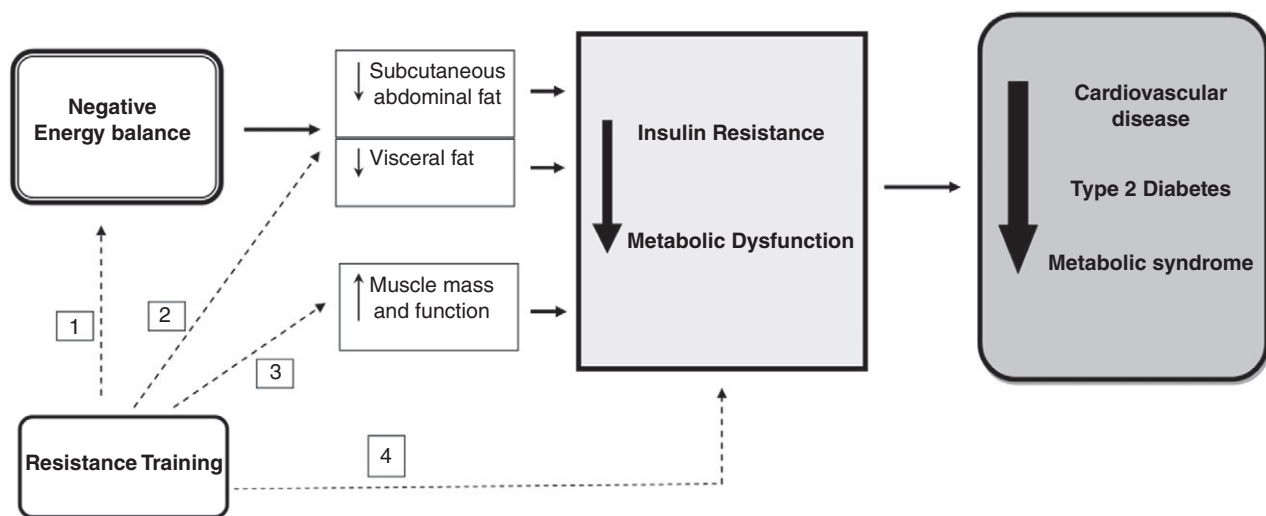


Figure 2 Possible schema for the relationship between visceral obesity and insulin resistance. Resistance training may (i) Promote a negative energy balance through increased energy expenditure and reduced food intake; (ii) Decrease fat mass and change body fat distribution; (iii) Increase muscle mass, strength and function; or (iv) Reduce insulin resistance/metabolic dysfunction over and above changes in body composition (28).

of the RT group were 29% higher than women who lost 15% of their weight but did not exercise (GR Hunter *et al.*, unpublished data). In addition, during the year following the weight loss those who participated in RT did not regain any of their visceral fat while participants who did not train regained over 70% of the visceral fat lost (35). Moreover, abdominally obese participants who completed a diet plus RT weight loss programme showed a significantly lower total body fat regain and a lower android to gynoid fat ratio after 12 weeks of free-living compared with similar participants who underwent diet alone weight loss (36). Hence RT has the potential to reduce metabolically active fat deposits through both immediate (during weight loss or weight maintenance) and delayed effects (on weight regain). While the visceral compartment has received the most attention, there is a need to examine the effect of RT on the contribution of the subcutaneous deposit to whole body insulin sensitivity.

Body composition and ageing

Biological ageing is typically associated with a progressive increase in body fat mass (FM) and a loss of fat-free mass (FFM), particularly skeletal muscle. Body fat in adults may double between the ages of 20 and 60 years (37,38), even in

those whose weight remains relatively constant, thereby increasing the risk of diabetes and sarcopenic obesity. Health risk is greatly increased beyond what would be expected from a simple adjustment in the ratio of stored energy between muscle and fat. Visceral fat increases by over 300% between the ages of 25 and 65 years, and this creates an increased risk for the development of T2DM and cardiovascular disease in women with normal body mass index values (<25 kg m⁻²) (39). The underlying reasons for this age-related increase in FM and loss of muscle mass/function are not well understood; however, it is likely that an age-related decrease in PA contributes to the problem. In Fig. 3, it is hypothesized that losses of muscle and muscle function is debilitating for PA, which leads to reduced PA and fat gain. Simultaneously, FFM loss leads to reduced resting energy expenditure that facilitates a positive energy balance and leads to further weight gain. RT has been shown to increase muscle size (40), decrease difficulty in activities of daily living (41,42), increase resting (40,43) and total energy expenditure (40), as well as reduce total and especially visceral (31,34). These data suggest that RT has a unique effect on T2DM by slowing sarcopenia and thus slowing gain in body fat, particularly visceral fat. Because RT is metabolically less demanding than AT, it is

