Exercise Training for Ameliorating Cardiovascular Risk Factors-focusing on Exercise Intensity and Amount

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Exercise training suppresses cardiovascular risk factors by improving lipid/glucose metabolism, insulin resistance and hypertension. There is an optimum amount and intensity of exercise that will suppress each risk factor, although limited information is currently available on this area. For instance, the level of high-density lipoprotein cholesterol seems to be dependent more on the amount than on the intensity of exercise training; the situation with exercise-induced suppression of triglyceride levels is less clear. It is notable that the ability of mild intensity or a low amount of exercise to suppress or prevent cardiovascular risk factors is largely unknown. This knowledge is essential for prescribing safe and effective exercise training to elderly people. At the same time, very few studies on the associations between cardiovascular risk factors in response to exercise can be found. For example, improvement in lipid metabolism or insulin resistance seems to be related to the weight loss seen during exercise training but weight-independent effects could also be observed. The optimal intensity and amount of exercise training for each cardiovascular risk factor and the relationships between the effects of exercise training on each risk factor need to be clarified in the near future in order to establish guidelines that can be applied to the majority of elderly people at risk of cardiovascular diseases.

Keywords: cardiovascular risk factors, exercise intensity, exercise amount, elderly people

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1. Introduction

It is known that obesity (especially increased abdominal fat), hypertension, hyperlipidemia, and insulin resistance are risk factors for cardiovascular disease (CVD) (DeFronzo, 1991; Carr, 2004; Rios, 1998; Goodpaster, 2005). Numerous reports have also shown that low cardiorespiratory fitness is a risk factor for CVD (Blair, 1991; Carnethon, 2003; Church, 2001; Lee, 1999; Farrell, 1998; Haddock, 1998; Lee, 2005). In general, exercise training ameliorates CVD risk factors through the effects shown in **Table 1**. Thus, exercise training would be expected to decrease the incidence of CVD in elderly people at high risk of CVD. The improvement in lipid metabolism and insulin resistance after exercise

training, however, is inconclusive since there are potentially confounding variables to be considered when examining the effects of exercise, such as exercise intensity and amount, and exercise-related weight loss and reduction of body fat (Cox, 1994). In particular, there is no clear evidence that low level exercise training with potentially limited weight loss, which is feasible for the elderly, is effective in improving insulin resistance and hyperlipidemia.

Associations between the improvement in CVD risk factors are often considered when investigating the efficacy of exercise training in protecting against arteriosclerosis-related diseases. However, it remains unresolved as to whether there are cause and effect relationships between the improved risk factors. For example, it remains controversial whether a

Table 1 CVD Risk Factor Improvement Effect by Exercise Training

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- 1) Decrease in weight (or body fat including intra-abdominal body fat) (Zelasko (1984), Poirier (2001))
 - 2) Improvement of aerobic capacity (Vo2max, etc.) (Samitz (1991))
 - 3) Improvement of fat metabolism (HDL-C increase, TG decrease) (Haskell WL (1984), Goldberg L (1987), Durstine (2001), Halbert (1999))
 - 4) Improvement of insulin resistance (fasting insulin concentration, HOMA-IR, and decrease of rate of glucose disappearance by insulin clamp technique) (Henriksson(1985), Borghouts (2000), Hawley (2004))
 - 5) Effect of hypotension (decrease of resting blood pressure) (Arroll (1992), Kelley (1999), Hagberg (2000), Fagard (1999), Stewart (2005))
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reduction in insulin resistance is dependent on weight loss after a program of exercise training that results in reductions in both insulin resistance and obesity, or whether there are other factors that cause both improvements.

Our aim here is to review studies examining the effect of exercise training on CVD risk factors, notably those that focus on the effect of the intensity and amount of exercise and those that focus on the relationships between improved CVD risk factors. This will, hopefully, clarify the hitherto unsolved problems that need to be addressed before an effective exercise prescription for the majority of elderly people at high risk of CVD can be established.

