Similar magnitude of post-exercise hyperglycemia despite manipulating resistance exercise intensity in type 1 diabetes individuals

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The aim of this study was to compare the glycemic and glucoregulatory hormone responses to low- and moderate-intensity morning resistance exercise (RE) sessions in type 1 diabetes (T1DM). Following maximal strength assessments (1RM), eight T1DM (HbA1c:72 ± 12 mmol/mol, age:34 ± 7 years, body mass index:25.7 ± 1.6 kg/m²) participants attended the research facility on two separate occasions, having fasted and taken their usual basal insulin but omitting rapid-acting insulin. Participants performed six exercises for two sets of 20 repetitions at 30%1RM during one session [low-intensity RE session (LOW)] and two sets of 10 repetitions at 60%1RM during another session [moderate-intensity RE session (MOD)], followed by 65-min recovery. Sessions were matched for total mass lifted (kg). Venous blood samples were taken before and after exercise. Data (mean ± SEM) were analyzed using analysis of variance (P ≤ 0.05). There were no hypoglycemic occurrences throughout the study. Blood glucose rose similarly between sessions during exercise (P = 0.382), remaining comparable between sessions throughout recovery (P > 0.05). There was no effect of RE intensity on metabolic acidosis (P > 0.05) or peak growth hormone responses (P = 0.644), but a tendency for greater catecholamine responses under LOW (individualized peak concentrations: adrenaline MOD 0.55 ± 0.13 vs LOW 1.04 ± 0.37 nmol/L, P = 0.155; noradrenaline MOD 4.59 ± 0.86 vs LOW 7.11 ± 1.82 nmol/L, P = 0.082). The magnitude of post-exercise hyperglycemia does not differ between equal volume low and moderate intensity RE sessions performed in the morning.

Regular performance of resistance exercise (RE), or weights training, is advocated to individuals with diabetes (American College of Sports Medicine, 2000; Colberg et al., 2010; Centers for Disease Control and Prevention Web Site, 2014), and research suggests that this could have a beneficial effect on health in people with both type 2 (Tresierras & Balady, 2009; Colberg et al., 2010) and type 1 diabetes (T1DM) (Durak et al., 1990; Ramalho et al., 2006). Prescription guidelines for RE are tailored to individual physical ability and/or fitness goals (Garber et al., 2011). For example, heavy loads (i.e., high intensity; 70 to ≥ 80%1RM) paired with a moderate/high number of repetitions (8–15 lifts) and multiple sets (2–4 sets) are undertaken when the aim is to elicit muscular hypertrophy. In contrast, light to moderate loads (i.e., low intensity; < 50%1RM) coupled with multiple high-repetition (15–20 lifts) sets (≤ 2 sets) is aimed towards training muscular endurance. Such an exercise session is best suited to novice and/or previously sedentary individuals or those with certain diabetic related complications or weight-bearing abilities (American College of Sports Medicine, 2000; Garber et al., 2011).

The effects of RE on blood glucose (BG) in T1DM are scant and responses vary between studies; earlier research demonstrates a net increase (Turner et al., 2015) or decrease (Yardley et al., 2013), or no change in BG (Yardley et al., 2012; Turner et al., 2015) in response to a single session of RE. It is unclear how specific RE session characteristics such as the load, volume, work to rest interval, contraction velocity (or pacing), etc., might affect glycemia in T1DM. Yet such knowledge is likely to facilitate the development of better glucose management routines for exercising T1DM individuals and ultimately favor the preclusion of exercise-induced glycemic disturbances – a primary cause of low exercise

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participation and adherence rates in T1DM (Brazeau et al., 2008).

