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COMMENTARY

Exercise intensity and insulin sensitivity: how low can you go?

J. A. Hawley · M. J. Gibala

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Abbreviations

$\dot{V}O_{2max}$	Maximum volume of O_2 uptake
$\dot{V}O_{2peak}$	Peak volume of O_2 uptake
SI	Insulin sensitivity index

Introduction

Readers of *Diabetologia* will be aware that the prevalence of diabetes mellitus has reached global epidemic proportions and now exerts major health consequences at both individual and public health levels [1, 2]. The majority of diabetic patients (over 90%) suffer from type 2 diabetes, a progressive metabolic disorder with a slow and insidious onset. While the true incidence of type 2 diabetes is likely to be under-reported, it has been estimated that by the year 2025, approximately 300 million people worldwide will be afflicted with this condition [3]. The proliferation of the rate of diagnosis of type 2 diabetes (and associated conditions) stems from the readiness of industrialised and developing nations alike to adopt a sedentary lifestyle in the face of excess energy intake [4–7]. While more than a half century of evidence from epidemiological, experimental and clinical trials pinpoints a positive correlation between dietary intake and disease risk, it has only recently been recognised that a physically inactive lifestyle rapidly initiates maladaptations that cause chronic disease [8–10]. Indeed, low cardiorespiratory fitness is a powerful and independent predictor of mortality in people with diabetes [11, 12], even after controlling for traditional risk factors such as age, hyperlipidaemia, smoking and hypertension.

While physical inactivity has emerged as a major risk factor for many chronic metabolic disorders [8], it is well accepted that regular physical activity (i.e. exercise training) has positive effects in terms of the prevention and treatment of almost all lifestyle-related disease states [13–16]. Of direct clinical importance is that the reductions in HbA_{1c} levels induced by exercise training are of a similar magnitude to those exerted by long-term drug or insulin therapy [17]. However, the precise intensity and volume of aerobic-based exercise needed to accrue health benefits has, in recent years, become a contentious issue, with the national health organisations of different countries failing to agree on the minimal recommended dose to prevent inactivity-related metabolic disorders [18–21]. With the recent finding that a low whole body maximal aerobic power (maximum volume of O_2 uptake [$\dot{V}O_{2max}$]) is closely associated with the coordinated downregulation of a subset of genes involved in oxidative phosphorylation in human diabetes [22], there is an urgent need for exercise physiologists to define the exercise prescription that will produce the most desired effects on targeted risk factors for preventing and/or treating patients with type 2 diabetes.

J. A. Hawley (✉)
Exercise Metabolism Group, School of Medical Sciences,
RMIT University,
Building 223.2.52, P.O. Box 71, Bundoora, VIC 3083, Australia
e-mail: john.hawley@rmit.edu.au

M. J. Gibala
Department of Kinesiology, McMaster University,
Hamilton, ON, Canada, L8S 4K1

Effect of the volume and intensity of exercise training on insulin sensitivity

It is not presently clear whether aerobic-based exercise training programmes of different intensities (in which intensity is measured as the percentage of $\dot{V}O_{2max}$, percentage of maximum heart rate [$\%HR_{max}$], percentage of heart rate reserve [$\%HR_{reserve}$] or percentage of peak power output [%PPO]) also differ in their ability to change insulin action. One of the first investigations into the effects of different exercise intensities on cardiorespiratory fitness and metabolic health was undertaken in the laboratory of J. Holloszy [23, 24]. These studies demonstrated the effect of 6 months of exercise of low to moderate intensity (walking 3–4 days/week for 30 min at ~40% of $HR_{reserve}$), immediately followed by 6 months of intense exercise (jogging 3–4 days/week for 30–45 min, at 75% of $HR_{reserve}$) on a variety of metabolic and clinical measures in older men and women (age ~63 years). After 6 months of low-intensity exercise training there were no changes in whole body insulin sensitivity (as determined by OGTT). However, after 6 months of the high-intensity programme, the insulin AUC during the OGTT was reduced by 30% [23]. Of note, while there was a modest improvement in peak O_2 uptake ($\dot{V}O_{2peak}$) after the first 6 months of training, values for this variable increased dramatically after completion of the high-intensity exercise protocol, such that, at the end of the 12 month intervention, it had risen by ~30%—an increase similar to that which may be expected in a younger, healthy population [24].