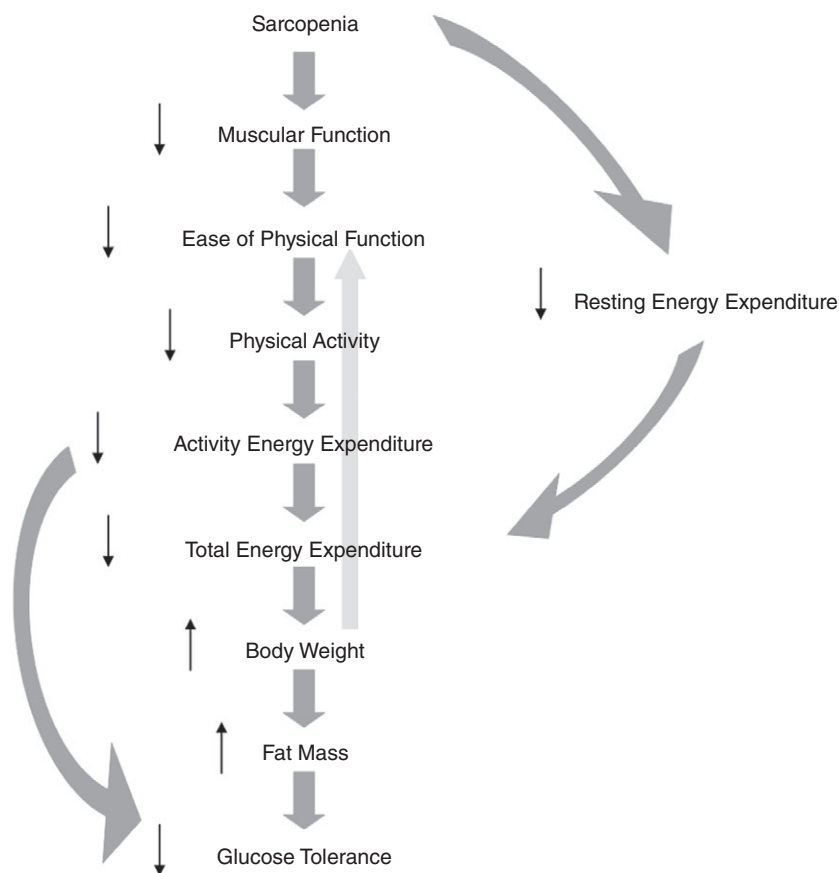


Figure 3 Model for sarcopenia influencing glucose tolerance. Dark grey arrows indicate unidirectional relationships. Light grey arrow indicates the negative feedback loop between an increased body weight and decreased capacity for physical activity.

hypothesized that a combination of AT and RT would induce unique benefits on risk for T2DM. AT would increase metabolic demand in skeletal muscle and improve insulin signalling while RT would be most productive in slowing the development of sarcopenic obesity.

Implications of exercise

A review of literature was conducted through MEDLINE databases using the following keywords (individually and in combination): RT, strength training, circuit training, diabetes, metabolic syndrome, overweight and obesity. Studies were excluded if (i) The publication date was prior to 1998; (ii) The study did not include T2DM participants with a mean BMI ≥ 25 kg m⁻²; (iii) There was no RT intervention

or the intervention was less than 6 weeks in duration; and (iv) A control group was not included as part of the research design. Based on these criteria, 13 studies were included for review.

