REVIEW ARTICLE

Sports Med 2000 Oct; 30 (4): 249-268
0112-1642/00/0010-0249/$20.00/0



# Strength Training in the Elderly
## Effects on Risk Factors for Age-Related Diseases

*Ben F. Hurley* and *Stephen M. Roth*

Department of Kinesiology, College of Health & Human Performance, University of Maryland, College Park, Maryland, USA

## Contents



## Abstract

Strength training (ST) is considered a promising intervention for reversing the loss of muscle function and the deterioration of muscle structure that is associated with advanced age. This reversal is thought to result in improvements in functional abilities and health status in the elderly by increasing muscle mass, strength and power and by increasing bone mineral density (BMD). In the past couple of decades, many studies have examined the effects of ST on risk factors for age-related diseases or disabilities. Collectively, these studies indicate that ST in the elderly: (i) is an effective intervention against sarcopenia because it produces substantial increases in the strength, mass, power and quality of skeletal muscle; (ii) can increase endurance performance; (iii) normalises blood pressure in those

with high normal values; (iv) reduces insulin resistance; (v) decreases both total and intra-abdominal fat; (vi) increases resting metabolic rate in older men; (vii) prevents the loss of BMD with age; (viii) reduces risk factors for falls; and (ix) may reduce pain and improve function in those with osteoarthritis in the knee region. However, contrary to popular belief, ST does not increase maximal oxygen uptake beyond normal variations, improve lipoprotein or lipid profiles, or improve flexibility in the elderly.

The well documented losses of muscular strength and muscle mass with age,[1-3] known as sarcopenia,[4] have important health consequences[5] because they are associated with an increased susceptibility to disability among the elderly,[6-9] an increased risk of falls[10] and hip fractures,[11] a decrease in bone mineral density (BMD)[12] and an increase in glucose intolerance.[13] These changes can predispose the elderly to an increased risk for a variety of diseases and disabilities.[7,8] Whether this results directly from losses of muscle mass or secondary to low strength levels, which then leads to disability and functional limitations, is unknown. Nevertheless, there has been an increasing body of evidence, in recent years, indicating that sarcopenia is associated with adverse public health and economic consequences and that strength training (ST) can delay or reverse some of these effects. Therefore, this review will discuss and critically evaluate the research literature on the effects of ST on risk factors for age-related diseases and disabilities that have been most commonly studied. For the purpose of this review, the topics will be limited to include the effects of ST on risk factors for sarcopenia, coronary heart disease (CHD), hypertension, diabetes mellitus, metabolic syndrome, obesity, osteoporosis and osteoarthritis. In addition, the prevention of disability will be discussed by focusing on the research literature that pertains to the effects of ST on the loss of flexibility with age and fall prevention.

## 1. Sarcopenia

### 1.1 Strength, Muscle Mass and Strength Training

The loss of strength with advanced age is associated with a deterioration of health status.[5,7,8] Whether the objective is to improve functional abilities or health status in the elderly, interventions should focus on preventing disability and progression of disease through modification of risk factors.[6] In addition to the more common goals of improving cardiovascular and metabolic function, these interventions should positively affect muscle mass, strength, power, flexibility and BMD.[6,14] Muscular strength alone is independently associated with functional ability in the elderly.[15,16] Because aerobic exercise training does not improve muscular force production in the elderly[17,18] and muscular strength may become more limiting for activities of daily living than cardiovascular function in frail elderly individuals,[19] ST may be the training modality of choice for some segments of the elderly population. The loss of muscle function is at least partially responsible for many functional limitations and disabilities in the elderly.[19]

There is a remarkable similarity between the losses in strength and body composition with aging and those resulting from physical inactivity.[20] There is also a comparable counteraction of these age-associated losses following a period of muscular activity.[20] Early reports that increases in strength levels following ST in the elderly were due entirely to neurological factors[21] were later refuted when direct assessments of muscle tissue were made from imaging techniques and/or muscle biopsies, showing that ST increases muscle mass in older men and women.[22-32]

We now have suggestive evidence for how much of the age-associated losses in strength and muscle mass can be reversed with ST. Strength losses assessed from isokinetic peak torque values occur at the rate of about 12 to 14% per decade after the age

of about 50 years,[2,3,33] and strength gains, assessed from 1 repetition maximum (1RM) values, of >30% occur within the first couple of months of heavy resistance ST in 65- to 75-year-old men and women.[34] Thus, about 2 months of ST essentially reverses at least 2 decades of strength loss with advanced aging. Similar reversals can be observed with muscle mass, which is lost at a rate of about 6% per decade after the age of 50 years[3] and increased by about 12% within the first couple of months of ST.[35] Thus, 2 decades of age-induced muscle mass loss can be reversed with only about 2 months of ST.

### 1.2 Mechanisms for Increases in Strength

The mechanisms responsible for ST-induced increases in strength in the elderly are not entirely understood, but increases in motor unit firing frequency and maximal motor unit recruitment rates[36-38] are likely contributors to the substantial increases in strength after short term ST. In addition, an improved efficiency of motor unit firing rates is a probable mechanism explaining the improved ability to maintain steady submaximal contractions with ST in older individuals.[39,40]

There is some evidence that the local insulin-like growth factor (IGF; somatomedin) system may be involved in the mechanism for ST-induced muscle hypertrophy in the elderly.[32,41] However, this does not appear to result in an increase in baseline serum levels of growth hormone or IGF-1.[42] Although it has been reported that specific training regimens may determine how blood levels of these anabolic factors respond,[43,44] there is no evidence that these responses are indicative of muscle IGF-1 levels (either mRNA levels for IGF-1, IGF-1 receptors, or IGF-1 binding proteins) or that they are related to muscle hypertrophy. Although investigators have often shown little or no effect of increased circulating IGF-1 on muscle hypertrophy,[45,46] Adams and McCue[47] and others[48-51] have demonstrated that local production or infusion of IGF-1 is directly related to muscle hypertrophy. Specific mechanisms for this relationship have been investigated recently.[51-53] Increased circulating IGF-1 appears to stimulate general increases in fat free mass (FFM), rather than increases in muscle mass specifically.[46,54] These data indicate the importance of assessing local IGF-1 production when assessing the effects of ST interventions on muscle hypertrophy. In this regard, a recent study demonstrated for the first time an increase in local (muscle) IGF-1 levels with ST.[32]

ST can stimulate muscle fibre hypertrophy in all fibre types in older individuals[55-57] and fibre type transitions have also been reported.[58] These results suggest that the muscles of older individuals can adapt to a ST stimulus, such that age-related muscle fibre atrophy may be completely reversed in some individuals.[55] Several investigations have demonstrated that satellite cell proportions increase following muscle stimulation or exercise in young[59,60] and older animals.[61,62] As satellite cells appear necessary for the hypertrophic response to muscle overload,[63,64] assessing the role of ST on satellite cell activation in humans is important. For a more detailed overview of potential mechanisms for increased strength and muscle hypertrophy with ST among the elderly, the reader is referred to our recent review on this topic.[65]

Regardless of what mechanisms are responsible for training-induced muscle hypertrophy in the elderly, it is clear that when older people maintain muscular activity, the losses in strength with age are reduced substantially.[66]

## 2. Coronary Heart Disease

### 2.1 Low Cardiovascular Fitness Levels

Cardiovascular fitness, as assessed by time to exhaustion on a treadmill test, is an important risk factor for all-cause mortality as well as mortality and morbidity associated with CHD in both men and women.[67,68] The relative risk (RR) of death associated with a lack of cardiovascular fitness (3.44 for men, 4.65 for women) is greater than or similar to the RR associated with cigarette smoking (2.60 for men, 2.08 for women), high cholesterol levels (2.21 for men, 2.69 for women), hypertension (1.74 for men, 3.24 for women), a family history of car-

diovascular disease (1.60 for men, 1.50 for women) and elevated fasting glucose levels (2.74 for men, 3.73 for women).[67] Furthermore, the RR of developing CHD from being physically inactive (RR = 1.9) is similar to the RR associated with cigarette smoking (2.5), hypertension (2.3) and hypercholesterolaemia (2.1).[68] Aging is associated with a marked decrease in cardiovascular fitness as indicated by a decline in maximal oxygen uptake ($\dot{V}O_{2max}$).[69] However, it appears that as much as half of this reduction may be caused by secondary factors, such as increased body fat and decreased physical activity.[69]

### 2.2 Strength Training and Cardiovascular Fitness

It is well established and obvious that aerobic exercise training leads to substantial improvements in cardiovascular fitness, but the effects of ST have been more controversial. It is thought by many investigators in the field that ST can improve cardiovascular fitness in elderly people, but whether this conclusion is actually supported by evidence depends on how cardiovascular fitness is defined and what magnitude of change constitutes a real improvement. For example, Frontera and co-workers[70] reported an increase of 5% in $\dot{V}O_{2max}$ measured on a cycle ergometer after 12 weeks of ST in healthy 60- to 72-year-old men. Although this change was statistically significant, it is substantially below the magnitude of changes reported with aerobic exercise training, and it is within the range of normal biological and/or methodological drifts reported from inactive control groups during similar time periods.[71,72] Furthermore, because noncyclists are usually unable to achieve true $\dot{V}O_{2max}$ on a cycle ergometer, it is unlikely that a true $\dot{V}O_{2max}$ was measured on these individuals. This is an important distinction because, in contrast to $\dot{V}O_{2max}$ measured on a treadmill using an appropriate protocol, cycle ergometer performance among noncyclists appears to be limited by noncardiovascular factors, such as leg strength or power and lactate threshold.[73,74]

A more recent report by Hagerman et al.[75] showed a 9% increase in a Bruce protocol treadmill peak $\dot{V}O_2$ test with ST. This was reduced to a 5.7% increase when peak $\dot{V}O_2$ values were corrected for gains in fat free mass.

