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Resistance Exercise Training Its Role in the Prevention of Cardiovascular Disease

Randy W. Braith, PhD; Kerry J. Stewart, EdD

The metabolic effects of reduced muscle mass, engendered by normal aging or decreased physical activity, lead to a high prevalence of obesity, insulin resistance, type 2 diabetes, dyslipidemia, and hypertension.¹⁻⁴ These risk factors are associated with abnormalities in cardiovascular structure and function such as arterial stiffness and impaired endothelial function. Skeletal muscle is the primary metabolic “sink” for glucose and triglyceride disposal and is an important determinant of resting metabolic rate. Accordingly, it has been hypothesized that resistance exercise training (RT) and subsequent increases in muscle mass may reduce multiple cardiovascular (CV) disease risk factors.⁵⁻⁸ The inclusion of RT as part of an exercise program for promoting health and preventing disease has been endorsed by the American Heart Association,⁹ American College of Sports Medicine,¹⁰ and the American Diabetes Association¹¹ as an integral part of an overall health and fitness program. Cross-sectional studies have shown that muscular strength is inversely associated with all-cause mortality¹² and the prevalence of metabolic syndrome,^{13,14} independent of cardiorespiratory fitness levels. To date, however, the evidence that RT reduces CV risk factors remains equivocal.

This review will critically evaluate whether RT modifies CV risk factors and improves characteristics of CV structure and function. The topics will be limited to the effects of RT on major and independent risk factors for CV disease including diabetes mellitus, hypertension, dyslipidemia, and advancing age.⁴ The quantitative relation between these risk factors and CV events has been elucidated by the Framingham Heart Study⁴ and other studies. The topics will also include 2 predisposing risk factors—obesity and physical inactivity—that are designated as major risk factors by the American Heart Association.^{1,2,4} To the extent possible, this review will examine the separate and independent effects of RT in studies that did not include a concomitant aerobic exercise component. However, in those instances where the data from RT studies are equivocal, studies that combined RT and aerobic exercise will be acknowledged to help the clinician formulate recommendations for their patients. Additionally, the review will focus mainly on primary prevention, for example, risk reduction in persons without established CV disease. Many low- to moderate-risk patients with established CV disease should be encouraged to incorporate

RT into their physical conditioning program, especially those who rely on their upper extremities for work or recreational pursuits. However, the safety and effectiveness of RT in other populations of CV patients (eg, women, older patients with low aerobic fitness, patients with severe left ventricular dysfunction) have not been well studied. Accordingly, these patient subsets may require more careful evaluation and initial monitoring, and RT guidelines and recommendations must be modified accordingly. Moreover, there is only a limited body of literature assessing the independent benefits of RT on CV risk factors in patients with established CV disease. Studies conducted in cooperation with comprehensive cardiac rehabilitation programs typically include the confounding influences of aerobic activity, initiation of vasoactive and lipid-lowering drugs, and nutritional education with subsequent dietary modifications.

Rationale for Resistance Training

There is overwhelming research evidence that RT prevents decline in skeletal muscle mass and function when the mechanical stimuli provided by tasks of daily living are not sufficient to offset these declines with aging.¹⁵⁻¹⁷ Adults who do not perform regular RT lose approximately 0.46 kg of muscle per annum from the fifth decade on.¹⁸ Furthermore, adults who do not perform RT experience a 50% reduction in type 2 muscle fibers, the fibers responsible for high levels of strength, by age 80 years.¹⁹ The profound beneficial effects of RT on the musculoskeletal system can contribute to the maintenance of functional abilities and prevent osteoporosis, sarcopenia, and accompanying falls, fractures, and disabilities.^{15,17,18} A comprehensive comparison of the chronic effects of RT versus aerobic exercise training in multiple organ systems is presented in Table 1.

Long-term adaptation to RT lowers cortisol response to acute stress,²⁰ increases total energy expenditure and physical activity in healthy^{18,21} and frail older adults,²² and relieves anxiety, depression, and insomnia in clinical depression.²³ RT has beneficial effects on bone density,^{18,24} osteoarthritic symptoms,^{22,25} hypertension,^{26,27} lipid profiles,²⁸ and exercise tolerance in coronary artery disease.²⁹ Conversely, the loss of skeletal muscle mass and contractile function that accompanies aging, for example, sarcopenia, is linked to peripheral insulin resistance, dyslipidemia, and increased adiposity.³⁰

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TABLE 1. Comparison of the Effects of Aerobic Training to Resistance Training on Health and Fitness Variables

Variable	Aerobic Exercise	Resistance Exercise
Bone mineral density	↑	↑ ↑ ↑
Body composition		
Fat mass	↓ ↓	↓
Muscle mass	↔	↑ ↑
Strength	↔	↑ ↑ ↑
Glucose metabolism		
Insulin response to glucose challenge	↓ ↓	↓ ↓
Basal insulin levels	↓	↓
Insulin sensitivity	↑ ↑	↑ ↑
Serum lipids		
High-density lipoprotein	↑ ↔	↑ ↔
Low-density lipoprotein	↓ ↔	↓ ↔
Resting heart rate	↓ ↓	↔
Blood pressure at rest		
Systolic	↓ ↓	↓
Diastolic	↓ ↓	↓
Physical endurance	↑ ↑ ↑	↑ ↑
Basal metabolism	↑	↑ ↑

↑ indicates increased; ↓, decreased; and ↔, negligible effect.

Sarcopenia is also accelerated in patients with chronic heart failure, a condition characterized by peripheral skeletal muscle abnormalities and muscle wasting. Several studies have shown beneficial effects of RT on muscle mass and strength in patients with chronic heart failure.^{31,32} However, the relative mitigating effects of RT on primary and secondary CV risk factors remains controversial.

Resistance Training and Diabetes

Diabetes mellitus, glucose intolerance, and insulin resistance are central features of coronary artery disease risk, being strongly related to hypertension and dyslipidemia, proinflammatory markers, thrombogenic factors, and endothelial dysfunction. These abnormalities increase with age and represent the early stages of CV disease that precede the clinical manifestations of CV disease.³³ Maintaining good glycemic control hinges on enhancing insulin availability or secretion and overcoming insulin resistance. Unfortunately, central obesity and physical inactivity hinder medical management and may hasten development of chronic complications, particularly in elderly people with long-standing diabetes. Even when glycemic control is near optimal with medication, reducing insulin resistance by any other means must be explored in view of these adverse consequences.