Recently, Houmard et al. [25] investigated whether vigorous-intensity exercise training would improve insulin action to a greater degree than moderate-intensity training. The participants in this study [25] comprised 154 sedentary, overweight men and women with pre-diabetes (i.e. dyslipidaemia considered as a pre-diabetic state). They were randomly assigned to either a control (no exercise) group or one of three exercise-trained groups for the study duration (6 months): (1) low-volume, moderate-intensity exercise (~20 km of jogging per week at 40–45% of $\dot{V}O_{2peak}$); (2) low-volume, high-intensity exercise (~20 km of jogging per week at 65–80% of $\dot{V}O_{2peak}$); and (3) high-volume, high-intensity exercise (32 km of jogging per week at 65–80% of $\dot{V}O_{2peak}$). Training volume was achieved by exercising for ~115 min (low-volume, high-intensity group) or ~170 min (low-volume, moderate-intensity group and high-volume, high-intensity group). Insulin action was measured during an IVGTT and reported as an insulin sensitivity index (SI). After the 6 month intervention, SI was increased significantly in all exercise-trained groups, but fell in the control group ($p<0.05$). However, exercise prescriptions that incorporated ~170 min/week improved SI significantly more than a programme of ~115 min/week, independently of exercise intensity. These workers concluded that exercise duration should be considered when designing exercise training interventions, with the intent of improving insulin sensitivity [25], and based upon an examination of the other dose–response variables, suggested that the theoretical minimal activity to maintain optimal health is about 10–20 km/week of walking or jogging (2,930–4,186 kJ) or other energy expenditure equivalent [26]. Other investigators [27, 28] have also reported that low-intensity exercise is just as effective as more intense exercise for improving insulin sensitivity in people with mild insulin resistance.

Boulé and co-workers [29] performed a meta-analysis on the effects of exercise training on cardiorespiratory fitness and metabolic heath in patients with type 2 diabetes. These workers analysed nine randomised trials that compared aerobic-based exercise programmes with control groups (overall $n=266$). Inclusion criteria included a mean exercise frequency of 3.4 sessions per week, lasting ~50 min per session, for a minimum of 20 weeks, with exercise intensities ranging from 50–75% of $\dot{V}O_{2max}$. There was a significant mean increase in $\dot{V}O_{2max}$ in exercise-trained groups (12%), with a 1% decrease in the control group ($p<0.003$) [29]. Of note, those studies that had employed higher exercise intensities tended to produce larger improvements in $\dot{V}O_{2max}$. Furthermore, exercise intensity predicted post-training improvements in HbA_{1c} level ($r=0.91$, $p=0.002$) to a larger extent than exercise volume ($r=0.46$, $p=0.26$). The findings of this meta-analysis 'provide support for encouraging individuals with type 2 diabetes who are already exercising at moderate intensity to consider increasing the intensity of their exercise because of possible additional benefits in both metabolic control and cardiorespiratory fitness'.

At this point, clinicians are likely to be confused by the apparent lack of consensus arising from the scientific literature: Is *intensity* or *volume* the most important determinant for the improved insulin action observed after exercise training? In this issue of the journal, Hansen and colleagues [30] provide evidence to suggest that, when matched for energy expenditure, low- to moderate-intensity exercise may be just as effective as moderate- to high-intensity exercise training in terms of lowering the HbA_{1c} levels of obese patients with type 2 diabetes.

The study's findings

Hansen and co-workers [30] compared the benefits of 6 months of aerobic-based, low- to moderate-intensity exercise training with those of a moderate- to high-intensity programme of the same duration. Fifty elderly (age ~60 years), obese (BMI 32 kg/m^2) male patients with type 2 diabetes commenced the study, and 37 participants completed three supervised exercise sessions per week of either 55 min at 50% of $\dot{V}O_{2peak}$ (low to moderate intensity) or 40 min at 75% of $\dot{V}O_{2peak}$ (moderate to high intensity). Each exercise session consisted of walking, cycling and indoor cross-country ski-type exercise and was adjusted (increased) after the first 2 months of training according to the target heart rate, which was reassessed at that time. Total energy expenditure during exercise training sessions was matched between groups. Before and after the intervention, a variety of blood, muscle and whole body measures were determined, including HbA_{1c} content, plasma glucose, insulin and lipid concentrations, muscle oxidative enzyme capacity, body composition and maximal work capacity.