2. Effect of Exercise Training on Improving CVD Risk Factors

It is generally held that exercise training improves CVD risk factors, as noted above. However, the improvements observed have not necessarily been consistent among studies and the amount and intensity of exercise are important confounding factors (Crouse, 1997).

2.1. Effect on Serum Lipid

In general, exercise training has a favorable effect on serum lipids such as high density lipoprotein cholesterol (HDL-C), which has a protective effect

against arteriosclerosis by removing cholesterol from the peripheral tissues, and triglycerides (TG) which are contained in very low density lipoprotein (VLDL) (Haskell, 1984; Goldberg, 1987). Low density lipoprotein cholesterol (LDL-C) decreases in some situations but seem less amenable to change induced by exercise than HDL-C and TG (Cox, 1994).

2.1.1. Effect on HDL-C

A cross-sectional study has indicated that there is a correlation between daily physical activity and serum HDL-C or TG levels. Hata, et al. (2000) report that persons who walk more than 6000 steps daily on average have a higher HDL-C of 3 mg/dl and a lower TG of 10 mg/dl than those who walk less than 2000 steps. In addition, their meta-analysis also indicated that more intense exercise such as jogging or swimming resulted in a 10 mg/dl reduction in TG and a 5 mg/dl elevation in HDL-C together with an approximately 300 kcal increase in energy expenditure per single bout of exercise. Interestingly, HDL-C was more significantly altered by exercise than any other lipoprotein measured (Szapary, 2003). Numerous studies have examined the effect of the intensity and amount of exercise on the change in HDL-C and some have suggested that there is a dose-dependent relationship between the amount of exercise and the increase in HDL-C, as shown in **Table 2** (Wood, 1983; Kraus, 2002; Kokkinos, 1995).

In contrast, most studies suggest (Kokkinos, 1998 excepted) that there is not a strong association

Table 2 Dose-dependent effect on HDL-C by regular exercise

presenter	subject	result
Kokkinos (1995)	2906 (mean age: 43±4yrs)	HDL-C increases by about 0.3mg/dl as weekly jogging increases every one mile.
Kraus (2002)	111 obese middle-aged women (mean age: 52±8yrs)	Weekly 20-mile and 6-month jogging increased 4.3mg/dl in HDL-C but weekly 12-mile jogging did not increase HDL-C.
Wood (1983)	25 middle-aged men (mean age: 45yrs)	When weekly running of more than 8 miles for one year was continued, the amount of weekly running showed a significant correlation with the change of HDL-C (r=0.48).

between exercise intensity and HDL-C increase (Spate-Douglas, 1999; Crouse, 1997; Kim, 2001). Resistance training is typically the form of high intensity training selected, although this form of exercise should be distinguished from endurance training. Joseph, et al. (1999) reported that resistance training resulted in a slight but significant increase of approximately 2 mg/dl in HDL-C (there was no significant change in TG) in middle-aged and elderly obese male subjects (aged 54-71yrs) after twelve weeks of resistance training. However, most studies showed no significant changes in HDL-C and TG concentrations after resistance training (Elliott, 2002; LeMura, 2000; Manning, 1991; Hurley, 1998). These findings suggest that exercise intensity is not strongly associated with the elevation of HDL-C. An increase in apolipoprotein (Apo) AI, which is the main constituent of HDL-C, is accompanied by an increase in HDL-C (Leon, 2002). Exercise intensity is not associated with the increase in HDL-C or ApoAI provided that energy expenditure is held constant (Crouse, 1995).