In a recent study, our group (Turner et al., 2015) demonstrated that the total mass lifted during a RE session (i.e., exercise volume) influenced the post-exercise BG profile across time, with one and two sets of RE increasing BG but with the addition of a third set post-exercise BG was returned to values similar to that of a resting control trial. So, 30 min of RE performed at – 70% one-repetition maximum elicits a hyperglycemic excursion for up to 1 h after exercise (Turner et al., 2015). However, despite clinical recommendations for people with T1DM to conduct RE, the occurrence of post-exercise hyperglycemia could detract from the host of possible health benefits gained through regular RE training because hyperglycemic per se could contribute to a worsening of glycemic control. Somewhat anecdotally, the administration of a small bolus of insulin can resolve the occurrence of exercise-induced hyperglycemia, although in clinical practice this strategy often increases the likelihood of hypoglycemia. Another approach is to examine the influence of manipulating exercise characteristics. While it has been shown that performance of moderate intensity aerobic exercise lowers BG (Rabasa-Lhoret et al., 2001), high-intensity or sprint exercise results in a significant counter-regulatory hormone response that attenuates the decline in glycemia during aerobic exercise (Guelfi et al., 2005; Bussau et al., 2006) and can in fact increases BG concentrations (Marliss & Vranic, 2002; Harmer et al., 2006; Fahey et al., 2012). Thus, the intensity of RE (i.e., the amount of mass lifted per repetition relative to maximal exertion) might play a role in explaining the magnitude of post-RE hyperglycemia in T1DM. Such knowledge is important to both developing strategies to improve glycemic stability during and after RE and accurately prescribing RE in this clinical cohort. Therefore, the aim of this study was to compare the acute glycemic, metabolic, and glucoregulatory responses to tightly controlled moderate and low intensity morning RE sessions matched for total mass lifted in T1DM individuals.

Research design and methods
Participants
Following UK Health Service Research Ethics Committee approval (Ref. 12/WA/0049), eight individuals with T1DM [6 male; 2 female, HbA1C 8.7 ± 1.1%/72 ± 12 mmol/mol, age 34 ± 7 years, body mass index (BMI) 25.7 ± 1.6 kg/m², body fat 23.0 ± 2.7%, T1DM duration 18 ± 5 years] volunteered and provided written informed consent for the study. Participants were free from any diabetes complications including hypoglycemia unawareness (Clarke et al., 1995) and were treated with a stable basal bolus insulin regimen composed of insulin glargine or detemir and insulin aspart for a 3 month before, but omitted rapid-acting insulin on the morning of testing. Participants reported food intake, insulin dosage, BG measurements, and levels of physical activity for 24 h preceding the session. Dietary intake (MOD 2358 ± 72 vs LOW 2368 ± 139 calories, P = 0.488) and insulin dosage (basal insulin: MOD 31 ± 4 vs LOW 31 ± 4 IU; bolus insulin: MOD 29 ± 3 vs LOW 29 ± 3 IU, P > 0.05) during the 24 h prior to exercise were replicated between experimental sessions of different exercise intensity. After a standardized 10-min warm-up of the main muscle groups, participants undertook one of two RE sessions followed by a 65 min period of passive recovery.

RE protocol
RE sessions were performed on the multi-gym Smith machine. Both exercise sessions involved six exercises performed at either a moderate-intensity (two sets of 10 repetitions at 60%1RM) (MOD) or a low-intensity session (two sets of 20 repetitions at 30%1RM) (LOW). Sets were completed in a circuit-based fashion, namely exercises were undertaken in the previously stated order (see Preliminary Testing) and completion of 10 (MOD) or 20 (LOW) repetitions of each six exercises marked the end of one set. In both protocols, successive exercises and sets were interspersed by 2 min of passive rest. Exercise repetitions were performed to a metronome at a pace of 2 s per concentric phase followed by a 2-s eccentric phase. The total session duration (including rest intervals) was 30 and 38 min for the MOD and LOW sessions, respectively. The total exercise time was 8 and 16 min for the
MOD and LOW sessions, respectively. The total mass lifted (i.e., exercise volume) was equivalent between sessions.

