Pediatric Diabetes 2014: 15(Suppl. 20): 203–223
doi: 10.1111/pedi.12176

Pediatric Diabetes

ISPAD Clinical Practice Consensus Guidelines 2014 Compendium

Exercise in children and adolescents with diabetes

Robertson K, Riddell MC, Guinhouya BC, Adolfsson P, Hanas R. Exercise in children and adolescents with diabetes.
Pediatric Diabetes 2014: 15 (Suppl. 20): 203–223.

Kenneth Robertson[a], Michael C Riddell[b], Benjamin C Guinhouya[c], Peter Adolfsson[d,e] and Ragnar Hanas[e,f]

[a]Greater Glasgow & Clyde Children's Diabetes Service, Royal Hospital for Sick Children, Glasgow, UK; [b]School of Kinesiology and Health Science, York University, Toronto, Canada; [c]EA 2694, Laboratory of Public Health, Faculty for Health engineering and management, USDL, University Lille Northern France, Lille, France; [d]Department of Pediatrics, Kungsbacka Hospital, Kungsbacka, Sweden; [e]Institute of Clinical Sciences, The Sahlgrenska Academy, University of Gothenburg, Gothenburg, Swedenand [f]Department of Pediatrics, NU Hospital Group, Uddevalla Hospital, Uddevalla, Sweden

Key words: adolescent – child – diabetes mellitus – exercise – physical activity

Corresponding author: Kenneth Robertson,
Royal Hospital for Sick Children,
Dalnair Street,
Yorkhill, Glasgow,
Scotland,
UK.
Tel: +44 141 201 0801;
fax: +44 141 201 0407;
e-mail: kjr@diabetes-scotland.org

Editors of the ISPAD Clinical Practice Consensus Guidelines 2014 Compendium: Carlo Acerini, Carine de Beaufort, Maria Craig, David Maahs, Ragnar Hanas.

This article is a chapter in the *ISPAD Clinical Practice Consensus Guidelines 2014 Compendium*. The complete set of guidelines can be found for free download at www.ispad.org. The evidence grading system used in the ISPAD Guidelines is the same as that used by the American Diabetes Association. See page 3 (the Introduction in *Pediatric Diabetes* 2014; 15 (Suppl. 20): 1-3).

Executive summary and Recommendations

- Tailor insulin regimen to activity (B). Multiple daily injections or a pump may be easier to combine with active exercise.
- Discuss the percentage reductions in insulin before exercise (C).
  - When exercise is planned at a time of peak insulin action, a marked reduction in dose should be made.
  - The pump needs to be disconnected or a temporary basal rate implemented at least 90 min before starting the exercise to give a reduced basal effect.
  - Do not inject the insulin in a site that will be heavily involved in muscular activity.
- Discuss type and amount of carbohydrate (CHO) required for specific activities (B).
- Any exercise is dangerous and should be avoided if pre-exercise blood glucose levels are high (>14 mmol/L, 250 mg/dL) with ketonuria (small or more)/ ketonemia (>0.5 mmol/L). Give approximately 0.05 U/kg or 5% of total daily dose(TDD, including all meal bolus doses and basal insulin/basal rate in pump) and postpone exercise until ketones have cleared (B).
- Consume up to 1.0–1.5 g of CHO per kilogram of body mass per hour of strenuous or longer duration exercise when circulating insulin levels are high, if pre-exercise insulin doses are not decreased (B).
- Meals with high content of CHOs should be consumed shortly after the exercise event, taking advantage of the period of heightened insulin sensitivity to help replenish glycogen content and limit post-exercise hypoglycemia.
- Alcohol inhibits gluconeogenesis so hypoglycemia is more likely if consumed (A).
- Dehydration is a risk unless sugar-free fluids also are consumed (E).
- Use of detailed records of physical activity, insulin, food, and glucose results is important for good diabetes control during spontaneous physical activity and/or exercise. New technologies, e.g., embedded into Smartphones may be of use (E).
- Hypoglycemia may not only be anticipated during or shortly after exercise, but is also possible up to 24 h afterwards, due to increased insulin sensitivity (A).

- ◦ Measure blood glucose before going to bed and decrease bedtime basal insulin (or pump basal) by 10–20% after an afternoon or evening exercise session if the exercise was more intense than usual or an activity not performed regularly.
- ◦ Short sprints added to aerobic training can minimize the risk of hypoglycemia
- ◦ Extra CHO after the activity is often the best option to prevent post-exercise hypoglycemia when short duration and high-intensity anaerobic activities are performed.
- ◦ A mixture between aerobic and anaerobic exercise (soccer, cycling, jogging, and swimming) will typically require extra CHO before, possibly during, and often after the activity.
- ◦ The rise in blood glucose after intense exercise may be prevented by giving a small additional dose of rapid-acting insulin at half-time or immediately after the exercise is finished – for example, a 50% correction bolus when levels are >15 mmol/L.

- Risk of post-exercise nocturnal hypoglycemia is high, and particular care should be taken if bedtime blood glucose level is <7.0 mmol/L (125 mg/dL) with NPH basal insulin. With basal analogs, the bedtime glucose level can be slightly lower without a substantial risk of night-time hypoglycemia but no specific value is a guarantee that hypoglycemia will be avoided (E).
- Patients who have proliferative retinopathy or nephropathy should avoid resistance-based exercises or anaerobic exercise that is more likely to result in high arterial blood pressure (E).
- Care should be taken that the blood glucose meter and test strips chosen are suitable for the environment where they will be used (C).
- High glycemic index snacks and hypoglycemia remedies should always be readily available at school (E).
- Careful advice on and planning of travel, exercise, and management is essential (E).
- A diabetes care plan containing written advice about exercise and sports should be provided for carers/teachers (E).
- Professionals should take opportunity to attend camps for children with diabetes (E).
- Continuous glucose monitoring (CGM) may have a role in helping to avoid hypoglycemia during and after exercise (C).
- New pump technologies such as low-glucose suspend and programmed low glucose management may also be useful in the future (E).

Introduction

It is clear that it is important to encourage children and adolescents to be physically active, to be less sedentary, to control their weight, and to develop healthy lifestyle habits that will be maintained. In children who already have diabetes, this will help to mitigate increased cardiovascular risk and in those who are not diabetic, physical activity will have an important role in prevention.

While this article is intended to address the issue of blood glucose regulation during various forms of sports and exercise, it is important for diabetes care professionals and parents to appreciate that the demands of day-to-day physical activity will also have to be considered if a young person is going to participate in any activity, which for them is unusually strenuous or prolonged.

In the 1950s, Joslin proposed that exercise is the third essential component in blood glucose regulation for persons with type 1 diabetes (T1D), after insulin and dietary management. Although most studies have shown little impact upon hemoglobin A1c (HbA1c) levels (1–3), and many only a brief improvement (4) unless the exercise was sustained for 6 months (5), a cross-sectional analysis of data on a larger group showed that the frequency of regular physical activity was associated with lower HbA1c without increasing the risk of severe hypoglycemia (6). Younk et al. have provided a useful review (7). The benefits of exercise go far wider and include weight control, reduced cardiovascular risk (8), and an improved sense of well-being (9).

There is growing evidence that the antecedents of cardiovascular risk begin early in diabetes (10) and studies have shown that exercise has a beneficial effects on various markers of vascular health including skin microvascular reactivity (11) and endothelial function (12). A systematic review of adult studies concluded that physical activity is associated with a marked decrease in cardiovascular and all-cause mortality in both men and women, even after adjusting for other relevant risk factors (8). Even if glucose targets, as measured by HbA1c are not achieved, regular physical activity is associated with reduced early mortality in the adult population (13).

For people with diabetes, post-meal low to moderate-intensity exercise can be a valuable way to minimize postprandial glycemic spikes (14). For some, participation in physical activity is somewhat sporadic and related to leisure, school, or work. For others, daily exercise is part of an overall training or conditioning program.

Children and adolescents with diabetes should derive many of the same health and leisure benefits as adults and should be allowed to participate with equal opportunities and with equal safety.

Diabetes should not limit the ability to excel in a chosen sport. Many famous athletes have proved this, e.g., Sir Steve Redgrave – five times Olympic gold

medal winning rower, Kris Freeman – Olympic cross-country skier (four winter Olympics), Gary Hall – five time Olympic Gold Medal swimmer, Zippora Karz – ballerina, Wasim Akram – Pakistani cricketer at international level, Brandon Morrow – Major League baseball player, Cliff Scherb – Ironman Triathlete, Scott Verplank – PGA Tour golfer, and female professional golfer Mimmi Hjorth and Emil Molin – NHL ice-hockey player. There is now even a professional cycling team (Team NovoNordisk), with all the riders having T1D, which holds the record for the Race Across America and has aspirations for a Tour de France ride in 2021.

The topic most commonly discussed with families with regard to exercise is avoidance of hypoglycemia, but prevention of acute hyperglycemia/ketoacidosis may become a concern as well (15).

The HELENA study has demonstrated, in a large multi-center cohort of European adolescents without diabetes, that muscular fitness and cardiorespiratory fitness are independently associated with metabolic risk of insulin resistance (16) and therefore of type 2 diabetes (T2D). Part of this study showed that self-reported physical activity correlates negatively with insulin resistance (after adjusting for confounders such as waist–hip ratio) but that higher cardiorespiratory fitness reduces the impact so insulin resistance was less in those with the higher fitness (17). These findings have been supported by a recent Dutch study (TRAILS) which also showed that increased childhood fatness is associated with increased cardiometabolic risk but that this is, to some extent, mitigated by fitness (18).

The relationship between physical activity, sedentary behavior, fitness, and glycemic control is complex, as suggested above, but several studies have found that children and adolescents with T1D are less fit than their non-diabetic peers, particularly if they are in poor glycemic control (19, 20).

Huge efforts are being made around the world to get children and adolescents to engage more in physical activity and to reduce sedentary behavior. A recent systematic review by MacMillan et al. has studied interventions aimed at youth with T1D (21).

Exercise physiology

Before considering the situation in T1D, it is useful to understand the 'normal' physiological responses to moderate-intensity aerobic exercise in the non-diabetic individual.

As shown in Fig. 1, non-diabetic individuals have a reduction in insulin secretion and an increase in glucose counterregulatory hormones facilitating an increase in liver glucose production that matches skeletal muscle glucose uptake during exercise. As a result of this precise autonomic and endocrine regulation, blood glucose levels remain stable under most exercise conditions (9).

Exercise has been shown to increase non-insulin dependent glucose uptake by muscle by the translocation of GLUT-4 receptors to the cell surface. Thus glucose uptake increases even when insulin levels are low (22).