Muscle contraction increases glucose uptake in skeletal muscle,³⁴ thereby forming the basis for recommending RT for individuals with abnormal glucose metabolism. Aerobic exercise uses large muscle groups for extended periods of time, but whole-body RT programs may provide equally high or higher recruitment of muscle mass over a comparable period of time. The American College of Sports Medicine has recommended the use of progressive RT as part of a well-

rounded exercise program for individuals with type 2 diabetes.³⁵ Similarly, in the absence of contraindications, the American Diabetes Association also recommends RT for those with type 2 diabetes.¹¹ These recommendations are supported by evidence that RT is an integral component in the therapeutic management of glycemic control in both young and older type 2 diabetics,^{28,36–38} particularly if the RT is performed in a supervised versus home-based program.³⁹

Glucose Tolerance and Insulin Sensitivity

A frequent postulate is that RT and subsequent increases in skeletal muscle mass may improve glucose and insulin responses to a glucose load.^{6,40} However, there are little data available showing that RT prevents type 2 diabetes. RT does not usually alter glucose tolerance or glycemic control regardless of age,^{5–7,16,41,42} unless baseline glucose tolerance is abnormal.^{36,38,40–44} Nevertheless, RT reduces acute insulin responses during an oral glucose tolerance test in healthy young, middle-aged, and older men in most studies.^{5,6,45} RT also reduces acute insulin responses during glucose tolerance testing in diabetic men^{36,38} and women^{36,38,44} and improves insulin sensitivity during hyperglycemic and hyperinsulinemic-euglycemic clamps in diabetic and/or insulin-resistant middle-aged^{6,46,47} and older men⁴² and diabetic middle-aged⁴⁸ and older women.⁴²

Glycemic Control

RT decreases glycosylated hemoglobin (HbA1c) levels in diabetic men^{28,36,37,43} and women,^{28,36,37} regardless of age. This effect is observed even in the absence of a lasting effect of RT on fasting glucose or insulin levels.³⁷ Improved glycemic control and decreased HbA1c levels are important for reducing the microvascular and macrovascular complications of diabetes. For example, the UK Prospective Diabetes Study⁴⁹ reported that each percentage point reduction in HbA1c was associated with a 35% reduction in microvascular complications, whereas the European Prospective Investigation of Cancer and Nutrition (EPIC)–Norfolk prospective population study showed that an increase of 1 percentage point in HbA1c was associated with a 28% increase in mortality risk, independent of other CV risk factors.⁵⁰ RT-induced improvements in glycemic control, however, appear to be intensity-dependent, with beneficial effects occurring when subjects train at 70% to 90% of the 1-repetition maximum strength (1-RM). One-repetition maximum is the maximal weight lifted in 1 attempt during strength testing. In studies where the RT intervention was less than 2 months and/or the exercise intensity was less than 50% of the 1-RM, improvements in HbA1c were modest or undetected.^{47,51,52}

Resistance Training and Hypertension

Adopting a healthy lifestyle is critical for the prevention of high blood pressure (BP) and is an indispensable part of the treatment of hypertension.⁵³ The American Heart Association⁹ and the American College of Sports Medicine¹⁰ have each endorsed moderate-intensity RT as a complement to aerobic exercise programs in the prevention, treatment, and control of hypertension.

Systemic Blood Pressure

The rationale for RT as an adjunct to aerobic exercise for controlling BP stems from multiple studies. Two meta-analyses of RT and hypertension are noteworthy.^{26,27} Inclusion criteria, consistent across both reviews, were (1) inclusion of a randomized nonexercise control group; (2) RT as the only intervention; (3) training for a minimum of 4 weeks; and (4) participants who were sedentary normotensive and/or hypertensive adults with no other concomitant disease. Kelley and Kelley²⁷ examined the effects of RT on resting BP in studies published between January 1966 and December 1998. A total of 11 studies met the inclusion criteria and represented initial and final BP assessments in 182 RT subjects and 138 control subjects. Decreases ($P \leq 0.05$) of approximately 3 mm Hg were found for both systolic and diastolic BP across all BP categories as the result of RT. These changes represented a 2% decrease for resting systolic BP and 4% for resting diastolic BP. No differences were found for changes in resting BP between studies that used conventional RT compared with a circuit RT protocol. A conventional RT protocol generally consists of lifting heavier weights with longer rest periods, whereas a circuit RT protocol consists of lifting lighter weights with shorter rest periods between exercises. By moving quickly between exercises and by using lighter weight with higher repetitions, circuit training introduces an aerobic component to the workout.⁵⁴

In the more recent meta-analysis, Cornelissen and Fagard²⁶ pooled data from studies published between 1996 and 2003 that included 9 randomized controlled trials involving 341 participants. The overall effect of RT was a decrease of 3.2 mm Hg ($P = 0.10$) in systolic BP and a decrease of 3.5 mm Hg ($P \leq 0.05$) in diastolic BP. Results from these meta-analyses are consistent with conclusions generated by narrative reviews.^{17,55,56} Although these reductions seem modest, a systolic BP reduction of 3 mm Hg in average populations has been estimated to reduce cardiac morbidity by 5% to 9%, stroke by 8% to 14%, and all-cause mortality by 4%.⁵⁷ The lack of data on the effects of RT on ambulatory BP warrants further investigation because this may be more indicative of future CV disease morbidity and mortality.⁵⁸

Control of BP is even more important in individuals who already have hypertension. Although there is general agreement that endurance training lowers resting BP in patients with mild to severe hypertension,^{27,57} there is a paucity of data on the effects of RT alone on BP in individuals with hypertension. Only 20% of the outcomes in the 2 meta-analysis reviews were based on a mean initial resting systolic BP >140 mm Hg, whereas only 13% had a mean initial resting diastolic BP >90 mm Hg.^{26,27} One study that used RT in combination with aerobic exercise in middle-aged hypertensive men for 10 weeks demonstrated reductions of 13 mm Hg for both systolic and diastolic BP.⁵⁹ Conversely, a similar program for 6 months in older adults with hypertension showed mean decreases in systolic and diastolic BP of 5.3 and 3.7 mm Hg, respectively.⁶⁰ The change in systolic BP, although significantly lower than study entry, was not different from values in the control group. Although any reduction in BP is desirable, the available studies do not answer the

question regarding the independent benefit of RT in persons initially classified as being hypertensive or prehypertensive.

Arterial Stiffness

With aging, hypertension, insulin resistance, and diabetes, there is increased arterial stiffness from degeneration of the arterial media, increased collagen and calcium content, and arterial dilation and hypertrophy. These factors lead to increased systolic BP and an increased risk of cardiac events.⁶¹ Several studies have shown that aerobic exercise is associated with reduced arterial stiffness in healthy subjects of all ages,⁶² competitive aerobic athletes,⁶³ patients with coronary artery disease,⁶⁴ and hemodialysis patients.⁶⁵ Moreover, when aerobic exercise is combined with RT, there is no evidence of increased arterial stiffness.⁶⁶ However, less is known about the independent effects of RT on arterial stiffness. Two cross-sectional studies have suggested that young and middle-aged men who participate in regular RT have greater arterial stiffness than age-matched sedentary control subjects.^{67,68} However, only 3 interventional studies have examined the effect of RT on arterial function.^{69–71} Miyachi et al⁷⁰ reported that RT 3 days per week for 4 months decreased carotid arterial compliance by 19% ($P < 0.05$) in young, healthy men who were novice weight trainers. Interestingly, carotid arterial compliance returned to baseline values within 2 months after RT was discontinued. Cortez-Cooper et al⁶⁹ reported that high intensity RT for 4 days per week for 3 months in young, healthy women who were novice weight trainers ($n = 23$; age 29 ± 1 years, mean \pm SD) increased carotid augmentation index (a measure of arterial wave reflection and arterial stiffness) from $-8 \pm 13\%$ to $1 \pm 18\%$ ($P < 0.05$), and carotid-femoral pulse-wave velocity increased ($P \leq 0.05$) from 791 ± 88 to 833 ± 96 cm/s. Paradoxically, neither study reported increases in systolic or diastolic BP secondary to RT.