Table 1 details the dose characteristics for the included studies. The majority of the studies had a duration of at least 4 months (43–48), with 2–3 non-consecutive days of PA per week (43–51). Eight studies (50,51) included an aerobic component, either as part of the independent variable or included in the combination exercise training protocol. All aerobic activities were completed between 60% and 85% of the participant's VO_{2max} or maximal heart rate and varied greatly in duration, ranging from 15 to 75 min d⁻¹ of PA (50,51). RT protocols were typically based on the determination of 1 RM (60–80%) for each

Table 1 Study characteristics for reviewed interventions involving obese adults with T2DM

Study	<i>n</i>	Intervention (<i>n</i>)	Frequency (d week ⁻¹)	Intensity	Duration	Length
Anderssen <i>et al.</i> (43)	188	EX (48) Diet (45) Diet + EX (58) CON (37)	3	60–80% max HR	60 min	12 months
Castaneda <i>et al.</i> (44)	62	RT (31) CON (31)	3	8 reps, 60–80% 1 RM	35 min; 3 sets (5 ex)	16 weeks
Cauza <i>et al.</i> (19)	39	AT (17) RT (22)	3	60% VO _{2peak} , 10–15 reps, or fatigue	up to 30 min 3–6 sets	4 months
Cuff <i>et al.</i> (45)	28	CON (9) AT (9) AT + RT (10)	3	60–75% HR reserve 60–75% HR reserve, 12 reps	75 min 75 min; 2 sets (5 ex)	16 weeks
Dunstan <i>et al.</i> (46)	29	RT (16) CON (13)	3	8–10 reps, 50–85% 1 RM	45 min, 3 sets (9 ex)	6 months
Dunstan <i>et al.</i> (49)	21	CT (11) CON (10)	3	10–15 reps, 50–55% 1 RM	2–3 sets	8 weeks
Honkola <i>et al.</i> (47)	38	CT (18) CON (20)	2	12–15 reps, moderate intensity	2 sets, 8–10 ex	5 months
Irving <i>et al.</i> (29)	27	CON (7) Low intensity (11) High intensity (9)	5 5	300–400 kcal per session (RPE 10–12) 3x/week (RPE 15–17); 2x/week (RPE 10–12)	Variable Variable	16 weeks
Lambers <i>et al.</i> (50)	46	CT (17) AT (18) CON (11)	3	10–15 reps, 60–85% 1 RM 60–85% HR	3 sets 50 min	3 months
Loimaala <i>et al.</i> (48)	49	CON (25) EX (24)	2 2	65–75% VO _{2peak} 10–12 reps, 70–80% 1 RM	30 min 3 sets, 8 ex	12 months
Maiorana <i>et al.</i> (51)	16	CT (8) CON (8)	3	45 s ex ⁻¹ ; 55–65% 1 RM; 70–85% max HR	1–3 sets, 7 ex	8 weeks
Sigal <i>et al.</i> (20)	251	AT (60) RT (64) AT + RT (64) CON (63)	3 N/A	60–75% VO _{2peak} 7–9 reps to fatigue AT + RT N/A	15–45 min 2–3 sets AT + RT N/A	26 weeks
Stewart <i>et al.</i> (30)	104	EX (51) CON (53)	3	10–15 reps (50% 1 RM) 60–90% max HR	2 sets (7 ex); 45 min CV	26 weeks

1 RM, 1 repetition maximum; 2x/week, 2 sessions per week; 3x/week, 3 sessions per week; AT, aerobic training; CON, control; CV, cardiovascular activity; CT, circuit training; ex, exercise; EX, exercise group; HR, heart rate; N/A, not applicable; reps, repetitions; RPE, rating of perceived exertion; RT, resistance training; T2DM, type 2 diabetes mellitus.