Exercise training-induced changes in HDL-C are affected by genetic polymorphisms of lipoprotein components such as ApoE, cholesteryl ester transfer protein (CETP), which alters HDL-C subtraction by transferring cholesterol from HDL₂ to HDL₃, and by genetic polymorphism of lipoprotein lipase (LPL), the enzyme that dissolves TG. Hagberg, et al. (1999), who determined whether the Apo E genotype affects

the exercise training-induced increase in HDL-C, reported that men with the Apo E2 genotype had a larger increase in plasma HDL-C with exercise training than men with the Apo E3 and E4 genotype. Wilund, et al. (2002) determined whether variation in the CETP gene locus contributed to the variability in the responses of HDL-C to endurance exercise training. They reported that HDL₂, which has the strongest effect in preventing arteriosclerosis among the HDL family, increased with endurance exercise training in CETP B1B1 but did not change in CETP B1B2 genotype individuals. In addition, Hagberg, et al. (1999), who used restriction fragment length polymorphism (RFLP) to examine the role of genotype in the response to exercise training, reported that LPL PvuII -/- individuals had larger increases in HDL-C than LPL PvuII +/- and +/+ individuals.

Familial factors also contribute significantly to the variability in the training response (Bouchard, 2001). However, Rice's results from the HERITAGE family study indicated that the percentage variance in the training responses attributable to familial factors ranged from 25% to 38% (Rice, 2002). Moreover, Leon, et al. (2002) reported that multivariate regression analysis including baseline variables and training responses only accounted for 15.5 % of the variability in the HDL-C response to training. Molecular studies are currently underway to identify the markers and genes associated with training responses.

2.1.2. Effect on TG

The reduction in blood TG levels after exercise is also well known (Haskell, 1984). As an acute response to exercise, the reduction in TG occurs about a day after one bout of exercise (Annuzzi, 1987; Sady, 1986; Cullinane, 1982) and persists for up to three days (Thompson, 1980). In these studies, the subjects took exercise for a long time (at least one-hour). Ferguson, et al. (1998) showed that the training subjects were required to consume at least 1000-kcal during acute exercise to increase the activity of LPL. This may explain why high-volume exercise is required before acute reductions in TG are seen in adolescents and athletes. However, it is unclear whether an acute decline in TG would also occur after exercise in sedentary or elderly people who are incapable of high volume exercise. By contrast, exercise intensity was not found to affect the TG acute effect (Crouse, 1995).

It is also known that a chronic effect of exercise is the suppression of post-prandial hypertriglyceridemia. Merrill, et al. (1989) found that running more than 30 miles per week suppressed the post-prandial TG response by 34 %. However, it is controversial whether prolonged training reduces the fasting TG concentration. A meta-analysis of the influence of exercise training on fasting TG levels showed that energy expenditure of 1200 to 2200 kcal per week dose-dependently reduced TG by 5 to 38 mg/dl (Durstine, 1991). However, Kelley's (2004) meta-analysis indicated that exercise training did not significantly reduce the fasting TG concentration (Kelley, 2004). Moreover, the decrease in TG was dispersed among individuals (Leon, 2001).

Clearly, both the acute and the chronic effects of exercise on TG levels need to be examined. Moreover, there are surprisingly few reports on the relationship between the amount and intensity of exercise and the change in TG levels. Further research is also necessary into the factors, including enzymatic activities, which affect the changes in HDL-C, TG and apoprotein.

2.2. Effect on Carbohydrate Metabolism

Insulin controls blood glucose levels (suppresses hyperglycemia) by promoting the uptake of post-prandial glucose by the liver, skeletal muscle, and fat tissue. Berson and Yalow (1970) defined insulin resistance as "a state (of a cell, tissue, system

or body) in which greater than normal amounts of insulin are required to elicit a quantitatively normal response." Insulin resistance has been defined accurately by a decreased rate of glucose disappearance (R_d) during a hyperinsulinemic-euglycemic clamp (DeFronzo, 1979). It is simply estimated by high levels of plasma insulin (either during fasting or during a glucose tolerance test) and homeostasis model assessment insulin resistance (HOMA-IR) [$\text{fasting plasma glucose (mg/dl)} \times \text{fasting plasma insulin } (\mu\text{U/ml}) / 405$] (Flier, 1993).

