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ORIGINAL ARTICLE

René Koopman · Ralph J. F. Manders
Antoine H. G. Zorenc · Gabby B. J. Hul
Harm Kuipers · Hans A. Keizer
Luc J. C. van Loon

A single session of resistance exercise enhances insulin sensitivity for at least 24 h in healthy men

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Abstract The aim of the present study was to determine whether a single session of resistance exercise improves whole-body insulin sensitivity in healthy men for up to 24 h. Twelve male subjects (23 ± 1 years) were studied over a period of 4 days during which they consumed a standardized diet, providing 0.16 ± 0.01 MJ·kg^{-1}·day^{-1} containing 15 ± 0.1 energy% (En%) protein, 29 ± 0.1 En% fat and 55 ± 0.3 En% carbohydrate. Insulin sensitivity was determined 24 h before and 24 h after a single resistance exercise session (8 sets of 10 repetitions at 75% of 1 repetition maximum for two leg exercise tasks) using an intravenous insulin tolerance test. Insulin sensitivity index was calculated by the decline in arterial blood glucose concentration following intravenous administration of a single bolus of human insulin (0.075 IU·kg^{-1} fat free mass). Basal glucose and insulin concentrations were not changed up to 24 h after the resistance exercise. However, a substantial 13 ± 5% improvement in whole-body insulin sensitivity was observed, 24 h after the resistance exercise ($P < 0.05$). This study shows that even a single session of resistance exercise improves whole-body insulin sensitivity for up to 24 h in healthy men, which is consistent with earlier observations following endurance exercise tasks.

R. Koopman (✉) · R. J. F. Manders · A. H. G. Zorenc
G. B. J. Hul · L. J. C. van Loon
Departments of Human Biology,
Nutrition Research Institute Maastricht (NUTRIM),
Maastricht University,
PO BOX 616, 6200 MD,
Maastricht, The Netherlands
E-mail: R.Koopman@HB.unimaas.nl
Tel.: +31-43-3882124
Fax: +31-43-3670976

H. Kuipers · H. A. Keizer · L. J. C. van Loon
Departments of Movement Sciences,
Nutrition Research Institute Maastricht (NUTRIM),
Maastricht University, Maastricht,
The Netherlands

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Introduction

Skeletal muscle tissue is responsible for most of the insulin-stimulated glucose uptake in humans. The capacity for insulin-mediated glucose uptake is directly related to total muscle mass and inversely associated with fat mass (Yki-Jarvinen and Koivisto 1983). In type 2 diabetes patients, insulin-stimulated glucose uptake is substantially impaired in liver and skeletal muscle tissue, leading to the development of a hyperinsulinemic and/or hyperglycemic state (DeFronzo et al. 1983). The development of insulin resistance (IR) and/or type 2 diabetes is strongly associated with the presence of obesity and physical inactivity (Eriksson and Lindgarde 1996; Hu et al. 2001; Mokdad et al. 2001). Weight loss (Goodpaster et al. 1999; Houmard et al. 2002; Niskanen et al. 1996), increased physical activity (Henriksen 2002) as well as pharmacological interventions (Hundal et al. 2000; Inzucchi et al. 1998; Saltiel and Olefsky 1996) have been shown to form effective strategies to improve insulin sensitivity.

Studies investigating the role of physical activity as a means to improve the insulin sensitivity generally apply endurance exercise as a model. Endurance exercise allows the use of a relatively large amount of muscle for a prolonged period of time, and, as such, forms a safe and effective means to elevate energy expenditure and promote weight loss (Dumortier et al. 2003). Prolonged endurance exercise training has been shown to improve insulin sensitivity in young (Dela et al. 1992), elderly (Kahn et al. 1990) and/or insulin-resistant subjects (Borghouts et al. 1999; Dela et al. 1994, 1995; Hughes et al. 1993; Perseghin et al. 1996; Rogers et al. 1988). However, even a single bout of moderate-to-high-intensity endurance exercise has been shown to acutely improve insulin sensitivity and/or glucose tolerance

(Devlin et al. 1987; Devlin and Horton 1985; Heath et al. 1983; Mikines et al. 1988; Perseghin et al. 1996). This effect has been reported to persist for a period ranging from 2 h (Mikines et al. 1988), 4–6 h (Wojtaszewski et al. 2000), 12–16 h (Devlin et al. 1987; Devlin and Horton 1985; Heath et al. 1983) to 48 h post-exercise (Mikines et al. 1988; Perseghin et al. 1996).

In contrast to endurance exercise, limited information is available on the potential of resistance exercise to affect insulin sensitivity and/or glucose tolerance (Smutok et al. 1993, 1994). Some studies have demonstrated that 6–12 weeks of progressive resistance training improves glucose tolerance (Craig et al. 1989; Fenicchia et al. 2004; Miller et al. 1984). These changes are generally attributed to the concomitant gain in skeletal muscle mass. Unfortunately, the few studies that investigated the more acute effects of resistance exercise have provided contradictory findings. Whereas some have reported an improved glucoregulatory response, 12–24 h after a single bout of resistance exercise (Fenicchia et al. 2004; Fluckey et al. 1994), others have failed to observe any change in insulin sensitivity (Chapman et al. 2002). The apparent discrepancy in the literature is likely due to the methods used to determine insulin sensitivity and/or glucose tolerance. Most studies have applied an oral glucose tolerance test (OGTT) as a surrogate measure of insulin sensitivity. However, the OGTT has been reported to have a questionable reproducibility (Ko et al. 1998). In contrast to the OGTT, the insulin tolerance test (ITT) directly measures insulin sensitivity (Hirst et al. 1993) and has been validated using the euglycaemic hyperinsulinemic clamp (Akinmokun et al. 1992). Therefore, in the present study we applied the ITT to investigate the short-term effects of a single resistance exercise session on whole-body insulin sensitivity.