Contradictory results were recently reported by Rakobowchuk and coworkers.⁷¹ By using similar vascular measurement techniques, they found that central arterial compliance was unaltered after 3 months of RT in young men ($n = 28$; age 23 ± 3.9 years, mean \pm SD). The discrepancy between studies may be explained by differences in the RT protocols. The 2 studies in which central arterial stiffness increased used RT protocols consisting of high-intensity super sets and an extremely high volume (up to 6 sets per exercise),^{69,70} both of which are not commonly recommended for the majority of the population and are usually performed by competitive athletes.^{9,10} Conversely, the study in which arterial stiffness was unchanged used a progressive training protocol that increased intensity but not the volume of exercise over 12 weeks.

Mechanisms for Change in Arterial Compliance

Studies reporting adverse effects of RT on the arterial system have only speculated about mechanisms responsible for the changes.^{67–70} The elastic properties of the arterial wall are determined by both structural components (eg, relative composition of elastin and collagen) and functional components (eg, vasoconstrictor tone exerted by the vascular smooth muscle cells). Because 3 to 4 months of RT are unlikely to

cause marked structural changes in the arterial wall, changes in the functional components of the arterial wall need to be considered. One potential mechanism is endothelial dysfunction manifested as a reduction in the bioavailability of nitric oxide. Recent evidence, however, indicates that 4 months of RT in healthy young men does not impair endothelium-dependent vasodilation in the brachial artery.⁷² Another mechanism for functional change in the arterial wall is increased sympathetic tone. There is evidence that RT increases resting humoral norepinephrine levels, a surrogate marker of sympathetic nervous system activity.⁷³ However, increased sympathetic nervous system vasoconstrictor tone is likely to be greater in peripheral muscular arteries than in central elastic arteries. Surprisingly, both studies reporting increases in stiffness of central conduit arteries after RT did not show changes in peripheral muscular arteries.^{69,70} Results from narrative^{15,17,55,56} and meta-analytical reviews^{26,27} do not support the contention the RT increases vascular resistance. Moreover, those findings are compatible with the absence of hypertension observed among isometric and power athletes.^{74,75}

Resistance Training and Obesity

Obesity is an important risk factor for CV disease, left ventricular dysfunction, congestive heart failure, stroke, and cardiac arrhythmias.³ Weight loss in obese patients can improve or prevent many of the obesity-related CV risk factors (ie, insulin resistance and type 2 diabetes mellitus, dyslipidemia, hypertension, and inflammation) and can improve diastolic function.³ Moreover, these benefits are often found after only modest weight loss ($\approx 5\%$ of initial weight) and continue to improve with increasing weight loss.³

Obesity Prevention

Epidemiological evidence supports the use of increased exercise in preventing age-associated weight and fat gains.¹ Exercise recommendations to treat or prevent obesity have focused mainly on aerobic activities.¹ However, RT is a behaviorally feasible and efficacious alternative to endurance exercise for weight control. For example, resting energy expenditure (REE) decreases with aging, and this decrease is closely correlated to losses in skeletal muscle mass.⁷⁶ RT increases muscle mass by a minimum of 1 to 2 kg in studies of sufficient duration.⁷⁷ Theoretically, a gain of 1 kg in muscle mass should result in an REE increase of approximately 21 kcal/kg of new muscle.⁷⁸ In practice, RT intervention studies report REE increases in the range of 28 to 218 kcal/kg of muscle.^{79–82} RT, when sustained over years or decades, translates into clinically important differences in daily energy expenditure and age-associated fat gains. However, even without a change in REE, maintenance of muscle mass through midlife years may prevent age-associated fat gains by promoting an active lifestyle.^{15,83,84}

Visceral Adipose Tissue

RT can reduce total body fat mass in men^{84,85} and women,^{83,85–87} independent of dietary caloric restriction. However, regional distribution of fat may be more important to health than the total amount of body fat. Excessive central obesity

and especially visceral adipose tissue have been linked with the development of hyperlipidemia, hypertension, insulin resistance and glucose intolerance, diabetes, and heart disease.^{15,17,88,89} Fat distributed in the arms and legs, however, appears to impose little or no risk.^{15,88,89} Although there may be a genetic predisposition for visceral adipose tissue, increasing age, high fat diets, and a sedentary lifestyle are also important determinants.

Several studies have demonstrated decreases in visceral adipose tissue after RT programs.^{84,85,87,90,91} Treuth and coworkers^{84,87} assessed body composition in older men by using dual-energy x-ray absorptiometry⁸⁴ and in older women by using computed tomography⁸⁷ and observed significant decreases in visceral fat after 16 weeks of RT. Ross et al^{90,91} used magnetic resonance imaging to measure regional fat losses after exercise combined with diet interventions. In their first study,⁹⁰ both diet plus aerobic exercise and diet plus RT elicited similar losses of visceral fat that were greater than losses of whole-body subcutaneous fat. In a follow-up study,⁹¹ they isolated the effects of endurance exercise training and RT by comparing the responses to diet alone and diet combined with each training modality in middle-aged obese men. All 3 groups lost significant amounts of total body fat, and all 3 groups experienced a significantly greater visceral fat loss compared with whole-body subcutaneous fat loss. The changes amounted to a 40% reduction in visceral fat in the RT and diet group, 39% in the endurance training and diet group, and a 32% reduction in the diet-only group.

One study has raised the possibility of gender specificity in visceral fat reduction in response to RT. Hunter et al⁸⁵ studied older women and men (age, 61 to 77 years) after 25 weeks of supervised RT. Both genders significantly increased muscle mass but men increased muscle more than women (2.8 versus 1.0 kg, respectively). Similar decreases in total body fat mass were found for the men (1.8 kg) and women (1.7 kg). However, women lost a significant amount of visceral adipose tissue (131 to 116 cm²), whereas the men did not (143 to 152 cm²). Similarly, women also lost a significant amount of subcutaneous adipose tissue (254 to 239 cm²), but men did not (165 to 165 cm²). Conversely, in a 6-month study of RT combined with aerobic exercise, men lost more visceral adipose tissue than women, but losses of total and subcutaneous adipose tissue were similar.⁶⁰ Although more research is needed to clarify these possible gender-specific responses, the overall available body of literature supports the use of RT, with or without aerobic exercise, and with or without diet modification, as an effective intervention that contributes to the reduction of abdominal obesity.