Evidence supporting the conclusion that true $\dot{V}O_{2max}$ does not change appreciably with ST, even when there is an increase in peak $\dot{V}O_2$, are the data of Hickson et al.[73] who found that 10 weeks of ST in young men resulted in a significant increase in peak $\dot{V}O_2$ tested on a cycle ergometer, but did not change true $\dot{V}O_{2max}$ measured during a maximal treadmill exercise test.[73] Similar conclusions were made from a study by Marcinik et al.[74] performed in our laboratory, in which time to exhaustion on a cycle ergometer was increased by 33% with circuit ST, despite no significant change in treadmill $\dot{V}O_{2max}$. Furthermore, most evidence indicates that ST, even when performed with short rest intervals, does not increase $\dot{V}O_{2max}$ much beyond normal biological or methodological variation.[71,72]

A mechanism has been reported that explains why ST fails to produce substantial improvements in aerobic capacity, even when training heart rates are maintained to the same level as high intensity aerobic exercise training.[72] The explanation is that ST stimulates a catecholamine surge approximately 7-fold higher than aerobic training in the same individual at the same oxygen cost. This results in heart rate elevations that are disproportionate to the low aerobic demands of the muscle during training, leading to an oxygen pulse during exercise below the threshold necessary to elicit the kind of cardiovascular adaptations observed with aerobic exercise training.[72]

ST may, however, elicit other more generalised adaptations that might benefit the cardiovascular systems of older men and women. Ades et al.[76] found that 12 weeks of ST increased treadmill walking endurance at 80% $\dot{V}O_{2max}$ by 38% in 65- to 79-year-old women, even though their $\dot{V}O_{2max}$ did not change.[76] Improvements in treadmill endurance time were significantly related to increased leg strength. Parker and co-workers[77] reported that 16 weeks of ST decreased heart rate, blood pressure (BP) and rate pressure product, as an index of myocardial oxygen uptake, significantly during a weight-

loaded submaximal treadmill walking test in 60- to 77-year-old women. Heart rate, BP and rate pressure product during short term resistive exercise are also lower after ST.[78] Thus, endurance performance may increase with ST despite little or no changes in $\dot{V}O_{2max}$. The specific mechanisms for these submaximal cardiovascular adaptations are not well understood, but possible explanations include changes in fibre type recruitment (i.e. greater rate of type I and a reduced rate of type II muscle fibre recruitment), less occlusion of blood flow and increased lactate threshold.[73,74]

### 2.3 Abnormal Plasma Lipoprotein-Lipid Profiles

Studies on the relationship of age, lipoprotein-lipid profiles, morbidity and mortality from cardiovascular disease in the elderly have produced conflicting results. For example, Wilson et al.,[79] in an 8-year follow-up of some 4900 men and women 20 to 79 years of age, found that total blood cholesterol levels actually declined with age. Others have suggested that this may be caused by disease rather than age,[80] although individuals with cardiovascular disease and cancer were excluded from the study of Wilson et al.[79] Nevertheless, declines in high-density-lipoprotein cholesterol (HDL-C) levels with age were still observed. The total cholesterol to HDL-C ratios increased slightly from young to middle-aged and stayed about the same in middle-aged to elderly men, whereas this ratio continued to increase from young to middle-aged and from middle-aged to the oldest women.

Studies investigating the predictive value of lipid profiles in the elderly have also produced mixed results. Krumholz et al.[81] concluded that neither high total cholesterol nor low HDL-C levels predict all-cause mortality, mortality caused by cardiovascular disease, or cardiovascular events in people >70 years of age. In contrast, Frost et al.[82] reported that lipid profiles do predict cardiovascular events for people over the age of 60 years, and Schaefer et al.[83] found that both low density lipoprotein-cholesterol (LDL-C) and HDL-C are important predictors of longevity. Thus, it appears that abnormal lipoprotein-lipid profiles confer increased cardiovascular disease risk in individuals at least until the age of 70 years.

### 2.4 Strength, Strength Training and Lipoprotein-Lipid Profiles

Two epidemiological studies have addressed the relationships among muscular strength, ST and plasma lipoprotein-lipid profiles. Kohl et al.[84] examined the association between muscular strength and serum lipoprotein-lipid levels in 1193 women and 5460 men. They found no association between strength and total or LDL-C for either men or women. However, there was a direct association between both upper and lower body strength and triglyceride levels in men, and an inverse relationship between muscular strength and HDL-C. In contrast, Tucker and Silvester[85] studied 8499 male employees of more than 50 companies, and observed a reduced risk of hypercholesterolaemia among individuals undergoing ST. However, only those who participated in ST 4 to 7 hours per week maintained this reduced risk when other potentially confounding variables were controlled.

Published reports on the effects of ST on plasma lipoprotein-lipid profiles have been limited preponderantly to adolescent, young and middle-aged individuals. Some of these studies have shown improvements in lipid profiles with ST in young[86,87] and middle-aged[71,88] individuals, but most of these studies either did not control for normal variations in lipoproteins, used individuals who were not at risk for cardiovascular disease, lacked proper dietary controls, or did not control for other factors that influence lipid profiles. When an attempt is made to control for at least some of these factors, most studies show no improvements in lipid profiles with ST.[89-94] This may be because of the methodological differences[95,96] or because bodyweight loss is needed to improve lipid profiles.[97]

To address the bodyweight loss issue, we recently studied the effects of 16 weeks of heavy resistance ST with or without bodyweight loss and found that ST does not elicit beneficial changes in lipoprotein-lipid levels in obese postmenopausal

women (ages 50 to 69 years) regardless of whether bodyweight loss is involved.[92] Similar findings of no improvements in lipid profiles in older women with ST have been reported by others.[91,94] Thus, we are not aware of any studies using elderly individuals that have demonstrated improvements in lipid profiles with ST.

### 2.5 Aerobic Training Versus Strength Training

Considering there are many more studies showing improved lipid profiles with aerobic exercise training than with ST in middle-aged and older individuals, it is quite surprising that out of the 5 published studies we could find that compared these 2 training modalities, none of them showed any differences in the effectiveness of aerobic exercise training compared with ST for improving lipoprotein-lipid profiles. In fact, all of these studies showed that neither training modality improved plasma lipoprotein-lipid profiles significantly.[89,93,98-100]

However, recent work by Hagberg et al.[101] suggests that genotype, particularly ApoE genotype, may be an important determinant as to whether aerobic exercise training improves lipoprotein-lipid profiles. It is conceivable that the same is true for lipoprotein-lipid responses to ST, but at the time of writing evidence for this is unavailable.

## 3. Hypertension

Resting BP increases with age, and elevated resting BP is a major cardiovascular disease risk factor in the elderly.[102] By age 60 to 70 years, approximately 50% of men and women are hypertensive.[102] Elevated BP remains a major cardiovascular disease risk factor in the elderly, except for those over 85 years of age.[102]

The limited information that is available on the effects of ST on resting BP in middle-aged and older individuals is conflicting. For example, while there is some evidence of a possible lowering effect of ST on BP,[71,103] not all studies have supported this finding. In the only previous study we could find addressing the effects of ST on BP in elderly men and women, Cononie and Graves[104] investigated the effects of 6 months of moderate resistance ST on resting BP in a group of 70- to 79-year-old men and women. There were no changes following ST in either systolic blood pressure (SBP) or diastolic blood pressure (DBP) in individuals characterised as having normal or elevated BP. A similar group of individuals performing 6 months of aerobic training in the same study showed significant BP reductions, but no significant differences in SBP when compared with the ST or control groups following training. Until recently this was the only study we were aware of that investigated the effects of ST on resting BP in older adults and reported no improvements.[104] Consequently, the conclusion from the American College of Sports Medicine Position Stand is that ST by itself does not consistently elicit significant reductions in BP in hypertensive individuals.[105]

However, the results from a recent study from our laboratory shows that heavy resistance, high volume ST can reduce resting BP in 65- to 73-year-old men and women whose average values are in the high normal range for resting BP.[106] The reductions were maintained for up to 48 hours following a ST session and were sufficient to shift mean values from the high normal to the normal category, as defined by the Joint National Committee on the Detection, Evaluation and Treatment of High Blood Pressure.[107] When the BP responses to ST were analysed by gender, men showed reductions in BP that were not observed in the women (i.e. 48 and 72 hours after a ST session for SBP and at 24 hours after a ST session for DBP). Because a large proportion of cardiovascular disease occurs in people with high normal BP,[108] this category shift has important implications, especially if corroborated by others, because overall cardiovascular morbidity and mortality is reduced in individuals who shift from the high normal to the normal BP category.[109]

Although most studies show significant reductions in resting BP as a result of aerobic exercise training, other studies comparing the effects of ST and aerobic training on resting BP have also shown no differences between aerobic and ST for the effectiveness in reducing resting BP.[93,110] Smutok

et al.[93] compared 20 weeks of moderate to heavy resistance ST to aerobic training and no exercise in 37 previously sedentary middle-aged men. No significant changes in resting BP were detected following training in any of the groups. Blumenthal et al.[110] studied 99 men and women with untreated mild hypertension (SBP 140 to 180mm Hg, DBP 90 to 105mm Hg) randomly assigned to 4 months of either aerobic exercise training, strength and flexibility training, or a control group. Despite significant within-group reductions of 7 to 9mm Hg in resting SBP and 5 to 6mm Hg in resting DBP following ST, there were no significant differences between any of the groups. There were also no significant group differences when comparing ambulatory BP readings before and after the training period. No studies could be found that independently assessed the effect of ST on the BP of older adults with essential hypertension.

## 4. Diabetes Mellitus

### 4.1 Glucose Intolerance and Insulin Resistance

Aging is associated with impaired glucose metabolism,[111,112] predisposing older men and women to the development of insulin resistance syndrome,[113] diabetes mellitus,[112] and CHD.[114] Since age-associated reductions in muscle mass are thought to be related to a deterioration in glucose metabolism,[13] it has been hypothesised that ST and subsequent increases in FFM may improve glucose and insulin responses to a glucose load in older populations.[93,115] However, ST does not usually change glucose tolerance,[71,115-117] or glycaemic control regardless of age, unless baseline glucose tolerance is abnormal.[93,118-122] Nevertheless, ST reduces insulin responses during an oral glucose tolerance test (OGTT) in young, middle-aged and older men in many,[71,115-117] but not all,[98] studies. In addition, ST studies have shown improvements in insulin sensitivity during hyperglycaemic and hyperinsulinaemic-euglycaemic clamps in healthy and diabetic middle-aged men[115,123,124] and women.[125]

In addition to being important in the aetiology of diabetes mellitus, glucose intolerance and insulin resistance are independent risk factors for CHD[114,126,127] and are also associated with hypertension,[128] elevated blood levels of LDL and reduced levels of HDL-C.[129] The prevalence of glucose intolerance increases with age,[13,130-133] because of insulin resistance.[132] This leads to hyperinsulinaemia[111,131] and may be caused by increased adiposity and/or decreased physical activity.[134-136] Evidence for this comes from Pacini et al.,[136] who found no independent association between age and glucose tolerance when the effects of obesity and physical activity were eliminated from consideration. Broughton and Taylor[134] reached a similar conclusion based on their analysis of previous studies. Glucose intolerance has also been found to be associated with age-related losses of muscle mass.[13] However, the age-associated deterioration in glucose tolerance and increase in hyperinsulinaemia are not observed in older athletes who do not participate in the type of exercise that leads to muscular hypertrophy,[137] whereas such deterioration is observed in older athletes who have substantially greater muscle mass.[138] Moreover, aerobic exercise training reduces plasma insulin responses to an oral glucose challenge in older individuals even though it does not increase muscle mass.[139] Thus, the evidence does not support the hypothesis that a reduced muscle mass with age is responsible for a deterioration in glucose tolerance.