Under conditions of intense exercise, catecholamines rise, tending to increase hepatic glucose production and limit glucose uptake into muscle, thereby promoting a brief and transient increase in glycemia, even in non-diabetic children. This increase is exaggerated

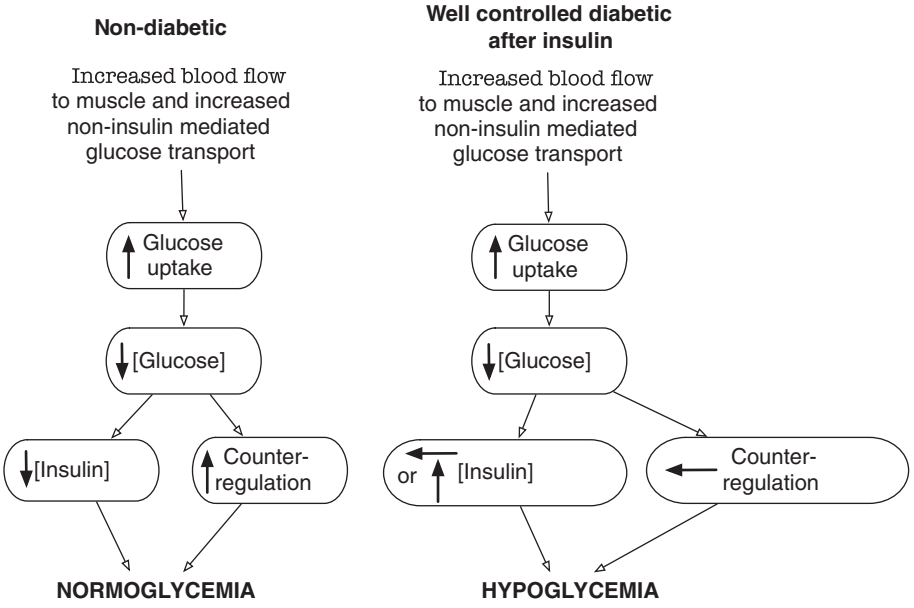

Fig. 1. Physiologic responses to exercise in the diabetic and non-diabetic individual (square brackets denote plasma concentration).

in children with T1D because of a failed increase in insulin secretion during the rise in glucose (23).

In T1D, the pancreas does not regulate insulin levels in response to exercise and there may be impaired glucose counterregulation, making normal fuel regulation nearly impossible. As a result, hypoglycemia or hyperglycemia commonly occurs during or soon after exercise.

Response to exercise

In real life, young people with diabetes have variable blood glucose responses to exercise. The blood glucose response to 60 min of intermittent exercise is reproducible in a child if the timing of exercise, the amount of insulin, and the pre-exercise meal remain consistent (24). Glucose production in healthy control subjects increases with exercise intensity and can be entirely attributed to increases in net hepatic glycogenolysis. In contrast, moderately controlled T1D subjects exhibit increased rate of glucose production both at rest and during exercise, which can be entirely accounted for by increased gluconeogenesis (25).

Young people with T1D have been found to have decreased aerobic capacity as measured by VO_2 max (percentage of maximal aerobic capacity), compared with non-diabetic control subjects (26) but this finding is contested by Adolfsson et al. in a detailed study of VO_2 max and endocrine response to different intensities of exercise (bicycle ergometer) in six reasonably well-controlled adolescents with diabetes and six non-diabetic controls of similar age. They found no significant differences except for higher growth hormone levels in those with diabetes (27). It is probably relevant that all participants in the latter study reported that they participated regularly in physical activity. Similarly, Cuenca García et al. compared 60 8–16 yr olds with diabetes with 37 sibling controls and found no difference in fitness or physical activity. They found that moderate to vigorous physical activity was associated with better metabolic control and accounted for 30–37% of the variance in HbA1c (28).

Total-body insulin-mediated glucose metabolism in adolescents correlates with the degree of glycemic control as assessed by the level of glycosylated hemoglobin (29). However, even in the same individual, it is possible for the blood glucose to be increased, decreased, or unchanged by exercise dependent upon circumstances as indicated in Table 1.

Factors affecting glucose response to exercise

Intrinsic characteristics of the exercise

Duration and intensity. It is especially important to plan for long duration or intense aerobic exercise, or else hypoglycemia is almost inevitable. Nearly all forms of activity lasting >30 min will be likely to require some adjustment to food and/or a reduction in insulin.

Most team and field sports and also spontaneous play in children are characterized by repeated bouts of intensive activity interrupting longer periods of low to moderate-intensity activity or rest. This type of activity has been shown to produce a lesser fall in blood glucose levels compared with continuous moderate-intensity exercise, both during and after the physical activity in young adults (30). The repeated bouts of high-intensity exercise stimulated higher levels of noradrenaline that likely increased blood glucose levels. A study in adults (31) suggested that late hypoglycemia was more common after intermittent high-intensity exercise but the opposite was found by another conducted in trained athletes with T1D (32).

Moderate-intensity exercise (40% of VO_2 max) followed by an intense cycling sprint at maximal intensity prevented a further decline in blood glucose for at least 2 h after the exercise (33). However, typical team games may last up to 90 min and the results may not be

Table 1. Factors that affect changes in blood glucose during exercise

Hypoglycemia	Glucose unchanged	Hyperglycemia
Hyperinsulinemia due to proximity to or excessive dose of administered insulin (both bolus and basal). Exercise prolonged – usually more than 30–60 min and/or no extra carbohydrate intake. Moderate-intensity aerobic exercise (50–75% maximal aerobic capacity). Non-familiarity with an activity, requiring greater energy expenditure than when in a trained state.	Insulin pre-exercise adjusted appropriately. Appropriate carbohydrate consumed.	Hypoinsulinemic state prior to and during exercise. The emotion of competition eliciting an adrenal response. Short, intermittent bouts of intense anaerobic activity eliciting an increase in adrenal response. Excessive carbohydrate consumed Post-exercise, when glucose production exceeds utilization.

applicable to this length of physical activity. The same research group, from Perth, Australia, further studied this phenomenon and concluded that short duration sprinting increases blood glucose levels via a disproportionate increase in glucose rate of appearance (Ra) relative to glucose rate of disappearance (Rd). This may explain the fact that levels of plasma epinephrine, norepinephrine, and GH rise only transiently after a 10 s sprint (23) (see also 'Type of Exercise').

Hypoglycemia in the morning before exercise does not diminish the glycemia-raising effect of an afternoon sprint in young adults with T1D, suggesting that sprinting is a useful strategy for opposing hypoglycemia, regardless of prior hypoglycemia (34).

Types of exercise. Anaerobic efforts last only a short time (sometimes only seconds) but may increase the blood glucose level dramatically due to the release of the hormones epinephrine and glucagon. This rise in blood glucose is usually transient, lasting typically 30–60 min, and can be followed by hypoglycemia in the hours after finishing the exercise. Aerobic activities tend to lower blood glucose both during (usually within 20–60 min after the onset) and after the exercise (9). Peak insulin sensitivity in recovery appears to be approximately 7–11 h after the end of exercise in adolescents with T1D (35).

The impact of activity type upon short- and long-term glycemia is scrutinized by Tonoli et al. in a meta-analysis across all age groups (36). They concluded that repeated short sprints added to aerobic training can minimize the risk of hypoglycemia and that aerobic training (as opposed to resistance training, mixed or high-intensity exercise) is likely to improve chronic glycemic control.

Although not yet studied in the pediatric population, resistance-based exercise (i.e., weight training) produces less drop in glycemia compared with aerobic exercise (37) and if both activities are to be done in one setting, it may be preferable from a glycemic management standpoint to do the resistance activity before the aerobic activity (37), perhaps because of augmented growth hormone release (38).

Timing of the exercise. Morning activity, done before insulin administration, may not result in hypoglycemia as circulating insulin levels are typically low and glucose counterregulatory hormones may be high (39). Indeed, severe hyperglycemia may occur with vigorous exercise in these circumstances, even precipitating ketoacidosis if insulin has been withheld for a long period of time (40).

Conditioning. Patients frequently report that the drop in blood glucose may be less with regular conditioning and familiarity with the sport, although no experimental evidence exists that tests this hypothesis. This may be because regular conditioning often is accompanied by a reduction in total daily insulin administration and a greater reliance on lipid as a fuel (41).

Degree of stress/competition involved in the activity. Catecholamines rise during high-intensity exercise, and their rise can contribute to exercise-associated hyperglycemia (42). The adrenal response during intense exercise or perhaps during stressful competition will raise blood glucose and may require corrective insulin administration (43).

Muscle mass/number of muscles used in the activity. Using more muscles during aerobic exercise produces a greater drop in blood glucose and weight-bearing activities tend to use more energy than non-weight-bearing activities.

Other factors involved in the glucose regulation during exercise

Metabolic control. Where control is poor and pre-exercise blood glucose level is high (>20 mmol/L), circulating insulin levels may be inadequate and the effect of counterregulatory hormones will be exaggerated, leading to a higher likelihood hyperglycemia and ketosis (40). In Ironman athletes with T1D, those with low (i.e., near normal) HbA1c had performance, equivalent to non-diabetic controls (44).

Blood glucose level. Some limited evidence exists to support a near normal blood glucose concentration during exercise for performance reasons. For example, high blood glucose has been found to reduce secretion of beta-endorphins during exercise, which has been associated with an increased rating of perceived exertion (RPE) during leg exercise (45). In fact, even baseline beta-endorphin levels were reduced in the diabetic subjects irrespective of blood glucose, and thus the resultant reduced tolerance of discomfort may compromise exercise performance in individuals with diabetes. Similarly, increases were found in RPE in adolescents with diabetes doing whole-body exercise (46), but the authors indicate that the higher response may be related to the lower peak mechanical power output often seen in these patients (47).

Even in well-controlled individuals, hyperglycemia at the time of exercise (adult study in controlled laboratory conditions) resulted in a shift from lipid to CHO oxidation (48).

Hypoglycemia has also been shown to compromise both sport performance and cognitive function in

youth with T1D (49). Thus a near normal glucose concentration may be optimal for overall sport performance.

Children with diabetes appear to have a normal aerobic and endurance capacity if good glycemic control is achieved (HbA1c <7.0%), even if they are slightly hyperglycemic at the time of exercise. In one study, physical working capacity in well-controlled prepubertal boys was not different from non-diabetic boys matched for age, weight, and physical activity patterns, even though the boys with diabetes exercised with considerably higher blood glucose concentrations (mean blood glucose 15 mmol/L at onset of exercise) (50). In line with this, cycling performances in adult males with T1D did not differ between glucose levels clamped at euglycemia vs. hyperglycemia (12 mmol/L, 220 mg/dL) (51). In contrast, aerobic capacity was lower and the fatigue rate higher in youth with T1D when glycemic control was less than optimal (i.e., HbA1c >7.5%) (26). Moreover, performance in sports like hockey, soccer, and sailing where a certain amount of cognitive function and precision is necessary may be better performed during normoglycemia compared with hyperglycemia, although studies have not yet been conducted to address this hypothesis. However, cognitive performance has been shown to be slower in youth with diabetes when their blood glucose is either hypoglycemic or hyperglycemic (52).