The results showed that the HbA_{1c} level declined (from 7.2% to 6.9%, $p<0.05$) following exercise training, but there was no interaction between exercise intensity and the reduction in HbA_{1c}. Plasma LDL-cholesterol concentrations also dropped following training, but there were no differences between the two groups. Furthermore, muscle measures (selected enzymes involved in oxidative metabolism and fibre composition) were not different between the two exercise groups. In contrast, after 2 months of training $\dot{V}O_{2peak}$ increased to a greater extent after medium- to high-intensity exercise than after low- to medium-intensity training (16% vs 9%, $p<0.05$), while the decline in trunk fat mass was also larger after the more intense exercise protocol (2.0 vs 1.0 kg respectively, $p<0.05$) at the completion of the study. The authors suggested that 'prolonged continuous low-to-moderate-intensity exercise training is equally effective when compared with more intense exercise training as a means to lower blood HbA_{1c} content, when exercise bouts are being matched for total energy expenditure.'

As noted previously, the issue of exercise intensity and disease risk remains contentious, and some authors have reported that moderate-intensity exercise training in the absence of dietary changes improves risk factors for metabolic syndrome to a greater extent than more vigorous exercise [31]. Other data, however, suggest that when matched for total work or energy expenditure, intermittent high-intensity aerobic interval training is superior to less intense exercise in inducing improvements in clinical outcomes and muscle metabolic markers. Tjønna et al. [32] studied patients with the metabolic syndrome (age ~52 years, BMI ~30 kg/m^2) who either performed a 16 week exercise programme consisting of continuous moderate-intensity exercise or aerobic interval training, or were assigned to a control group. Participants who performed the interval training warmed up for 10 min at 70% of HR_{max}, before undertaking four 4 min work bouts at 90% of HR_{max} with a 3 min active recovery at 70% of HR_{max}, followed by a 5 min cool-down period (a total exercise time of 40 min). To match training volume and energy expenditure, the moderate-intensity group exercised for 47 min at 70% of HR_{max} during each session. Both training groups exercised on three occasions per week. Compared with the continuous training protocol, the higher intensity interval training regimen induced superior responses for a number of whole body and muscle measures, including insulin sensitivity (determined from the HOMA index), $\dot{V}O_{2max}$, skeletal muscle markers of mitochondrial biogenesis and insulin signalling, as well as endothelial function. This same group has also reported that intense interval training is superior to continuous moderate intensity training for inducing specific health-related benefits in obese individuals [33] and patients with heart failure [34], and the general conclusion given by the authors is that high-intensity exercise training programmes may yield more favourable results than programmes employing low- to moderate-intensity exercise.

Hansen et al. [30] raise the important issue of psychosocial determinants of behaviour, citing work showing that moderate, as opposed to high-intensity exercise is associated with greater patient adherence to a training programme [35]. While there are equivocal data regarding the influence of exercise intensity on adherence/compliance [36], it should be noted that the overwhelming barrier to regular exercise participation is lack of time [37]. This finding is ubiquitous; regardless of age, ethnicity, sex or health status, individuals report that a lack of time is the primary reason for their failure to exercise on a regular basis [38, 39]. Given that time constraints are cited as a common barrier to exercise training, innovations in exercise prescription that induce clinically meaningful health benefits despite a minimal time commitment represent a valuable strategy to encourage physical activity participation and reduce the economic burden and numerous pathologies associated with an inactive lifestyle [40].