It has commonly been believed, based on the notion that improvements in glucose metabolism were dependent on reductions in body fat[140] and increases in $\dot{V}O_{2max}$,[141] that only aerobic exercise training should be recommended for improving glucose homeostasis.[142] However, ST has been observed to improve glucose metabolism in individuals with normal[71,93] and abnormal[122] glucose metabolism, even when body fat or $\dot{V}O_{2max}$ is not changed. Some individuals with impaired glucose tolerance become normalised following ST.[93] Glucose metabolism is improved in both young[117,118] and older[116,143] individuals with ST, most often in the form of blunted plasma insulin responses to an

OGTT, but a blunted glucose response has also been demonstrated in middle-aged individuals.[93,122]

Improvements in glucose tolerance[144,145] or glycaemic control[146-148] have been shown preponderantly in ST studies involving middle-aged individuals with impaired glucose tolerance and/or type 2 diabetes mellitus, but most other studies show no change in glucose tolerance with ST in young,[23,149] middle-aged[86,150] and older[149,151] men, as well as older women.[153] Similarly to these findings, Hersey et al.[98] found that ST did not improve glucose responses to an OGTT in healthy 70- to 79-year-old men and women, but in contrast to others who found improved insulin responses, they also found no improvement in insulin responses. Other ST studies using hyperinsulinaemic-euglycaemic clamps and intravenous insulin tolerance tests have shown improvements in glucose uptake and insulin sensitivity in normally glucose-tolerant middle-aged[115] and elderly[153] men, middle-aged women,[125] as well as individuals with type 2 diabetes mellitus.[124]

Heavy resistance ST increased insulin action and reduced hyperinsulinaemia in postmenopausal women with or without bodyweight loss,[125] and increased glucose uptake by 23% in middle-aged and older (50 to 70 years) men.[115] A potential mechanism that explains how ST might improve glucose tolerance and/or insulin sensitivity has not yet been reported. However, the reduced plasma insulin response to an OGTT with ST appears to result from an increase in insulin clearance, not a decrease in insulin secretion.[143]

### 4.2 Aerobic Training Versus Strength Training

We could only find 3 studies that compared the effects of aerobic exercise training to ST on glucose homeostasis in middle-aged to older men and women.[93,98,122] Hersey et al.[98] found that only aerobic training lowered plasma insulin responses to an OGTT, whereas Smutok et al.[93,122] observed from our laboratory that both aerobic and moderate to heavy resistance ST reduced glucose and insulin responses to an OGTT. There are a number of potentially important differences among these studies. Hersey and co-workers[98] studied older persons than Smutok and co-workers[93,122] (70- to 79-*vs* 50- to 70-year-olds). Hersey and co-workers[98] also studied both men and women, whereas Smutok et al.[93,122] studied only men. The resistance used and the relative increases in both upper and lower body muscular strength were also much greater in the Smutok et al.[93,122] study compared with those in the Hersey et al.[98] study. Thus, it is possible that older individuals may require heavy resistance ST to improve their glucose and insulin responses to an OGTT.

To further address these issues, we recently studied the effects of a heavy resistance ST programme on glucose and insulin responses to an OGTT and compared these responses in young (20 to 30 years) and older (65 to 75 years) men and women who received the same relative training stimulus.[152] Until this investigation, no other studies had compared glucose-stimulated glucose and insulin responses with ST between older men and older women using the same relative training protocol. This comparison has potentially important health implications for elderly people, because elevated blood glucose levels are associated with a higher RR of mortality in women than in men.[67] The results demonstrate that the effects of ST on insulin response to an OGTT may be different for older men than for older women. In response to ST, men had reduced insulin responses at several time points with no changes in glucose responses to an OGTT, whereas women showed nonsignificant trends for increases in glucose and insulin responses at the same time points during the OGTT.[152]

Thus, substantial evidence exists to support the conclusion that ST can improve glucose homeostasis in men, and that ST may be just as effective as aerobic exercise training as an intervention against insulin resistance in men, but this does not appear to be the case in women.

## 5. Metabolic Syndrome

Abdominal obesity is thought to be the first step in a series of events that leads to insulin resistance, glucose intolerance, abnormal lipoprotein-lipid profiles and hypertension.[131,154,155] This constella-

tion of risk factors for cardiovascular disease, diabetes mellitus and hypertension has been called many names, including syndrome X, the deadly quartet, the Reaven syndrome, the insulin resistance syndrome, the atherothrombogenic syndrome,[149] the plurimetabolic syndrome,[97] the abdominal obesity syndrome and the metabolic syndrome.[131,154,155] Although there may be a genetic predisposition for abdominal obesity, increasing age, high fat diets and a sedentary lifestyle are also thought to be important determinants.[154,155]

### 5.1 Intra-Abdominal Fat

ST can reduce total body fat stores in older men and women, even when individuals are not undergoing restriction of energy intake.[31] However, since abdominal obesity is more consistently related to a metabolic profile predictive of cardiovascular disease risk than is general obesity,[97] a more important issue may be the effects of ST on abdominal visceral fat depots. Aging is associated with a preferential deposition of fat in the abdominal region, especially in men.[156] Abdominal obesity may increase the risk for cardiovascular disease independent of other cardiovascular disease risk factors,[157] but it is also closely associated with other risk factors.[154,155]

There is very little information available on the effects of ST on visceral fat in older individuals. Using dual energy x-ray absorptiometry, Treuth et al.[31] observed reductions in truncal fat mass of middle-aged and older (50 to 70 years) men after 16 weeks of total body ST. In a follow-up study using computed tomography, Treuth et al.[94] found significant ST-induced reductions in intra-abdominal fat in older women. However, diet cannot be ruled out as a factor that could have affected the results from ST studies addressing intra-abdominal fat.

Although no studies measuring abdominal fat have controlled diet adequately throughout the entire training programme, Ross and co-workers[158,159] have performed some of the best controlled studies assessing the effects of diet and exercise training. They used magnetic resonance imaging to provide a direct measurement of fat tissue in studies comparing regional fat losses with diet combined with either aerobic exercise training or ST. In their first study,[158] there were no differences between the 2 groups (i.e. diet and aerobic exercise training *vs* diet and ST) for losses in either whole body subcutaneous fat or visceral fat, but within each group there was a significantly greater visceral fat loss compared with subcutaneous fat loss. In a follow-up study[106] they isolated the effects of aerobic exercise training and ST by comparing the responses to diet alone and diet combined with each training modality in 33 middle-aged obese men. All 3 groups lost a substantial amount of whole body subcutaneous and visceral fat, and all 3 groups experienced a significantly greater visceral fat loss compared with whole body subcutaneous fat loss. The changes amounted to a 39% reduction in visceral fat in the diet and aerobic training group, a 40% reduction in the diet and ST group and a 32% reduction in the diet only group. These differences among the groups were not significant. We estimated that the low volume ST programme required less than a third of the energy required for the aerobic training programme. When comparing losses from the abdominal subcutaneous fat depot to those from the gluteal-femoral (leg) region, there was a preferential loss from the abdominal region in the 2 training and diet groups, but no preferential losses in the diet-only group. Both training and diet groups had the advantage of maintaining whole body skeletal muscle tissue, whereas the diet only group lost muscle tissue.

It is somewhat surprising that the aerobic training programme did not result in significantly greater fat losses compared with ST because the caloric expenditure associated with ST sessions is substantially less than generally occurs during aerobic training sessions. It is unclear exactly what accounts for this discrepancy in energy balance, but one possible explanation could be increases in resting metabolic rate (RMR) with ST.

### 5.2 Resting Metabolic Rate

Aging is associated with a loss of FFM and an increase in fat mass.[156] FFM loss is accompanied

by a decline in RMR, which can lead to obesity.[160] Although FFM is by far the major determinant of RMR, explaining approximately 60 to 70% of the interindividual variability,[161] the age-related reduction in $Na^+$-$K^+$ pump activity[162] also contributes to the decline in RMR with age.

In a cross-sectional study, Poehlman et al.[163] found higher RMR values in ST athletes compared with untrained controls. Investigators who have examined the effect of ST on RMR have shown mixed results, whether men[164-167] and women[168-171] were studied separately or combined.[144,172,173] Most,[144,165,169,171,172] but not all,[170,173] studies have demonstrated an increase in RMR in older individuals in response to ST. In contrast, investigations in young individuals have found a consistent lack of change in RMR with ST.[164,166-168,173] One study that compared age responses of RMR to ST showed no change in RMR for either young (age 26 years) or older (age 70 years) individuals.[173] This finding could be related to the fact that neither group increased FFM in response to ST. Pratley et al.,[165] from our group, studied 50- to 65-year-old men before and after 16 weeks of heavy resistance ST and observed a 2.6% increase in FFM, a 7.7% increase in RMR and a 36% increase in resting plasma noradrenaline levels. We concluded that the increases in FFM and the increased activity of the sympathetic nervous system may be responsible for the training-induced increase in RMR.[165] Campbell et al.[144] also found increases in FFM and RMR in older men and women with heavy resistance ST. However, they concluded that the increase in RMR was caused by an increase in the metabolic activity of lean tissue and not an increase in FFM, since the increase in FFM was reported to be caused by an increase in body water. Ryan et al.[169] observed a significant rise in RMR in postmenopausal women aged 50 to 69 years as a result of heavy resistance ST with and without bodyweight loss. In another study, during bodyweight loss from a very low energy diet, lean body mass and RMR were preserved by the addition of a high volume ST programme.[174]

However, other studies failed to show increases in RMR with ST. Taaffe et al.,[170] for example, reported that neither low- nor high-intensity ST altered RMR significantly. In addition, Treuth et al.[171] found that increases in RMR as a result of ST in postmenopausal women were not significant when increases in FFM were taken into account. Similar findings were reported by Van Etten et al.,[166] who observed no significant increases in metabolic rate with ST during sleep. Differences in training regimens and testing conditions may explain some of the discrepancies on this issue. As has been previously well established for aerobic training, both Van Etten et al.[166] and Treuth et al.[171] reported that ST significantly increased fat oxidation.

It has been suggested that increases in RMR and energy expenditure from physical activity outside of training[22,175] could explain fat losses that sometimes occur with ST[31,94,158,171,176] by increasing the total energy expenditure[144] outside of training. However, we recently observed no changes in energy expenditure from physical activity outside of training following a 6-month ST programme in 65- to 75-year-old men and women.[177] In addition, we found that changes in RMR in response to ST were affected by gender, but not by age. When young (20 to 30 years) and older (65 to 75 years) men were pooled as a group, there was a significant increase in RMR with training, whereas young (20 to 30 years) and older (65 to 75 years) women showed no change. When all groups were combined, there was again an increase in RMR. Furthermore, when RMR was corrected for FFM there was still a significant gender effect, with the men showing a significant increase in RMR, while women still showed no change, despite having similar increases in total body FFM. Others have observed increases in RMR with ST when older men and women are pooled together,[144,165,169,171,172] but the finding that this change is not significantly different from young men and women had not been reported previously. The finding of a lack of change in RMR in women is consistent with some previous studies in both young[168] and older[170] women and may be related

to differences in sympathetic nervous system activity.[165]

## 6. Osteoporosis

### 6.1 Fracture Risk Increases with Age

Osteoporosis is one of the most prevalent conditions in postmenopausal women and the prevalence of osteoporosis also increases with age in men, though it is a greater public health concern in women. The morbid events associated with osteoporosis are fractures that primarily occur in the neck of the femur, the vertebrae and the forearm in older men and women. The loss of BMD after menopause in women results in a doubling of hip fracture risk for every 5 years of age past the age of 50 years.[178] A third of 80-year-old women will have had a hip fracture and a third of those affected will have had 2 hip fractures.[178] The end result is that osteoporosis affects 25 million people, with most of them women, and osteoporosis is the primary cause of 1.5 million fractures annually.[179]

### 6.2 Bone Mineral Density

One major risk factor for hip fracture is low BMD. BMD decreases markedly in women in the 2 to 5 years immediately after menopause and continues to decline at a slower rate thereafter.[179] As a result, maintenance or enhancement of BMD in older persons, especially older women, is a major public health concern. Determining whether various forms of physical activity enhance BMD at critical skeletal sites in postmenopausal women has been a major area of research in recent years.