Blood sampling and analysis
From an antecubital vein, blood was sampled at rest, and 0, 5, 20, 35, and 65 min (recovery phase) after cessation of exercise. Blood was obtained via a 1-mL LH (lithium heparin) syringe (RAPIDlyte, Siemens AG, Munich, Germany) and used to determine HbA1C (on one occasion at baseline; Roche Cobas Integra 800 analyzer, Roche Diagnostics Corp., Indianapolis, Indiana, USA), and BG, pH, lactate, and extra cellular fluid base excess (Becf) on a metabolic analyzer (GEM Premier 3000, Instrumentation Laboratories, Warrington, UK). Blood was drawn into a 10-mL syringe (BD Luer-Lok tip®, BD, Oxford, UK) and immediately decanted into lithium heparinized vacutainer tubes (BD Vacutainer®, BD), then centrifuged at 2.4 RCF for 5 min (BOECO Centrifuge S-8, Boeckel+Co., Hamburg, Germany). Plasma aliquots were pipetted into 1.5-mL microcentrifuge tubes and stored at −80 °C for later determination of catecholamines (ELISA, Eagle Biosciences Inc., Nashua, New Hampshire, USA), growth hormone (GH), interleukin-6 (IL-6), cortisol (ELISA; RnD Systems, Minneapolis, USA), and insulin (Invitron, Monmouth, UK). Thresholds for hypoglycemia and hyperglycemia were ≤ 3.5 and > 10.9 mmol/L, respectively (Yardley et al., 2013).

Statistical analysis
Data are reported as mean ± SEM. Total exercise volume (or mass lifted, in kg) was calculated by multiplying the mass lifted during each repetition by the number of repetitions completed over the duration of the exercise session. Net incremental BG area under the curve (BGIAUC) was determined using the trapezoidal integrative method (Gannon et al., 1989). Ratings of perceived exertion during RE were reported on the OMNI-RE scale (Robertson et al., 2003). Statistical analysis was performed using PASW Statistics software (IBM PASW version 18, IBM, New York, New York, USA), with significance set at P ≤ 0.05. Data were analyzed using repeated-measures analysis of variance on two factors (session×time), with Fisher’s Least Significant Difference (LSD) pairwise comparisons used to examine within-session changes from baseline. Where a significant effect of session was found, paired samples t-tests were used to perform pairwise post hoc comparison between experimental sessions for each time point.

Results
Performance
Total mass lifted during RE was similar between sessions (volume: MOD 3675 ± 651 vs LOW 3725 ± 674 kg, P = 0.124). Intensity was twofold greater under MOD than LOW (load: 59 ± 1%1RM, P < 0.001), meaning mass lifted per minute was significantly greater under MOD than LOW (MOD 459 ± 81 vs LOW 232 ± 42 kg/min, P = 0.027).

BG
The BG responses to exercise are presented in Fig. 1(a). Pre-exercise fasting BG concentrations were similar between sessions (MOD 11.2 ± 1.3 vs LOW 11.2 ± 1.2 mmol/L, P = 0.995). For absolute BG responses, there was no session × time interaction (P = 0.393) or effect of experimental session (P = 0.768), but there was a significant effect of time (P = 0.041). BG rose to similar concentrations immediately after exercise (MOD +1.5 ± 0.8 vs LOW +2.2 ± 0.9 mmol/L, P = 0.382). During recovery, BGIAUC was similar between experimental sessions (MOD 109.8 ± 55.8 vs LOW 171.8 ± 69.4 mmol/65 min/L, P = 0.304). From an observational perspective, after 65 min of recovery from exercise, a similar number of participants were observed to experience a ≥ 2 mmol/L

Resistance exercise intensity in type 1 diabetes

Fig. 1. Blood glucose responses to MOD (diamonds) and LOW (squares) sessions. Data presented are means ± SEM. Transparent sample points indicate significant changes from rest within each session (P < 0.05).
rise from pre-exercise BG concentrations under LOW ($n=5$) and MOD ($n=6$), and two (LOW) or one (MOD) participants experienced exercise-induced BG excursions of $\geq 4$ mmol/L from pre-exercise. There were no occasions of hypoglycemia (BG $\leq 3.5$ mmol/L) experienced by any participant under either experimental session.