A high-intake of fat and obesity impair insulin-mediated glucose uptake (Vessby, 2000). Insulin resistance and/or the compensatory hyperinsulinemia will lead to dyslipidemia (i.e. high TG and low HDL-C) and high blood pressure, which plays an important role in causing type 2 diabetes or/and CHD (Reaven, 1988).

2.2.1. Effect on Prevalence of Diabetes

It is well known that daily physical activity protects against impaired carbohydrate metabolism. Hu, et al. (1999) showed during an 8 year of follow up study that the intensity and amount of physical activity related to the incidence of type 2 diabetes. Three Randomized Control Trial (RCT) studies carried out in different countries indicated that the incidence of type 2 diabetes in high risk subjects with impaired glucose tolerance (IGT) was reduced by 41% to 58% through lifestyle intervention that included alterations to diet and physical activity (Pan, 1997; Tuomilehto, 2001; Knowler, 2002). **Table 3** shows a summary of the three RCT study data.

2.2.2. Effect on Carbohydrate Metabolism

There are many reports that exercise training improves carbohydrate metabolism in type 2 diabetes, including improving glycemic control (i.e. lowering HbA_{1c}). Boule, et al. (2001) reported that the weighted mean post-intervention HbA_{1c} was lower by -0.66% in the exercise groups compared with the control groups, although no significantly greater change in body mass was found by their meta-analysis when exercise groups were compared with control groups.

Although there is little evidence on the impact of the amount and intensity of aerobic exercise on glycemic control, the decrease in HbA_{1c} may have a stronger association with exercise intensity than with exercise amount. Kelley's (2001) meta-analysis

Table 3 Primary Preventive Clinical Test of main type 2 diabetes

Study	Da Qing IGT and Diabetes Study, China (Pan (1997))	Finnish Diabetes Prevention Study (Tuomilehto (2001))	Diabetes Prevention Program, USA (Knowler (2002))
Number of subjects	520	522	3234
Rate of women (%)	47	67	68
Mean age	45±9	55±7	51±11
Mean body mass index	25.8±3.8	31.2±4.6	34.0±6.7
Period of intervention (year)	6	3.2	2.8
Group	Control group Eating group (25-30kcal/day, limiting fat intake (25-30% of total calories) Exercise group (exercise for 1-2hrs/day) Eating and exercising group	Control group Lifestyle intervention group (limiting fat intake (MAX 30% of total calories), targeting 5% decrease in the original weight by exercise of more than 30min/day)	Control group Medication group Lifestyle intervention group (to decrease by 5-7% the original weight by order-made exercise and dietary)
Result (decrease rate of development of type 2 diabetes)	Eating group:31% Exercising group: 46% Eating+Exercising group:42%	Lifestyle intervention group: 58%	Medication group: 31% Lifestyle intervention group: 58%

Body Mass Index is calculated by (weight (kg)/height² (m²)).

Criteria of obesity is more than 25(kg/m²).

showed that it is uncertain whether there is a dose-response effect of exercise on improved glucose control in Type 2 diabetes. Boule's (2003) meta-analysis of the improvements in glycemic control after aerobic training indicated that exercise

intensity predicted the post-intervention improvement in HbA_{1c} ($r = -0.91$, $p = 0.002$) to a larger extent than did exercise duration.

Lately, there have been an increasing number of studies on the usefulness of resistance training in

improving glycemic control and insulin resistance in diabetic patients. Resistance training alone (Dunstan, 2005; Castaneda, 2002; Dunstan, 2002; Ishii, 1998; Honkola, 1997) and combined with aerobic training (Cuff, 2003; Maiorana, 2002) was found to be effective in improving glycemic control and insulin resistance. Resistance training is also known as high intensity training although its exercise form is different from endurance training. The studies of resistance training may suggest that exercise intensity is strongly associated with the improvement of carbohydrate metabolism in diabetic patients. However, to the best of our knowledge, the amount and intensity of resistance training required to elicit an improvement in carbohydrate metabolism has never been studied. Further research on the design of a safe resistance-training program for the purposes of preventing and caring for type 2 diabetes is necessary.