Obesity Reduction

Studies of the efficacy of RT in the context of total body weight loss have had mixed results. Studies that use more severe caloric intake restriction have not shown gains in muscle mass,^{92,93} whereas RT studies with less severe caloric restriction have shown muscle mass gains with only modest losses in body weight.^{87,90,91,94} RT studies that attempt to maintain caloric balance during the intervention typically do not observe major changes in body weight in either gender, despite significant reductions in fat mass and percent body

TABLE 2. Summary of Guidelines for Resistance Training for Disease Prevention

Exercise mode	Resistance exercise consists of weight lifting. Machines are preferred for safety and ease of use; hand-held weights, barbells, and elastic bands can also be used.
No. of exercises	8 to 10 exercises covering the major muscle groups; chest, shoulders, arms, back, abdomen, thigh, lower legs
Intensity	Resistance (weight) is set at 30% to 40% of 1 repetition maximum for upper body and 50% to 60% for lower-body exercises. One repetition maximum is the highest weight lifted 1 time. If testing is not available, use a weight that can be lifted for 8 to 10 repetitions; increase weight when 15 repetitions can be done easily.
Duration	Resistance training consisting of a single set of 8 to 10 exercises takes about 20 minutes
Frequency	Resistance exercise should be done at least twice per week.
Precautions	Risk/benefit ratio of resistance exercise is very favorable. Contraindications to resistance training are the same as those for aerobic exercise. Treatment for systolic BP >160 mm Hg or diastolic BP >100 mm Hg should be initiated before starting any type of exercise program. Avoid extended breath-holding to minimize exaggerated BP response.

fat.^{15,84,85,87} In essence, body weight does not change much because loss of fat mass is generally offset by the gain in muscle mass. Conversely, endurance training-induced decreases in fat mass are more likely to be associated with reductions in body weight because there is no offsetting gain in muscle mass.

Resistance Training and Dyslipidemia

There is a dearth of well-controlled studies investigating the effect of RT intervention on lipid metabolism in individuals with normal lipoprotein-lipid profiles and those who are hypercholesterolemic. Cross-sectional evidence regarding the relation between muscle strength, RT, and plasma lipoprotein-lipid profiles is contradictory. Tucker and Silvester^{95,96} studied 8499 male employees of more than 50 companies and observed a reduced risk of hypercholesterolemia among individuals participating in RT programs. However, only those individuals who participated in RT >4 hours per week maintained this reduced risk when confounding variables were controlled. In contrast, Kohl et al⁹⁷ studied 1193 women and 5460 men and reported no significant association between muscle strength and total or low-density lipoprotein cholesterol for either gender. However, there was a direct association between both upper and lower body strength and triglyceride levels in men.

Most interventional studies have failed to adequately control for normal variations in lipoproteins and lacked proper dietary controls and/or lacked statistical power. When these factors are controlled, most studies show no improvement in lipid profiles after RT in either middle-aged or older adults.^{40,87,98,99} However, the lack of significant lipoprotein-lipid changes with RT may be due to the fact that total cholesterol values for most study groups has been ≤ 200 mg/dL at study entry. Individuals with normal lipoprotein-lipid profiles may require greater exercise stimulus and energy expenditure coupled with significant reductions in body weight to further improve lipid profiles. Alternatively, Shoup and Durstine¹⁰⁰ postulated that for changes in blood lipids to occur, lipoprotein lipase levels need to increase or remain elevated postexercise, or associated hepatic lipase must be suppressed to reduce conversion of high-density lipoprotein cholesterol (HDL-C) subfraction HDL₂-C to HDL₃-C. It is possible that the RT stimulus in most studies does not generate these cellular changes.

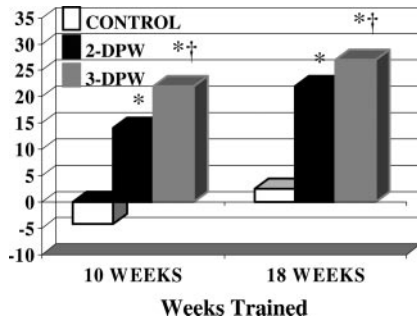
Screening and Precautions

The risk-to-benefit ratio of RT is highly favorable for most healthy individuals. The hemodynamic response to aerobic exercise is an increase in heart rate, with a progressive rise in systolic BP with little or no change in diastolic BP, and a widening of the pulse pressure.⁹ These responses result primarily in a volume load on the heart. Conversely, RT causes a marked rise in both systolic and diastolic BP and consequently mean blood pressures, with less of a rise in heart rate compared with aerobic exercise.¹⁰¹ Thus, RT imposes primarily a pressure load on the heart. Because of the considerably lower response of heart rate during RT, this mode of exercise generally results in a lower rate-pressure product compared with aerobic exercise. Among the many studies of RT in healthy adults, there have been no reported cardiovascular complications. The American College of Sports Medicine⁹⁵ and the American Heart Association⁹ indicate that the contraindications to RT are similar to those for endurance exercise. Thus, the same screening criteria used for healthy adults before participation in endurance exercise would apply.

Selected individuals should consult their healthcare practitioners before beginning a vigorous exercise program. For those at moderate risk or higher, such as men age 45 years and over, women age 55 years and over, those with major risk factors for arteriosclerosis, and those with diabetes at any age, the American College of Sports Medicine recommends a medical history and physical examination including an exercise stress test before initiating a vigorous exercise program.⁹⁵ Because of the marked rise in BP with RT, those with uncontrolled hypertension (systolic BP ≥ 160 mm Hg and/or diastolic BP ≥ 100 mm Hg) should be controlled to lower levels before starting an exercise program.¹⁰² High-intensity RT should also be avoided by individuals who have active proliferative retinopathy or moderate or worse nonproliferative diabetic retinopathy. To minimize excessive BP responses, individuals should be told to avoid extended breath-holding during their workouts.^{35,80,103}

Resistance Training Exercise Prescription

A summary of RT guidelines is presented in Table 2. RT of all major muscle groups can be accomplished through the use of expedient programs. Indeed, adherence rates in RT interventional studies are high and due, in part, to the minimal



Relative changes (%) in peak isometric strength during knee extension after 10 and 18 weeks of variable resistance training. Group 2 trained 2 days per week (DPW) ($n=50$); group 3 trained 3 DPW ($n=41$); control did not train. Data represent mean \pm SEM. *2-DPW and 3-DPW > control ($P \leq 0.01$); †3-DPW > 2-DPW ($P \leq 0.05$).