Methods

Subjects

Twelve healthy male volunteers with no history of having participated in any regular exercise program were recruited for the present study. Subject characteristics are shown in Table 1. All subjects were informed of the nature and possible risks of the experimental procedures before their informed consent was obtained, the latter after approval by the Medical Ethical Committee of the Academic Hospital Maastricht, The Netherlands.

Pre-testing

All subjects reported to the laboratory in the morning after an overnight fast, for measurement of body composition, assessed using the hydrostatic weighing method. Residual lung volume was measured by the helium-dilution technique using a spirometer (Volugraph 2000, Mijnhart, Bunnik, The Netherlands). Body weight was measured with a digital balance with an accuracy of 0.001 kg (E1200, August Sauter GmbH, Albstadt, Germany). Body fat percentage was calculated using Siri's equation (Siri 1956). Fat free mass (FFM) was calculated by subtracting fat mass from total body mass.

Table 1 Subjects characteristics

Age (years)	23.0 ± 1.0
Weight (kg)	74.3 ± 2.8
Height (m)	1.79 ± 0.02
BMI ($kg \cdot m^{-2}$)	23.1 ± 0.7
% bodyfat (%)	17.1 ± 2.2
Fat free mass (kg)	61.2 ± 1.8
Fat mass (kg)	13.1 ± 1.9
HbA_{1C} (%)	5.23 ± 0.08
1RM leg press (kg)	198 ± 8
1RM leg press (kg BW^{-1})	2.67 ± 0.08
1RM leg extension (kg)	108 ± 4
1RM leg extension (kg BW^{-1})	1.46 ± 0.05

Values are expressed as means ± SEM

Thereafter, subjects participated in an exercise trial to become familiarized with the exercise protocol and the equipment. The proper lifting technique was demonstrated and practiced for each of the two lower-limb exercises (leg press and leg extension) and for the three upper-body exercises (chest press, shoulder press and lat-pulldown). Thereafter maximum strength was estimated using the multiple repetitions testing procedure (Mayhew et al. 1993). In another session, at least 1 week before the first experimental trial, subjects' 1 repetition maximum (1RM) was determined (Kraemer and Fry 1995). After warming up, the load was set at 90–95% of the estimated 1RM, and increased after each successful lift until failure. A 5-min resting period between subsequent attempts was allowed. A repetition was valid if the subject was able to complete the entire lift in a controlled manner without assistance.

Standardized diet and activity prior to testing

All subjects received a standardized diet for 4 days, i.e. the day prior to the ITT (on day 1), the ITT (on day 2), the resistance exercise session (on day 3), and the second ITT (on day 4), which were performed at exactly 8.30 a.m. in the morning after an overnight fast. Subjects were provided with a pre-weighed amount of food products, beverages, and instant meals and were allowed to drink water ad libitum. They were also instructed to take all main meals (breakfast, lunch, and dinner) and between-meal snacks at predetermined time intervals during each day. Subjects were asked to record their food intake during the entire testing period. Energy intake averaged 0.16 ± 0.01 $MJ \cdot kg\ bodyweight^{-1} \cdot day^{-1}$ containing 15 ± 0.1 Energy% (En%) of protein, 29 ± 0.1

En% of fat and 55 ± 0.3 En% of carbohydrate. Energy intake did not differ significantly between days, even though subjects were free to increase or decrease the size of all meals and snacks. All volunteers were instructed to refrain from any sort of heavy physical exercise during the entire period except for the resistance exercise session.

Experimental trials

An overview of the study protocol is provided in Fig. 1. One week after the subjects completed their 1RM-test, insulin sensitivity was measured using a short ITT. Twenty-four hours after the ITT, subjects exercised for ~1 h using the resistance exercise machines. The resistance exercise was followed 24 h later with a second ITT.

Insulin tolerance test

The day before and after the resistance exercise session, subjects arrived at the laboratory by car or public transportation (to avoid disturbances in insulin sensitivity due to physical activity: i.e. cycling, stair walking) at 8.00 a.m. after an overnight fast. A Teflon catheter was inserted into an antecubital vein for insulin infusion and a second Teflon catheter was inserted retrogradely in a dorsal vein of the contralateral hand which the subject had to hold in a hot-box (60°C), for arterial blood sampling. After 30 min of bed rest in an inclined position, a basal blood sample was collected ($t = -5$). Another blood sample ($t = 0$) was obtained before administration of a single intravenous dose of human insulin (Actrapid, Novo Nordisk A/S, Bagsværd, Denmark) of 0.075 IU·kg^{-1} fat free mass. Thereafter, blood samples were collected every 2 min until $t = 16$ min. Thereafter, the test was terminated and subjects consumed a standardized breakfast.