2.2.3. Effect on Insulin Resistance (Sensitivity)

It is well known that physical activity and exercise training reduce insulin resistance (Ross, 2004; Dumortier, 2003; Boudou, 2003). In fact, some cross-sectional studies have shown that a low level of physical activity is associated with insulin resistance (Saito, 1992; Harding, 2001). However, there have been few exercise intervention studies of the intensity and amount of exercise required for improving insulin resistance and the findings of these studies were disparate. Borghouts, et al. (1999) reported that improvements in insulin sensitivity in healthy young adults after four weeks' endurance training were more pronounced with high intensity training. Resistance training also has been shown to be effective in the improvement of insulin resistance in elderly people and menopausal women (Reynolds, 2004; Ryan, 2000; Ryan, 1996). These results would indicate that exercise intensity is related to an improvement in insulin resistance. By contrast, Houmard's findings in obese individuals suggested that total exercise duration per week and not exercise intensity should be considered when designing training programs with the intent of improving insulin action (Houmard, 2004). Mayer, et al. (1998) studied exercise training by 1467 African American females and found that the amount and intensity of exercise were associated with higher insulin sensitivity. Thus, both the amount and the intensity of exercise may be associated with the improvement in insulin resistance, although the relative contribution of each factor is unknown at

present.

Another factor relating to the improvement in insulin resistance after exercise training is age. Short (2003), who examined the impact of exercise training at 70-80 % maximum heart rate (HR max) on improving insulin resistance in subjects of 21-87yrs, showed that increase in insulin sensitivity is adversely correlated with age ($r = -0.41$). It is possible that, for a given level of exercise intensity, the effect of exercise training on the improvement in insulin sensitivity declines with age.

2.3. Effect on Resting Blood Pressure

A large-scale epidemiological survey indicated that moderately vigorous physical activity led to a reduced risk of hypertension (Paffenbarger, 1997; Hernelahti, 2004) while the volume of physical activity (including light physical activity) was not associated with hypertension (Hernelahti, 2004).

The findings of exercise intervention studies have been inconsistent with regard to the effect of endurance training intensity in lowering blood pressure. Cooper, et al. (2000) reported that the magnitude of the hypotensive effect of moderate intensity exercise was not as great as that found in studies of higher intensity exercise among hypertensives (systolic blood pressure (SBP): -1.9 mmHg, diastolic blood pressure (DBP): -2.2 mmHg). On the contrary, Motoyama, et al. (1997) reported that SBP (-15 ± 8 mm Hg) and DBP (-9 ± 9 mm Hg) decreased significantly after 3 months of lactate threshold (LT) level intensity exercise training in elderly hypertensive subjects. Braith, et al. (1994) reported that SBP decreased similarly in both moderate intensity (70 % heart rate reserve (HRR)) and high intensity (85 % HRR) training groups of elderly people after 6 months. Halbert's meta-analysis of 29 studies of exercise training by the elderly reported that exercise training of more than 70% maximum oxygen consumption (VO_2 max) intensity did not have any additional impact on reducing BP (Halbert, 1997). These reports may suggest that high intensity training is not necessarily required in order to lower resting BP in elderly people, although it is unclear if low intensity training (e.g. less than LT intensity) also has a hypotensive effect.