In this regard, a growing body of evidence suggests that many of the adaptations normally associated with traditional endurance training can be induced more rapidly than was previously thought with a surprisingly small volume of high-intensity interval training (for review see [41]). Recent work in this area has examined metabolic, cardiovascular and performance adaptations in participants who performed high-intensity, short-duration interval training using the Wingate Test, a 30 s maximal cycling bout that leads to high levels of muscular fatigue. A typical training session consists of four to six 'all-out' sprints lasting 30 s separated by 4 min of recovery (2–3 min of very intense exercise per training session and a total time investment of ~20 min), undertaken three times a week for a 2–6 week period. After only six sessions (2 weeks) of this exercise protocol, there are marked increases (15–35%) in muscle oxidative capacity, measured as the maximal activity or protein content of mitochondrial enzymes such as citrate synthase and cytochrome oxidase [42, 43]. Canadian researchers recently compared the efficacy of two widely divergent (in terms of exercise intensity, volume and energy expenditure) protocols: a low-volume interval training regimen (four to six sprints of 30 s duration each session) performed 3 days/week, or a 40–60 min continuous cycling protocol (~65% $\dot{V}O_{2peak}$) performed 5 days/week [44, 45]. Despite the lower weekly training time (~1.5 vs ~4.5 h) and a 90% lower exercise training volume in the high-intensity interval-trained cohort, both groups showed similar increases in markers of mitochondrial capacity [44] and vascular structure and function [45]. In agreement with these findings, Babraj et al. [46] have recently reported that 2 weeks of high-intensity training (comprising four to six cycle sprints of 30 s duration at each session, a total session length of 15 min and six sessions in total) improved insulin sensitivity by 23% in young, healthy men. These workers concluded: 'This novel time-efficient training paradigm can be used as a strategy to reduce metabolic risk factors in young and middle aged sedentary populations who otherwise would not adhere to time consuming traditional aerobic exercise regimes.' Whether these results translate into similar improvements in glucose control in patient populations remains to be determined. Indeed, studies of the efficacy of high-intensity, short-duration interval training in people with insulin resistance and type 2 diabetes are urgently needed to address this question. A large proportion of patients with type 2 diabetes suffer from comorbidities, such as osteoarthrosis, cardiac problems and other disease states, that confer a low tolerance for exercise of any sort. Accordingly, increasing the level of physical activity for the majority of patients with diabetes is likely to be problematic, regardless of intensity!

So, how low can you go?

It remains to be determined whether high-intensity, low-volume interval training protocols can confer all of the health-related benefits associated with less intense, more prolonged traditional endurance training programmes. Studies to date that have used intense exercise regimens have been of a relatively short duration, and it needs to be clearly established whether the early adaptation profile induced by such regimens is similar to less intense programmes when training is maintained over a longer period (e.g. several months to several years). It is possible that the time course for selected physiological adaptations differs between training protocols; the very intense nature of interval training may stimulate an early response and rapid changes to muscle metabolism, whereas the adaptations induced by traditional endurance training may occur more slowly, and possibly promote greater clinical improvements. While high-intensity training models are time-efficient, they do require extremely high levels of participant motivation. Given the extreme nature of the exercise, it is unlikely that such protocols could be safely adopted at the population level. Clearly, a comprehensive evaluation of the physiological adaptations induced by different interval training strategies in a wide range of diseased and healthy populations will ultimately permit evidence-based recommendations that may provide an alternative to current exercise prescriptions for time-challenged individuals. In the meantime, it seems prudent to recommend that, for patients with insulin resistance or type 2 diabetes, the minimal dose of physical activity needed to maintain or improve health is equivalent to ~4,000 kJ/week of low- to moderate-intensity exercise. However, for patients who only show modest improvements in clinical and metabolic outcomes at this level of activity-induced energy expenditure, an increase in the intensity of exercise may be considered because of the potential additional benefits in both metabolic control and cardiorespiratory fitness.