Table 2 Outcomes of exercise interventions for obese adults with T2DM

Study	Metabolic	Cardiovascular	Physical function	Body composition
Anderssen <i>et al.</i> (43)	N/A	N/A	N/A	NS
Castaneda <i>et al.</i> (44)	RT: ↓ levels of HbA _{1c}	RT: ↓ SBP	RT: ↑ strength	RT: ↑ LBM
Cauza <i>et al.</i> (19)	RT: ↓ levels of fasting plasma glucose, HbA _{1c} , insulin resistance, and total cholesterol AT: NS	RT: ↓ SBP, DBP AT: ↓ SBP, DBP	RT: ↑ upper and lower extremity strength AT: ↑ lower extremity strength	RT ↓ fat mass, body fat %, total fat; ↑ LBM AT ↓ fat mass, body fat %, total fat
Cuff <i>et al.</i> (45)	NS	N/A	AT: ↑ VO _{2peak} AT + RT: ↑ VO _{2peak}	NS
Dunstan <i>et al.</i> (46)	RT: ↓ levels of HbA _{1c}	RT: ↓ SBP, DBP	RT: ↑ upper and lower extremity strength	RT: ↓ body weight, waist circumference, body fat
Dunstan <i>et al.</i> (49)	NS	NS	N/A	CT: ↓ BMI
Honkola <i>et al.</i> (47)	CT: ↓ levels of LDL, HbA _{1c}	CT: ↓ DBP	CT: ↑ abdominal, back, biceps, quadriceps strength	CT: ↓ body weight
Irving <i>et al.</i> (29)	N/A	N/A	High intensity: ↑ VO _{2peak} Low intensity: ↑ VO _{2peak}	High intensity: ↓ BMI, body weight, waist circumference, fat mass, amount of abdominal (subcutaneous, visceral) fat
Lambers <i>et al.</i> (50)	CT: ↓ levels of HbA _{1c} , total cholesterol AT: NS	N/A	CT: ↑ upper and lower extremity strength	NS
Loimaala <i>et al.</i> (48)	EX: ↓ levels of HbA _{1c}	EX: ↓ SBP, resting HR	EX: ↑ VO _{2peak} , lower extremity and abdominal strength	NS
Maiorana <i>et al.</i> (51)	CT: ↓ levels of HbA _{1c} , fasting plasma glucose	CT: ↓ resting HR	CT: ↑ VO _{2peak} , total strength	CT: ↓ body fat %
Sigal <i>et al.</i> (20)	AT: ↓ levels of HbA _{1c} RT: ↓ levels of HbA _{1c} AT + RT: ↓ levels of HbA _{1c}	NS	N/A	AT: ↓ BMI, body weight, waist circumference, fat mass, abdominal subcutaneous fat RT: ↓ waist circumference, abdominal subcutaneous fat
Stewart <i>et al.</i> (30)	EX: ↑ levels of HDL	EX: ↓ DBP	EX: ↑ VO _{2peak} , total strength	EX: ↓ BMI, body weight, waist circumference, body fat %, abdominal subcutaneous and visceral fat; ↑ LBM

AT, aerobic training; BMI, body mass index; CT, circuit training; DBP, diastolic blood pressure; EX, exercise group; HDL, high-density lipoprotein; HR, heart rate; LBM, lean body mass; LDL, low-density lipoprotein; N/A, not applicable; NS, not significant; RT, resistance training; SBP, systolic blood pressure; T2DM, type 2 diabetes mellitus.

exercise. When details were provided, training programmes utilized 5–10 exercises with 8 exercises being the most common strategy. Each exercise was performed in 2–4 sets of 5–15 repetitions, often to fatigue (45–51). The protocols for all interventions followed the general guidelines set by the ACSM and American Diabetes Association (18,20,52).

Table 2 presents the benefits and outcomes from studies comparing different types of training interventions. Exercise interventions promote increases in physical strength and aerobic fitness; two variables that have been highly correlated to improved glucose tolerance. An acute bout of exercise will begin to affect metabolism through the insulin-independent increase of glucose uptake, as well as increases in insulin-dependent glucose transport and glycogen synthesis. The primary mediator for this change in glucose transport is the activation of adenosine

monophosphate-activated protein kinase (AMPK) by muscle contraction. Repeated activation of AMPK can increase muscle oxidative capacity and VO_{2max} (53). The increased fitness levels (objectively measured using VO_{2max}) presented after an AT intervention would suggest increased levels of AMPK and therefore, increased insulin sensitivity. AT interventions and combinations of AT and RT consistently increased VO_{2peak} (29,30,45,48,51), with studies also showing concomitant improvements in insulin resistance (48,51). Conversely, the direct targeting of large areas of skeletal muscle, as specifically seen in the increased strength parameters during RT interventions (50,51), would increase the amount of muscle contraction and improve skeletal muscle insulin sensitivity (50,51). The effects of exercise on glucose transport can last for up to 48 h, requiring T2DM patients to be physically active at least 3–4 d week⁻¹ in order to maintain stable glucose uptake.