Type and timing of insulin injections. When regular (soluble) insulin has been injected prior to exercise, the most likely time for hypoglycemia will be 2–3 h after injection, when insulin levels peak. For rapid-acting insulin analogs, peak insulin action, and therefore the greatest hypoglycemia risk, is between 40 and 90 min (53).

It should be noted, however, that exercise can dramatically increase skin and systemic blood flow and insulin and glucose delivery to skeletal muscle (54). Exercise has been shown to increase rapid-acting insulin absorption rate (55), thereby likely hastening the peak insulin action. Basal insulin absorption, on the other hand, does not appear to be impacted significantly by long-acting insulin (glargine), although exercise can still promote a drop in glycemia compared with basal insulin injected at rest (56).

To help prevent hypoglycemia during prolonged exercise, reductions in bolus and/or basal insulin are typically required. The normal recommendation is to reduce rapid-acting analog prior to exercise lasting longer than 30 min. The impact of the scale of the reduction was studied in adults by Bracken et al. by reducing the pre-exercise insulin to 75, 50, or 25% of the normal dose. Interestingly, although the largest reduction was associated with higher post-exercise glucose, there was no difference in the production of ketones (57). This is a reassuring message and is helpful when encouraging young people to experiment to find what scale of reduction works for them.

We have found no studies on the timing of basal insulins (NPH, glargine, or detemir) and exercise in children but Arutchelvam et al. found that insulin detemir was associated with less hypoglycemia during and post-exercise than insulin glargine (58).

When playing in morning or in all-day tournaments, a long-acting basal insulin given once daily in the evening can be substituted for one with shorter action (NPH) to reduce the basal insulin effect the next day while exercising. An alternative is to split the TDD in half and take half the long-acting basal insulin in the evening and then lower the second dose in the morning by 20–50% to compensate for the increased activity.

Type and timing of food. A meal containing CHOs, fats, and protein should be consumed roughly 3–4 h prior to competition to allow for digestion and to maximize endogenous energy stores (59). This is especially important for longer duration activities. Glycogen stores can be enhanced with a CHO beverage (1–2 g CHO/kg) approximately 1 h prior; this also helps to supplement energy stores and provide adequate fluids for hydration (60). As this CHO loading is insulin dependent, and this will not be covered by the preceding meal bolus, it might be reasonable to try giving 50% of the bolus that would be dictated by the insulin to CHO ratio at this time and monitoring the response. Next time, a little more or a little less insulin may be necessary based on the measured glucose response. While the experimental evidence is laid out above, it is often impractical to have a meal 3–4 h prior to exercise and 1–2 h is more likely. This makes the individualization via trial and error especially important.

If extra CHO is necessary for a short duration activity, then it may be useful to have 'fast acting' CHOs in a beverage form. An isotonic beverage containing 6% simple sugar (i.e., sucrose, fructose, and dextrose) provides optimal absorption compared with other more concentrated beverages with more than 8% glucose, such as juice or carbonated drinks that delay gastric absorption and cause stomach upset (60). One study, however, found that both 8 and 10% isotonic beverages were well-tolerated and helped to prevent the drop in blood glucose level during exercise in adolescents with T1D (61). The amount of CHO should be matched as closely as possible to the amount of CHO utilized during exercise, if a reduction in insulin is not performed. In general, approximately 1.0–1.5 g CHO/kg body weight/h should be consumed during exercise performed during peak insulin action in young adults

with diabetes (60), depending upon type of activity (see Table 2) (62). The requirements will be lower if the pre-meal bolus for the meal before the exercise is lowered or the exercise is performed several hours after the bolus dose has been given (0.3–0.5 g CHO/kg body mass/h).

Extra CHOs together with adjustments of insulin doses are especially important when the activity is of longer duration than 60 min (63). That CHO beverages perform best pre-exercise in minimizing the drop in blood glucose was affirmed by Dube et al. who studied a variety of nutritional strategies (breakfast and pre-exercise) in adolescents. They found that including protein in the breakfast (before morning exercise) was associated with less hypoglycemia during and after exercise (64).

Because insulin sensitivity remains elevated for hours post-exercise, CHO stores must be replenished quickly to lower the risk of hypoglycemia during the first few hours after the activity (CHO reloading).

Short duration and high-intensity anaerobic activities (such as weight lifting, sprints, board diving, and baseball) may not require CHO intake prior to the activity, but may produce a delayed drop in blood sugar. For activities of these types, extra CHO after the activity may be needed.

Longer duration, lower intensity aerobic activities such as soccer (often described as a mixture between aerobic and anaerobic exercise), cycling, jogging, and swimming will require extra CHO before, possibly during and often after the activity.

Currently, no evidence-based guidelines exist on the amount and timing of increased CHO intake to limit post-exercise hypoglycemia. However, reductions in basal insulin, low-glycemic-index snacks (with no bolus), or reduced boluses at post-exercise meals will usually reduce the problem. A snack of complex CHO, fat, and protein at bedtime may limit nocturnal hypoglycemia caused by daytime exercise (65, 66).

Table 2. Exercise exchanges of 100 kcal (420 kJ) in children of various body masses. Assuming that, on average, 60% of total energy is provided by carbohydrate, one exchange is equivalent to 60 kcal or 15 g carbohydrate

	Body mass (kg)		
Activity	20	40	60
Basketball (game)	30	15	10
Cross-country skiing	40	20	15
Cycling			
10km/h	65	40	25
15 km/h	45	25	15
Figure skating	25	15	10
Ice hockey (ice time)	20	10	5
Running			
8 km/h	25	15	10
12 km/h	—	10	10
Snowshoeing	30	15	10
Soccer	30	15	10
Swimming			
30 m/min breast stroke	55	25	15
Tennis	45	25	15
Walking			
4 km/h	60	40	30
6 km/h	40	30	25

Tables of carbohydrate intake guidelines for a variety of sports are provided in a recent review (62). This table shows the estimated number of minutes that a certain activity lasts to require 15 g of extra carbohydrate to keep the blood glucose from falling. For example, a 40 kg child should consume 15 g of carbohydrate for every 15 min of basketball, whereas a 60 kg child should consume 15 g of carbohydrate for every 10 min of basketball.
If the pre-meal or basal insulin dose is lowered, less extra carbohydrate than the table shows is probably needed.

Absorption of insulin.

Choice of injection site. As mentioned above, when an extremity (arm or leg) has been injected with insulin and is then exercised vigorously, the increased blood flow to the limb is likely to result in more rapid absorption and metabolic effect of the insulin (67). This may be especially marked if the injection site is hypertrophied. Thus, a cyclist may achieve more consistent response by choosing to inject in an arm or the abdomen rather than a leg before an event.

Ambient temperature. High temperature will increase insulin absorption and low temperature the converse (68). The latter may be a consideration in long distance swimming. Heat also induces additional stress on the cardiovascular system, resulting in greater energy expenditure and potential for a faster drop in blood glucose levels.

Altitude. There is likely to be no altitude effect on insulin during recreational activities such as piste skiing but de Mol et al. studied eight complication free young people with diabetes, climbing above 5000 m and found that despite high energy expenditure, insulin requirement increased. Further, they found that glucose levels (and insulin requirement) correlated directly with the symptoms of acute mountain sickness (69).

Most absorption studies were done with regular insulin. The effect is less pronounced with rapid-acting analogs (70). An intense 30-min period of exercise did not increase the absorption rate of glargine in adults with T1D (56).

Normal day-to-day exercise

Daily physical activities should be a part of the normal routine for both health benefits and for consistency in blood glucose management.

The habitual physical activity of children encompasses activities performed during their leisure time as well as more structured activities in the framework of exercise, sports, or some school-related activities, such as physical education lessons. It was found that the spontaneous activity of children is by nature sporadic and intermittent, with bouts (95% of the time) of very intense activity not exceeding 15 s, and only 0.1% of active periods for more than 1 min (71). These activity periods are interspersed by rest periods that are shorter than 4 min. This particular form of spontaneous physical activity is deemed to be consistent with the biological needs of children (72) and necessary for their appropriate growth and development (71).

On average, at least 60 min of cumulative activity is recommended by most organizations with at least 20 min daily of vigorous activity. Guidelines also state that for health benefits, children (aged 5–11 yr) and youth (aged 12–17 yr) should minimize the time that they spend being sedentary each day (73). Sedentary time (i.e., screen time) is linked to elevated HbA1c levels in children and adolescents with T1D (74). Some groups of schoolchildren and teenagers with diabetes have been found to be more physically active than their non-diabetic friends (75) but others less so (76). In one recent study of children and adolescents with T1DM in Brazil increased physical activity was associated with the best glucose control (77), although this relationship between activity participation and glycemic control is not always found (74).

Regular and accustomed exercise is easier to manage because it is part of the daily routine. However, adjustments may still be necessary for sporadic extra physical activity.

Whatever level of involvement in exercise and sport that a child or adolescent with diabetes adopts, it is good practice to keep careful notes of what they do (i.e., timing and intensity of physical activity), what CHO has been taken and the blood glucose response before, during, and afterwards. Advice from the diabetes care team will be general in the first instance, but accurate record keeping will allow much more individualized and fruitful consultation. Emerging technologies, e.g., apps on Smartphones may have a role to play in improving record keeping.

Where exercise is performed regularly, insulin sensitivity is generally enhanced. A positive association between glycemic control (i.e., HbA1c) and aerobic fitness or reported physical activity exists in youth with T1D, suggesting that either increased aerobic capacity may improve glycemic control or that good metabolic control maximizes exercise (26, 28). An inverse relationship was observed between HbA1c level and the maximal work load in a study in diabetic adolescents (78). The lack of evidence on improving HbA1c with exercise may be related to a tendency to reduce insulin doses too aggressively for exercise or the overconsumption of CHOs in an effort to avoid hypoglycemia (79).

Training

The management of diabetes may vary according to the phase of training so when endurance is being built with long moderate-intensity work, the insulin regimen and additional CHO may be quite different from that required when the concentration is on power and high-intensity training (80). See the 'Duration and intensity' section above for more detail on the possible effect of short, high-intensity work on glycemia.

Exercise causes enhanced muscle insulin sensitivity (81) and increased activation of non-insulin sensitive glucose transporters (GLUT-4) (22, 82). Insulin sensitivity was similar directly and 15 h after exercise but decreased to near untrained levels after 5 d in non-diabetic adults (83). During and immediately after exercise performed in the late afternoon and from 7 to 11 h in recovery, the insulin sensitivity is elevated in adolescents with T1D (35). In contrast, exercise performed earlier in the day results in heightened insulin sensitivity thought 11 h of recovery in adolescents with T1D, without an obvious biphasic response in sensitivity (84).

In practical life, exercise for >1 h appears to lead to increased insulin sensitivity and therefore an increased risk for hypoglycemia for 12–24 h (35), often occurring during evening after exercise (85). This may be because of several factors including the change in insulin sensitivity, a reduction in glucose counterregulation and the problem of a fixed basal regimen (86). This means that adolescents who only exercise on occasion can have real difficulties in managing their basal insulins. If hypoglycemia is frequent, then it may be better to limit vigorous exercise every other day rather than daily, if possible. If not, a strategy for altering basal insulins to cope with the widely varying insulin sensitivity is needed. Younger children more often exercise every day to some extent, which results in less post-exercise fluctuations in blood glucose.