Blood samples were taken for blood glucose measurement (see Analysis section). In addition, blood glucose concentrations were directly monitored using a blood glucose monitor (Precision Xtra, MediSense, Amersfoort, The Netherlands) at $t = 0$, 6, 12, 16 min. If blood glucose < 2.5 mmol l^{-1} at $t = 16$ a 10 ml bolus of 20% dextrose (Baxter B.V., Utrecht, The Netherlands) was injected to prevent severe hypoglycaemia. Blood glucose was checked again at 22 min and 30 min after the insulin injection. No subjects reported symptoms of hypoglycaemia during the ITT, and/or during the 20–30 min after insulin administration.

Resistance exercise

The day after the first ITT subjects arrived at the laboratory by car or public transportation at 8.00 a.m., in an overnight fasted state. Subjects performed a general warm-up of 5 min using a Stairmaster, followed by three sets of ten repetitions on three resistance exercise machines targeting the upper-body (chest press, shoulder press and lat-pulldown, Jimsa Benelux BV, Rotterdam, The Netherlands). The latter were included to provide a whole-body warm-up and to reduce the risk of injury. Thereafter, the resistance exercise session targeted the legs, with eight sets of ten repetitions on the horizontal leg press machine (Technogym BV, Rotterdam, The Netherlands) and eight sets of ten repetitions on the leg extension machine (Technogym). Both exercises were performed at 75% of the subjects' individual 1RM with 2-min rest intervals between sets and in total required ~40 min to complete. All subjects were verbally encouraged during the test to complete the entire protocol. At the end of the exercise session, subjects consumed a standardized breakfast. Energy expenditure during the exercise session was not measured. Based on indirect calorimetry measurements during similar resistance exercise protocols, others have shown energy expenditure rates ranging between 14 kJ·min^{-1} and 27 kJ·min^{-1} (Ballor et al. 1988; Burleson et al. 1998; Pichon et al. 1996).

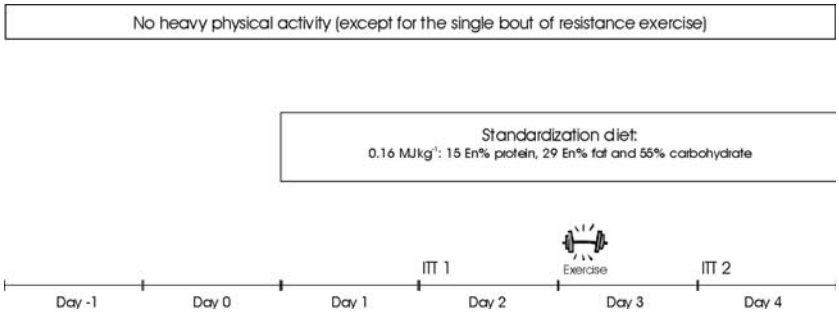

Fig. 1 Overview of the study design. All subjects received a standardized diet during the entire study period; prior to the day preceding the first insulin tolerance test (*ITT*) (day 1), the ITT (day 2), the resistance exercise session (day 3), and the second ITT (day 4), which were performed at exactly 8.30 a.m. in the morning after an overnight fast. All volunteers were instructed to refrain from any sort of heavy physical activity during the entire period (6 days) except for the resistance exercise session

Analysis

Blood samples (4 ml) were collected in tubes containing a glycolytic inhibitor (sodium fluoride) and anticoagulant (potassium oxalate), and placed on ice. After centrifugation at 1,000*g* and 4°C for 5 min aliquots of plasma were frozen immediately in liquid nitrogen and stored at −80°C until analyses. Plasma glucose (Uni Kit III, 07367204, Roche, Basel, Switzerland) concentrations were analyzed with the COBAS FARA semi-automatic analyzer (Roche). Plasma insulin was measured by radioimmunoassay (HI-11K, Linco Research Inc., St. Charles, MO, USA). To determine basal fasting blood HbA1c content a 3 ml blood sample was collected in EDTA containing tubes and analyzed by high-performance liquid chromatography (Bio-Rad Diamat, Munich, Germany).

Calculation of insulin sensitivity

The decline in blood glucose between 4 min and 16 min during the ITT was used to determine the insulin sensitivity (Borghouts et al. 1999). Linear regression was used to calculate the slope of the decline in log transformed blood glucose concentration against time during the first 4–16 min (Akinmokun et al. 1992; Borghouts et al. 1999; Hirst et al. 1993). The slope was multiplied by −100 to derive the rate constant (K_{ITT}) which is equivalent to the percentage decline in blood glucose per minute (Hirst et al. 1993).

Insulin sensitivity was also estimated by the homeostasis model assessment or HOMA-(IR) index which is calculated by dividing the product of fasting plasma glucose ($mmol \cdot l^{-1}$) and insulin concentrations ($mU \cdot l^{-1}$) by 22.5 (Matthews et al. 1985).

Statistics

All data are expressed as means ± SEM. IS_{index} and K_{ITT} calculated from data obtained during ITT1 and ITT2 were compared using a two-tailed, paired *t*-test. In addition, simple regression analysis was performed to calculate correlations between basal insulin, glucose, IS_{index} and HOMA index. Statistical significance was set at $P < 0.05$.

Results

Resistance exercise

Mean 1RM measured during the pre-testing was 198 ± 8 kg on the horizontal leg press and 108 ± 4 kg on the leg extension. Therefore, average weights used during the resistance exercise were 148 ± 6 kg and 81 ± 3 kg for the leg press and leg extension, respectively. All subjects completed eight sets with ten repetitions on the leg press. However, during the sixth set, two subjects could not finish all ten repetitions, after which weight was reduced down to 65% of the individual 1RM. Ten subjects completed eight sets of ten repetitions on the leg extension. Two subjects were not able to finish the last two sets due to dizziness.