**Glucoregulatory hormones**

There were no significant effects of time ($P=0.294$) or session ($P=0.412$) for plasma insulin concentrations (Table 1). For plasma adrenaline (AD), noradrenaline (NA) and GH responses during recovery (see Table 1), there were significant effects of time ($P > 0.05$) but no effect of session ($P > 0.05$) or session $\times$ time interactions ($P > 0.05$). Individualized peak concentrations of AD (MOD $0.55 \pm 0.13$ vs LOW $1.04 \pm 0.37$ nmol/L, $P=0.155$), NA (MOD $4.59 \pm 0.86$ vs LOW $7.11 \pm 1.82$ mmol/L, $P=0.082$) tended to be greatest under LOW, whereas GH concentrations (MOD $3.52 \pm 0.80$ vs LOW $3.66 \pm 0.93$ ng/mL, $P=0.644$) were similar between sessions. There was a significant session $\times$ time interaction ($P=0.016$) and effect of time ($P=0.031$) for cortisol responses (Table 1), with lower cortisol concentration observed under MOD at the end of recovery ($P < 0.05$). For plasma IL-6 (Table 1), there were no significant effects of time ($P=0.750$) or session ($P=0.217$), with similar individualized peak concentrations between sessions (MOD $2.3 \pm 0.6$ vs LOW $3.0 \pm 1.0$ pg/mL, $P=0.195$).

**Physiological and perceptual strain**

No significant differences were observed in blood lactate, pH and extra cellular fluid base excess ($B_{ec}$) responses to exercise (see Fig. 2(a)). Individualized peak blood lactate concentrations (MOD $9.8 \pm 2.4$ vs LOW $10.6 \pm 1.9$ mmol/L, $P=0.381$) and nadir blood pH (occurring immediately after RE) (MOD $7.28 \pm 0.02$ vs LOW $7.28 \pm 0.02$, $P=0.946$) were similar between sessions. Following completion of RE, perceptual ratings were similar between sessions (MOD $7 \pm 1$ vs LOW $8 \pm 1$, $P=0.269$), meaning that participants perceived exertion during RE ranged from “somewhat hard to hard.”

**Discussion**

The aim of this study was to compare the acute glycemic, metabolic, and glucoregulatory responses to moderate- and low-intensity morning RE sessions of equal volume in T1DM individuals. The results from this study are the first to demonstrate that performing a low-intensity RE session does not attenuate the magnitude of post-exercise hyperglycemia compared with that of moderate-intensity RE session matched for total mass lifted.

Participants commenced both exercise sessions mildly hyperglycemic ($\sim 11$ mmol/L) and avoided hypoglycemia during and also throughout recovery, with BG rising to similar concentrations of $13.4 \pm 1.8$ and $12.7 \pm 1.5$ mmol/L during low and moderate intensity RE, respectively. During 1 h of recovery from the low and moderate intensity RE sessions, BG climbed to similar concentrations of $3.1 \pm 1.1$ and $2.0 \pm 0.9$ mmol/L greater than baseline (pre-exercise) values, respectively, noting that (a) participants replicated the same pre-exercise diet and insulin adjustments across the experimental sessions; (b) circulating insulin concentrations were similar before performing RE; and (c) insulin levels remained comparable between sessions throughout recovery. Interestingly, previous work in our laboratory demonstrated that when T1DM participants implemented the same pre-exercise glucose management routine as in the present study, insulin glargine levels remained comparable with baseline levels during 1 h
after 15, 30, and 45 min of RE at an intensity of ∼ 70% 1RM, with the avoidance of hypoglycemia during and soon after exercise (Turner et al., 2015). The binding of catecholamines to β-adrenoceptors augments hepatic glycogenolysis and inhibits glucose uptake (Howlett et al., 1999; Watt & Hargreaves, 2002). The resultant greater increment in glucose production over uptake is a major factor in the development of post-exercise hyperglycemia in T1DM individuals (Sigal et al., 1999). The low-intensity RE session elicited a threefold and fourfold increase in AD and NAD concentrations, respectively, whereas the moderate intensity RE session produced a twofold increase in AD and threefold increase in NAD. These significant increases in catecholamines reflect increased sympathoadrenal medullary activity, which was likely a component in the occurrence of post-exercise hyperglycemia (Sigal et al., 1999; Harmer et al., 2006). The tendency for greater sympathetic activity in response to low- over moderate-intensity RE without significant differences in the magnitude of post-exercise hyperglycemia highlights the complex relationship between RE characteristics and exercise-induced changes in glycemic regulation.