Meals with high content of CHOs should be consumed shortly after the exercise event to take advantage of the period of heightened insulin sensitivity to help replenish glycogen content and limit post-exercise hypoglycemia. However, the insulin dose will need to be reduced (in relation to the normal insulin to CHO ratio for the individual) to avoid hypoglycemia. Adding protein to the post-exercise meal increases the glucose uptake and enhances

glycogen resynthesis in healthy individuals (64) (87). Added proteins will also stimulate the muscle recovery post-exercise.

It is well beyond the scope of this article to offer sport-specific training advice but such information is readily available – see:

- Diabetes Exercise and Sports Association (www.diabetes-exercise.org), an international organization that provides guidance and networking between novices, health professionals, and experienced diabetic athletes.
- www.runsweet.com where a combination of contributions from sportsmen and sportswomen are interspersed with expert advice.

Choice of insulin regimen

In developed health care environments, it is now the norm to commence insulin therapy with a multi-injection regiment or even an insulin pump. While for most children and adolescents, the choice of insulin regimen will not be influenced heavily by their exercise habits, for those who are regularly active either multiple daily injections or insulin pump therapy should be considered to allow for manipulations in insulin delivery prior to and following the activity.

In one small study of young adults with T1D, insulin pump usage was associated with better glycemic control in early recovery from vigorous exercise than MDI (i.e., less hyperglycemia) (88).

Twice daily injections

It may be difficult to maintain very strict blood glucose control on these regimens especially with different levels of exercise throughout the week, but the essential requirements of taking various forms of CHO before, during, and after exercise may be even more important than for more adjustable regimens. In these situations, tables of Exercise Carbohydrate equivalents may be a useful starting point (59).

Three injections insulin regimen

In these situations, normally a mixed insulin is given before breakfast, then a split-evening insulin regimen with rapid analog before the evening meal, and a longer acting insulin at bedtime. Again this regimen must be accompanied by appropriate CHO advice for moderate exercise, e.g., dancing or swimming two or three evenings per week or at weekends.

Multi-injection regimens or insulin pumps

These regimens afford greater flexibility for serious training and competitive events. Both pre-exercise bolus and basal rates can be reduced before, during, and after exercise to help increase hepatic glucose production and limit hypoglycemia (see below).

A further variation is to use a long-acting insulin in conjunction with an insulin pump allowing partial replacement of the basal dose (once or twice daily long-acting injections) and therefore a longer period off the pump with lesser risk of ketosis.

The choice of insulin regimen is always influenced by many different factors including the availability of various insulins (and pumps), professional and personal expertise, and in the ideal world should be influenced by the nature of the sport. There is no doubt that being able to reduce the training day into manageable 'chunks' of 4–6 h makes control of blood glucose much more straightforward, with the potential to move training/competitive periods around in the day and being able to adjust the appropriate bolus (and perhaps basal) insulin doses (89). In general, if basal rates are to be reduced for exercise, then the reduction should occur approximately 90 min before the onset of the activity to allow the circulating insulin levels to drop sufficiently before the exercise starts (59).

Hypoglycemia

In adults, the autonomic and counterregulatory response to hypoglycemia the following day has been shown to be blunted by repeated episodes of low- or moderate-intensity exercise (90, 91). The same phenomenon is likely to be true for children. Glucose requirements for maintaining stable glucose levels in adolescents with diabetes are elevated during and shortly after exercise, as well as from 7 to 11 h after exercise (35). In adults, repeated episodes of hypoglycemia in a sedentary state result in an attenuated counterregulatory response to subsequent exercise and increases the risk for hypoglycemia. Hence, two to three times more exogenous glucose may be needed to maintain euglycemia during exercise following a previous exposure to hypoglycemia (92). In laboratory studies of diabetic adolescents who received their usual insulin dose and then performed 75 min walking on a treadmill, 86% had hypoglycemia if their starting blood glucose was less than 6.6 mmol/L (120 mg/dL). In the same study, it was noted that 15 g CHO was frequently insufficient to restore blood glucose to normal (93). In another study (94), 45% of children with T1D had blood glucose levels drop below 4.0 mmol/L (72 mg/dL) during 60 min of moderate cycling performed in the fed state when insulin was unadjusted for the activity. By consuming additional CHO (drinking 6–8% glucose solution) at a rate that equalled CHO utilization during exercise (approximately 1 g of CHO/kg body mass/h), the drop in blood glucose during exercise could be prevented (94).

The use of CGM and appropriate response to falling glucose may help to attenuate or avoid hypoglycemia during and after exercise (95).

If a child with diabetes is feeling unwell during exercise with signs and symptoms of hypoglycemia, glucose tablets or other form of quick-acting CHO should be given as for treatment of hypoglycemia, even if blood glucose cannot be measured to confirm hypoglycemia.

To treat hypoglycemia with a rise in BG of approximately 3–4 mmol/L (55–70 mg/dL), approximately 9 g of glucose is needed for a 30 kg child (0.3 g/kg) and 15 g for a 50-kg child. See the Hypoglycemia article for further advice and references.

On outward-bound or activity holidays all the responsible adults (and also peers) should be alert to the possibility of hypoglycemia. Strict guidance should be given that no person with diabetes should exercise or go off alone, or 'decide' not to have regular snacks when they are provided.

A sensible rule is that if young people with diabetes are together on holiday, they should stay in groups of at least four, so that two can accompany each other if they need to alert adult helpers to the occurrence of an accident or hypoglycemia.

Glucose tablets, glucose gel, or some form of rapidly absorbed sugar should always be carried by young people who exercise or, at a minimum, kept within a reasonable distance of the activity.

See Table 3 (96) for further advice on how to avoid hypoglycemia when exercising.

Table 3. Summary recommendations for avoiding hypoglycemia in physically active young people with diabetes (adapted from reference 96)

- Arrive at a good level of metabolic control: neither with hyperglycemia nor ketonuria. Eventually measure blood glucose concentration before the activity.
- Always carry some sugar.
- Increase the intensity and duration of the activity in a progressive fashion.
- In the few hours preceding the exercise, ingest slowly absorbing carbohydrates in order to replete the liver and muscle glycogen reserves.
- In the case of unforeseen physical activity, increase glucose consumption immediately before, during, and after the activity
- In the case of foreseen activity, decrease the insulin dose during and after intense muscular activity.
- Do not inject the insulin at a site that will be heavily involved in the muscular activity.
- When physical activity is planned at a time of peak insulin action, a marked reduction in the insulin dose should be made.
- If the activity is of the prolonged endurance type, be certain to ingest glucose-sweetened water or carbohydrates just before, during, and after the exercise.
- Measure the blood glucose before retiring on the evening after major physical activity, in order to avoid hypoglycemia during the night.
- Evaluate the effect after every modification in insulin dose and every change in nutritional status.
- Make the people accompanying you aware of the procedures and treatment of severe hypoglycemia (glucagon injection)

Late hypoglycemia

As mentioned above, hypoglycemia can occur several hours after exercise, especially when this has been prolonged and of moderate or high intensity (97). This is due to the late effect of increased insulin sensitivity and delay in replenishing liver and muscle glycogen stores and perhaps due to failed glucose conterregulatory hormone responses during the night (86). A single bout of exercise can increase glucose transport into skeletal muscle tissue for at least 16 h post-exercise in non-diabetic and diabetic subjects (30). In a controlled study, twice as many youngsters aged 11–17 yr had a hypoglycemic event on the night after an exercise day compared with the night after a sedentary day (when the basal overnight insulin was not altered) (85). CGM may be a valuable tool for determining the blood glucose response and hypoglycemia risk during and after exercise (98, 99). One adult study has shown that the likelihood of late hypoglycemia was greater after intermittent high-intensity than moderate-intensity exercise despite the blood glucose and catecholamine levels being higher after the former (31). Again in adults, use of CGMS in conjunction with a low-glucose suspend (i.e., threshold suspend) function on insulin pumps may reduce the duration and severity of hypoglycemia with exercise in laboratory conditions (100). It should be noted, however, that if a recent episode of exercise-associated hypoglycemia occurred, low-glucose suspend technology may not be as effective in ameliorating hypoglycemia risk (101).

Taplin et al. attempted to reduce hypoglycemia after a 60 min bout of exercise in 16 youths with T1D on insulin pumps by either reducing their basal insulin by 20% for 6 h or by giving 2.5 mg of oral terbutaline. Although the latter did reduce overnight hypoglycemia, it was associated with overnight hyperglycemia. The basal reduction was not only effective but also associated with some late high glucose results. The authors accept that reducing insulin in this way is not possible for patients using intermittent insulin injections (102).

Insulin adjustments

Competitive athletes may be tempted to reduce their insulin doses too much to avoid hypoglycemia and their metabolic control may suffer as a result (79). Careful monitoring and experiential adjustments are

essential. In a group of young people aged 10–18 yr, those attending a competitive sport of at least 6 h of exercise per week had a lower HbA1c (75).

In one study, cross-country skiers with T1D were able to carry on for several hours without hypoglycemia when reducing the pre-meal dose by 80%, compared with only 90 min if the dose was reduced by 50% (103). Some people find that lowering their pre-meal insulin dose may cause an initial rise in their blood glucose which impairs their performance. In such a case, it is probably better to rely on extra CHO intake just before the onset of exercise rather than dose reduction for best performance.

See Table 4 (104) for examples on adjustments of pre-exercise bolus doses in order to avoid hypoglycemia. There is a greater need for reduction of rapid-acting insulin when the dose is given within 1 h of the exercise, while the need of reduction is greater for later exercise (3 h post-meal) when using regular insulin. (53).

For evening exercise, it may be sensible to reduce the rapid analog before the evening meal by 25–75%, as well as taking 10–15 g of fast acting CHO before the activity.

Advice about reducing basal insulin by 10–20% (e.g., a reduction in overnight long-acting/basal insulin or basal rate in pump or reductions in subsequent mealtime boluses), and/or extra low glycemic index snacks following the activity is prudent.

With all-day or unusual activities such as camps, long-distance walking, skiing, water sports, etc. consider a 30–50% reduction of long-acting insulin the night before and on the day of the activity, or a 30–50% reduction in the pump's basal insulin throughout the day and the night following the activity. High excitement amusement parks and fairs may be more likely to raise BG because of adrenalin surges.

Table 4. Examples of percent reduction in pre-meal insulin bolus for a carbohydrate-containing meal, in order to strictly avoid hypoglycemia (with either pump or multiple daily injections) for low-, moderate-, and high-intensity exercise lasting 30 or 60 min in duration are given in the table. Note however, that this study was in adults and did not consider the effect of additional carbohydrate intake before or during the exercise. Moreover, the adjustments also were associated with an increased number of episodes of hyperglycemia pre-exercise and post-exercise (104)

	Duration of exercise and recommended reduction in insulin	
Intensity of exercise	30 min	60 min
Low (~25% VO_2 max)	25%	50%
Moderate (~50% VO_2 max)	50%	75%
Heavy (~75% VO_2 max)	75%	—

%VO_2 max = percentage of maximal aerobic capacity.