In fact, resistance training as a typical form of high intensity training is not recommended for

the treatment of hypertension (Pescatello, 2004). The acute and chronic influences of resistance training on blood pressure have been examined in normotensive individuals. The results suggest that resistance training is not associated with chronic elevations in blood pressure, but could become part of a non-pharmacological intervention strategy to prevent and combat high blood pressure (Schwartz, 1995). In general, blood pressure elevates during resistance exercise. In normotensive individuals, the mean blood pressures declined during the hour after resistance training (Fisher, 2001) and the average values for 24-h ambulatory blood pressure pre- and post-training did not differ (Gilders, 1991). Cornelissen, et al. (2005) determined the influence of resistance training in healthy normotensive adults by meta-analysis and found that the weighted net change (SBP/DBP) in blood pressure averaged $-3.2/-3.5$ mmHg. Kelley (2000) also reported that resistance training resulted in decreases of approximately 3 mmHg in both resting SBP and resting DBP. It is concluded that moderate intensity resistance training is not harmful for normotensive subjects. However, additional studies are needed in hypertensive subjects before resistance exercise can be considered as a safe and effective regimen of therapy.

The dose-response relationship between exercise training and the change in blood pressure has been examined by some studies. Ishikawa, et al. (2003) reported that the magnitude of reductions in SBP was greater in the 61 to 90 min/wk group than in the 30 to 60 min/wk group, but there were no greater reductions in SBP with further increases in exercise volume. Halbert, et al. (1997) reported that increasing exercise frequency to more than three sessions per week did not have any additional impact on reducing blood pressure. Thus, the amount of exercise may have comparatively little effect on the reduction of blood pressure.

3. Correlation between Risk Factor Improvements

As mentioned above, little is known about the cause and effect relationships between exercise and the improvements in individual CVD risk factors. Previous reports have only examined the correlations between exercise training-induced improvements in risk factors (Miyatake, 2002; Mourier, 1997; Arden, 2004; Despres, 1991; Leon, 2000; Poirier,

2001; Miyatake, 2003). Most of these reports focussed on the correlation between the improvement in fat metabolism and insulin resistance and the improvement in body composition and aerobic capacity after training. Moreover, few studies have examined whether the concurrent improvements in insulin resistance and fat metabolism observed with exercise training are independent of exercise-induced changes in body weight and aerobic capacity (Lamarche, et al., 1992).

3.1. Correlation between Reduction of Obesity and Improvement of Fat Metabolism/Insulin Resistance

Some studies have reported that an improvement in lipid metabolism and insulin resistance after exercise training relates to a reduction in body fat (or visceral fat). Miyatake, et al. (2002) has shown using stepwise multiple regression analysis that the change in visceral fat area was the major determinant for the change in HOMA-IR ($r=0.51$) in obese middle-aged males. Mourier, et al. (1997) reported that the change in visceral abdominal fat was associated with the improvement in insulin sensitivity ($r=0.84$) in type 2 diabetic patients after aerobic training at 75% VO_2 peak. Arden, et al. (2004) found in the HERITAGE family study (17-65 yrs) that greater fat loss was associated with increases in HDL-C and decreases in TG after exercise training.

It is controversial whether exercise training itself improves blood lipid levels or insulin resistance regardless of weight loss. Some studies have shown that aerobic training results in a decrease in TG (Weintraub, 1989) and a modest increase in HDL-C (Thompson, 1988) without weight loss in healthy subjects. Duncan, et al. (2003) reported that in obese, previously sedentary subjects (mean BMI $28.9 \pm 4.6 \text{ kg/m}^2$), walking exercise positively affected markers of glucose and fat metabolism even in the absence of weight loss. Most reports, however, suggest that weight loss by exercise training and/or caloric restriction should be encouraged to improve the CHD risk factor profile of obese men (Ross, 2000; Katznel, 1997). Wood, et al. (1988) reported that the changes in plasma lipids and lipoproteins induced by decreasing energy intake or by increasing exercise were not significantly different in obese persons. These reports suggest that improvements in lipid metabolism and insulin resistance are strongly

Figure 1 Hypothetical safe therapeutic margin in exercise prescription for improving cardiovascular risk factors

associated with the loss of body weight/fat. Exercise training itself may be effective in ameliorating CHD risk factors; however it is more important for obese persons to reduce obesity to improve fat metabolism and insulin resistance.