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References

1. Jönsson B (2002) Revealing the cost of type II diabetes in Europe. Diabetologia 45:S5–S12
2. Zimmet P, Alberti KG, Shaw J (2001) Global and societal implications of the diabetes epidemic. Nature 414:782–787
3. King H, Aubert RE, Herman WH (1998) Global burden of diabetes, 1995–2025: prevalence, numerical estimates, and projections. Diabetes Care 21:1414–1431
4. Dwyer T, Magnussen CG, Schmidt MD et al (2009) Decline in physical fitness from childhood to adulthood associated with increased obesity and insulin resistance in adults. Diabetes Care 32:683–7
5. Healy GN, Wijndaele K, Dunstan DW et al (2008) Objectively measured sedentary time, physical activity, and metabolic risk: the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Diabetes Care 31:369–371
6. McNaughton SA, Dunstan DW, Ball K, Shaw J, Crawford D (2009) Dietary quality is associated with diabetes and cardio-metabolic risk factors. J Nutr 139:734–42
7. Misra A, Khurana L (2008) Obesity and the metabolic syndrome in developing countries. J Clin Endocrinol Metab 93:S9–S30
8. Booth FW, Gordon SE, Carlson CJ, Hamilton MT (2000) Waging war on modern chronic diseases: primary prevention through exercise biology. J Appl Physiol 88:774–787
9. Booth FW, Laye MJ, Lees SJ, Rector RS, Thyfault JP (2008) Reduced physical activity and risk of chronic disease: the biology behind the consequences. Eur J Appl Physiol 102:381–390