It should be obvious from the above that individuals vary greatly in their response to different types of exercise so the most important thing is for patients and families to be aware of the broad themes and to use this knowledge coupled with good record keeping to find what works for them.

Insulin pumps

For certain types of exercise (like contact sports), it may be appropriate to disconnect prior to the start of the activity and remain disconnected for up to 1–2 h during an event. In these situations, patients may require a 50% bolus correction afterwards (i.e., 50% of the missed basal insulin while disconnected), if needed, to reduce any resulting post-exercise hyperglycemia. To get a significant lowering of the basal insulin effect during the exercise, the pump needs to be disconnected at least 60 min before starting the exercise (105), but many centers advise that the pump should not be disconnected for more than 2 h. The safer option may be to set a temporary basal rate 90 min before the activity (50–80% reduction depending upon the intensity and duration of the activity), lasting until the end of exercise.

Even if the pump is removed during exercise, hypoglycemia can still occur for several hours after the end of the activity (106).

After a short period of intense exercise (≥80% VO_2 max), marked catecholamine responses lead to hyperglycemia which lasts for approximately 2 h post-exercise in adults with T1D (43). Even when pre-exercise plasma glucose was normal, there ensued a post-exercise hyperglycemia which lasted for 2 h post-exhaustion in pump patients (107). This reaction may be exaggerated if the pump has been disconnected during the exercise. The rise in blood glucose may be prevented by giving a small additional dose of rapid-acting insulin at half-time or immediately after the exercise is finished, i.e., before the shower.

New insulin pump technology may offer better opportunities to avoid hypoglycemia associated with exercise. The ASPIRE study, considered the use of low-glucose suspend (LGS) technology to turn off an insulin pump for 2 h once a CGM sensor detected a blood glucose value less than 70 mg/dL (3.9 mmol/L). Subjects, including adolescents, were randomized to sensor augmented pump therapy with or without low-glucose suspend turned on in a crossover study. After overnight fasting, subjects exercised until hypoglycemia intervened. The LGS group duration of hypoglycemia was less (100).

In a further development, predictive low glucose management algorithms built into pumps are being designed to turn off the insulin delivery when the blood glucose reaches a certain point and turns it back

on again at a predetermined level. In *in silico* and live testing on adolescents exercising, this method delivered promising results with 80% of the experiments where the glucose fell below threshold resulting in the prevention of hypoglycemia (108).

Ketones

In situations of under-insulinization, whether through systematically poor control or intercurrent illness, any exercise is likely to be dangerous because of the effect of uninhibited action of the counterregulatory hormones. In one study in adults, patients exercising with a blood glucose of >20 mmol/L (260 mg/dL) and ketonuria experienced a rise in blood glucose over 40 min (109).

The rapid production of ketone bodies coupled with impaired muscle glucose uptake will lead not only to under-performance, but may precipitate ketoacidotic abdominal pain and vomiting. Thus it is important for families to be warned about not participating in exercise if blood glucose is high and ketones (small or more) are present in the urine (9, 89, 109) or the level of beta-hydroxybutyrate (BOHB, 'blood ketones') in blood is >0.5 mmol/L.

It is a relatively common misconception that no insulin is needed when prolonged exercise is to be undertaken. This could be a dangerous error unless insulin cover is being provided by a long-acting product, and under carefully monitored conditions.

Blood ketone testing (measuring BOHB) provides additional information to urine ketone testing (110). This method is excellent for rapid detection and exact measurement of ketone levels and is preferable, when available. During resolution of ketosis, blood BOHB normalizes sooner than urine ketones (111). Blood BOHB >0.5 mmol/L is abnormal in children with diabetes (112, 113).

Patients can be reassured that reducing insulin down to 25% of pre-exercise doses does not make later ketosis more likely (57).

What to eat and drink

When insulin is not reduced to accommodate for exercise, it is usually necessary to consume extra CHO in order to avoid hypoglycemia. This is dependent upon type and duration of activity.

The amount of CHO needed depends largely on the mass of the child and the activity performed as well as the level of circulating insulin (53). Up to 1.5 g CHO/kg of body mass/h of strenuous exercise may be needed.

Numerous charts indicating CHO replacement for specific exercises based on duration of activity and body size are found in references 114, 115 and for youth specifically in a review by Riddell and Iscoe (116).

The growing use of CGM may offer opportunities for better tailoring of food intake before, during, and after exercise through the use of more precise algorithms (95).

It is worth reminding adolescents and young adults about the effect of alcohol upon the ability to respond to exercise and falling blood glucose (see chapters on Nutrition and Adolescence). Alcohol impairs the glucose counterregulation in subjects with diabetes by inhibiting gluconeogenesis (but not glycogenolysis) (117–120). Accordingly, hypoglycemia (especially night time) becomes more likely and is best avoided when participating in exercise, especially as alcohol may also impair performance.

While not confined to people with diabetes, the risk of dehydration should be borne in mind lest too much focus be kept upon glucose control. Even a 1% decrease in body mass due to dehydration may impair performance (121). In practice, both needs can often be met by using specially formulated drinks, but if dehydration is a risk, sugar free fluids should also be taken. Fluid intake should match sweat and hyperventilation losses, such that there is no change in body weight pre- versus post-exercise. Fluid intake may need to be as great as 1.3 L/h in adolescents exercising in hot and humid environments (122).

Monitoring

Blood glucose monitoring is the key for the active child with diabetes so that trends in glycemic responses can be identified. Records should include notes of their blood glucose, the timing, duration, and intensity of exercise, as well as the strategies used to maintain glucose concentrations in the normal range. Measurements of glucose should be taken before, during, and after the end of exercise with particular attention paid to the direction of change in glycemia.

It can be especially useful, where a young person is involved in multiple sports or different types of training/competition for them to keep records in a structure that allows similar elements (e.g., all the gym sessions or competition days) to be looked at together.

Monitoring several hours after exercise and before bed is particularly critical on days where strenuous activities occur, as nocturnal hypoglycemia is common. It remains controversial whether certain bedtime BG levels predict nocturnal hypoglycemia and predictions are particularly difficult after exercise. In one hospital-based study where 34% had night-time hypoglycemia using a twice daily regimen NPH as basal insulin, a bedtime blood glucose of less than 7 mmol/L (125 mg/dL) suggested particular risk for nocturnal hypoglycemia (123), while another study using long-acting basal analogs or pumps found a lower frequency of 13% but no threshold for nocturnal hypoglycemia risk after exercise in the afternoon (85).

CGM has proven to be a valuable adjunct to blood glucose monitoring in both the prevention and early detection of exercise-induced hypoglycemia (98, 99) and during a sports camp detected significantly more episodes of hyperglycemia and hypoglycemia than frequent blood glucose testing (124). With CGM it was also shown that exercise-induced hypoglycemia could be reduced by using value and trend information along with a new CHO intake program (95).

Caution should be taken when using BG meters in extreme temperatures (125). Meters using glucose dehydrogenase may give more accurate readings at high altitude. In circumstances where control solution is used to check the meter, e.g., on a long hike, further criteria apply with the solution only being accurate between 15 and 30°C. In cold environments such as skiing, keeping a meter and strips inside several layers of clothes close to the body will usually avoid inaccurate readings.

Special care should be taken at high altitude where the symptoms of hypoglycemia may be confused with those of hypoxia/altitude sickness. Taking acetazolamide to prevent or treat altitude sickness may contribute to an increased risk of ketoacidosis in a person with diabetes (126). However, in another report, 73% of the participants with diabetes used acetazolamide without side effects (127).

School activities and diabetes camps

While this article is aimed principally at the practicalities of managing intense and/or prolonged physical activity, it is clear that the advice can be tailored for more moderate exercise. In the normal school week, most young people will have at least one period of physical education, and how they deal with avoiding hypoglycemia will be dependent upon all of the factors mentioned above.

Some earlier studies have shown that school time may be one of the highest providers of activity to youth (128–131). This is particularly relevant as the school environment has the potential to encourage physical activity in youth through physical education lessons, extracurricular activities (structured physical activity), and during recess or lunchtime (discretionary physical activity).

For many, all that will be required for a 30 min recess break is a small snack of 10–15 g CHO, e.g., a fruit or fruit juice, dried fruit, a cereal, fruit or granola bar or sports bar. This may also be a convenient opportunity to allow a treat such as chocolate or a few sweets. Chocolate contains fat which will cause the sugar to be absorbed more slowly (132). This can make it more suitable for low-grade longer-lasting activity, e.g., hiking, swimming, or long walks. However, the extra calories will not benefit a child with weight problems.

Where a multi-injection regimen or a pump is being used, a reduction in the pre-exercise bolus or setting a temporary basal rate may be appropriate (see Table 3 below).

For pump patients, a short period of disconnection may be best to allow free activity.

For longer periods of physical activity (>60 min), a reduction in basal insulin by 30–50% should be considered, along with CHO snacks being provided.

Activity weeks are now a common part of the school curriculum and many young people with diabetes also have the opportunity to attend dedicated diabetes camps. These two situations differ mainly in the expertise available, with the latter usually being managed and monitored by diabetes professionals with advice about adjustments of insulin and food on-site.

Clinical professionals can gain much more insight into the day-to-day management of diabetes by participating in diabetes camps and in some countries this is now a training requirement.

The benefits of spending a week being active in the open air are obvious and self-esteem is often improved, and where the activity is shared with others with diabetes, there are real opportunities to learn better ways of coping. Camps for children with diabetes that include counseling on nutrition and insulin adjustments for exercise can result in improved glycemic control (133–135).

Insulin doses may have to be reduced substantially to prevent hypoglycemia in a camp setting, especially in children not accustomed to physical activity, and it is wise to begin with a 20–25% reduction in TDD (136). A more recent study by Miller et al. was conducted on 256 children aged 7–15 yr attending a week long summer camp (137). They reduced all children's insulin by 10% (55% were on pumps). Sixty percent of them had at least one episode of hypoglycemia on the first day. While, overall, insulin doses did not decrease further during the camp, the number of hypoglycemic episodes decreased. There was a difference between pumps and injections with children using injections requiring around an extra 8% reduction. They also noted that the older children were more likely to have hypoglycemic episodes. So it would seem that consideration of these factors may be wise before recommending the scale of insulin reduction.

When being physically active for a prolonged period, for example, on a skiing trip or an outward bound camp, insulin sensitivity will increase after 1–2 d which will probably call for substantially lowered insulin doses (decreased by 20% or sometimes even 50%, especially if not used to hard physical exercise). The increased insulin sensitivity will continue for at least a couple of days after returning home (81).