time requirement for full participation. Most studies report that RT 3 days per week elicits superior strength gains when compared with training regimens of lower frequency.^{77,104} However, if training intensity remains high (7 to 10 repetitions performed to momentary muscular failure), RT only 2 days per week produces approximately 80% of the strength benefits reported by studies using traditional 3 days-per-week routines (Figure).¹⁰⁴ Using scientific evidence and expert opinion, the American Heart Association, with endorsement of the American College of Sports Medicine, has promulgated RT guidelines for individuals with and without CV disease.^{9,95} The guidelines for those without CV disease are summarized briefly herein. RT is recommended a minimum of 2 days per week, with progression to 3 days per week. A typical workout should consist of 8 to 10 exercises to cover the major muscle groups, which includes the chest, shoulders, arms, back, abdomen, thighs, and lower legs. The resistance or weight lifted should be moderate, which is defined as 30% to 40% of 1 RM for upper body exercises and 50% to 60% of 1 RM for lower body exercises. If maximal strength testing is not available, the individual can, through trial and error, use a weight that can be lifted for a minimum of 8 to 10 repetitions. When 12 to 15 repetitions can be accomplished with little difficulty, the weight is increased. This progressive resistance strategy meets the requirements of the overload principle, which is the basis for improvement in strength. Furthermore, by using moderate weight and gradually increasing the workload in stages, there is less risk of musculoskeletal injury while maintaining effectiveness of the workout.

Summary

Although randomized controlled trials among diverse populations are needed to further examine the role of RT in reducing CV risk factors, the following conclusions can be made regarding the mitigating effects of RT on the risk of cardiovascular disease:

(1) RT does not appear to alter glucose tolerance or glycemic control regardless of age, unless baseline glucose tolerance is abnormal. Nonetheless, most studies show that RT improves insulin action either through reductions in acute insulin responses during an oral glucose tolerance test or

increased glucose uptake during glycemic clamp procedures. Moreover, RT significantly decreases HbA1c in diabetic men and women regardless of age, and this effect is observed even in the absence of a lasting effect of RT on fasting glucose.

(2) In healthy, normotensive persons, RT elicits reductions of approximately 3 mm Hg for both systolic and diastolic BP. Future studies are needed in individuals initially classified as hypertensive or prehypertensive to determine the extent to which RT lowers BP when it is elevated at baseline. Until these studies are performed, an RT program combined with aerobic exercise should be recommended for lowering BP in hypertensive adults.

(3) There is some evidence that RT can increase central arterial stiffness during high-intensity and high-volume training regimens, but an explanation for this effect has not been determined. No studies have found increased BP or peripheral vascular resistance secondary to RT.

(4) There is good evidence that RT reduces total body fat mass in men and women, independent of dietary caloric restriction. There is also good evidence that RT reduces visceral adipose tissue in older men and women.

(5) There is little evidence that RT improves lipoprotein-lipid profiles. However, total cholesterol values for most study groups have been ≤ 200 mg/dL at study entry. Individuals with normal lipoprotein-lipid profiles may require greater exercise stimulus and energy expenditure coupled with significant reductions in body weight in order to further improve lipid profiles.

(6) Although RT by itself may have limited beneficial effects on CV disease risk factors, this mode of exercise is beneficial in the prevention and management of musculoskeletal injuries and disorders, osteoporosis, and sarcopenia. RT also reduces susceptibility to falls and prevents or delays impaired physical function in frail and elderly persons.

(7) Although performing RT by itself rather than in combination with aerobic exercise appears to contribute to some aspects of CV disease reduction, the available data do not permit accurate estimation of the magnitude of the risk reduction. Thus, for the individual without existing cardiac disease whose goal is to improve their CV health and prevent disease, there is little evidence herein to challenge existing exercise guidelines that call for moderate-intensity RT to be performed in combination with aerobic exercise.

Disclosures

None.

References

1. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults—the evidence report. National Institutes of Health. *Obes Res.* 1998;6(suppl 2):51S–209S.
2. Fletcher GF, Balady G, Blair SN, Blumenthal J, Caspersen C, Chaitman B, Epstein S, Sivarajan Froelicher ES, Froelicher VF, Pina IL, Pollock ML. Statement on exercise: benefits and recommendations for physical activity programs for all Americans: a statement for health professionals by the Committee on Exercise and Cardiac Rehabilitation of the Council on Clinical Cardiology, American Heart Association. *Circulation.* 1996; 94:857–862.
3. Klein S, Burke LE, Bray GA, Blair S, Allison DB, Pi-Sunyer X, Hong Y, Eckel RH. Clinical implications of obesity with specific focus on cardiovascular disease: a statement for professionals from the American Heart Association Council on Nutrition, Physical Activity, and Metab-