Because BMD is related to the strength of the proximal muscle groups[12,180] and because positive associations have been reported between BMD and muscle strength[181-183] and between bone mass and FFM,[184] ST has been hypothesised as an exercise intervention of choice for preserving age-associated losses in BMD. In this regard, Bevier et al.[185] in a cross-sectional study reported that muscular strength was a better predictor of BMD in older men and women than $\dot{V}O_{2max}$. Similarly, a number of studies have documented that those who participate in ST have markedly enhanced BMD compared with their sedentary peers.[186,187]

In 50- to 70-year-old women, Nelson and coworkers[175] found that heavy resistance ST essentially maintained BMD (0.9 and 1.0% increases at femoral neck and lumbar spine, respectively), whereas BMD decreased by 2.5% at the femoral neck and 1.8% in the lumbar spine in the control group. The elevated values for both femoral neck and the lumbar spine BMD were significantly different in the ST intervention group compared with the losses in the control group. Notelovitz and coworkers[188] found that 1 year of ST combined with estrogen replacement therapy increased spine, radial midshaft and total body BMD more in surgically postmenopausal women than did estrogen replacement therapy alone. Pruitt and colleagues[189] reported that 9 months of ST increased BMD at the lumbar spine, but not the femoral neck and distal wrist, in women with an average age of 54 years.

There appears to be a great deal of conflicting results in the literature on the effects of age and gender on BMD in response to ST, even from our own laboratory. For example, we reported that 50- to 70-year-old men increase femoral neck BMD and tend to increase lumbar spine BMD with 16 weeks of heavy resistance ST.[190,191] However, in a more recent study (Ryan AS, Ivey FM, Hurlbut DE, et al., unpublished data), we observed significant increases in BMD in several regions in young men and young women (20- to 30-year-olds), but no significant changes were observed in any regions in older men or in older women. Other studies have indicated that ST increased,[192,193] did not change[194] or decreased[195] BMD in premenopausal women and increased[175] or maintained[181,189] BMD in postmenopausal women. Ward's triangle BMD increased after 16 weeks of ST in elderly men,[196] but lumbar spine and femoral neck BMD have shown inconsistent responses in middle-aged and older men, with some studies showing increases[190,191] and other studies showing no significant changes in these regions.[196]

Because of the many conflicting reports it is difficult to make any definitive conclusions about the

effects of ST on specific regions of BMD. However, there does seem to be evidence from the best controlled studies that ST can at least prevent some of the losses in BMD that occur over time in older persons. Even among the studies that show significant increases in BMD with ST, the magnitude of these changes are not usually substantial enough to markedly reduce an older person's risk of bone fracture once a fall occurs.[197] It has been estimated that increases in BMD of >20% would be required to provide adequate protection against bone fractures resulting from falls.[197] Given the fact that most studies report <5% increase in BMD from any exercise training modality, it is more likely that exercise training could play a role in the prevention of falls rather than the prevention of fractures once a fall occurs.

## 7. Osteoarthritis

Osteoarthritis, the most common form of arthritis, is characterised by a progressive loss of articular cartilage around the affected joint leading to pain and functional disability.[198] The prevalence of osteoarthritis increases with age and is seen most often in older women.[199] Muscle atrophy and weakness have been hypothesised to contribute to the disability and pain of patients with osteoarthritis.[200-202] Thus, ST is thought to reduce functional instability and pain in older osteoarthritis patients by preventing sarcopenia and by improving the strength and function of the surrounding connective tissue, which is often damaged by the disease.[203] Early research indicated that both moderate[204,205] and heavy[206,207] resistance training resulted in improvements in pain and disability; however, possible methodological and research design flaws may have affected the results in these studies.[208]

Recent evidence, however, provides similar support for a role for ST in osteoarthritis therapy programmes. Schilke et al.[209] demonstrated that an 8-week ST programme improved strength and mobility and decreased joint pain and stiffness in patients with knee osteoarthritis. Further, improvements in both the Osteoarthritis Screening Index and the Arthritis Impact Measurement Scale indicated a significant decrease in arthritis activity in patients who underwent ST. Ettinger et al.[210] assessed 365 community-dwelling adults with knee osteoarthritis and self-reported physical disability. The individuals underwent either an aerobic exercise training programme, a ST programme or a health education programme. Significant but modest improvements were reported for self-reported disability, knee pain and other indices of physical function and strength in individuals from both the aerobic and ST programmes. The researchers concluded that both moderate-intensity aerobic and ST were generally well tolerated and effective therapies for older individuals with knee osteoarthritis.[210] Most recently, Rogind et al.[211] examined the role of a general physical activity programme that included progressive ST exercises in patients with severe knee osteoarthritis, and reported improved quadriceps strength, decreased pain, improved walking speed and decreased Algofunctional Index scores (an indicator of pain, discomfort and dysfunction). The researchers concluded that including strengthening exercises in a physical activity programme was beneficial for patients with severe knee osteoarthritis, although possible adverse effects such as knee joint effusions were discussed.[211]

*In summary*, research provides evidence for the benefits of ST in patients with knee osteoarthritis, although the appropriate level of resistance and possible long term consequences of ST in these patients are unclear. Further work is necessary to determine the most appropriate ST programme for osteoarthritis patients. Nevertheless, specific guidelines for the use of exercise in the treatment of osteoarthritis have been discussed recently.[212]

## 8. Prevention of Disability

### 8.1 Loss of Flexibility

The loss of flexibility or joint range of motion (ROM) with age is well documented[146,213,214] and is related to physical dysfunction and a decline in health status.[215,216] This loss in flexibility may be associated with difficulty in climbing stairs, getting up from a chair or bed and the need for walking

aids.[215] Much of this loss is thought to be caused by inactivity, suggesting that increasing muscular activity might at least delay losses in flexibility.[217,218] Kligman and Pepin[219] concluded that older adults who maintain high levels of muscular strength and flexibility are rarely candidates for long term healthcare.

It has often been concluded that as long as ST exercises are performed through the full ROM and both the agonist and antagonist muscle groups are exercised, ST will improve flexibility;[220,221] however, we were unable to find any published data to support this conclusion. Despite this potential link between muscular strength and flexibility, little information is available from well controlled published studies concerning the impact of ST on flexibility in older adults. Furthermore, most studies that have investigated the effects of ST on flexibility have included stretching exercises in the training programme, and thus the independent effects of ST on flexibility have not been studied appropriately. In a study from our laboratory,[222] joint ROM was assessed in older individuals (mean 61 years) before and after they performed either strength and flexibility training, flexibility-only training, or no training for 10 weeks. Despite performing all exercises through the full ROM, training both the agonist and antagonist muscle groups, and performing stretching exercises before and after every ST session, as is recommended for improving flexibility,[220,221] no significant improvements in flexibility were demonstrated in the individuals who performed both strength and flexibility training compared with the inactive control group. Moreover, there was significantly greater improvement in shoulder abduction in the individuals who performed the flexibility-only training compared with the strength and flexibility training and to the inactive control groups.[222]

Other investigations that have assessed whether ST can improve flexibility either used only young individuals,[223,224] did not indicate if stretching exercises were incorporated,[225] included aerobic exercise in the training programme,[226-228] used only low-resistance exercise,[228] or did not control for other factors that can affect flexibility.[225,228] These methodological differences may explain why investigators have reported increases,[217,224,227] no change[223,225] or losses[225] in flexibility with ST. Thus, there is no definitive evidence that a total body ST programme in itself improves flexibility in older adults. To the contrary, based on our work on this topic,[222] it is more likely that ST inhibits flexibility. However, because of the limitations of this and other previous studies, well-designed studies on the effects of heavy resistance ST on flexibility in older adults are needed. Nevertheless, based on the evidence available currently, it does not appear that ST alone should be used for the purpose of improving flexibility, even when training of the agonist and antagonist muscle groups and when exercises are performed through the full ROM. Brief warm-up and cool-down stretching may not be enough to improve flexibility significantly, particularly in the shoulder region.[214,222,225,228] Therefore, prolonged stretching should be an integral part of any properly designed ST programme.[229]

### 8.2 Fall Prevention

Cognitive impairment, visual deficits, environmental conditions and medication use may combine with physical activity–related risk factors such as neuromuscular, gait and balance impairments to increase the risk of falls.[230] Although many authors have emphasised the importance of regular exercise for prevention of falls in the elderly, only very limited data are available on this topic[7] and the information available is not conclusive. Nevertheless, ST can improve strength,[26] muscular power,[231] walking mechanics[22]and walking speed[22] in the elderly, all important risk factors for falls.

## 9. Conclusion

Table 1 provides an overview comparing the effects of aging to the effects of ST on indicators of muscle function and health status. The following conclusions can be made about the effects of ST on risk factors for age-related diseases or disabilities:

(i) Approximately 2 decades worth of age-associated losses in strength and muscle mass can

**Table I.** Effects of aging and strength training on risk factors for age-related diseases

| Disease/risk factors | Effects of aging | Reference | Effects of strength training | Reference |
|---|---|---|---|---|
| Sarcopenia | | | | |
| muscular strength | ↓↓↓ | 2, 3 | ↑↑↑ | 34 |
| muscle mass | ↓↓ | 156 | ↑↑ | 22-32 |
| muscle power | ↓↓ | 33 | ↑ | 231 |
| muscle quality | ↓ | 2, 3 | ↑ | 35 |
| Coronary heart disease | | | | |
| $\dot{V}O_{2max}$ | ↓↓ | 69 | ↔ | 71-74 |
| endurance performance | ↓↓ | 69 | ↑ | 76-78 |
| plasma lipoprotein-lipid profile | ↓ or ↔ | 79-83 | ↔ | 89-94 |
| Hypertension | ↑ | 102 | ↓ or ↔ | 71, 93, 104, 108, 110 |
| Diabetes | | | | |
| glucose intolerance | ↑ | 13, 130-133 | ↓ or ↔ | 71, 93, 98, 116, 122, 143, 149, 151, 152 |
| insulin resistance | ↑ | 132 | ↓ men, ↔ women | 67, 98, 115, 124, 125, 152, 153 |
| Abdominal obesity syndrome | | | | |
| total body fat | ↑↑ | 156 | ↓ | 31 |
| intra-abdominal fat | ↑ | 154, 155 | ↓ or ? | 31, 94, 158, 159 |
| resting metabolic rate | ↓ | 161, 162 | ↑ men, ↔ women | 144, 165, 169-173, 177 |
| Osteoporosis | | | | |
| bone mineral density | ↓ | 12, 179 | ↑ or ↔ | 175, 181, 186-191, 196 (unpublished data)[a] |
| risk of falls | ↑ | 10 | ↓ | 22, 175 |
| Loss of flexibility | ↑ | 146, 213, 214 | ↔ or ↑ | 217, 220-225, 227, 228 |
| Osteoarthritis | ↑ | 199 | ↓ | 203, 208-211 |

a Ryan AS, Ivey FM, Hurlbut DE, et al., unpublished data.