Plasma analysis

All subjects showed normal fasting plasma glucose (5.42 ± 0.13 $mmol \cdot l^{-1}$) and insulin concentrations (5.35 ± 0.43 $mU \cdot l^{-1}$), before the start of the first ITT. Basal plasma glucose concentrations were similar before the ITT1 and ITT2 trial (5.42 ± 0.13 vs. 5.45 ± 0.10 $mmol \cdot l^{-1}$, respectively). Basal plasma insulin concentrations did not differ between the ITT1 and ITT2 (5.35 ± 0.43 $mU \cdot l^{-1}$ and 5.51 ± 0.35 $mU \cdot l^{-1}$, respectively). The HOMA-(IR) index before ITT1 and ITT2 averaged 1.29 ± 0.11 and 1.33 ± 0.08, respectively (NS). An overview of the plasma data is presented in Table 2.

Intravenous insulin tolerance test

During the first ITT, the administration of insulin resulted in a decline in plasma glucose concentrations from 5.27 ± 0.11 $mmol \cdot l^{-1}$ at $t = 4$ min to 3.00 ± 0.18 $mmol \cdot l^{-1}$ at $t = 16$ min. During the ITT, 24 h after the resistance exercise (ITT2), plasma glucose concentrations were reduced from 5.26 ± 0.10 $mmol \cdot l^{-1}$ at $t = 4$ min to 2.73 ± 0.19 $mmol \cdot l^{-1}$ at $t = 16$ min. Plasma glucose levels at $t = 16$ min were significantly lower in ITT2 compared to ITT1 ($P < 0.05$). The decline in blood glucose concentration is presented in Fig. 2.

Calculated rate constants for the glucose disappearance (K_{ITT}, % min^{-1}) between 4 min and 16 min following insulin injection during a short ITT before, and 24 h after resistance exercise are shown in Fig. 3a. K_{ITT} values were significantly increased with 13.4 ± 4.8% 24 h post-exercise compared to pre-exercise values (5.1 ± 0.4%·min^{-1} vs. 5.8 ± 0.5%·min^{-1}, $P < 0.05$). The individual K_{ITT} values of blood glucose after insulin administration during the ITT before and 24 h after resistance exercise are shown in Fig. 3b.

Table 2 Blood parameters

	ITT1	ITT2
Basal glucose ($mmol \cdot l^{-1}$)	5.44 ± 0.12	5.45 ± 0.10
Glucose at $t = 16$ ($mmol \cdot l^{-1}$)	3.00 ± 0.18	2.73 ± 0.19[a]
K_{ITT} (% · min^{-1})	5.07 ± 0.37	5.77 ± 0.53[a]
Basal insulin ($mU \cdot l^{-1}$)	5.35 ± 0.43	5.51 ± 0.35
HOMA-(IR)-index	1.29 ± 0.11	1.33 ± 0.08

Values are expressed as means ± SEM
[a]Significantly different from ITT1, $P < 0.05$

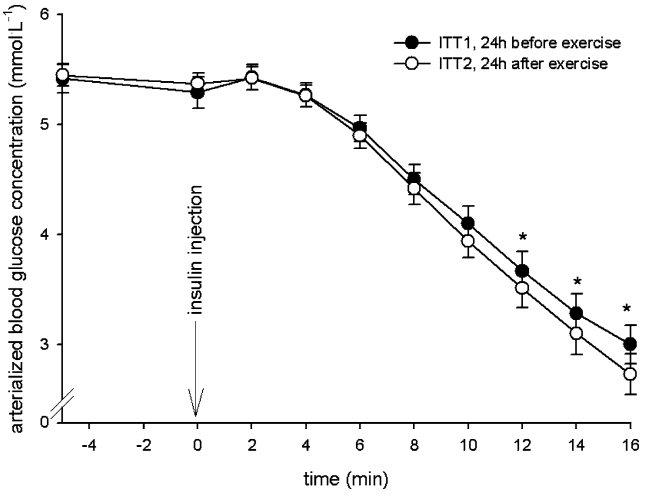

Fig. 2 Plasma glucose concentrations following injection of a bolus of insulin 0.075 IU·kg^{-1} fat free mass 24 h before and 24 h after a single bout of resistance exercise. Values are expressed as means ± SEM. * Significantly different from basal blood glucose concentrations during *ITT1* and *ITT2* ($P < 0.01$)