Where young people will be cared for by non-clinical professionals (e.g., teachers), it is vital that

both the adults and the child/adolescent are provided with appropriate verbal and written information as well as emergency contact telephone numbers.

The emergence of 'cloud technology' will afford even better opportunities to support children and young people participating in camps and activities away from home but care will be required not to overstep and impinge upon the development of independence.

Special mention should be made of the need to plan ahead. Activities often last longer than anticipated so extra snacks and hypoglycemia remedies should always be carried. Diabetes educators may meet with parents, school, and support staff to ensure that a child's participation can be planned properly.

While very rare, it may occasionally be advisable for a diabetes team to recommend to a school that a young person should not go on a school activity week. For example, safety might be compromised if the person with diabetes has exhibited dangerous behavior such as frequent omission of insulin or episodes of disabling hypoglycemia. The negative experience from handling a difficult child and the impact upon the others in the group might prejudice the prospects for future children with diabetes.

Miscellaneous advice for unusual activities

Everything possible should be done to support a young person with diabetes who has serious sporting aspirations, or simply wants to understand how best to manage their control while participating. However, diabetes care teams have a duty of care and there are occasions when medical 'certification' is required before participation is allowed. Examples include diving and boxing. It would be negligent to provide such certification without careful consideration of the overall control and knowledge of the participant, as well as the possible impact of any other health factors such as diabetes complications. It may be possible to use a little leverage here to persuade the young person that it is in their interest to work with the team to improve their self-management.

Participation in almost any sport or exercise is likely to be safer in company, but for the person with diabetes this is even more important. At very least, one companion should know something about diabetes and how to recognize and manage hypoglycemia. Every participant in a sports team should be aware of a person with diabetes and know where to find the person's hypoglycemia remedies.

It is good practice to have 'Diabetes ID' somewhere on the body – preferably in the form of a durable bracelet or necklace.

Taking account of diabetes in other extreme situations may be lifesaving, e.g., the signs and symptoms of exhaustion and hypothermia could easily be confused with hypoglycemia. It is always safer to assume that the latter is making some contribution and to check blood glucose or treat expectantly.

Diving clubs in the UK, as well as in many other countries, have allowed individuals with diabetes to dive under certain carefully controlled circumstances (138), while in Australia, New Zealand, and Norway, only people with diet-controlled diabetes are allowed to dive (139). The suggested age limit in the UK is $\geq$18 yr ($\geq$16 yr if taking part in a special training program) (140). In the USA, the same age limits apply, and teenagers are only allowed to dive after counseling by a physician and with letter stating they understand how to care for their diabetes during a dive. This letter is usually only provided to teenagers diving with their parents and after completing diving certification (140) (http://www.diversalertnetwork.org/news/download/SummaryGuidelines.pdf). In all countries where recreational scuba diving is allowed when diagnosed with T1D, the individual has to be declared as 'fit to dive' by a physician and this should also be continuously reevaluated (140). Specifically, the individual should have had no severe hypoglycemic episodes in the last 12 months.

A large number of dives performed by individuals with diabetes has been reported where no deaths, episodes of decompression illness, or hypoglycemia occurred (141), even in 16- to 17-yr old adolescents (142). In another report, hypoglycemic events were present in very small numbers, with no adverse outcome (143). Divers Alert Network (DAN) found 1.5% of participants having diabetes in a group of 1180 divers in Project Dive Exploration (144). In this report, 4 of 101 accidents involved diabetes that could indicate that individuals with diabetes are exposed to a higher risk than healthy individuals.

Repetitive episodes of hypoglycemia should be avoided during days before diving, as this could blunt the hormonal response during subsequent exercise or hypoglycemia (92).

The use of downloaded data regarding 2 wk of home glucose measurements made it possible to detect those who are suitable for diving.

In order to prevent episodes of hypoglycemia during the dive, a monitoring schedule is recommended with assessment of glucose levels via finger pricking 60, 30, and 10 min pre-dive and immediately post-dive (145). The same result was found when analyzing data from a CGM before, during, and after dive (146).

Those individuals with T1D who are permitted to dive should be trained to signal 'L' (low) for hypoglycemia (signal performed with the hand while diving). For safety reasons they should also be trained to use a fructose/glucose gel for oral ingestion below the surface, if signs of hypoglycemia are present during dive (146).

Type 2 diabetes

As opposed to the situation in T1D, there is no question that exercise has a direct and important part in the treatment of T2D. Exercise results in changes in body composition, reducing the amount of fat and increasing the amount of lean tissue: muscle, fibers, and bone. This increases the metabolic rate, reduces blood pressure and low-density lipoprotein (LDL) cholesterol, and increases high-density lipoprotein (HDL), reducing the risk of cardiovascular morbidity and mortality (147). The vast majority of studies on T2D and exercise have been done in adults, but there is every reason to believe that the results are applicable to adolescents as well.

Affected individuals and family members of adolescents in whom T2D has been diagnosed have lifestyles characterized by minimal physical activity (148) and fitness (149).

A twice-per-week 16-wk resistance training program significantly increased insulin sensitivity in overweight adolescents independent of changes in body composition (150).

Large clinical trials in adults with impaired glucose tolerance demonstrate that lifestyle interventions including exercise can reduce the incidence of T2D (151).

In a meta-analysis it was found that exercise training reduced HbA1c by an amount that should decrease the risk of diabetic complications. This effect was not mediated primarily by weight loss (152).

The incidence of hypoglycemia in T2D is lower than in T1D, partly because counterregulatory mechanisms are much less affected, but patients taking insulin or sulfonylurea medication (especially long-acting preparations) may require reduction in doses (153, 154).

Diabetes complications

Competitive sports are generally safe for anyone with T1D who is in good metabolic control and without long-term complications (155). However, patients who have proliferative retinopathy or nephropathy should avoid exercise conditions that can result in high arterial blood pressures (systolic pressures >180 mm Hg), such as lifting heavy weights (or any tasks in which a Valsalva manoeuvre is involved) or performing high-intensity sprints (156) or a cold bath after a sauna. Patients with complications should be monitored with ambulatory blood pressure measurement during exercise. Patients with peripheral neuropathy should be careful to avoid blisters and cuts and should avoid running and other sports that involve excessive wear of legs and feet (156). See reference 155 for more detailed advice on diabetes complications and exercise, and (157) for a more complete lists of sport-specific advice.

Diabetes and bone

The relationship between diabetes and osteopenia has been known since the 1950s but there has been much conflicting evidence. More recent studies have confirmed that children and adolescents with T1D do appear to have reduced bone mineral density compared with their non-diabetic peers (inversely correlated with HbA1c) (158). Whether or not this is, in turn, influenced by physical activity is interesting given the widespread evidence that children generally are not meeting the published targets for activity. Salvatoni et al. (159) studied 57 children and adolescents with diabetes and 57 controls and followed them with accelerometers to assess activity. Like others, they found that bone mineral density was less in diabetes but they also found a direct correlation between the average time per week spent doing physical activity and bone mineral content. Their findings were confirmed by Heilman et al. (160) who found the most significant reductions in bone mineral content and bone mineral density in boys with diabetes and that the boys were also the least active.

Contrary evidence was presented in 2010 by Maggio et al. who found that bone mineral density was normal during growth in 32 children with diabetes but that markers of bone turnover were decreased (161). Further support for abnormal bone metabolism in diabetes was demonstrated by Hamed et al. in 2011 when they studied 36 children and adolescents with diabetes and 15 controls and found that the group with diabetes had higher phosphate and pararthyroid hormone levels with significantly lower levels of calcium, IGF-1, and 25(OH)D. They also showed total body osteopenia-osteoporosis in 94.4% (total body) (162).

A prospective study by Maggio et al. in 2012 looked at the impact of two 90 min sessions per week of weight-bearing exercise for 9 months (ball games, jumping, rope-skipping, and gymnastics) upon bone mineral density in 27 diabetic and 32 healthy children(163). After the intervention the cohort of diabetic and healthy children randomized to exercise had similar measures of bone mineral density and these were significantly different from the non-intervention group.

Conflicts of interest

The authors have declared no conflicts of interest.

References

1. Roberts L, Jones TW, Fournier PA. Exercise training and glycemic control in adolescents with poorly controlled type 1 diabetes mellitus. J Pediatr Endocrinol 2002: 15: 621–627.