$\dot{V}O_{2max}$ = maximal oxygen uptake; ↓ = decrease; ↓↓ = moderate to large decrease; ↓↓↓ = very large decrease; ↑ = increase; ↑↑ = moderate to large increase; ↑↑↑ = very large increase; ↔ = little or no change or conflicting evidence; **?** = unknown or too little data available to conclude.

be regained within the first couple of months of heavy resistance ST.

(ii) ST fails to produce substantial changes in $\dot{V}O_{2max}$, but can improve endurance performance.

(iii) There is little or no evidence that ST can improve mean values of lipoprotein-lipid profiles. It is possible that people with certain genotypes may be able to improve their profiles with ST, but no evidence for this exists at the present time.

(iv) There is no evidence that ST can reduce BP in elderly hypertensives, but there is now evidence for normalising BP in the high normal category.

(v) Some studies show no changes in glucose tolerance with ST and others show reductions in plasma glucose in response to oral glucose tolerance tests. However, most studies show that ST can improve insulin action either through reductions in insulin responses from oral glucose tolerance tests or increased glucose uptake from glycaemic clamp studies.

(vi) There is evidence for reductions in total body fat and intra-abdominal fat with ST. However, no studies that have reported this have been able to completely rule out the effects of diet.

(vii) There is some evidence that ST can increase RMR in older men, but there is little evidence for this effect in women. An explanation for this gender difference has not been determined, but differences in sympathetic neural activity in response to ST has been suggested.

(viii) The evidence for increases in BMD with ST is mixed, but there is good evidence for the effectiveness of ST in preventing age-associated losses in BMD.

(ix) Although there is little or no evidence for ST preventing falls, there is strong evidence for reduction in several risk factors for falls.

(x) There is no evidence from properly controlled studies that ST improves flexibility, even when both the agonist and antagonist muscle groups are trained through the full ROM. There is suggestive evidence that ST in itself may even worsen flexibility. Therefore, prolonged stretching should be a part of any well designed ST programme.

(xi) There is some evidence that ST may reduce symptoms of osteoarthritis in the knee, but better controlled studies are needed.

## Acknowledgements

S.M. Roth was supported by NIA Grant AG-00268. Some of the research outlined from the authors' laboratory in the review was partially supported by NIH research contract AG-42148 (B.F. Hurley).

## References

1. Larsson L, Grimby G, Karlsson J. Muscle strength and speed of movement in relation to age and muscle morphology. J Appl Physiol 1979; 46: 451-6
2. Lindle R, Metter E, Lynch N, et al. Age and gender comparisons of muscle strength in 654 women and men aged 20-93. J Appl Physiol 1997; 83: 1581-7
3. Lynch NA, Metter EJ, Lindle RS, et al. Muscle quality I: age-associated differences in arm *vs* leg muscle groups. J Appl Physiol 1999; 86 (1): 188-94
4. Butler R. Did you say 'sarcopenia'? Geriatrics 1993; 48: 11-2
5. Young A, Skelton D. Applied physiology of strength and power in old age. Int J Sports Med 1994; 15: 149-51
6. Buchner D, Wagner E. Preventing frail health. Clin Geriatr Med 1992; 8: 1-17
7. Rantanen T, Guralnik JM, Ferrucci L, et al. Coimpairments: strength and balance as predictors of severe walking disability. J Gerontol A Biol Sci Med Sci 1999; 54A: M172-6
8. Rantanen T, Guralnik JM, Foley D, et al. Midlife hand grip strength as a predictor of old age disability. JAMA 1999; 281: 558-60
9. Rantanen T, Guralnik JM, Sakari-Rantala R, et al. Disability, physical activity, and muscle strength in older women: The women's health and aging study. Arch Phys Med Rehab 1999; 80: 130-5
10. Campbell A, Borrie M, Spears G. Risk factors for falls in a community-based prospective study of people 70 years and older. J Gerontol 1989; 44: M112-7
11. Aniansson A, Zetterberg C, Hadberg M, et al. Impaired muscle function with aging: a background factor in the incidence of fractures of the proximal end of the femur. Clin Orthop Relat Res 1984; 191: 193-200
12. Sinaki M, McPhee M, Hodgson S, et al. Relationship between bone mineral density of spine and strength of back extensors in healthy postmenopausal women. Mayo Clin Proc 1986; 61: 116-22
13. Bloesch D, Schultz Y, Breitenstein E, et al. Thermogenic response to an oral glucose load in man: comparison between young and elderly subjects. J Am Coll Nutr 1988; 7: 471-83
14. Bassey E, Fiatarone M, O'Neil E, et al. Leg extensor power and functional performance in very old men and women. Clin Sci 1992; 82: 321-7
15. Hyatt R, Whitelaw M, Bhat A, et al. Association of muscle strength with functional status of elderly people. Age Ageing 1990; 19: 330-6
16. Buchner D, de Lateur BJ. The importance of skeletal muscle strength to physical function in older adults. Ann Behav Med 1991; 13: 91-8
17. Thompson R, Crist D, Marsh M, et al. Effects of physical exercise for elderly patients with physical impairments. J Am Geriatr Soc 1988; 36: 130-5
18. Klitgaard H, Mantoni M, Schiaffino S, et al. Function, morphology and protein expression of ageing skeletal muscle: a cross-sectional study of elderly men with different training backgrounds. Acta Physiol Scand 1990; 140: 41-54
19. Pendergast D, Fisher N, Calkins E. Cardiovascular, neuromuscular and metabolic alterations with age leading to frailty. J Gerontol 1993; 48: 61-7
20. Bortz W. Disuse and aging. JAMA 1982; 248: 1203-8
21. Moritani T, deVries H. Potential for gross muscle hypertrophy in older men. J Gerontol 1980; 35: 672-82
22. Fiatarone M, O'Neill E, Ryan N, et al. Exercise training and nutritional supplementation for physical frailty in very elderly people. N Engl J Med 1994; 330: 1769-75
23. Brown A, McCartney N, Sale D. Positive adaptation to weight-lifting in the elderly. J Appl Physiol 1990; 69: 1725-33
24. Charette S, McEvoy L, Pyka G, et al. Muscle hypertrophy response to resistance training in older women. J Appl Physiol 1991; 70: 1912-6
25. Fiatarone M, Marks E, Ryan N, et al. High-intensity strength training in nonagenarians. JAMA 1990; 263: 3029-34
26. Frontera W, Meredith C, O'Reilly K, et al. Strength condition in older men: skeletal muscle hypertrophy and improved function. J Appl Physiol 1988; 63: 1038-44
27. Grimby G, Aniansson A, Hedberg M, et al. Training can improve muscle strength and endurance in 78- to 84-yr-old men. J Appl Physiol 1992; 73: 2517-23
28. Roman W, Fleckenstein J, Stray-Gundersen J, et al. Adaptations in the elbow flexors of elderly males after heavy-resistance training. J Appl Physiol 1993; 74: 750-4
29. Staron R, Karapondo D, Kraemer W, et al. Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. J Appl Physiol 1994; 76: 1247-55
30. Hurley B, Redmond R, Pratley R, Trueth M, Rogers M, Goldberg A. Effects of strength training on muscle hypertrophy and muscle cell disruption in older men. Int J Sports Med 1995; 16: 380-6
31. Treuth M, Ryan A, Pratley R, et al. Effects of strength training on total and regional body composition in older men. J Appl Physiol 1994; 77: 614-20
32. Singh MA, Ding W, Manfredi TJ, et al. Insulin-like growth factor I in skeletal muscle after weight-lifting exercise in frail elders. American Journal of Physiology 1999; 277 (1 Pt 1): E135-43
33. Metter EJ, Conwit R, Tobin J, et al. Age-associated loss of power and strength in the upper extremities in women and men. J Gerontol A Biol Sci Med Sci 1997; 52: B267-76