- olism: endorsed by the American College of Cardiology Foundation. *Circulation*. 2004;110:2952–2967.
4. Wilson PW, D'Agostino RB, Levy D, Belanger AM, Silbershatz H, Kannel WB. Prediction of coronary heart disease using risk factor categories. *Circulation*. 1998;97:1837–1847.
 5. Hurley BF, Hagberg JM, Goldberg AP, Seals DR, Ehsani AA, Brennan RE, Holloszy JO. Resistive training can reduce coronary risk factors without altering VO₂max or percent body fat. *Med Sci Sports Exerc*. 1988;20:150–154.
 6. Miller JP, Pratley RE, Goldberg AP, Gordon P, Rubin M, Treuth MS, Ryan AS, Hurley BF. Strength training increases insulin action in healthy 50- to 65-year-old men. *J Appl Physiol*. 1994;77:1122–1127.
 7. Poehlman ET, Dvorak RV, DeNino WF, Brochu M, Ades PA. Effects of resistance training and endurance training on insulin sensitivity in nonobese, young women: a controlled randomized trial. *J Clin Endocrinol Metab*. 2000;85:2463–2468.
 8. Smutok MA, Reece C, Kokkinos PF, Farmer CM, Dawson PK, DeVane J, Patterson J, Goldberg AP, Hurley BF. Effects of exercise training modality on glucose tolerance in men with abnormal glucose regulation. *Int J Sports Med*. 1994;15:283–289.
 9. Pollock ML, Franklin BA, Balady GJ, Chaitman BL, Fleg JL, Fletcher B, Limacher M, Pina IL, Stein RA, Williams M, Bazzarre T. AHA Science Advisory: resistance exercise in individuals with and without cardiovascular disease: benefits, rationale, safety, and prescription: an advisory from the Committee on Exercise, Rehabilitation, and Prevention, Council on Clinical Cardiology, American Heart Association; Position paper endorsed by the American College of Sports Medicine. *Circulation*. 2000;101:828–833.
 10. Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. American College of Sports Medicine position stand: exercise and hypertension. *Med Sci Sports Exerc*. 2004;36:533–553.
 11. Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C. Physical activity/exercise and type 2 diabetes. *Diabetes Care*. 2004;27:2518–2539.
 12. Fitzgerald SJ, Barlow CE, Kampert JB, Morrow JR, Jackson AW, Blair SN. Muscular fitness and all-cause mortality: prospective observations. *J Physical Activity Health*. 2004;1:7–18.
 13. Jurca R, Lamonte MJ, Barlow JB, Kampert JB, Church TS, Blair SN. Association of muscular strength with incidence of metabolic syndrome in men. *Med Sci Sports Exerc*. 2005;37:1849–1855.
 14. Jurca R, Lamonte MJ, Church TS, Earnest CP, Fitzgerald SJ, Barlow CE, Jordan AN, Kampert JB, Blair SN. Associations of muscle strength and fitness with metabolic syndrome in men. *Med Sci Sports Exerc*. 2004;36:1301–1307.
 15. American College of Sports Medicine Position Stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. *Med Sci Sports Exerc*. 1998;30:975–991.
 16. Banz WJ, Maher MA, Thompson WG, Bassett DR, Moore W, Ashraf M, Keefer DJ, Zemel MB. Effects of resistance versus aerobic training on coronary artery disease risk factors. *Exp Biol Med (Maywood)*. 2003;228:434–440.
 17. Hurley BF, Roth SM. Strength training in the elderly: effects on risk factors for age-related diseases. *Sports Med*. 2000;30:249–268.
 18. Nelson ME, Fiatarone MA, Morganti CM, Trice I, Greenberg RA, Evans WJ. Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures: a randomized controlled trial. *JAMA*. 1994;272:1909–1914.
 19. Larsson L. Histochemical characteristics of human skeletal muscle during aging. *Acta Physiol Scand*. 1983;117:469–471.
 20. Fabbri A, Giannini D, Aversa A, De Martino MU, Fabbri E, Franceschi F, Moretti C, Frajese G, Isidori A. Body-fat distribution and responsiveness of the pituitary-adrenal axis to corticotropin-releasing-hormone stimulation in sedentary and exercising women. *J Endocrinol Invest*. 1999;22:377–385.
 21. Vincent KR, Braith RW, Feldman RA, Kallas HE, Lowenthal DT. Improved cardiorespiratory endurance following 6 months of resistance exercise in elderly men and women. *Arch Intern Med*. 2002;162:673–678.
 22. Fiatarone M, Singh N. The exercise prescription. In: Fiatarone M, Singh N, eds. *Exercise, Nutrition, and the Older Woman*. Boca Raton, Fla: CRC Press; 2000:37–104.
 23. Singh NA, Clements KM, Fiatarone MA. A randomized controlled trial of progressive resistance training in depressed elders. *J Gerontol A Biol Sci Med Sci*. 1997;52:M27–M35.
 24. Braith RW, Mills RM, Welsch MA, Pollock MH, Keller J. Training reverses steroid induced osteoporosis after heart transplantation. *J Am Coll Cardiol*. 1996;28:1471–1477.
 25. Ettinger WH Jr, Burns R, Messier SP, Applegate W, Rejeski WJ, Morgan T, Shumaker S, Berry MJ, O'Toole M, Monu J, Craven T. A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis: the Fitness Arthritis and Seniors Trial (FAST). *JAMA*. 1997;277:25–31.
 26. Cornelissen VA, Fagard RH. Effect of resistance training on resting blood pressure: a meta-analysis of randomized controlled trials. *J Hypertens*. 2005;23:251–259.
 27. Kelley GA, Kelley KS. Progressive resistance exercise and resting blood pressure: a meta-analysis of randomized controlled trials. *Hypertension*. 2000;35:838–843.
 28. Honkola A, Forsen T, Eriksson J. Resistance training improves the metabolic profile in individuals with type 2 diabetes. *Acta Diabetol*. 1997;34:245–248.
 29. McCartney N. Role of resistance training in heart disease. *Med Sci Sports Exerc*. 1998;30:S396–S402.
 30. Baumgartner RN, Koehler KM, Gallagher D, Romero L, Heymsfield SB, Ross RR, Garry PJ, Lindeman RD. Epidemiology of sarcopenia among the elderly in New Mexico. *Am J Epidemiol*. 1998;147:755–763.
 31. Pu CT, Johnson MT, Forman DE, Hausdorff JM, Roubenoff R, Foldvari M, Fielding RA, Singh MA. Randomized trial of progressive resistance training to counteract the myopathy of chronic heart failure. *J Appl Physiol*. 2001;90:2341–2350.
 32. Selig SE, Carey MF, Menzies DG, Patterson J, Geerling RH, Williams AD, Bamroongsuk V, Toia D, Krum H, Hare DL. Moderate-intensity resistance exercise training in patients with chronic heart failure improves strength, endurance, heart rate variability, and forearm blood flow. *J Card Fail*. 2004;10:21–30.
 33. Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, Wiedmeyer HM, Byrd-Holt DD. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults: the Third National Health and Nutrition Examination Survey, 1988–1994. *Diabetes Care*. 1998;21:518–524.
 34. Holloszy JO, Hansen PA. Regulation of glucose transport into skeletal muscle. *Rev Physiol Biochem Pharmacol*. 1996;128:99–193.
 35. Albright A, Franz M, Hornsby G, Kriska A, Marrero D, Ullrich I, Verity LS. American College of Sports Medicine position stand: exercise and type 2 diabetes. *Med Sci Sports Exerc*. 2000;32:1345–1360.
 36. Castaneda C, Layne JE, Munoz-Orians L, Gordon PL, Walsmith J, Foldvari M, Roubenoff R, Tucker KL, Nelson ME. A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. *Diabetes Care*. 2002;25:2335–2341.
 37. Dunstan DW, Daly RM, Owen N, Jolley D, De Courten M, Shaw J, Zimmet P. High-intensity resistance training improves glycemic control in older patients with type 2 diabetes. *Diabetes Care*. 2002;25:1729–1736.
 38. Ibanez J, Izquierdo M, Arguelles I, Forga L, Larrion JL, Garcia-Unciti M, Idoate F, Gorostiaga EM. Twice-weekly progressive resistance training decreases abdominal fat and improves insulin sensitivity in older men with type 2 diabetes. *Diabetes Care*. 2005;28:662–667.
 39. Dunstan DW, Daly RM, Owen N, Jolley D, Vulikh E, Shaw J, Zimmet P. Home-based resistance training is not sufficient to maintain improved glycemic control following supervised training in older individuals with type 2 diabetes. *Diabetes Care*. 2005;28:3–9.
 40. Smutok MA, Reece C, Kokkinos PF, Farmer C, Dawson P, Shulman R, DeVane-Bell J, Patterson J, Charabogous C, Goldberg AP. Aerobic versus strength training for risk factor intervention in middle-aged men at high risk for coronary heart disease. *Metabolism*. 1993;42:177–184.
 41. Fluckey JD, Hickey MS, Brambrink JK, Hart KK, Alexander K, Craig BW. Effects of resistance exercise on glucose tolerance in normal and glucose-intolerant subjects. *J Appl Physiol*. 1994;77:1087–1092.
 42. Ryan AS, Hurlbut DE, Lott ME, Ivey FM, Fleg J, Hurley BF, Goldberg AP. Insulin action after resistive training in insulin resistant older men and women. *J Am Geriatr Soc*. 2001;49:247–253.
 43. Eriksson J, Taimela S, Eriksson K, Parviainen S, Peltonen J, Kujala U. Resistance training in the treatment of non-insulin-dependent diabetes mellitus. *Int J Sports Med*. 1997;18:242–246.
 44. Fenicchia LM, Kanaley JA, Azevedo JL Jr, Miller CS, Weinstock RS, Carhart RL, Ploutz-Snyder LL. Influence of resistance exercise training on glucose control in women with type 2 diabetes. *Metabolism*. 2004;53:284–289.