2. Sarnblad S, Ekelund U, Aman J. Physical activity and energy intake in adolescent girls with type 1 diabetes. Diabet Med 2005: 22: 893–899.
3. Ligtenberg PC, Blans M, Hoekstra JB, van der Tweel I, Erkelens DW. No effect of long-term physical activity on the glycemic control in type 1 diabetes patients: a cross-sectional study. Neth J Med 1999: 55: 59–63.
4. Ruzic L, Sporis G, Matkovic BR. High volume-low intensity exercise camp and glycemic control in diabetic children. J Paediatr Child Health 2008: 44: 122–128.
5. Aouadi R, Khalifa R, Aouidet A et al. Aerobic training programs and glycemic control in diabetic children in relation to exercise frequency. J Sports Med Phys Fitness 2011: 51: 393–400.
6. Herbst A, Bachran R, Kapellen T, Holl RW. Effects of regular physical activity on control of glycemia in pediatric patients with type 1 diabetes mellitus. Arch Pediatr Adolesc Med 2006: 160: 573–577.
7. Younk L, Tate D, Davis SN. Physical activity in adolescents with type 1 diabetes: is more better for glycemic control? Pediatr Diabetes 2009: 10: 231–233.
8. Nocon M, Hiemann T, Muller-Riemenschneider F, Thalau F, Roll S, Willich SN. Association of physical activity with all-cause and cardiovascular mortality: a systematic review and meta-analysis. Eur J Cardiovasc Prev Rehabil 2008: 15: 239–246.
9. Riddell MC, Perkins BA. Type 1 diabetes and vigorous exercise: applications of exercise physiology to patient management. Can J Diabetes 2006: 30: 63–71.
10. Margeirsdottir HD, Larsen JR, Brunborg C, Overby NC, Dahl-Jorgensen K. High prevalence of cardiovascular risk factors in children and adolescents with type 1 diabetes: a population-based study. Diabetologia 2008: 51: 554–561.
11. Roche DM, Edmunds S, Cable T, Didi M, Stratton G. Skin microvascular reactivity in children and adolescents with type 1 diabetes in relation to levels of physical activity and aerobic fitness. Pediatr Exerc Sci 2008: 20: 426–438.
12. Seeger JPH, Thijssen DHJ, Noordam K, Cranen MEC, Hopman MTE, Nijhuis-van der Sanden MWG. Exercise training improves physical fitness and vascular function in children with type 1 diabetes. Diabetes Obes Metab 2011: 13: 382–384.
13. Reddigan JI, Riddell MC, Kuk JL. The joint association of physical activity and glycaemic control in predicting cardiovascular death and all-cause mortality in the US population. Diabetologia 2012: 55: 632–635.
14. Manohar C, Levine JA, Nandy DK et al. The effect of walking on postprandial glycemic excursion in patients with type 1 diabetes and healthy people. Diabetes Care 2012: 35: 2493–2499.
15. Nordfeldt S, Ludvigsson J. Fear and other disturbances of severe hypoglycaemia in children and adolescents with type 1 diabetes mellitus. J Pediatr Endocrinol 2005: 18: 83–91.
16. Artero EG, Ruiz JR, Ortega FB et al. Muscular and cardiorespiratory fitness are independently associated with metabolic risk in adolescents: the HELENA study. Pediatr Diabetes 2011: 12: 704–712.
17. Jiménez-Pavón D, Ruiz JR, Ortega FB et al. Physical activity and markers of insulin resistance in adolescents: role of cardiorespiratory fitness levels – the HELENA study. Pediatr Diabetes 2013: 14: 249–258.
18. Brouwer SI, Stolk RP, Liem ET, Lemmink KAPM, Corpeleijn E. The role of fitness in the association between fatness and cardiometabolic risk from childhood to adolescence. Pediatr Diabetes 2013: 14: 57–65.
19. Lukacs A, Mayer K, Juhasz E, Varga B, Fodor B, Barkai L. Reduced physical fitness in children and adolescents with type 1 diabetes. Pediatr Diabetes 2012: 13: 432–437.
20. Williams BK, Guelfi KJ, Jones TW, Davis EA. Lower cardiorespiratory fitness in children with type 1 diabetes. Diabet Med 2011: 28: 1005–1007.
21. MacMillan FKA, Mutrie N, Matthews L, Robertson K, Saunders DH. A systematic review of physical activity and sedentary behaviour intervention studies in youth with type 1 diabetes: study characteristics, intervention design and efficacy. Pediatr Diabetes 2014: 15: 175–189.
22. Thorell A, Hirshman MF, Nygren J et al. Exercise and insulin cause GLUT-4 translocation in human skeletal muscle. Am J Physiol 1999: 277 (4 Pt 1): E733–E741.
23. Fahey AJ, Paramalingam N, Davey RJ, Davis EA, Jones TW, Fournier PA. The effect of a short sprint on postexercise whole-body glucose production and utilization rates in individuals with type 1 diabetes mellitus. J Clin Endocrinol Metab 2012: 97: 4193–4200.
24. Temple MY, Bar-Or O, Riddell MC. The reliability and repeatability of the blood glucose response to prolonged exercise in adolescent boys with IDDM. Diabetes Care 1995: 18: 326–332.
25. Petersen KF, Price TB, Bergeron R. Regulation of net hepatic glycogenolysis and gluconeogenesis during exercise: impact of type 1 diabetes. J Clin Endocrinol Metab 2004: 89: 4656–4664.
26. Komatsu WR, Gabbay MAL, Castro ML et al. Aerobic exercise capacity in normal adolescents and those with type 1 diabetes mellitus. Pediatr Diabetes 2005: 6: 145–149.
27. Adolfsson P, Nilsson S, Albertsson-Wikland K, Lindblad B. Hormonal response during physical exercise of different intensities in adolescents with type 1 diabetes and healthy controls. Pediatr Diabetes 2012: 13: 587–596.
28. Cuenca-Garcia M, Jago R, Shield JPH, Burren CP. How does physical activity and fitness influence glycaemic control in young people with type 1 diabetes? Diabet Med 2012: 29: e369–e376.
29. Arslanian S, Nixon PA, Becker D, Drash AL. Impact of physical fitness and glycemic control on in vivo insulin action in adolescents with IDDM. Diabetes Care 1990: 13: 9–15.
30. Guelfi KJ, Jones TW, Fournier PA. The decline in blood glucose levels is less with intermittent high-intensity compared with moderate exercise in individuals with type 1 diabetes. Diabetes Care 2005: 28: 1289–1294.

31. Maran A, Pavan P, Bonsembiante B et al. Continuous glucose monitoring reveals delayed nocturnal hypoglycemia after intermittent high-intensity exercise in nontrained patients with type 1 diabetes. Diabetes Technol Ther 2010: 12: 763–768.
32. Iscoe KE, Riddell MC. Continuous moderate-intensity exercise with or without intermittent high-intensity work: effects on acute and late glycaemia in athletes with Type 1 diabetes mellitus. Diabet Med 2011: 28: 824–832.
33. Bussau VA, Ferreira LD, Jones TW, Fournier PA. The 10-s maximal sprint: a novel approach to counter an exercise-mediated fall in glycemia in individuals with type 1 diabetes. Diabetes Care 2006: 29: 601–606.
34. Davey RJ, Paramalingam N, Retterath AJ et al. Antecedent hypoglycaemia does not diminish the glycaemia-increasing effect and glucoregulatory responses of a 10 s sprint in people with type 1 diabetes. Diabetologia 2014: 57: 1111–1118.
35. McMahon SK, Ferreira LD, Ratnam N et al. Glucose requirements to maintain euglycemia after moderate-intensity afternoon exercise in adolescents with type 1 diabetes are increased in a biphasic manner.[see comment]. J Clin Endocrinol Metab 2007: 92: 963–968.
36. Tonoli C, Heyman E, Roelands B et al. Effects of different types of acute and chronic (training) exercise on glycaemic control in type 1 diabetes mellitus: a meta-analysis. Sports Med 2012: 42: 1059–1080.
37. Yardley JE, Kenny GP, Perkins BA et al. Effects of performing resistance exercise before versus after aerobic exercise on glycemia in type 1 diabetes. Diabetes Care 2012: 35: 669–675.
38. Yardley JE, Sigal RJ, Riddell MC, Perkins BA, Kenny GP. Performing resistance exercise before versus after aerobic exercise influences growth hormone secretion in type 1 diabetes. Appl Physiol Nutr Metab 2014: 39: 262–265.
39. Ruegemer JJ, Squires RW, Marsh HM et al. Differences between prebreakfast and late afternoon glycemic responses to exercise in IDDM patients. Diabetes Care 1990: 13: 104–110.
40. Berger M, Berchtold P, Cuppers HJ et al. Metabolic and hormonal effects of muscular exercise in juvenile type diabetics. Diabetologia 1977: 13: 355–365.
41. Galassetti P, Riddell MC. Exercise and type 1 diabetes (T1DM). Compr Physiol 2013: 3: 1309–1336.
42. Kreisman SH, Halter JB, Vranic M, Marliss EB. Combined infusion of epinephrine and norepinephrine during moderate exercise reproduces the glucoregulatory response of intense exercise. Diabetes 2003: 52: 1347–1354.
43. Marliss EB, Vranic M. Intense exercise has unique effects on both insulin release and its roles in glucoregulation: implications for diabetes. Diabetes 2002: 51 (Suppl. 1): S271–S283.
44. Baldi JC, Cassuto NA, Foxx-Lupo WT, Wheatley CM, Snyder EM. Glycemic status affects cardiopulmonary exercise response in athletes with type I diabetes. Med Sci Sports Exerc 2010: 42: 1454–1459.
45. Wanke T, Auinger M, Formanek D et al. Defective endogenous opioid response to exercise in type I diabetic patients. Metabolism 1996: 45: 137–142.
46. Riddell MC, Bar-Or O, Gerstein HC, Heigenhauser GJ. Perceived exertion with glucose ingestion in adolescent males with IDDM. Med Sci Sports Exerc 2000: 32: 167–173.
47. Bar-Or O, Ward S. Rating of Perceived Exertion in Children. In: Bar-Or O, ed. Advances in Pediatric Sports Science. Champaign, IL: Human Kinetics, 1989, pp 151–168.
48. Jenni S, Oetliker C, Allemann S et al. Fuel metabolism during exercise in euglycaemia and hyperglycaemia in patients with type 1 diabetes mellitus – a prospective single-blinded randomised crossover trial. Diabetologia 2008: 51: 1457–1465.
49. Kelly D, Hamilton JK, Riddell MC. Blood glucose levels and performance in a sports cAMP for adolescents with type 1 diabetes mellitus: a field study. Int J Pediatr 2010 (available from http://www.ncbi.nlm.nih.gov/pubmed/20811595).
50. Heyman E, Briard D, Gratas-Delamarche A, Delamarche P, De Kerdanet M. Normal physical working capacity in prepubertal children with type 1 diabetes compared with healthy controls. Acta Paediatr 2005: 94: 1389–1394.
51. Stettler C, Jenni S, Allemann S et al. Exercise capacity in subjects with type 1 diabetes mellitus in eu- and hyperglycaemia. Diabetes Metab Res Rev 2006: 22: 300–306.
52. Gonder-Frederick LA, Zrebiec JF, Bauchowitz AU et al. Cognitive function is disrupted by both hypo- and hyperglycemia in school-aged children with type 1 diabetes: a field study. Diabetes Care 2009: 32: 1001–1006.
53. Tuominen JA, Karonen SL, Melamies L, Bolli G, Koivisto VA. Exercise-induced hypoglycaemia in IDDM patients treated with a short-acting insulin analogue. Diabetologia 1995: 38: 106–111.
54. Wasserman DH, Kang L, Ayala JE, Fueger PT, Lee-Young RS. The physiological regulation of glucose flux into muscle in vivo. J Exp Biol 2011: 214 (Pt 2): 254–262.
55. Koivisto VA, Felig P. Effects of leg exercise on insulin absorption in diabetic patients. N Engl J Med 1978: 298: 79–83.
56. Peter R, Luzio SD, Dunseath G et al. Effects of exercise on the absorption of insulin glargine in patients with type 1 diabetes. Diabetes Care 2005: 28: 560–565.
57. Bracken RM, West DJ, Stephens JW, Kilduff LP, Luzio S, Bain SC. Impact of pre-exercise rapid-acting insulin reductions on ketogenesis following running in type 1 diabetes. Diabet Med 2011: 28: 218–222.
58. Arutchelvam V, Heise T, Dellweg S, Elbroend B, Minns I, Home PD. Plasma glucose and hypoglycaemia following exercise in people with type 1 diabetes: a comparison of three basal insulins. Diabet Med 2009: 26: 1027–1032.
59. Chu L, Hamilton J, Riddell MC. Clinical management of the physically active patient with type 1 diabetes. Phys Sportsmed 2011: 39: 64–77.
60. Riddell MC, Iscoe KE. Physical activity, sport, and pediatric diabetes. Pediatr Diabetes 2006: 7: 60–70.
61. Perrone C, Laitano O, Meyer F. Effect of carbohydrate ingestion on the glycemic response of type 1 diabetic adolescents during exercise. Diabetes Care 2005: 28: 2537–2538.