34. Lemmer JT, Hurlbut DE, Martel GF, et al. Age and gender responses to strength training and detraining. Med Sci Sports Exerc 2000; 32: 1505-12
35. Tracy BL, Ivey FM, Hurlbut D, et al. Muscle quality II: effects of strength training in 65-75 year old men and women. J Appl Physiol 1999; 86 (1): 195-201
36. Leong B, Kamen G, Pattern C, et al. Maximal motor discharge rates in the quadriceps muscles of older weight lifters. Med Sci Sports Exerc 1999; 31: 1638-44
37. Patten C, Kamen G. Adaptations in human motor unit discharge behavior to strength training [abstract]. Soc Neurosci 1996; 22: 130
38. Enoka RM. Neural adaptations with chronic physical activity. J Biomech 1997; 30: 447-55
39. Keen DA, Yue GH, Enoka RM. Training-related enhancement in the control of motor output in elderly humans. J Appl Physiol 1994; 77: 2648-58
40. Laidlaw DH, Kornatz KW, et al. Strength training improves the steadiness of slow lengthening contractions performed by old adults. J Appl Physiol 1999; 87: 1786-95
41. Chen Y, Bornfeldt KE, Arner A, et al. Increase in insulin-like growth factor I in hypertrophying smooth muscle. Am J Physiol 1994; 266: 224-9
42. Craig B, Brown R, Everhart J. Effects of progressive resistance training on growth hormone and testosterone levels in young and elderly subjects. Mech Ageing Dev 1989; 49: 159-69
43. Kraemer W, Marchitelli L, Gordon S, et al. Hormonal and growth factor responses to heavy resistance exercise protocols. J Appl Physiol 1990; 69: 1442-50
44. Kraemer W, Fleck S, Dziados J, et al. Changes in hormonal concentrations after different heavy-resistance exercise protocols in women. J Appl Physiol 1993; 75: 594-604
45. Taaffe DR, Jin IH, Vu TH, et al. Lack of effect of recombinant human growth hormone (GH) on muscle morphology and GH-insulin-like growth factor expression in resistance-trained elderly men. J Clin Endocrinol Metab 1996; 81: 421-5
46. Yarasheski KE, Campbell JA, Smith K, et al. Effect of growth hormone and resistance exercise on muscle growth in young men. Am J Physiol 1992; 262 (3 Pt 1): E261-7
47. Adams GR, McCue SA. Localized infusion of IGF-I results in skeletal muscle hypertrophy in rats. J Appl Physiol 1998; 84: 1716-22
48. Czerwinski S, Martin J, Bechtel, PJ. Modulation of IGF mRNA abundance during stretch-induced skeletal muscle hypertrophy and regression. J Appl Physiol 1994; 76: 2026-30
49. Yang S, Alnaqeeb M, Simpson H, et al. Changes in muscle fibre type, muscle mass and IGF-I gene expression in rabbit skeletal muscle subjected to stretch. J Anat 1997; 190: 613-22
50. Coleman ME, DeMayo F, Yin KC. Myogenic vector expression of insulin-like growth factor I stimulates muscle cell differentiation and myofiber hypertrophy in transgenic mice. J Biol Chem 1995; 270: 12109-16
51. Musaro A, McCullagh KJA, Naya FJ, et al. IGF-1 induces skeletal myocyte hypertrophy through calcineurin in association with GATA-2 and NF-ATc1. Nature 1999; 400: 581-5
52. Napier JR, Thomas MF, Sharma M, et al. Insulin-like growth factor-1 protects myoblasts from apoptosis but requires other factors to stimulate proliferation. J Endocrinol 1999; 163: 63-8
53. Semsarian C, Wu MJ, Ju YK. Skeletal muscle hypertrophy is mediated by a $Ca^{2+}$-dependent calcineurin signalling pathway. Nature 1999; 400: 576-81
54. Grindland RE, Roy RR, Edgerton VR. Interactive effects of growth hormone and exercise on muscle mass in suspended rats. Am J Physiol 1994; 267: R316-22
55. Larsson L. Physical training effects on muscle morphology in sedentary males at different ages. Med Sci Sports Exerc 1982; 14: 203-6
56. Pyka G, Lindenberger E, Charette SL, et al. Muscle strength and fiber adaptations to a year-long resistance training program in elderly men and women. J Gerontol 1994; 49: M22-7
57. Martel GF, Roth SM, Ivey FM, et al. Effects of strength training on muscle fiber characteristics in young and older men and women [abstract]. Med Sci Sports Exerc 1999; 31: S325
58. Hakkinen K, Newton RU, Gordon SE. Changes in muscle morphology, electromyographic activity, and force production characteristics during progressive strength training in young and older men. J Gerontol A Biol Sci Med Sci 1998; 53 (6): B415-23
59. Darr KC, Schultz E. Exercise-induced satellite cell activation in growing and mature skeletal muscle. J Appl Physiol 1987; 63: 1816-21
60. Snow MH. Satellite cell response in rat soleus muscle undergoing hypertrophy due to surgical ablation of synergists. Anatomical Record 1990; 227: 437-46
61. Jacobs SM, Wokke JJ, Bar PR, et al. Satellite cell activation after muscle damage in young and adult rats. Anat Rec 1995; 242: 329-36
62. McCormick KM, Thomas DP. Exercise-induced satellite cell activation in senescent soleus muscle. J Appl Physiol 1992; 72: 888-93
63. Phelan JN, Gonyea WJ. Effect of radiation on satellite cell activity and protein expression in overloaded mammalian skeletal muscle. Anat Rec 1997; 247: 179-88
64. Rosenblatt JD, Yong D, Parry DJ. Satellite cell activity is required for hypertrophy of overloaded adult rat muscle. Muscle Nerve 1994; 17: 608-13
65. Roth SM, Ferrell RE, Hurley BF. Strength training for the prevention and treatment of sarcopenia. J Nutr Health Aging 2000. In press
66. Greig C, Botella J, Young A. The quadriceps strength of healthy elderly people remeasured after eight years. Muscle Nerve 1993; 16: 6-10
67. Blair S, Kohl H, Paffenbarger R, et al. Physical fitness and all-cause mortality: a prospective study of healthy men and women. JAMA 1989; 262: 2395-401
68. Powell K, Thompson P, Caspersen C, et al. Physical activity and the incidence of coronary heart disease. Ann Rev Public Health 1987; 8: 253-87
69. Heath G, Hagberg J, Ehsani A, et al. A physiological comparison of young and older endurance athletes. J Appl Physiol 1981; 51: 634-40
70. Frontera W, Meredith CN, O'Reilly K, et al. Strength training and determinants of $\dot{V}O_{2max}$ in older men. J Appl Physiol 1990; 68: 329-33
71. Hurley B, Hagberg J, Goldberg A, et al. Resistive training can reduce coronary risk factors without altering $\dot{V}O_{2max}$ or percent body fat. Med Sci Sports Exerc 1988; 20: 150-4
72. Hurley B, Seals D, Ehsani A, et al. Effects of high intensity strength training on cardiovascular function. Med Sci Sports Exerc 1984; 16: 483-8
73. Hickson R, Rosenkoetter M, Brown M. Strength training effects on aerobic power and short-term endurance. Med Sci Sports Exerc 1980; 12: 336-9
74. Marcinik E, Potts J, Schlabach G, et al. Effects of strength training on lactate threshold and endurance performance. Med Sci Sports Exerc 1991; 23: 739-43
75. Hagerman F, Walsh S, Staron R, et al. Effects of high-intensity resistance training on untrained older men. I. Strength, car-

diovascular, and metabolic responses. J Gerontol A Biol Sci Med Sci 2000; 55 (7); B336-46
76. Ades P, Ballor D, Ashikaga T, et al. Weight training improves walking endurance in healthy elderly persons. Ann Intern Med 1996; 124: 568-72
77. Parker N, Hunter G, Treuth M. Effects of strength training on cardiovascular responses during a submaximal walk an a weight-loaded walking test in older females. J Card Rehab 1996; 16: 56-62
78. McCartney N, McKelvie R, Martin J, et al. Weight-training–induced attenuation of the circulatory response of older males to weight lifting. J Appl Physiol 1993; 74: 1056-60
79. Wilson P, Anderson K, Harris T, et al. Determinants of change in total cholesterol and HDL-C with age: the Framingham study. J Gerontol 1994; 49 (6): M252-7
80. Satler L, Green C, Wallace R, et al. Coronary artery disease in the elderly. Am J Cardiol 1989; 63: 245-8
81. Krumholz H, Seeman T, Merrill S, et al. Lack of association between cholesterol and coronary heart disease mortality and morbidity and all-cause mortality in persons older than 70 years. JAMA 1994; 272: 1335-40
82. Frost P, Davis B, Burlando A, et al. Serum lipids and incidence of coronary heart disease. Circulation 1996; 94: 2381-8
83. Schaefer E, Moussa P, Wilson W, et al. Plasma lipoproteins in healthy octogenarians: lack of reduced high density lipoprotein cholesterol levels: results from the Framingham heart study. Metabolism 1989; 38 (4): 293-6
84. Kohl HI, Gordon N, Scott C, et al. Musculoskeletal strength and serum lipid levels in men and women. Med Sci Sports Exerc 1992; 24: 1080-7
85. Tucker L, Silvester L. Strength training and hypercholesterolemia: an epidemiologic study of 8499 employed men. Am J Health Promot 1996; 11: 35-41
86. Boyden T, Pamenter R, Going S, et al. Resistance exercise training is associated with decreases in serum low-density lipoprotein cholesterol levels in premenopausal women. Arch Intern Med 1993; 153: 97-100
87. Fripp R, Hodgson J. Effect of resistive training on plasma lipid and lipoprotein levels in male adolescents. J Paediatr 1987; 11: 926-31
88. Johnson C, Stone M, Lopez-S A, et al. Diet and exercise in middle-aged men. J Am Diet Assoc 1982; 81: 695-701
89. Blumenthal J, Matthews K, Fredrikson M, et al. Effects of exercise training on cardiovascular function and plasma lipid, lipoprotein, and apolipoprotein concentrations in premenopausal and postmenopausal women. Arterioscler Thromb 1991; 11: 912-7
90. Kokkinos P, Hurley B, Smutok M, et al. Strength training does not improve lipoprotein-lipid profiles in men at risk for CHD. Med Sci Sports Exerc 1991; 123: 1134-9
91. Manning J, Dooly-Manning C, White K, et al. Effects of a resistance training program on lipoprotein-lipid levels in obese women. Med Sci Sports Exerc 1991; 23: 1222-6
92. Rhea P, Ryan A, Nicklas B, et al. Effects of strength training with and without weight loss on lipoprotein-lipid levels in postmenopausal women. Clin Exerc Physiol 1999; 1: 138-44
93. Smutok M, Reece C, Kokkinos P, et al. Aerobic *vs* strength training for risk factor intervention in middle-aged men at high risk for coronary heart disease. Metabolism 1993; 42: 177-84
94. Treuth M, Hunter G, Kekes-Szabo T, et al. Reduction in intra-abdominal adipose tissue after strength training in older women. J Appl Physiol 1995; 78 (4): 1425-31
95. Hurley B. Effects of resistive training on lipoprotein-lipid profiles: a comparison to aerobic exercise training. Med Sci Sports Exerc 1989; 21: 689-93
96. Kokkinos P, Hurley B. Strength training and lipoprotein-lipid profiles: a critical analysis and recommendations for further study. Sports Med 1990; 9: 266-72
97. Despres J. Visceral obesity, insulin resistance, and dyslipidemia: contribution of endurance exercise training to the treatment of the plurimetabolic syndrome. Exerc Sport Sci Rev 1997; 25: 271-300
98. Hersey III W, Graves J, Pollock M, et al. Endurance exercise training improves body composition and plasma insulin responses in 70-to 79-year-old men and women. Metabolism 1994; 43: 847-54
99. Lee A, Craig B, Lucas J, et al. The effect of endurance training, weight training and a combination of endurance and weight training upon the blood lipid profile of young male subjects. J Appl Sports Sci Res 1990; 4: 68-75
100. Wosornu D, Bedford D, Ballantyne D. A comparison of the effects of strength and aerobic exercise training on exercise capacity and lipids after coronary artery bypass surgery. Eur Heart J 1996; 17: 854-63
101. Hagberg JM, Ferrell RE, Katzel LI, et al. Apolipoprotein E genotype and exercise training-induced increases in plasma high-density lipoprotein (HDL)- and $HDL_2$-cholesterol levels in overweight men. Metabolism 2000; 48: 943-5
102. Kaplan N. Clinical hypertension. 5th ed. Baltimore (MD): Williams and Wilkins, 1990
103. Stone M, Wilson G, Blessing D, et al. Cardiovascular responses to short-term Olympic style weight-training in young men. Can J Appl Sport Sci 1983; 8: 134-9
104. Cononie CC, Graves JEPML. Effect of exercise training on blood pressure in 70-to 79-yr-old men and women. Med Sci Sports Exerc 1991; 23: 505-11
105. Hagberg JM, Blair S, Ehsani AA. Position stand: Physical activity, physical fitness, and hypertension. Med Sci Sports Exerc 1993; 25: i-x
106. Martel GF, Hurlbut MS, Lott ME, et al. Strength training normalizes resting blood pressure in 65 to 73 year-old men and women with high normal blood pressure. J Am Geriatr Soc 1999; 47: 1215-21
107. The sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Arch Intern Med 1997 Nov; 157 (21): 2413-46
108. Kannel WB. Blood pressure as a cardiovascular risk factor: prevention and treatment. JAMA 1996; 275: 1571-6
109. National High Blood Pressure Education Pressure Education Program. National High Blood Pressure Education Program Working Group Report on Primary Prevention of Hypertension. Bethesda (MD): US Department of Health and Human Services, National Heart Lung, and Blood Institute, 1993: NIH publication no.: 93-2669
110. Blumenthal J, Siegel W, Appelbaum M. Failure of exercise to reduce blood pressure in patients with mild hypertension. JAMA 1991; 266: 2098-101
111. Davidson M. The effect of aging on carbohydrate metabolism: a review of the English literature and a practical approach to the diagnosis of diabetes mellitus in the elderly. Metabolism 1979; 28: 687-705
112. DeFronzo RA. Glucose tolerance and aging: evidence for tissue insensitivity to insulin. Diabetes 1979; 28: 1095-101
113. Hjermann I. The metabolic cardiovascular syndrome: syndrome X, Reaven's syndrome, insulin resistance syndrome,