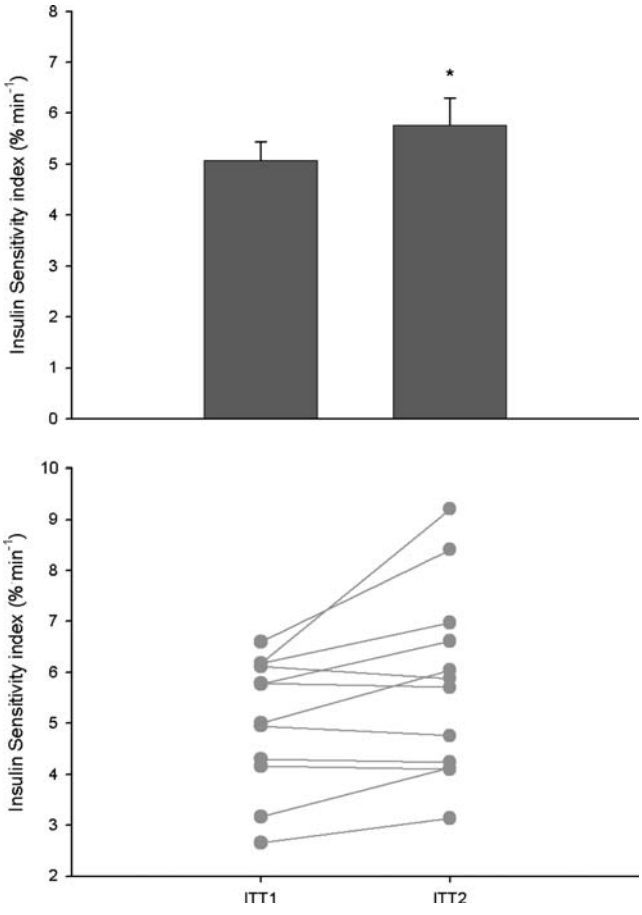

Fig. 3 Mean rate constants for the disappearance (K_{ITT}) of blood glucose following insulin injection during a short insulin tolerance test (*ITT*) before, and 24 h after resistance exercise **(a)**. Values are expressed as means ± SEM. * Significantly different from values observed during ITT1 ($P < 0.05$). Individual results showing K_{ITT} values of blood glucose after insulin administration during the ITT before and 24 h after resistance exercise (**b**)

Discussion

It has been well established that a single bout of endurance exercise improves whole-body insulin sensitivity for up to 24 h. In the present study, we extend on those findings by showing that a single session of resistance exercise can also effectively increase whole-body insulin sensitivity for up to 24 h in healthy males.

Endurance exercise training has been reported to improve whole-body insulin sensitivity in young (Dela et al. 1992), elderly (Kahn et al. 1990) and insulin-resistant subjects (Borghouts et al. 1999; Dela et al. 1994, 1995; Hughes et al. 1993; Perseghin et al. 1996; Rogers et al. 1988). The latter is attributed to the concomitant induction of weight loss (Dumortier et al. 2003) and the upregulation of skeletal muscle GLUT-4 expression (Cox et al. 1999). Besides the more prolonged adaptive response to endurance training, it has been firmly established that even an acute bout of endurance exercise elevates whole-body insulin sensitivity for a prolonged period, ranging from 2 (Mikines et al. 1988), 4–6 (Wojtaszewski et al. 2000), 12–16 (Devlin et al. 1987; Devlin and Horton 1985; Heath et al. 1983) to up to 48 h following cessation of exercise (Mikines et al. 1988; Perseghin et al. 1996). The latter is generally attributed to attenuated muscle GLUT-4 translocation (Thorell et al. 1999) as well as increased GLUT-4 expression in muscle tissue (Kraniou et al. 2000). Factors thought to play a major regulatory role in this process include muscle lipid content (van Loon 2004) in relation with physical inactivity, AMPK activation (Fryer et al. 2002; Hayashi et al. 2000; Winder 2001), muscle glycogen content and subsequent activation of glycogen synthase activity (Bogardus et al. 1983; Derave et al. 2000; Garcia-Roves et al. 2003; Perseghin et al. 1996; Wojtaszewski et al. 1997, 2000), which enhance insulin signalling downstream of the receptor (Goodyear and Kahn 1998; Henriksen 2002).

In addition to prolonged endurance training, resistance exercise interventions have also been reported to improve glucose tolerance and/or whole-body insulin sensitivity (Craig et al. 1989; Dunstan et al. 2002; Fenicchia et al. 2004; Holten et al. 2004; Ishii et al. 1998; Miller et al. 1984). The latter is generally attributed to a concomitant gain in skeletal muscle tissue, which improves whole-body glucose disposal capacity (Craig et al. 1989; Fenicchia et al. 2004; Miller et al. 1984). Besides this increase in lean muscle tissue, resistance exercise also improves functional capacity, thereby supporting a more active and healthy lifestyle. However, in the development of most exercise intervention programs, the focus generally lies on the implementation of endurance exercise, because of its acute stimulating effect on whole-body insulin sensitivity. Consequently, endurance exercise sessions are generally implemented at the expense of resistance exercise when designing such intervention programs. In the present study, we specu-

lated that a single resistance exercise session could also acutely improve whole-body insulin sensitivity. The latter would imply that there is no need to restrict the inclusion of resistance exercise when designing effective lifestyle intervention programs.