10. Olsen RH, Krogh-Madsen R, Thomsen C, Booth FW, Pedersen BK (2008) Metabolic responses to reduced daily steps in healthy nonexercising men. JAMA 19:1261–1263
11. Kohl HW, Gordon NF, Villegas JA, Blair SN (1992) Cardiorespiratory fitness, glycemic status, and mortality risk in men. Diabetes Care 15:184–192
12. Wei M, Gibbons LW, Kampert JB, Nichaman MZ, Blair SN (2000) Low cardiorespiratory fitness and physical inactivity as predictors of mortality in men with type 2 diabetes. Ann Intern Med 132:605–611
13. Goodyear LJ, Kahn BB (1998) Exercise, glucose transport, and insulin sensitivity. Annu Rev Med 49:235–261
14. Hawley JA (2004) Exercise as a therapeutic intervention for the prevention and treatment of insulin resistance. Diabetes Metab Res Rev 20:383–393
15. Hawley JA, Lessard SJ (2008) Exercise training-induced improvements in insulin action. Acta Physiol (Oxf) 192:127–135
16. Pedersen BK, Saltin B (2006) Evidence for prescribing exercise as therapy in chronic disease. Scand J Med Sci Sports 16:3–63
17. Snowling NJ, Hopkins WG (2006) Effects of different modes of exercise training on glucose control and risk factors for complications in type 2 diabetic patients: a meta-analysis. Diabetes Care 29:2518–2527
18. American College of Sports Medicine Position (1998) American College of Sports Medicine Position Stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness and flexibility in healthy adults. Med Sci Sports Exerc 30:975–991
19. Centers for Disease Control and Prevention (1996) Physical activity and health: report of the Surgeon General (S/N 017-023-00196-5). Centers for Disease Control and Prevention, US Department of Health and Human Services, Atlanta
20. National Institutes of Health Consensus Panel (1995) Physical activity and cardiovascular health. NIH Consens Statement 13:1–33
21. Pate RR, Pratt M, Blair SN et al (1995) Physical activity and public health. A recommendation from the Centers for Disease Control and Prevention and the American College of Sports Medicine. JAMA 273:402–407
22. Mootha VK, Lindgren CM, Eriksson KF et al (2003) PGC-1α-responsive genes involved in oxidative phosphorylation are coordinately downregulated in human diabetes. Nat Genet 34:267–273
23. Seals DR, Hagberg JM, Hurley BF, Ehsani AA, Holloszy JO (1984) Effects of endurance training on glucose tolerance and plasma lipid levels in older men and women. JAMA 252:645–649
24. Seals DR, Hagberg JM, Hurley BF, Ehsani AA, Holloszy JO (1984) Endurance training in older men and women. I. Cardiovascular responses to exercise. J Appl Physiol 57:1024–1029
25. Houmard JA, Tanner CJ, Slentz CA, Duscha BD, McCartney JS, Kraus WE (2004) Effect of the volume and intensity of exercise training on insulin sensitivity. J Appl Physiol 96:101–106
26. Slentz CA, Houmard JA, Kraus WE (2007) Modest exercise prevents the progressive disease associated with physical inactivity. Exerc Sport Sci Rev 35:18–23
27. Braun B, Zimmermann MB, Kretchmer N (1995) Effects of exercise intensity on insulin sensitivity in women with non-insulin-dependent diabetes mellitus. J Appl Physiol 78:300–306
28. Kang J, Robertson RJ, Hagberg JM et al (1996) Effect of exercise intensity on glucose and insulin metabolism in obese individuals and obese NIDDM patients. Diabetes Care 19:341–349
29. Boulé NG, Kenny GP, Haddad E, Wells GA, Sigal RJ (2003) Meta-analysis of the effect of structured exercise training on cardiorespiratory fitness in type 2 diabetes mellitus. Diabetologia. 46:1071–1081
30. Hansen D, Dendale P, Jonkers RAM et al (2009) Continuous low- to moderate-intensity exercise is equally as effective as moderate- to high-intensity exercise training to lower blood HbA_{1c} content in obese, type 2 diabetes patients. Diabetologia. doi:10.1007/s00125-009-1354-3
31. Johnson JL, Slentz CA, Houmard JA et al (2007) Exercise training amount and intensity effects on metabolic syndrome (from Studies of a Targeted Risk Reduction Intervention through Defined Exercise). Am J Cardiol 100:1759–1766
32. Tjønna AE, Lee SJ, Rognmo Ø et al (2008) Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome: a pilot study. Circulation 118:346–354
33. Schjerve IE, Tyldum GA, Tjønna AE et al (2008) Both aerobic endurance and strength training programmes improve cardiovascular health in obese adults. Clin Sci (London) 115:283–293
34. Wisløff U, Støylen A, Loennechen JP et al (2007) Superior cardiovascular effect of aerobic interval training versus moderate continuous training in heart failure patients: a randomized study. Circulation 115:3086–3094
35. Perri MG, Anton SD, Durning PE et al (2002) Adherence to exercise prescriptions: effects of prescribing moderate versus higher levels of intensity and frequency. Health Psychol 21:452–458
36. King AC, Haskell WL, Young DR, Oka RK, Stefanick ML (1995) Long-term effects of varying intensities and formats of physical activity on participation rates, fitness, and lipoproteins in men and women aged 50 to 65 years. Circulation 91:2596–2604
37. Dishman RK, Sallis JF, Orenstein DR (1985) The determinants of physical activity and exercise. Public Health Rep 100:158–171
38. Godin G, Desharnais R, Valois P, Lepage P, Jobin J, Bradet R (1994) Differences in perceived barriers to exercise between high and low intenders: observations among different populations. Am J Health Promot 8:279–285
39. Trost SG, Owen N, Bauman AE, Sallis JF, Brown W (2002) Correlates of adults' participation in physical activity: review and update. Med Sci Sports Exerc 34:1996–2001
40. Gibala MJ (2007) High-intensity interval training: a time-efficient strategy for health promotion? Curr Sports Med Rep 6:211–213
41. Gibala MJ, McGee S (2008) Metabolic adaptations to short-term high-intensity interval training: a little pain for a lot of gain? Exerc Sports Sci Rev 36:58–63
42. Burgomaster KA, Hughes SC, Heigenhauser GJF, Bradwell SN, Gibala MJ (2005) Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity. J Appl Physiol 98:1895–1990
43. Gibala MJ, Little JP, van Essen M et al (2006) Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. J Physiol 575:901–911
44. Burgomaster KA, Howarth KR, Rakobowchuk M, Phillips SM, MacDonald MJ, McGee S, Gibala MJ (2008) Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. J Physiol 586:151–160
45. Rakobowchuk M, Tanguay S, Burgomaster KA, Howarth KR, Gibala MJ, MacDonald MJ (2008) Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow mediated dilation in healthy humans. Am J Physiol Regul Integr Comp Physiol 295:R236–R242
46. Babraj JA, Vollaard NB, Keast C, Guppy FM, Cottrell G, Timmons JA (2009) Extremely short duration high intensity interval training substantially improves insulin action in young healthy males. BMC Endocr Disord 9:3