The magnitude of the GH response was similar between different intensity RE sessions, with two to threefold rise in baseline concentrations appearing after exercise. Considering that a rise in portal vein GH concentration has been associated with increased rates of

Fig. 2. (a) Blood lactate (b) blood pH and (c) extracellular fluid base excess responses to MOD (diamonds) and LOW (squares) sessions. Data presented are means ± SEM. Transparent sample points indicate significant changes from rest within each session ($P < 0.05$). The asterisk (*) indicates a statistically significant difference ($P < 0.05$) between MOD and LOW.
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hepatic glycogenolysis (Yuen et al., 2013), while venous GH infusion can directly impair glucose uptake (Fowelin et al., 1995; Yuen et al., 2013), the marked exercise-induced appearance of GH could have contributed to a rise in BG in response to exercise. Considering that the exercise-induced rise in GH was similar between RE sessions but there was a tendency for greater catecholamine responses to low than moderate intensity RE, it is unexpected that the magnitude of post-exercise glycemia was unaffected by adjusting the intensity of exercise. Notably, it is possible that hyperglycemia itself might have suppressed the appearance of GH in response to exercise as evidenced in a previous study by Jenni et al. (2010).

Indeed, GH can inhibit the contraction of free fatty acids to hepatic glucose production (Yuen et al., 2013) whereas AD has the opposite effect and can also suppress the appearance of GH (Howlett et al., 1999). It cannot be determined from this study what interactions might have occurred between these counter-regulatory hormones in response to exercise. It is also unclear why the time course changes in GH during recovery were different between LOW and MOD sessions (i.e., prolonged increase in GH values under MOD, yet later peak concentrations during recovery under LOW; Table 1). Temporal changes in GH after exercise have been shown to indicate differences in oxygen utilization during exercise (VanHelder et al., 1987). Interestingly, in those without diabetes, 12 times as much energy (kcal) is required to perform one repetition at 80%1RM when compared with one repetition at 20%1RM, despite a fourfold increase in mechanical work (Hunter et al., 1988). Furthermore, rates of muscle glycogenolysis are raised by increasing the intensity of RE relative to repetition maximum (Robergs et al., 1991). Together, these findings suggest that altering the intensity of RE could affect fuel utilization during and after RE.

IL-6 might play a role in the balance between glucose uptake and production (Fischer, 2006); in those without diabetes, IL-6 infusion stimulates insulin-independent glucose uptake possibly via enhancing GLUT4 expression and activation of AMP-activated kinase in skeletal muscle (Carey et al., 2006), and IL-6 has also been evidenced to increase endogenous glucose production (Stouthard et al., 1995). Our findings demonstrate that neither of our RE sessions increased the appearance of this myokine (Table 1), which is perplexing because previous findings from our lab demonstrate increased appearance of IL-6 in T1DM following RE (Turner et al., 2014). Hyperglycemia has been shown to attenuate the IL-6 response in T1DM to cycling exercise (Jenni et al., 2010). Yet our previous work demonstrated significant increases in plasma IL-6 in fasted, moderately hyperglycemic (∼11 mmol/L) T1DM participants with reasonable glycemic control, at 60 min after greater volume (>5713 to >8286 kg) and higher intensity sessions of RE (70%1RM) than the present study (Turner et al., 2014). Mass lifted and intensity of exercise could provide a clue as to why we observed no change in IL-6 appearance in the present study because a dose-dependent relationship between total mass lifted during a RE session and IL-6 appearance has been demonstrated elsewhere (Fischer, 2006; Phillips et al., 2010), with post-exercise IL-6 values of 5.2–7.4 pg/mL observed after participants lifted more than fourfold greater mass (i.e., 13 160–17 729 kg) than the present study at intensities of 65% to 85%1RM (Phillips et al., 2010). Interestingly, with increased IL-6 concentrations, significant improvements in insulin sensitivity were also observed under higher volume RE sessions (Phillips et al., 2010). Thus, it is possible that pre-exercise hyperglycemia suppressed the IL-6 response to RE, but more likely that the exercise volume in this study was insufficient to stimulate IL-6 production. The findings from this study suggest that IL-6 did not contribute to any exercise-induced change in BG or alteration in glucocorticoid activity (Tsigos et al., 1997).