So far, the few studies that investigated the acute effects of resistance exercise on insulin sensitivity have provided contradictory findings (Chapman et al. 2002; Fenicchia et al. 2004; Fluckey et al. 1994). In the present study, we show that a single resistance exercise session (mainly consisting of 16 sets of leg-exercise using 75% of the individual 1RM) improves whole-body insulin sensitivity by as much as 13.4 ± 4.8% when measured 24 h after exercise. However, a large inter-subject variation was observed. Whereas five subjects did not show a measurable increase in plasma glucose clearance 24 h after resistance exercise, the other seven subjects showed improvements in insulin sensitivity ranging between 13% and 49%. The average increase in whole-body insulin sensitivity following resistance exercise seems to be of a similar magnitude as the ~20% improvement reported following an acute (~60 min) bout of endurance exercise (Mikines et al. 1988; Perseghin et al. 1996). Previous studies, investigating the acute effects of resistance exercise on insulin sensitivity, have applied either oral (Fenicchia et al. 2004; Fluckey et al. 1994) or intravenous (Chapman et al. 2002) glucose tolerance tests (OGTT and IVGTT, respectively) to estimate insulin sensitivity. These studies, which use the glucose and/or insulin response following glucose administration as a representative of insulin action, have provided discrepant findings. Fluckey et al. (1994) reported no change in integrated glucose concentrations following glucose ingestion 18 h after resistance exercise, but observed a lower insulin response (Fluckey et al. 1994). Fenicchia et al. (2004) reported a lower glucose response 12–24 h after resistance exercise in female type 2 diabetes patients, without observing any difference in insulin levels. In that study as well as in a study by Chapman et al. (2002) no changes in glucose or insulin responses were observed 15 h after a single bout of resistance exercise in the healthy, non-obese, normoglycaemic controls (Chapman et al. 2002; Fenicchia et al. 2004). Clearly, in the literature the acute effects of resistance exercise on whole body insulin sensitivity have always remained equivocal.

The discrepancy in these earlier findings could likely be attributed to the use of glucose tolerance tests, which do not measure insulin sensitivity directly. In accordance, the OGTT has been reported to have a questionable reproducibility as a measure of whole-body insulin sensitivity (Ko et al. 1998). Therefore, in the present study, we applied an intravenous ITT 24 h before and 24 h after performing a resistance exercise session. We observed a greater arterial blood glucose clearance rate following an intravenous dose of insulin 24 h after resistance exercise compared with pre-exercise values (Fig. 3). During the ITT, insulin sensitivity index was calculated by the observed decline in arterial blood glucose following insulin administration. In contrast to the OGTT, the ITT provides a reproducible method that more directly assesses insulin sensitivity (Hirst et al. 1993), and has been validated using the euglycaemic hyper-insulinemic clamp (Akinmokun et al. 1992). Other factors that might explain the discrepant findings in the literature include the intensity and/or duration of the resistance exercise session, the standardization of dietary intake and daily physical activities as well as the population studied. The resistance exercise protocol used both by Fluckey et al. (1994) and Chapman et al. (2002) may not have been of sufficient intensity to invoke a large significant improvement in whole body insulin sensitivity as observed in the present study. Furthermore, many of the earlier studies did not standardize food intake and/or physical activity prior to the OGTTs (Fenicchia et al. 2004; Fluckey et al. 1994). In the present study, both food intake and physical activity were strictly standardized to reduce any confounding effects of diet on insulin sensitivity.

In the present study we show that resistance exercise acutely stimulates insulin sensitivity. Similar to the reported effects of a single bout of endurance exercise, this is likely attributed to attenuated muscle GLUT-4 translocation (Thorell et al. 1999) and/or elevated GLUT-4 expression (Kraniou et al. 2000). Though information on the metabolic demand imposed upon by resistance exercise is scarce, we speculate that, resistance exercise activates AMPK and substantially reduces muscle glycogen and/or IMTG content. More research is warranted to elucidate the exact mechanisms responsible for the observed increase in insulin sensitivity following exercise. In conclusion, an acute bout of intense resistance exercise substantially improves whole-body insulin sensitivity for up to 24 h after cessation of exercise. Therefore, the present data indicate that both endurance as well as resistance type exercise tasks stimulate insulin sensitivity. As such, there should be no restriction in combining the benefits of both types of exercise in future lifestyle intervention programs.

References

Akinmokun A, Selby PL, Ramaiya K, Alberti KG (1992) The short insulin tolerance test for determination of insulin sensitivity: a comparison with the euglycaemic clamp. Diabet Med 9:432–437

Ballor DL, Katch VL, Becque MD, Marks CR (1988) Resistance weight training during caloric restriction enhances lean body weight maintenance. Am J Clin Nutr 47:19–25

Bogardus C, Thuillez P, Ravussin E, Vasquez B, Narimiga M, Azhar S (1983) Effect of muscle glycogen depletion on in vivo insulin action in man. J Clin Invest 72:1605–1610

Borghouts LB, Backx K, Mensink MF, Keizer HA (1999) Effect of training intensity on insulin sensitivity as evaluated by insulin tolerance test. Eur J Appl Physiol 80:461–466

Burleson MA Jr, O'Bryant HS, Stone MH, Collins MA, Triplett-McBride T (1998) Effect of weight training exercise and treadmill exercise on post-exercise oxygen consumption. Med Sci Sports Exerc 30:518–522

Chapman J, Garvin AW, Ward A, Cartee GD (2002) Unaltered insulin sensitivity after resistance exercise bout by postmenopausal women. Med Sci Sports Exerc 34:936–941

Cox JH, Cortright RN, Dohm GL, Houmard JA (1999) Effect of aging on response to exercise training in humans: skeletal muscle GLUT-4 and insulin sensitivity. J Appl Physiol 86:2019–2025

Craig BW, Everhart J, Brown R (1989) The influence of high-resistance training on glucose tolerance in young and elderly subjects. Mech Ageing Dev 49:147–157

DeFronzo RA, Ferrannini E, Koivisto V (1983) New concepts in the pathogenesis and treatment of noninsulin-dependent diabetes mellitus. Am J Med 74:52–81