3.2. Relationships between Improvement of Aerobic Capacity and Improvement of Fat Metabolism and Insulin Resistance

The relationship between the improvement of aerobic capacity and the improvement of fat metabolism and insulin resistance has been examined in many studies. Gan (2003) excepted, there is a clear dissociation between the improvement in cardiorespiratory fitness and the improvement in the metabolic risk profile after endurance training programs (Despres, 1991; Leon, 2000; Poirier, 2001). Regular exercise training is likely to generate beneficial metabolic effects regardless of the improvement of aerobic function.

3.3. The Relationship between the Reduction of Obesity and Blood Pressure

In a meta-analysis, Neter, et al. (2003) estimated

the effect of weight reduction on blood pressure and indicated that SBP and DBP reductions were -1.05 mmHg and -0.92 mmHg, respectively, when expressed per kilogram of weight loss. Miyatake, et al. (2003), who examined the link between a reduction in blood pressure (BP) and daily exercise, reported that there was a positive correlation ($r=0.47$) between the reduction in DBP and the decrease in visceral fat area measured with computed tomography (CT) scanning. Blumenthal, et al. (2000), however, reported that both weight loss and aerobic exercise were associated with a reduction in blood pressure. From these reports, it can be concluded that exercise training alone may have an antihypertensive effect, although weight loss is important for reducing blood pressure. Little is known about how much exercise training and weight loss, respectively, contribute to lowering blood pressure.

3.4. Relationships between the Improvement in Aerobic Capacity and the Reduction in Blood Pressure

Some studies have shown that an increase in aerobic fitness does not coincide with a reduction

Figure 2 An illustration of the intensity and amount of exercise which is optimal for the improvement of each CVD risk factor (it does not show quantitative relationships between each areas.)

in blood pressure (Grant, 1992; Sedgwick, 1989). There are fewer reports on the relationship between the changes in aerobic capacity and blood pressure than on the relationship between the improvements in aerobic capacity and fat metabolism and/or insulin resistance. Future investigations are necessary, therefore, on the relationship between exercise and the reduction in blood pressure.

4. Exercise Training for Elderly People

4.1. Exercise Prescription for Elderly People

Many studies have indicated that exercise training for elderly people is also effective in reducing insulin resistance (Seal, 1984; Kirman, 1993; Dengel, 1998; Reynolds 4th, 2002; Evans, 2005), improving lipid metabolism (Seal, 1984; Schuit, 1998; Fahlman, 2002; Park, 2003), and lowering blood pressure (Jessup, 1998, 1999; Kelley, 2001; Ohkubo, 2001). Thus, daily exercise training is expected to lower the incidence of CVD.

More than half of older people complain of barriers to physical activity. The common barriers are health issues such as heart and orthopedic problems (Lim, 2005). Consequently, little of the current research on the impact of physical activity on strength and function in elderly people has included elderly participants who have more barriers to physical

activity (Taunton, 1997). Given that the elderly person is more fragile and has more problems than the middle-aged participant, the exercise intensity of the program is usually lower. With elderly people who are frail and/or have physical-medical limitations, exercise intensity may be decreased (Pollock, 1994). The American College of Sports Medicine (2004) has stated key practices for promoting physical activity, especially in older adults with chronic disease or low fitness and in those with low levels of physical activity. The key practices include the management of risk by commencing physical activity at low intensity and gradually increasing it. The amount of exercise may also be low because the elderly are affected by the drawbacks of physical overloading and the safety margin of an exercise dose tends to decline with aging. However, there are few studies on the efficacy of a low amount or a low intensity of exercise training in elderly people.