The decline in resting cortisol concentrations following RE, irrespective of exercise intensity, reduces the possibility that post-exercise changes in BG were related to this glucoregulatory hormone. These findings are in line with our previous study in T1DM in which cortisol concentrations remained similar to baseline values during 1 h after performance of one, two, and three sets of RE at ~70%1RM (Turner et al., 2015). Resting hyperglycemia (Haff et al., 2003) and/or usual diurnal patterns in circadian rhythm (Kanaley et al., 2001) offer plausible reasons as to why cortisol might not have increased in response to morning RE in our cohort. It is a limitation that cortisol concentrations were not measured outside of exercising days. We speculate that the differences in the magnitude of decline in plasma cortisol during recovery between our RE session was likely attributed to daily changes in circadian decline (Kanaley et al., 2001) as, although sessions were all performed in the morning, session days were not standardized.

There was a trend for the greater appearance of catecholamines after LOW over MOD RE exercise. Quite possibly, if the sample size (n = 8) in this study was larger there might have been a significant difference in catecholamine responses between sessions (P < 0.05), that is, in the case of a type 2 statistical error. Although not statistically significant, it is interesting to explore why catecholamine hormone responses to RE were slightly greater under LOW than MOD. A possible reason for this response was a subtle difference in exercise session design. For instance, it is important to recognize that the following RE characteristics were fixed across all sessions: (a) rest intervals between exercises and sets (120 s); (b) the duration of each repetition (4 s); and (c) the total mass lifted (∼3600–3700 kg). This meant that a further 8 min of accumulative
exercise time was performed during LOW than MOD. Therefore participants had half the amount of rest to time spent exercising during the LOW RE session (i.e., MOD: 3 s rest for every 1 s of exercise vs LOW: 1.5 s rest for every 1 s of exercise) despite that participants lifted double the amount of mass per minute during MOD when compared with LOW. It was somewhat counterintuitive to observe a tendency for a greater catecholamine hormone response to low- over moderate-intensity RE because these adrenal hormones share a strong relationship with glucose regulation and post-exercise BG concentrations remained statistically similar between different RE sessions. One theory for the slightly greater increased catecholamine concentrations under LOW is that the appearance of circulatory catecholamines trends linearly with fixed intensity and increasing duration exercise (Galbo et al., 1975), and free plasma catecholamines significantly diminish within 2–3 min of secretion (Goldstein et al., 2003). Considering these findings, there was most likely a slower rate but protraction of catecholamine production during LOW over MOD, which could be attributed to the longer accumulative exercise time, coupled with short (2 min) but similar rest intervals to MOD. Further research is required to determine the impact of altering the exercise to rest interval during RE on glycemia in T1DM because it could be possible that increasing the rest interval might help prevent exercise-induced hyperglycemia.

Interestingly, under both exercise sessions, there were succinct reductions in blood pH (nadir pH 7.28) and extracellular fluid base excess (nadir B base ≈ −6.0 mEq/L), which reflect the 10-fold increase in post-exercise blood lactate concentrations, and is indicative of the significant anaerobic component (Cerretelli & Samaja, 2003) to RE. From these results, we show that a reduction in the absolute mass lifted per repetition by ~50% did not alleviate exercise-induced metabolic stress when matching total mass lifted. This finding helps reconcile similarities in ratings of perceptual difficulty (i.e., “somewhat hard to hard”) between moderate- and “low-intensity” RE. However, the similarity in blood lactate accumulation between different RE session is paradoxi-cal when considering that catecholamines tended to be further raised under LOW than MOD; AD has powerful effects on muscle glycogenolysis by binding to β-adrenergic receptors on the skeletal muscle membrane initiating a cascade of events that augment glycogen breakdown (via activation of phosphorylase a) and resulting in increased lactate appearance (Podolin et al., 1991). It cannot be determined from this study design whether the contribution of lactate to endogenous glucose production by hepatic gluconeogenesis differed between low- and moderate-intensity RE – albeit the sparing effect that lactate could have on muscle glycogen utilization could be of benefit to the T1DM individual in preventing the onset of post-exercise hypoglycemia. Furthermore, the presence of AD is not essential to glycolytic activity because muscle glycolytic turnover has been shown to occur independent of the conversion of phosphorylase b to a (Ren & Hultman, 1990). Thus, there are complex relationships between counter-regulatory hormones and manipulations of RE session characteristics warranting further work to improve understanding of the glycemic impact and metabolic stress caused by RE in T1DM individuals.