Dela F, Mikines KJ, von Linstow M, Secher NH, Galbo H (1992) Effect of training on insulin-mediated glucose uptake in human muscle. Am J Physiol 263:E1134–E1143

Dela F, Ploug T, Handberg A, Petersen LN, Larsen JJ, Mikines KJ, Galbo H (1994) Physical training increases muscle GLUT4 protein and mRNA in patients with NIDDM. Diabetes 43:862–865

Dela F, Larsen JJ, Mikines KJ, Ploug T, Petersen LN, Galbo H (1995) Insulin-stimulated muscle glucose clearance in patients with NIDDM. Effects of one-legged physical training. Diabetes 44:1010–1020

Derave W, Hansen BF, Lund S, Kristiansen S, Richter EA (2000) Muscle glycogen content affects insulin-stimulated glucose transport and protein kinase B activity. Am J Physiol Endocrinol Metab 279:E947–E955

Devlin JT, Horton ES (1985) Effects of prior high-intensity exercise on glucose metabolism in normal and insulin-resistant men. Diabetes 34:973–979

Devlin JT, Hirshman M, Horton ED, Horton ES (1987) Enhanced peripheral and splanchnic insulin sensitivity in NIDDM men after single bout of exercise. Diabetes 36:434–439

Dumortier M, Brandou F, Perez-Martin A, Fedou C, Mercier J, Brun JF (2003) Low intensity endurance exercise targeted for lipid oxidation improves body composition and insulin sensitivity in patients with the metabolic syndrome. Diabetes Metab 29:509–518

Dunstan DW, Daly RM, Owen N, Jolley D, De Courten M, Shaw J, Zimmet P (2002) High-intensity resistance training improves glycemic control in older patients with type 2 diabetes. Diabetes Care 25:1729–1736

Eriksson KF, Lindgarde F (1996) Poor physical fitness, and impaired early insulin response but late hyperinsulinaemia, as predictors of NIDDM in middle-aged Swedish men. Diabetologia 39:573–579

Fenicchia LM, Kanaley JA, Azevedo JL Jr, Miller CS, Weinstock RS, Carhart RL, Ploutz-Snyder LL (2004) Influence of resistance exercise training on glucose control in women with type 2 diabetes. Metabolism 53:284–289

Fluckey JD, Hickey MS, Brambrink JK, Hart KK, Alexander K, Craig BW (1994) Effects of resistance exercise on glucose tolerance in normal and glucose-intolerant subjects. J Appl Physiol 77:1087–1092

Fryer LG, Foufelle F, Barnes K, Baldwin SA, Woods A, Carling D (2002) Characterization of the role of the AMP-activated protein kinase in the stimulation of glucose transport in skeletal muscle cells. Biochem J 363:167–174

Garcia-Roves PM, Han DH, Song Z, Jones TE, Hucker KA, Holloszy JO (2003) Prevention of glycogen supercompensation prolongs the increase in muscle GLUT4 after exercise. Am J Physiol Endocrinol Metab 285:E729–E736

Goodpaster BH, Kelley DE, Wing RR, Meier A, Thaete FL (1999) Effects of weight loss on regional fat distribution and insulin sensitivity in obesity. Diabetes 48:839–847

Goodyear LJ, Kahn BB (1998) Exercise, glucose transport, and insulin sensitivity. Annu Rev Med 49:235–261

Hayashi T, Hirshman MF, Fujii N, Habinowski SA, Witters LA, Goodyear LJ (2000) Metabolic stress and altered glucose transport: activation of AMP-activated protein kinase as a unifying coupling mechanism. Diabetes 49:527–531

Heath GW, Gavin JR III, Hinderliter JM, Hagberg JM, Bloomfield SA, Holloszy JO (1983) Effects of exercise and lack of exercise on glucose tolerance and insulin sensitivity. J Appl Physiol 55:512–517

Henriksen EJ (2002) Invited review: effects of acute exercise and exercise training on insulin resistance. J Appl Physiol 93:788–796

Hirst S, Phillips DI, Vines SK, Clark PM, Hales CN (1993) Reproducibility of the short insulin tolerance test. Diabet Med 10:839–842

Holten MK, Zacho M, Gaster M, Juel C, Wojtaszewski JF, Dela F (2004) Strength training increases insulin-mediated glucose uptake, GLUT4 content, and insulin signaling in skeletal muscle in patients with type 2 diabetes. Diabetes 53:294–305

Houmard JA, Tanner CJ, Yu C, Cunningham PG, Pories WJ, MacDonald KG, Shulman GI (2002) Effect of weight loss on insulin sensitivity and intramuscular long-chain fatty acyl-CoAs in morbidly obese subjects. Diabetes 51:2959–2963

Hu FB, Manson JE, Stampfer MJ, Colditz G, Liu S, Solomon CG, Willett WC (2001) Diet, lifestyle, and the risk of type 2 diabetes mellitus in women. N Engl J Med 345:790–797

Hughes VA, Fiatarone MA, Fielding RA, Kahn BB, Ferrara CM, Shepherd P, Fisher EC, Wolfe RR, Elahi D, Evans WJ (1993) Exercise increases muscle GLUT-4 levels and insulin action in subjects with impaired glucose tolerance. Am J Physiol 264:E855–E862