atherothrombogenic syndrome. J Cardiovasc Pharmacol 1992; 20 Suppl. 8: S5-10
114. Stout R. Overview of the association between insulin and atherosclerosis. Metabolism 1984; 34: 7-12
115. Miller J, Pratley R, Goldberg A, et al. Strength training increases insulin action in healthy 50-65 year old men. J Appl Physiol 1994; 77: 1122-7
116. Craig B, Everhart J, Brown R. The influence of high-resistance training on glucose tolerance in young and elderly subjects. Mech Ageing Dev 1989; 49: 147-57
117. Miller W, Sherman W, Ivy J. Effect of strength training on glucose tolerance and post-glucose insulin response. Med Sci Sports Exerc 1984; 16: 539-43
118. Durak E, Jovanovis-Petersol L, Peterson C. Randomized crossover study of effect of resistance training on glycemic control, muscular strength, and cholesterol in type I diabetic men. Diabetes Care 1990; 13: 1039-42
119. Eriksson J, Taimela S, Eriksson K, et al. Resistance training in the treatment of non-insulin-dependent diabetes mellitus. Int J Sports Med 1997; 18: 242-6
120. Jovanovic-Peterson L, Durak EP, Peterson CM. A twelve session exercise program and its effects on physical conditioning and glucose metabolism in type I diabetes mellitus [abstract]. Int J Sports Med 1989; 10: 377
121. Peterson CM, Jones RL, DuPuis A, et al. Feasibility of improved blood glucose control in patients with insulin-dependent diabetes mellitus. Diabetes Care 1979; 2: 329-35
122. Smutok MA, Reece C, Kokkinos PF, et al. Effects of exercise training modality on glucose tolerance in men with abnormal glucose regulation. Int J Sports Med 1994; 15 (6): 283-9
123. Eriksson J, Tuominen T, Valle S, et al. Aerobic endurance exercise or circuit-type resistance training for individuals with impaired glucose tolerance? Horm Metab Res 1998; 30: 37-41
124. Ishii T, Yamakita T, Sato T, et al. Resistance training improves insulin sensitivity in MIDDM subjects without altering maximal oxygen uptake. Diabetes Care 1998; 21: 1353-5
125. Ryan A, Pratley R, Goldberg A, et al. Resistive training increases insulin action in postmenopausal women. J Gerontol A Biol Sci Med Sci 1996; 51 (5): M199-205
126. Barret-Conner E, Wingard D, Criqui M, et al. Is border-line fasting hyperglycemia a risk factor for cardiovascular death? J Chronic Dis 1984; 37: 773-9
127. Despres J, Lamarche B, Mauriege P, et al. Hyperinsulinemia as an independent risk factor for ischemic heart disease. N Engl J Med 1996; 334: 952-7
128. Pollare T, Lithell H, Berne C. Insulin resistance is a characteristic feature of primary hypertension independent of obesity. Metabolism 1990; 39: 167-74
129. Zavaroni I, Bonoro E, Pagliara M, et al. Risk factors for coronary artery disease in healthy persons with hyperinsulinemia and normal glucose tolerance. N Engl J Med 1989; 320: 702-6
130. Harris M, Hadden W, Knowler W, et al. Prevalence of diabetes and impaired glucose tolerance and plasma glucose levels in US. population aged 20-74 yr. Diabetes 1987; 36: 523-34
131. Muller D, Elahi D, Tobin J, et al. The effect of age on insulin resistance and secretion: a review. Semin Nephrol 1996; 16: 289-98
132. Rowe J, Minaker K, Palotta J, et al. Characterization of the insulin resistance of aging. J Clin Invest 1983; 71: 1581-7
133. Shimokata H, Muller D, Fleg J, et al. Age as independent determinant of glucose tolerance. Diabetes 1991; 40: 44-51
134. Broughton D, Taylor R. Review: deterioration of glucose tolerance with age: the role of insulin resistance. Age Ageing 1991; 20: 221-5
135. Helmrich S, Ragland D, Leung R, et al. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. N Engl J Med 1991; 325: 147-52
136. Pacini G, Valerio A, Beccaro F, et al. Insulin sensitivity and beta-cell responsivity are not decreased in elderly subjects with normal OGTT. J Am Geriatr Soc 1988; 36: 317-23
137. Seals D, Hagberg J, Allen W, et al. Glucose tolerance in young and older athletes and sedentary men. J Appl Physiol 1984; 56: 1521-5
138. Hurley B, Hagberg J, Seals D, et al. Glucose tolerance and lipid-lipoprotein levels in middle-aged powerlifters. Clin Physiol 1987; 7: 11-9
139. Seals D, Hagberg J, Hurley B, et al. Effects of endurance training on glucose tolerance and plasma lipid levels in older men and women. JAMA 1984; 252: 645-9
140. National Institute of Health. Consensus development conference on diet and exercise in non-insulin-dependent diabetes mellitus. Diabetes Care 1987; 10: 639-44
141. Koivisto V, Yki-Jarvinen H, DeFronzo R. Physical training and insulin sensitivity. Diabetes Metab Rev 1986; 1: 445-81
142. Council on Exercise of the American Diabetes Association. Exercise and NIDDM. Diabetes Care 1990; 13: 785-9
143. Joseph LO, Farrell PA, Davey SL, et al. Effect of resistance training with or without chromium picolinate supplementation on glucose metabolism in older men and women. Metabolism 1999; 48: 546-53
144. Campbell W, Crim M, Young V, et al. Increased energy requirements and changes in body composition with resistance training in older adults. Am J Clin Nutr 1994; 60: 167-75
145. Castro M, McCann D, Shaffrath J, et al. Peak torque per unit cross-sectional area differs between strength-trained and untrained young adults. Med Sci Sports Exerc 1995; 27 (3): 397-403
146. Bell R, Hoshizaki T. Relationships of age and sex with joint range of motion of seventeen joint actions in humans. Can J Appl Sport Sci 1981; 6: 202-6
147. Benestad A. Trainability of old men. Acta Med Scand 1965; 178: 321-7
148. Brock D, Guralnick J, Brody J. Handbook of the biology of aging. In: Schneider E, Rowe J, editors. Demography and epidemiology of aging in the US. San Diego: Academic Press, 1990: 3-23
149. Anderssen S, Hjermann I, Urdal P, et al. Improved carbohydrate metabolism after physical training and dietary intervention in individuals with the atherothrombogenic syndrome. Oslo diet and exercise study (ODES): a randomized trial. J Intern Med 1996; 240 (4): 203-9
150. Brown M, Hagberg J. Does exercise training play a role in the treatment of essential hypertension. J Cardiovasc Risk 1995; 2: 296-302
151. Bloomfield S, Williams N, Lamb D, et al. Nonweightbearing exercise increases lumbar bone mineral density in healthy postmenopausal women. Am J Phys Med Rehab 1993; 72: 204-9
152. Lott ME, Hurlbut DE, Ryan AS, et al. Effects of strength training on glucose homeostasis in older men and women [abstract]. Med Sci Sports Exerc 1998; 30: S196
153. Zachwieja J, Toffolo G, Cobelli C, et al. Resistance exercise and growth hormone administration in older men: effects on insulin sensitivity and secretion during a stable-label intravenous glucose tolerance test. Metabolism 1996; 45: 254-60
154. Buemann B, Tremblay A. Effects of exercise training on abdominal obesity and related metabolic complications. Sports Med 1996; 3: 191-212