The changes in body composition as a result of exercise intervention have the most far-reaching effects on an individual's health. Adipose tissue has a large impact on the pathology of inflammation through its production and release of pro-inflammatory markers, such as interleukin-1 and interleukin-6, tumour necrosis factor alpha and C-reactive protein (54). Subclinical inflammation has been identified as an important factor associated with the increased risk of cardiovascular disease, metabolic syndrome and T2DM. Previous research has indicated that modest and habitual exercise decreases the cytokines associated with insulin resistance (53). RT, AT and combination interventions consistently report decreased fat deposition including FM, body fat percentage, total fat and abdominal subcutaneous fat along with subsequent decreases in glycated haemoglobin (HbA_{1c}) and fasting plasma glucose (19,26,29,30,46,51), supporting the relationship between adipose tissue and glucose tolerance. Cardiovascular risk factors were indirectly measured in several exercise intervention studies. RT interventions showed decreases in total cholesterol (47,50), low-density lipoprotein cholesterol (47) and triglycerides (47), as well as decreased diastolic and systolic blood pressure (46–48). The cardiovascular benefits of RT could be instrumental in preparing an overweight individual to sustain more demanding aerobic activity.

Challenges for obese, type 2 diabetes mellitus adults to follow exercise guidelines: barriers to exercise

A range of physical, psychological and psychosocial factors could reduce the rate of exercise adoption and adherence in

obese patients with T2DM (Fig. 4). Encouraging exercise adoption and compliance is difficult in any population; however, in people with chronic disease the problem is exacerbated by barriers specific to the disease. The situation is compounded by evidence that demonstrates that taking medication is more easily maintained than regular exercise and is therefore perceived as a more manageable alternative (55).

The factors that potentially prevent the adoption of, or adherence to exercise in people with obesity and T2DM, include attitudes and beliefs about exercise and the disease (56), previous experience of exercise as a method of managing the disease (57), and the complication or intrusiveness of the disease itself. Common barriers specific to RT include the fear of injury, low self-efficacy of a novel or unfamiliar mode of exercise and the assumption that exercise will lead to increase in muscle mass, which in turn will increase body weight. Obese, T2DM individuals are also disadvantaged by physical (i.e. body weight) and cardiovascular constraints as a function of excess body weight and the effort involved in moving a large mass. This movement challenge is accentuated during weight-bearing tasks (e.g. jogging, running) and creates an increased risk of injury and pain-related intolerance (58). Additionally, participation in high-intensity exercise commonly results in some discomfort and associated increases in core temperature, sweating and hyperventilation. However, there is the potential to resolve each of these issues by prescribing RT and thereby limiting weight-bearing exercise or moving the body over a given distance. If appropriate recommendations and guidelines are followed when prescribing RT, musculoskeletal adaptation can be improved, and the risk of muscle soreness and injury reduced. Such an approach

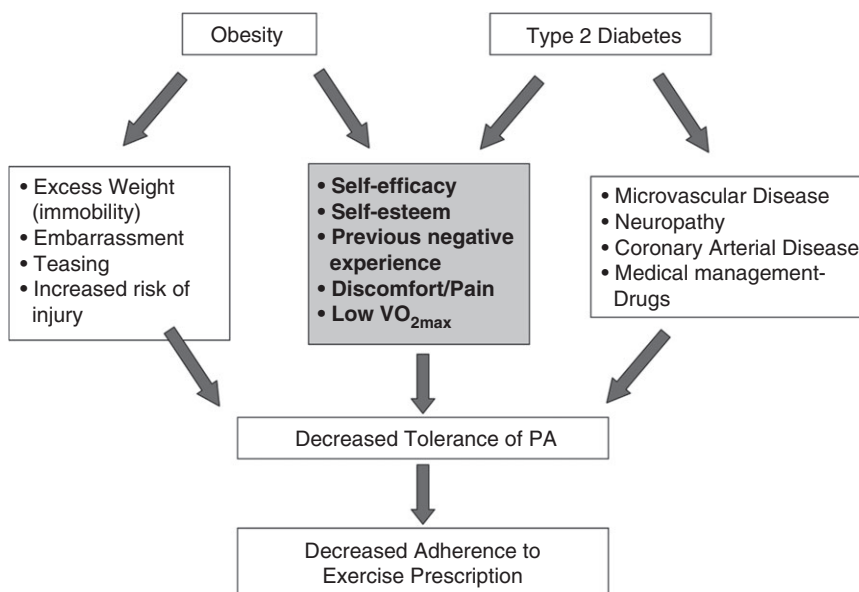


Figure 4 Barriers to physical activity (PA) in obese individuals. Adapted from Lyell *et al.* (68).

may be an appropriate forerunner to the introduction or extension of AT based on individual capacity.