Hundal RS, Krssak M, Dufour S, Laurent D, Lebon V, Chandramouli V, Inzucchi SE, Schumann WC, Petersen KF, Landau BR, Shulman GI (2000) Mechanism by which metformin reduces glucose production in type 2 diabetes. Diabetes 49:2063–2069

Inzucchi SE, Maggs DG, Spollett GR, Page SL, Rife FS, Walton V, Shulman GI (1998) Efficacy and metabolic effects of metformin and troglitazone in type II diabetes mellitus. N Engl J Med 338:867–872

Ishii T, Yamakita T, Sato T, Tanaka S, Fujii S (1998) Resistance training improves insulin sensitivity in NIDDM subjects without altering maximal oxygen uptake. Diabetes Care 21:1353–1355

Kahn SE, Larson VG, Beard JC, Cain KC, Fellingham GW, Schwartz RS, Veith RC, Stratton JR, Cerqueira MD, Abrass IB (1990) Effect of exercise on insulin action, glucose tolerance, and insulin secretion in aging. Am J Physiol 258:E937–E943

Ko GT, Chan JC, Woo J, Lau E, Yeung VT, Chow CC, Cockram CS (1998) The reproducibility and usefulness of the oral glucose tolerance test in screening for diabetes and other cardiovascular risk factors. Ann Clin Biochem 35(Pt 1):62–67

Kraemer WJ, Fry AC (1995) Strength testing; development and evaluation of methodology. In: Maud PJ, Foster C (eds) Physiological assessment of human fitness. Human Kinetics, Leeds, pp 115–133

Kraniou Y, Cameron-Smith D, Misso M, Collier G, Hargreaves M (2000) Effects of exercise on GLUT-4 and glycogenin gene expression in human skeletal muscle. J Appl Physiol 88:794–796

van Loon LJ (2004) Use of intramuscular triacylglycerol as a substrate source during exercise in humans. J Appl Physiol 97:1170–1187

Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC (1985) Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 28:412–419

Mayhew JL, Piper FC, Ware JS (1993) Anthropometric correlates with strength performance among resistance trained athletes. J Sports Med Phys Fitness 33:159–165

Mikines KJ, Sonne B, Farrell PA, Tronier B, Galbo H (1988) Effect of physical exercise on sensitivity and responsiveness to insulin in humans. Am J Physiol 254:E248–E259

Miller WJ, Sherman WM, Ivy JL (1984) Effect of strength training on glucose tolerance and post-glucose insulin response. Med Sci Sports Exerc 16:539–543

Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP (2001) The continuing epidemics of obesity and diabetes in the United States. JAMA 286:1195–1200

Niskanen L, Uusitupa M, Sarlund H, Siitonen O, Paljarvi L, Laakso M (1996) The effects of weight loss on insulin sensitivity, skeletal muscle composition and capillary density in obese non-diabetic subjects. Int J Obes Relat Metab Disord 20:154–160

Perseghin G, Price TB, Petersen KF, Roden M, Cline GW, Gerow K, Rothman DL, Shulman GI (1996) Increased glucose transport-phosphorylation and muscle glycogen synthesis after exercise training in insulin-resistant subjects. N Engl J Med 335:1357–1362

Pichon C, Hunter GR, Morris M, Bond RL, Metz J (1996) Blood pressure and heart rate response and metabolic cost of circuit versus traditional weight training. J Strength Cond Res 10:153–156

Rogers MA, Yamamoto C, King DS, Hagberg JM, Ehsani AA, Holloszy JO (1988) Improvement in glucose tolerance after 1 wk of exercise in patients with mild NIDDM. Diabetes Care 11:613–618

Saltiel AR, Olefsky JM (1996) Thiazolidinediones in the treatment of insulin resistance and type II diabetes. Diabetes 45:1661–1669

Siri WE (1956) The gross composition of the body. Adv Biol Med Physiol 4:238–280

Smutok MA, Reece C, Kokkinos PF, Farmer C, Dawson P, Shulman R, DeVane-Bell J, Patterson J, Charabogos C, Goldberg AP et al (1993) Aerobic versus strength training for risk factor intervention in middle-aged men at high risk for coronary heart disease. Metabolism 42:177–184

Smutok MA, Reece C, Kokkinos PF, Farmer CM, Dawson PK, DeVane J, Patterson J, Goldberg AP, Hurley BF (1994) Effects of exercise training modality on glucose tolerance in men with abnormal glucose regulation. Int J Sports Med 15:283–289

Thorell A, Hirshman MF, Nygren J, Jorfeldt L, Wojtaszewski JF, Dufresne SD, Horton ES, Ljungqvist O, Goodyear LJ (1999) Exercise and insulin cause GLUT-4 translocation in human skeletal muscle. Am J Physiol 277:E733–E741

Winder WW (2001) Energy-sensing and signaling by AMP-activated protein kinase in skeletal muscle. J Appl Physiol 91:1017–1028

Wojtaszewski JF, Hansen BF, Kiens B, Richter EA (1997) Insulin signaling in human skeletal muscle: time course and effect of exercise. Diabetes 46:1775–1781

Wojtaszewski JF, Hansen BF, Gade J, Kiens B, Markuns JF, Goodyear LJ, Richter EA (2000) Insulin signaling and insulin sensitivity after exercise in human skeletal muscle. Diabetes 49:325–331

Yki-Jarvinen H, Koivisto VA (1983) Effects of body composition on insulin sensitivity. Diabetes 32:965–969