The insulin and dietary routine adopted by all participants did not elicit clinical hypoinsulinemia during the study because insulin concentrations were approximately comparable with those typically observed in individuals without diabetes, although it is conceded that these values could represent a relative hypoinsulinemic-hyperglycemic state for this particular clinical cohort. Earlier work from our group demonstrated that this routine for exercise did not expose T1DM individuals to acute ketonemia (Turner et al., 2015). We acknowledge that subtle yet non-significant alterations in circulating insulin levels might have influenced the observed time course changes in glycemia within and between sessions, but under the present conditions it seems fair to conclude that altering exercise intensity in this manner does not affect the glycemic response to post-absorptive RE. A limitation in this study was the brief (albeit intensive) window of monitoring participant glycemia. Previous research by Yardley et al. (2013) has shown that a three-set session of RE could put T1DM individuals at greater risk of later post-exercise and nocturnal hypoglycemia, and T1DM individuals have been observed to experience late-onset hypoglycemia following performance of high-intensity intermittent exercise (Campbell et al., 2014). Further research is required to determine whether the metabolic responses to exercise within this cohort might differ from those of better glycemic control (i.e., HbA1c < 7.0%), given that hyperglycemia might have altered the acute counter-regulatory hormone responses to these RE sessions (as previously discussed).

The findings from this study are important to T1DM individuals and practitioners because current exercise guidelines for this cohort lack information pertaining to the acute metabolic stress and glycemic impact resulting from performance of different RE sessions, and this lack of awareness could compromise exercise safety. From the scant amount of research in this area, it is difficult to identify the optimal balance between acute exercise safety and chronic impact, but these findings taken together with our prior research (Turner et al., 2015) suggest that individuals should be judicious of possible hyperglycemia soon after low- to high-volume and intensity morning RE sessions, and that low- to moderate-intensity RE sessions can result in significant metabolic stress.
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Perspective

In conclusion, the magnitude of post-exercise hyperglycemia, acid–base disturbance, and perceptual difficulty were similar in response to moderate- and low-intensity RE sessions where total mass lifted was matched between sessions. Despite different exercise intensities, the longer exercise duration of the low-intensity RE session may be responsible for comparable if not greater counter-regulatory hormone responses, leading to similar post-exercise changes in BG. The lighter masses lifted with low-intensity RE (i.e., low resistance coupled with high repetitions) is likely to be more suited to less physically active TIDM individuals, and it might be prudent to prescribe longer rest intervals between sets of exercises when performing this form of RE.

Key words: Blood glucose, patient, weights training, catecholamines, growth hormone, interleukin-6.

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Author contributions

D. Turner, the guarantor, takes full responsibility for the integrity of contents in the manuscript, contributed to the formation and design of study, researched and analyzed data, and wrote the manuscript. S. Luzio contributed to conception and design of the study, biochemical analysis, and reviewed/edited manuscript. B. J. Gray assisted in researching data and reviewed/edited manuscript. S. C. Bain aided in participant recruitment, study design, and reviewed/edited manuscript. S. Hanley, A. Richards, D. C. Rhyderch, and RMartin all assisted in researching data. M. Ayles assisted in data analysis. M. D. Campbell reviewed/edited the manuscript. L. P. Kilduff reviewed/edited the manuscript. D. J. West contributed to conception and design of the study and reviewed/edited manuscript. R. M. Bracken contributed to the formation and design of the study, assisted in researching and analyzing data, and had a primary role in reviewing/editing of the manuscript.

All authors hereby declare that none of the data within this manuscript has been previously published nor are these data under consideration for publication elsewhere.

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