155. Despres J. Abdominal obesity as important component of insulin-resistance syndrome. Nutrition 1993; 9: 452-9
156. Cohn S, Vaswani A, Zanzid I, et al. Changes in body chemical composition with age measured by total-body neutron activation. Am J Physiol 1976; 25: 85-95
157. Donahue R, Abbort R, Bloom E, et al. Central obesity and coronary heart disease in men. Lancet 1987; I: 822-4
158. 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
159. Ross R, Rissanen J, Pedwell H, et al. Influence of diet and exercise on skeletal muscle and visceral adipose tissue in men. J Appl Physiol 1996; 81: 2445-55
160. Poehlman E, Goran M, Gardner A, et al. Determinants of decline in resting metabolic rate in aging females. Am J Physiol 1993; 264 (3 Pt 1): E450-5
161. Tataranni P, Ravussin E. Variability in metabolic rate: biological sites of regulation. Int J Obes 1995; 19 Suppl. 4: S102-6
162. Poehlman E, Toth M, Webb G. Sodium-potassium pump activity contributes to the age-related decline in resting metabolic rate. J Clin Endocrinol Metab 1993; 76: 1054-7
163. Poehlman ET, Gardner AW, Ades PA, et al. Resting energy metabolism and cardiovascular disease risk in resistance-trained and aerobically trained males. Metabolism 1992; 41: 1351-60
164. Broeder C, Burrhus K, Svanenick L, et al. The effects of either high-intensity resistance or endurance training on resting metabolic rate. Am J Clin Nutr 1992; 55: 802-10
165. Pratley R, Nicklas B, Rubin M, et al. Strength training increases resting metabolic rate and norepinephrine levels in healthy 50- to 65-yr-old men. J Appl Physiol 1994; 76: 133-7
166. Van Etten L, Westerterp K, Verstaooebm FT. Effect of weight-training on energy expenditure and substrate utilization during sleep. Med Sci Sports Exerc 1994; 27: 188-93
167. Van Etten L, Westertep K, Vestappen F, et al. Effect of an 18-wk weight-training program on energy expenditure and physical activity. J Appl Physiol 1997; 82: 298-304
168. Cullinen K, Caldwell M. Weight training increases fat-free mass and strength in untrained young women. J Am Diet Assoc 1998; 98: 414-8
169. Ryan A, Pratley R, Elahi D, et al. Resistive training increases fat-free mass and maintains RMR despite weight loss in postmenopausal women. J Appl Physiol 1995; 79: 818-23
170. Taaffe D, Pruitt L, Reim J, et al. Effect of sustained resistance training on basal metabolic rate in older women. J Am Geriatr Soc 1995; 43: 465-71
171. Treuth M, Hunter G, Weinsier R, et al. Energy expenditure and substrate utilization in older women after strength training: 24-h calorimeter results. J Appl Physiol 1995; 78 (6): 2140-6
172. Ballor DL, Harvey-Berino JR, Ades PA, et al. Contrasting effects of resistance and aerobic training on body composition and metabolism after diet-induced weight loss. Metabolism 1996; 45: 179-83
173. Rall LC, Meydani SN, Kehayias JJ, et al. The effect progressive resistance training in rheumatoid arthritis: increased strength with changes in energy balance or body composition. Arthritis Rheum 1996; 39 (3): 415-26
174. Bryner RW, Ullrich IH, Sauers J, et al. Effects of resistance *vs* aerobic training combined with an 800 calorie liquid diet on lean body mass and resting metabolic rate. J Am Col Nutr 1999; 18: 115-21
175. Nelson M, Fiatarone M, Morganti C, et al. Effects of high-intensity strength training on multiple risk factors for osteoporotic fractures: a randomized controlled trial. JAMA 1994; 272: 1909-14
176. Ross R, Pedwell H, Rissanen J. Effects of energy restriction and exercise on skeletal muscle and adipose tissue in women as measured by magnetic resonance imaging. Am J Clin Nutr 1995; 61: 1179-85
177. Lemmer JT, Ivey FM, Ryan AS, et al. Effect of strength training on RMR and physical activity: age and gender comparisons. Med Sci Sports Exerc 2000. In press
178. Cummings S, Kelsey J, Nevitt M. Epidemiology of osteoporosis and osteoporotic fractures. Epidemiol Rev 1985; 7: 178-205
179. Lindsay R. Osteoporosis. Chicago (IL): National Osteoporosis Foundation, 1992
180. Zimmermann CL, Smidt GL, Brooks JS, et al. Relationship of extremity muscle torque and bone mineral density in postmenopausal women. Phys Ther 1990; 70: 302-9
181. Ryan AS, Treuth MS, Hunter GR, et al. Resistive training maintains bone mineral density in postmenopausal women. Calcif Tissue Int 1998; 62: 295-9
182. Calmels P, Vico L, Alexandre C, et al. Cross-sectional study of muscle strength and bone mineral density in a population of 106 women between the ages of 44 and 87 years: relationship with age and menopause. Eur J Appl Physiol 1995; 70: 180-6
183. Hughes V, Frontera W, Dallal G, et al. Muscle strength and body composition: associations with bone density in older subjects. Med Sci Sports Exerc 1995; 27: 967-74
184. Aloia JF, Vaswani A, Flaster MaR, et al. To what extent is bone mass determined by fat-free or fat mass? Am J Clin Nutr 1995; 61: 1110-4
185. Bevier W, Wiswell R, Pyka G, et al. Relationship of body composition, muscle strength, and aerobic capacity to bone mineral density in older men and women. J Bone Miner Res 1989; 4: 421-32
186. Haapasalo H, Kannus P, Sievanen H, et al. Long-term unilateral loading and bone mineral density and content in female squash players. Calcif Tissue Int 1994; 54: 249-55
187. Karlsson M, Vergnaud P, Delmas P, et al. Indicators of bone formation in weight lifters. Calcif Tissue Int 1995; 56: 177-80
188. Notelovitz M, Martin D, Probart C. Estrogen therapy and variable-resistance weight training increase bone mineral in surgically menopausal women. J Bone Miner Res 1991; 6: 583-90
189. Pruitt LA, Jackson RD, Bartels RL, et al. Weight training effects on bone mineral density in early postmenopausal women. J Bone Miner Res 1992; 7: 179-85
190. Menkes A, Mazel S, Redmond R, et al. Strength training increases regional bone mineral density and bone remodeling in middle-aged and older men. J Appl Physiol 1993; 74: 2478-84
191. Ryan A, Treuth M, Rubin M, et al. Effects of strength training on bone mineral density: hormonal and bone turnover relationships. J Appl Physiol 1994; 77 (4): 1678-84
192. Snow-Harter C, Bouxsein M, Lewis B, et al. Effects of resistance and endurance exercise on bone mineral status of young women. J Bone Miner Res 1992; 7: 761-9
193. Lohman T, Going S, Pamenter R, et al. Effects of resistance training on regional and total bone mineral density in premenopausal women: a randomized prospective study. J Bone Miner Res 1995; 10: 1015-24
194. Gleeson P, Protas E, LeBlanc A, et al. Effects of weight lifting on bone mineral density in premenopausal women. J Bone Miner Res 1990; 5: 153-8
195. Rockwell JC, Sorensen AM, Baker S, et al. Weight training decreases vertebral bone density in premenopausal women: a prospective study. J Clin Endocrinol Metab 1990; 71: 988-93

196. Yarasheski KE, Campbell JA, Kohrt WM. Effect of resistance exercise and growth hormone on bone density in older men. Clin Endocrinol 1997; 47: 223-9
197. Courtney A, Wachtel E, Myers E, et al. Effects of loading rate on strength of the proximal femur. Calcif Tissue Int 1994; 55: 53-8
198. Davis MA, Ettinger WH, Neuhaus JM, et al. Knee osteoarthritis and physical functioning: evidence from the NHANES I epidemiologic follow-up study. J Rheumatol 1990; 18: 591-8
199. Gilliland B. Degenerative joint disease. In: Eugene B, Isselbacher K, Petersdorf R, et al., editors. Harrison's principles of internal medicine. New York: McGraw Hill Book Company, 1987: 1456-8
200. Ekdahl C, Andersson S, Svensson B. Muscle function of the lower extremities in rheumatoid arthritis and osteoarthrosis: a descriptive study in patients in a primary health care district. J Clin Epidemiol 1989; 42: 947-54
201. O'Reilly S, Jones A, Doherty M. Muscle weakness in osteoarthritis. Curr Opin Rheumatol 1997; 9: 259-62
202. Fisher NM, Pendergast DR. Effects of a muscle exercise program on exercise capacity in subjects with osteoarthritis. Arch Phys Med Rehab 1994; 75: 792-7
203. Pothier B, Allen M. Kinesiology and the degenerative joint. Rheum Dis Clin North Am 1991; 16: 989-1002
204. Adler S. Self care in the management of the degenerative knee joint. Physiotherapy 1985; 71: 58-60
205. Chamberlain M, Care G, Harfield B. Physiotherapy in osteoarthritis of the knees: a controlled trial of hospital versus home exercises. Int Rehab Med 1982; 4: 101-6
206. Marks R. The effect of isometric quadriceps strength training in mid-range for osteoarthritis of the knee. Arthritis Care Res 1993; 6: 52-6
207. Quirk A, Newman R, Newman K. An evaluation of interterential therapy, shortwave diathermy and exercise in the treatment of osteo-arthritis of the knee. Physiotherapy 1985; 71: 55-7
208. Marks R. Quadriceps strength training for osteo-arthritis of the knee: A literature review and analysis. Physiotherapy 1993; 79: 13-8
209. Schilke JM, Johnson GO, Housh TJ, et al. Effects of muscle-strength training on the functional status of patients with osteoarthritis of the knee joint. Nurs Res 1996; 45: 68-72
210. Ettinger WJ, Burns R, Messier S, et al. A randomized trial comparing aerobic exercise and resistance exercise with a health education program in older adults with knee osteoarthritis. JAMA 1997; 277: 25-31
211. Rogind H, Bibow-Nielson B, Jensen B, et al. The effects of a physical training program on patients with osteoarthritis of the knees. Arch Phys Med Rehab 1998; 79: 1421-7
212. Minor MA. Exercise in the treatment of osteoarthritis. Rheum Dis Clin North Am 1999; 25: 397-415
213. Bassey E, Morgan K, Dallosso H, et al. Flexibility of the shoulder joint measured as range of abduction in a large representative sample of men and women over 65 years of age. Eur J Appl Physiol 1989; 58: 353-60
214. Germain N, Blaire S. Variability of shoulder flexion with age, activity and sex. Am Correct Ther J 1983; 37 (6): 156-60
215. Bergstrom G, Aniansson A. Functional consequences of joint impairment at age 79. Scand J Rehab Med 1985; 17: 183-90
216. Gehlsen G, Whaley M. Falls in the elderly: part II, balance, strength, and flexibility. Arch Phys Med Rehab 1990; 71: 739-41
217. Chapman E, deVries H, Swezey R. Joint stiffness: effects of exercise on young and old men. J Gerontol 1972; 27 (2): 218-21
218. Voorhips L, Lemmunk K, Van Heuvellon M, et al. The physical condition of elderly women differing in habitual physical activity. Med Sci Sports Exerc 1993; 25: 1152-7
219. Kligman E, Pepin E. Prescribing physical activity for older patients. Geriatrics 1992; 47: 33-47
220. Lombardi V. Beginning weight training. Dubuque: WC Brown Publishers, 1989
221. Stone M, Fleck S, Triplett N, et al. Health and performance related potential of resistance training. Sports Med 1991; 11: 210-31
222. Girouard C, Hurley B. Does strength training inhibit gains in range of motion from flexibility training in older adults? Med Sci Sports Exerc 1995; 27 (10): 1444-9
223. Gardner G. Effect of isometric and isotonic exercise on joint motion. Arch Phys Med Rehab 1966; 47: 24-30
224. Weltman A, Janney C, Rians C, et al. The effects of hydraulic resistance strength training in pre-pubertal males. Med Sci Sports Exerc 1986; 18: 629-38
225. Massey B, Chauder N. Effects of systematic, heavy resistive exercise on range of joint movement in young male adults. Res Q 1956; 27: 41-51
226. Marcinik E, Hodgdon J, Mittleman K, et al. Aerobic/calisthenic and aerobic/circuit weight training programs for Navy men: a comparative study. Med Sci Sports Exerc 1985; 17: 482-7
227. Morey M, Cowper P, Fuessner J, et al. Evaluation of a supervised exercise program in a geriatric population. J Am Geriatr Soc 1989; 37: 348-54
228. Raab D, Agre J, McAdam M, et al. Light resistance and stretching exercise in elderly women: effect upon flexibility. Arch Phys Med Rehab 1988; 69: 268-72
229. American College of Sports Medicine. Exercise and physical activity for older adults. Med Sci Sports Exerc 1998; 30: 992-1009
230. Myers A, Young Y, Langlois J. Prevention of falls in the elderly. Bone 1996; 18 (1 Suppl.): 87S-101S
231. Jozsi AC, Campbell WW, Joseph L, et al. Changes in power with resistance training in older and younger men and women. J Gerontol A Biol Sci Med Sci 1999; 54 (11): M591-6

Correspondence and offprints: Dr *Ben F. Hurley*, Department of Kinesiology, University of Maryland, College Park, MD 20742, USA.
E-mail: bh24@umail.umd.edu