4.2. Low Intensity Training for Elderly People

Low intensity (e.g. 50 % VO_2 max) exercise training has also been shown to be effective in enhancing aerobic capacity in some studies. De Vito, et al. (1997) reported that exercise training at 50% HRR did not result in a significant improvement in VO_2 max. However, most studies reported that aerobic capacity was improved even by low intensity training. Poster, et al. (1992) reported that VO_2 max was increased by 8.5% by low to moderate intensity (70 %HR max) exercise training. It also has been found that low (30-40% HRR) and moderate intensity (60-70 %HRR) training produced similar improvements in VO_2 max (Stevenson, 1990; Foster, 1989). However, there are only a few reports that low intensity training by elderly people produces improvements in fat metabolism, insulin resistance, and resting blood pressure (Sunami, 1999; Stewart, 2005). Moreover, the effect of low intensity training (i.e. training which the subjects may feel is 'light') with perceived exertion as an indicator of somatic stress (Borg, 1970) has never been studied. The amount of exercise may need to be low for elderly people who are incapable of high volume training, as noted above. When designing a training program for the elderly who have barriers to high volume exercise, a confounding problem is quantification of the dose-response relationships between the volume

of exercise and the improvements in risk factors. It is currently unclear how many bouts of exercise are required to ameliorate individual risk factors in the elderly.

There are a few reports on the correlation between the favorable effects of exercise on cardiovascular risk factors in elderly people (i.e. the correlation between weight loss and the improvement in aerobic capacity and fat metabolism and insulin resistance in exercise training). Katzel, et al. (1995) indicated that there was a significant correlation between the loss of body fat and the improvement in blood TG and HDL-C levels and insulin resistance. On the contrary, there was no significant association between the change in VO_2 max and the reduction in insulin response during an oral glucose tolerance test (OGTT) after exercise training (Pratray, 2000). However, there are few reports on the relationships between these changes after low intensity exercise training which feasible for elderly people (Stewart, 2005). It is necessary to establish an exercise prescription which is not only effective in ameliorating CVD risk factors such as dyslipidemia, insulin resistance, hypertension but which is also feasible for the majority of elderly people. Future research is needed to determine the efficacy of low-intensity and low-amount exercise training and to determine the relationships between exercise and the improvements in CVD risk factors.

5. Conclusion

Generally, exercise training is effective in improving cardiovascular risk factors such as hyperlipidemia (e.g. low levels of HDL-C and high levels of TG), insulin resistance (abnormalities in carbohydrate metabolism), and hypertension. The improvements are affected by the amount and intensity of training (**Figure 2**) and by the degree of reduction in obesity and the increase in aerobic capacity. Other as yet unknown factors may also exist.

The increase in HDL-C, which is protective from arteriosclerosis, is affected more by the amount rather than the intensity of training. Resistance training, which is a typical form of high intensity training but which differs from endurance training, is not effective in elevating HDL-C. The improvement in insulin resistance is associated with both the intensity and the amount of exercise. On the

contrary, an improvement in glycemic control in diabetes is associated more with exercise intensity than exercise amount. Moderate to high intensity exercise training (e.g. more than LT intensity) has been reported to be effective in the protection from hypertension. Nevertheless, it is unclear whether low intensity training (e.g. less than LT intensity) also has a hypotensive effect. An excessive amount of training is not necessarily required to reduce blood pressure. Most of the previous studies examining the relationships between exercise and the improvements in cardiovascular risk factors after exercise training suggest that the improvement in fat metabolism and insulin resistance is associated more with weight and/or fat loss rather than with enhanced aerobic capacity. A reduction in blood pressure through exercise training is also affected more by a reduction in body weight and/or fat than by improved aerobic capacity, although exercise training itself can also have a hypotensive effect regardless of weight loss.

Since the physical condition of the elderly population is variable, there are limited studies on exercise training in elderly subjects including those who are not physically active. In particular, there have been few reports on the improvements in CHD risk factors after low-intensity/amount exercise training which is feasible for the majority of elderly people. In order to establish an exercise prescription that is optimal for the elderly, it is necessary to investigate the effect of exercise designed for the elderly on the risk factors for CHD.

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