45. Reynolds TH, Supiano MA, Dengel DR. Resistance training enhances insulin-mediated glucose disposal with minimal effect on the tumor necrosis factor- α system in older hypertensives. *Metabolism*. 2004; 53:397–402.
46. Eriksson J, Tuominen J, Valle T, Sundberg S, Sovijarvi A, Lindholm H, Tuomilehto J, Koivisto V. Aerobic endurance exercise or circuit-type resistance training for individuals with impaired glucose tolerance? *Horm Metab Res*. 1998;30:37–41.
47. Ishii T, Yamakita T, Sato T, Tanaka S, Fujii S. Resistance training improves insulin sensitivity in NIDDM subjects without altering maximal oxygen uptake. *Diabetes Care*. 1998;21:1353–1355.
48. Ryan AS, Pratley RE, Goldberg AP, Elahi D. Resistive training increases insulin action in postmenopausal women. *J Gerontol A Biol Sci Med Sci*. 1996;51:M199–M205.
49. Manley S. Haemoglobin A1c: a marker for complications of type 2 diabetes: the experience from the UK Prospective Diabetes Study (UKPDS). *Clin Chem Lab Med*. 2003;41:1182–1190.
50. Khaw KT, Wareham N, Luben R, Bingham S, Oakes S, Welch A, Day N. Glycated haemoglobin, diabetes, and mortality in men in Norfolk cohort of European prospective investigation of cancer and nutrition (EPIC-Norfolk). *BMJ*. 2001;322:1–6.
51. Dunstan DW, Puddey IB, Beilin LJ, Burke V, Morton AR, Stanton KG. Effects of a short-term circuit weight training program on glycaemic control in NIDDM. *Diabetes Res Clin Pract*. 1998;40:53–61.
52. Maiorana A, O'Driscoll G, Goodman C, Taylor R, Green D. Combined aerobic and resistance exercise improves glycemic control and fitness in type 2 diabetes. *Diabetes Res Clin Pract*. 2002;56:115–123.
53. Whelton PK, He J, Appel LJ, Cutler JA, Havas S, Kotchen TA, Roccella EJ, Stout R, Vallbona C, Winston MC, Karimbakas J. Primary prevention of hypertension: clinical and public health advisory from the National High Blood Pressure Education Program. *JAMA*. 2002;288: 1882–1888.
54. Stewart KJ. Weight training in coronary artery disease and hypertension. *Prog Cardiovasc Dis*. 1992;35:159–168.
55. Schwartz RS, Hirth VA. The effects of endurance and resistance training on blood pressure. *Int J Obes Relat Metab Disord*. 1995;19(suppl 4):S52–S57.
56. Stone MH, Fleck SJ, Triplett NT, Kraemer WJ. Health- and performance-related potential of resistance training. *Sports Med*. 1991; 11:210–231.
57. Whelton SP, Chin A, Xin X, He J. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. *Ann Intern Med*. 2002;136:493–503.
58. Verdecchia P. Prognostic value of ambulatory blood pressure: current evidence and clinical implications. *Hypertension*. 2000;35:844–851.
59. Kelemen MH, Efron MB. Exercise training combined with antihypertensive drug therapy. *JAMA*. 1990;263:2766–2771.
60. Stewart KJ, Bacher AC, Turner KL, Fleg JL, Hees PS, Shapiro EP, Tayback M, Ouyang P. Effect of exercise on blood pressure in older persons: a randomized controlled trial. *Arch Intern Med*. 2005;165: 756–762.
61. Henry RM, Kostense PJ, Spijkerman AM, Dekker JM, Nijpels G, Heine RJ, Kamp O, Westerhof N, Bouter LM, Stehouwer CD. Arterial stiffness increases with deteriorating glucose tolerance status: the Hoorn Study. *Circulation*. 2003;107:2089–2095.
62. Tanaka H, DeSouza CA, Seals DR. Absence of age-related increase in central arterial stiffness in physically active women. *Arterioscler Thromb Vasc Biol*. 1998;18:127–132.
63. Edwards DG, Lang JT. Augmentation index and systolic load are lower in competitive endurance athletes. *Am J Hypertens*. 2005;18:679–683.
64. Edwards DG, Schofield RS, Magyari PM, Nichols WW, Braith RW. Effect of exercise training on central aortic pressure wave reflection in coronary artery disease. *Am J Hypertens*. 2004;17:540–543.
65. Mustata S, Chan C, Lai V, Miller JA. Impact of an exercise program on arterial stiffness and insulin resistance in hemodialysis patients. *J Am Soc Nephrol*. 2004;15:2713–2718.
66. Hayashi K, Sugawara J, Komine H, Maeda S, Yokoi T. Effects of aerobic exercise training on stiffness of central and peripheral arteries in middle-aged sedentary men. *Jpn J Physiol*. 2005;55:235–239.
67. Bertovic DA, Waddell TK, Gatzka CD, Cameron JD, Dart AM, Kingwell BA. Muscular strength training is associated with low arterial compliance and high pulse pressure. *Hypertension*. 1999;33:1385–1391.
68. Miyachi M, Donato AJ, Yamamoto K, Takahashi K, Gates PE, Moreau KL, Tanaka H. Greater age-related reductions in central arterial compliance in resistance-trained men. *Hypertension*. 2003;41:130–135.
69. Cortez-Cooper MY, DeVan AE, Anton MM, Farrar RP, Beckwith KA, Todd JS, Tanaka H. Effects of high intensity resistance training on arterial stiffness and wave reflection in women. *Am J Hypertens*. 2005; 18:930–934.
70. Miyachi M, Kawano H, Sugawara J, Takahashi K, Hayashi K, Yamazaki K, Tabata I, Tanaka H. Unfavorable effects of resistance training on central arterial compliance: a randomized intervention study. *Circulation*. 2004;110:2858–2863.
71. Rakobowchuk M, McGowan CL, de Groot PC, Bruinsma D, Hartman JW, Phillips SM, MacDonald MJ. Effect of whole body resistance training on arterial compliance in young men. *Exp Physiol*. 2005;90: 645–651.
72. Rakobowchuk M, McGowan CL, de Groot PC, Hartman JW, Phillips SM, MacDonald MJ. Endothelial function of young healthy males following whole body resistance training. *J Appl Physiol*. 2005;98: 2185–2190.
73. Pratley R, Nicklas B, Rubin M, Miller J, Smith A, Smith M, Hurley B, Goldberg A. Strength training increases resting metabolic rate and norepinephrine levels in healthy 50- to 65-year-old men. *J Appl Physiol*. 1994;76:133–137.
74. Colliander EB, Tesch PA. Blood pressure in resistance-trained athletes. *Can J Sport Sci*. 1988;13:31–34.
75. Longhurst JC, Stebbins CL. The isometric athlete. *Cardiol Clin*. 1992; 10:281–294.
76. Vaughan L, Zurlo F, Ravussin E. Aging and energy expenditure. *Am J Clin Nutr*. 1991;53:821–825.
77. Fleck SJ, Kraemer WJ. *Designing Resistance Training Programs*. 2nd ed. Champaign, Ill: Human Kinetics; 1997.
78. Weinsier RL, Schutz Y, Bracco D. Reexamination of the relationship of resting metabolic rate to fat-free mass and to the metabolically active components of fat-free mass in humans. *Am J Clin Nutr*. 1992;55: 790–794.
79. Broeder CE, Burrhus KA, Svanevik LS, Wilmore JH. The effects of either high-intensity resistance or endurance training on resting metabolic rate. *Am J Clin Nutr*. 1992;55:802–810.
80. Campbell WW, Crim MC, Young VR, Evans WJ. Increased energy requirements and changes in body composition with resistance training in older adults. *Am J Clin Nutr*. 1994;60:167–175.
81. Ryan AS, Pratley RE, Elahi D, Goldberg AP. Resistive training increases fat-free mass and maintains RMR despite weight loss in postmenopausal women. *J Appl Physiol*. 1995;79:818–823.
82. Taaffe DR, Pruitt L, Reim J, Butterfield G, Marcus R. Effect of sustained resistance training on basal metabolic rate in older women. *J Am Geriatr Soc*. 1995;43:465–471.
83. Schmitz KH, Jensen MD, Kugler KC, Jeffery RW, Leon AS. Strength training for obesity prevention in midlife women. *Int J Obes Relat Metab Disord*. 2003;27:326–333.
84. Treuth MS, Ryan AS, Pratley RE, Rubin MA, Miller JP, Nicklas BJ, Sorkin J, Harman SM, Goldberg AP, Hurley BF. Effects of strength training on total and regional body composition in older men. *J Appl Physiol*. 1994;77:614–620.
85. Hunter GR, Bryan DR, Wetzstein CJ, Zuckerman PA, Bamman MM. Resistance training and intra-abdominal adipose tissue in older men and women. *Med Sci Sports Exerc*. 2002;34:1023–1028.
86. Prabhakaran B, Dowling EA, Branch JD, Swain DP, Leutholtz BC. Effect of 14 weeks of resistance training on lipid profile and body fat percentage in premenopausal women. *Br J Sports Med*. 1999;33: 190–195.
87. Treuth MS, Hunter GR, Kekes-Szabo T, Weinsier RL, Goran MI, Berland L. Reduction in intra-abdominal adipose tissue after strength training in older women. *J Appl Physiol*. 1995;78:1425–1431.
88. Hunter GR, Kekes-Szabo T, Snyder SW, Nicholson C, Nyikos I, Berland L. Fat distribution, physical activity, and cardiovascular risk factors. *Med Sci Sports Exerc*. 1997;29:362–369.
89. Williams MJ, Hunter GR, Kekes-Szabo T, Snyder S, Treuth MS. Regional fat distribution in women and risk of cardiovascular disease. *Am J Clin Nutr*. 1997;65:855–860.
90. Ross R, Rissanen J. Mobilization of visceral and subcutaneous adipose tissue in response to energy restriction and exercise. *Am J Clin Nutr*. 1994;60:695–703.
91. Ross R, Rissanen J, Pedwell H, Clifford J, Shragge P. Influence of diet and exercise on skeletal muscle and visceral adipose tissue in men. *J Appl Physiol*. 1996;81:2445–2455.