There is also evidence to demonstrate that individuals with insulin resistance have a reduced physical work capacity compared with individuals who do not (59). Therefore, a more appropriate strategy for obese individuals with T2DM is to progressively increase exercise intensity and load, which can also facilitate compliance (60). High-intensity weight-bearing exercise has the potential to be over-prescribed and lead to a reduction in self-reported pleasure in overweight individuals (61). The change in perceived tolerance and pleasure (i.e. affective response) of PA negatively influences compliance (62). A variety of mild-to moderate-intensity exercise modalities that can be easily maintained and performed at regular intervals has been recommended. Typically, the literature has recommended that short bouts of high-intensity exercise be avoided as they may prove difficult to maintain in the long term (63); however, more research is required in this area. In short, if prescribed correctly and with appropriate supervision, RT is a useful alternative to AT as it has the potential to overcome some of the common barriers to exercise and simultaneously provides metabolic and physiological benefits.

Future research

An increasing body of research has helped to substantiate the importance of different exercise modalities in relation to the health and fitness of obese, T2DM populations. However, the previous focus has been primarily on the efficacy of exercise on metabolic improvement compared with dietary or pharmaceutical interventions or a combination of diet and exercise (64–66), without considering the factors that might prevent compliance to the intervention (27). Because of the reduced exercise compliance commonly seen in obese, T2DM individuals, there is an urgent need to explore strategies that successfully increase PA and exercise and maximize compliance. This is specifically true when considering RT interventions, an area that is still relatively undeveloped. There is evidence to suggest that despite some commonality regarding barriers to RT and AT exercise, the prescription of RT, particularly in a circuit training context could overcome some of these barriers. In addition, RT has been shown to affect certain body composition measurements, specifically total and abdominal subcutaneous fat; however, less research has addressed the consequences of RT on visceral fat. Therefore, more research is needed to better understand the relationships between RT, visceral and subcutaneous fat deposits, and resulting changes to insulin sensitivity.

Specific populations, including different ethnic groups have varying risks of cardiovascular and metabolic consequences associated with excess adiposity; however, to date

the majority of intervention studies have been undertaken with Caucasian participants in developed countries. For example, Asian Indians and South Asians have less muscle mass and high body fat, and a disproportionately high prevalence of diabetes and cardiovascular disease and may benefit significantly from RT (67). Similarly, interventions have primarily focused on the impact of exercise training in a specific age range (35–55 years). Although T2DM was once considered an adult complication of obesity, the age of onset has steadily declined with both physical and socioeconomic implications. It is critical that appropriate exercise modalities are incorporated into the management of obesity and T2DM from a young age. Conversely, sarcopenic obesity affects individuals who are typically over the age of 50 years. The benefits for lean body mass as a function of regular participation in RT mean that this area is critical to the successful intervention in those suffering from sarcopenic obesity. However, there is a dearth of definitive evidence regarding the use of different exercise modalities in the older adult with multiple health conditions.

Conclusion

Aerobic training and RT interventions provide benefits to features of metabolic and cardiovascular health, and fitness. Because RT does not require a high level of cardio-respiratory fitness to commence, this form of exercise represents a logical starting point in a weight management programme, particularly as muscular strength and endurance are required in all activities of daily living. Current evidence suggests that additional improvements are possible in interventions with combined modalities (RT and AT), therefore both components are necessary for optimal results. As untrained, obese and diabetic individuals are commonly challenged during aerobic-only tasks because of low fitness levels, RT should be a foundational component of exercise prescription for such individuals and thereby optimize for the chance of later engagement in aerobic activity.

Conflict of Interest Statement

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