92. Donnelly JE, Jacobsen DJ, Jakicic JM, Whatley JE. Very low calorie diet with concurrent versus delayed and sequential exercise. *Int J Obes Relat Metab Disord.* 1994;18:469–475.
93. Wadden TA, Vogt RA, Andersen RE, Bartlett SJ, Foster GD, Kuehnel RH, Wilk J, Weinstock R, Buckenmeyer P, Berkowitz RI, Steen SN. Exercise in the treatment of obesity: effects of four interventions on body composition, resting energy expenditure, appetite, and mood. *J Consult Clin Psychol.* 1997;65:269–277.
94. Marks BL, Ward A, Morris DH, Castellani J, Rippe JM. Fat-free mass is maintained in women following a moderate diet and exercise program. *Med Sci Sports Exerc.* 1995;27:1243–1251.
95. Franklin BA, ed. *American College of Sports Medicine Guidelines for Exercise Testing and Prescription.* 7th ed. Baltimore, Md: Lippincott Williams & Wilkins; 2006.
96. Tucker LA, Silvester LJ. Strength training and hypercholesterolemia: an epidemiologic study of 8499 employed men. *Am J Health Promot.* 1996;11:35–41.
97. Kohl HW III, Gordon NF, Scott CB, Vaandrager H, Blair SN. Musculoskeletal strength and serum lipid levels in men and women. *Med Sci Sports Exerc.* 1992;24:1080–1087.
98. Manning JM, Dooly-Manning CR, White K, Kampa I, Silas S, Kesselhaut M, Ruoff M. Effects of a resistive training program on lipoprotein–lipid levels in obese women. *Med Sci Sports Exerc.* 1991;23:1222–1226.
99. Vincent KR, Braith RW, Bottiglieri T, Vincent HK, Lowenthal DT. Homocysteine and lipoprotein levels following resistance training in older adults. *Prev Cardiol.* 2003;6:197–203.
100. Shoup EE, Durstine JL. Acute circuit weight lifting and its effects on postheparin lipoprotein lipase activity. *Med Sci Sports Exerc.* 1991;23:4–9.
101. McKelvie RS, McCartney N, Tomlinson C, Bauer R, MacDougall JD. Comparison of hemodynamic responses to cycling and resistance exercise in congestive heart failure secondary to ischemic cardiomyopathy. *Am J Cardiol.* 1995;76:977–979.
102. Miller ER III, Jehn ML. New high blood pressure guidelines create new at-risk classification: changes in blood pressure classification by JNC 7. *J Cardiovasc Nurs.* 2004;19:367–373.
103. Keul J, Dickhuth HH, Simon G, Lehmann M. Effect of static and dynamic exercise on heart volume, contractility, and left ventricular dimensions. *Circ Res.* 1981;48(supp 1):I-162–I-170.
104. Braith RW, Graves JE, Pollock ML, Leggett SL, Carpenter DM, Colvin AB. Comparison of 2 vs 3 days/week of variable resistance training during 10- and 18-week programs. *Int J Sports Med.* 1989;10:450–454.

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