62. Riddell MC, Bar-Or O. Handbook of Exercise in Diabetes: Children and Adolescents. 2nd edn. Alexandria, VA: American Diabetes Association, 2002: xvii: 699.
63. Grimm JJ, Ybarra J, Berne C, Muchnick S, Golay A. A new table for prevention of hypoglycaemia during physical activity in type 1 diabetic patients. Diabetes Metab 2004: 30: 465–470.
64. Dube M-C, Lavoie C, Galibois I, Weisnagel SJ. Nutritional strategies to prevent hypoglycemia at exercise in diabetic adolescents. Med Sci Sports Exerc 2012: 44: 1427–1432.
65. Kalergis M, Schiffrin A, Gougeon R, Jones PJ, Yale JF. Impact of bedtime snack composition on prevention of nocturnal hypoglycemia in adults with type 1 diabetes undergoing intensive insulin management using lispro insulin before meals: a randomized, placebo-controlled, crossover trial. Diabetes Care 2003: 26: 9–15.
66. Hernandez JM, Moccia T, Fluckey JD, Ulbrecht JS, Farrell PA. Fluid snacks to help persons with type 1 diabetes avoid late onset postexercise hypoglycemia. Med Sci Sports Exerc 2000: 32: 904–910.
67. Frid A, Ostman J, Linde B. Hypoglycemia risk during exercise after intramuscular injection of insulin in thigh in IDDM. Diabetes Care 1990: 13: 473–477.
68. Berger M, Cuppers HJ, Hegner H, Jorgens V, Berchtold P. Absorption kinetics and biologic effects of subcutaneously injected insulin preparations. Diabetes Care 1982: 5: 77–91.
69. de Mol P, de Vries ST, de Koning EJP, Gans ROB, Tack CJ, Bilo HJG. Increased insulin requirements during exercise at very high altitude in type 1 diabetes. Diabetes Care 2011: 34: 591–595.
70. Rave K, Heise T, Weyer C et al. Intramuscular versus subcutaneous injection of soluble and lispro insulin: comparison of metabolic effects in healthy subjects. Diabet Med 1998: 15: 747–751.
71. Bailey RC, Olson J, Pepper SL, Porszasz J, Barstow TJ, Cooper DM. The level and tempo of children's physical activities: an observation study. Med Sci Sports Exerc 1995: 27: 1033–1041.
72. Rowland T. The biological basis of physical activity. Med Sci Sports Exerc 1998: 30: 392–399.
73. Tremblay MS, Leblanc AG, Janssen I et al. Canadian sedentary behaviour guidelines for children and youth. Appl Physiol Nutr Metab 2011: 36: 59–64 65–71.
74. Galler A, Lindau M, Ernert A, Thalemann R, Raile K. Associations between media consumption habits, physical activity, socioeconomic status, and glycemic control in children, adolescents, and young adults with type 1 diabetes. Diabetes Care 2011: 34: 2356–2359.
75. Bernardini AL, Vanelli M, Chiari G et al. Adherence to physical activity in young people with type 1 diabetes. Acta Biomed Ateneo Parmense 2004: 75: 153–157.
76. Sundberg F, Forsander G, Fasth A, Ekelund U. Children younger than 7 years with type 1 diabetes are less physically active than healthy controls. Acta Paediatr 2012: 101: 1164–1169.
77. Miculis CP, De Campos W, Boguszweski MC. Correlation between glycemic control and physical activity level in type 1 diabetes adolescents and children. J Phys Act Health 2014 (available from http://www.ncbi.nlm.nih.gov/pubmed/24508755).
78. Poortmans JR, Saerens P, Edelman R, Vertongen F, Dorchy H. Influence of the degree of metabolic control on physical fitness in type I diabetic adolescents. Int J Sports Med 1986: 7: 232–235.
79. Ebeling P, Tuominen JA, Bourey R, Koranyi L, Koivisto VA. Athletes with IDDM exhibit impaired metabolic control and increased lipid utilization with no increase in insulin sensitivity. Diabetes 1995: 44: 471–477.
80. Yardley JE, Sigal RJ, Perkins BA, Riddell MC, Kenny GP. Resistance exercise in type 1 diabetes. Can J Diabetes 2013: 37: 420–426.
81. Borghouts LB, Keizer HA. Exercise and insulin sensitivity: a review. Int J Sports Med 2000: 21: 1–12.
82. Gulve EA, Spina RJ. Effect of 7-10 days of cycle ergometer exercise on skeletal muscle GLUT-4 protein content. J Appl Physiol 1995: 79: 1562–1566.
83. Mikines KJ, Sonne B, Tronier B, Galbo H. Effects of acute exercise and detraining on insulin action in trained men. J Appl Physiol 1989: 66: 704–711.
84. Davey RJ, Howe W, Paramalingam N et al. The effect of midday moderate-intensity exercise on postexercise hypoglycemia risk in individuals with type 1 diabetes. J Clin Endocrinol Metab 2013: 98: 2908–2914.
85. Tsalikian E, Mauras N, Beck RW et al. Impact of exercise on overnight glycemic control in children with type 1 diabetes mellitus. J Pediatr 2005: 147: 528–534.
86. Tamborlane WV. Triple jeopardy: nocturnal hypoglycemia after exercise in the young with diabetes. J Clin Endocrinol Metab 2007: 92: 815–816.
87. Berardi JM, Price TB, Noreen EE, Lemon PWR. Postexercise muscle glycogen recovery enhanced with a carbohydrate-protein supplement. Med Sci Sports Exerc 2006: 38: 1106–1113.
88. Yardley JE, Iscoe KE, Sigal RJ, Kenny GP, Perkins BA, Riddell MC. Insulin pump therapy is associated with less post-exercise hyperglycemia than multiple daily injections: an observational study of physically active type 1 diabetes patients. Diabetes Technol Ther 2013: 15: 84–88.
89. Perkins BA, Riddell MC. Type 1 diabetes and exercise using the insulin pump to maximum advantage. Can J Diabetes 2006: 30: 72–80.
90. Sandoval DA, Guy DLA, Richardson MA, Ertl AC, Davis SN. Effects of low and moderate antecedent exercise on counterregulatory responses to subsequent hypoglycemia in type 1 diabetes. Diabetes 2004: 53: 1798–1806.
91. Bao S, Briscoe VJ, Tate DB, Davis SN. Effects of differing antecedent increases of plasma cortisol on counterregulatory responses during subsequent exercise in type 1 diabetes. Diabetes 2009: 58: 2100–2108.
92. Galassetti P, Tate D, Neill RA, Morrey S, Wasserman DH, Davis SN. Effect of antecedent hypoglycemia on counterregulatory responses to subsequent euglycemic exercise in type 1 diabetes. Diabetes 2003: 52: 1761–1769.

93. Tansey MJ, Tsalikian E, Beck RW et al. The effects of aerobic exercise on glucose and counterregulatory hormone concentrations in children with type 1 diabetes. Diabetes Care 2006: 29: 20–25.
94. Riddell MC, Bar-Or O, Ayub BV, Calvert RE, Heigenhauser GJ. Glucose ingestion matched with total carbohydrate utilization attenuates hypoglycemia during exercise in adolescents with IDDM. Int J Sport Nutr 1999: 9: 24–34.
95. Riddell MC, Milliken J. Preventing exercise-induced hypoglycemia in type 1 diabetes using real-time continuous glucose monitoring and a new carbohydrate intake algorithm: an observational field study. Diabetes Technol Ther 2011: 13: 819–825.
96. Dorchy H, Poortmans JR. Juvenile diabetes and sports. In: Bar-Or O, ed. The Child and Adolescent Athlete. Oxford: Blackwell Science, 1996.
97. MacDonald MJ. Postexercise late-onset hypoglycemia in insulin-dependent diabetic patients. Diabetes Care 1987: 10: 584–588.
98. Adolfsson P, Lindblad B. Glucose monitoring during various types of physical exercise in adolescents with diabetes. Am J Physiol Endocrinol Metab 2002: 15 (Suppl. 4): 1(Poster).
99. Riddell M, Perkins BA. Exercise and glucose metabolism in persons with diabetes mellitus: perspectives on the role for continuous glucose monitoring. J Diabetes Sci Technol 2009: 3: 914–923.
100. Garg S, Brazg RL, Bailey TS et al. Reduction in duration of hypoglycemia by automatic suspension of insulin delivery: the in-clinic ASPIRE study. Diabetes Technol Ther 2012: 14: 205–209.
101. Garg SK, Brazg RL, Bailey TS et al. Hypoglycemia begets hypoglycemia: the order effect in the ASPIRE in-clinic study. Diabetes Technol Ther 2014: 16: 125–130.
102. Taplin CE, Cobry E, Messer L, McFann K, Chase HP, Fiallo-Scharer R. Preventing post-exercise nocturnal hypoglycemia in children with type 1 diabetes. J Pediatr 2010: 157: 784–8.e1.
103. Sane T, Helve E, Pelkonen R, Koivisto VA. The adjustment of diet and insulin dose during long-term endurance exercise in type 1 (insulin-dependent) diabetic men. Diabetologia 1988: 31: 35–40.
104. Rabasa-Lhoret R, Bourque J, Ducros F, Chiasson JL. Guidelines for premeal insulin dose reduction for postprandial exercise of different intensities and durations in type 1 diabetic subjects treated intensively with a basal-bolus insulin regimen (ultralente-lispro). Diabetes Care 2001: 24: 625–630.
105. Frohnauer M, Liu K, Devlin J. Adjustment of basal lispro insulin in CSII to minimize glycemic fluctuations caused by exercise. Diab Res Clin Pract 2000: 50 (Suppl. 1): S80(Abstract).
106. Admon G, Weinstein Y, Falk B et al. Exercise with and without an insulin pump among children and adolescents with type 1 diabetes mellitus. Pediatrics 2005: 116: e348–e355.
107. Mitchell TH, Abraham G, Schiffrin A, Leiter LA, Marliss EB. Hyperglycemia after intense exercise in IDDM subjects during continuous subcutaneous insulin infusion. Diabetes Care 1988: 11: 311–317.
108. Danne T, Tsioli C, Kordonouri O et al. The PILGRIM Study: in silico modeling of a predictive low glucose management system and feasibility in youth with type 1 diabetes during exercise. Diabetes Technol Ther 2014: 21 (available from http://www.ncbi.nlm.nih.gov/pubmed/24447074).
109. Wahren J, Felig P, Hagenfeldt L. Physical exercise and fuel homeostasis in diabetes mellitus. Diabetologia 1978: 14: 213–222.
110. Guerci B, Tubiana-Rufi N, Bauduceau B et al. Advantages to using capillary blood beta-hydroxybutyrate determination for the detection and treatment of diabetic ketosis. Diabetes Metab 2005: 31 (4 Pt 1): 401–406.
111. Laffel L. Ketone bodies: a review of physiology, pathophysiology and application of monitoring to diabetes. Diabetes Metab Res Rev 1999: 15: 412–426.
112. Samuelsson U, Ludvigsson J. When should determination of ketonemia be recommended? Diabetes Technol Ther 2002: 4: 645–650.
113. Laffel LMB, Wentzell K, Loughlin C, Tovar A, Moltz K, Brink S. Sick day management using blood 3-hydroxybutyrate (3-OHB) compared with urine ketone monitoring reduces hospital visits in young people with T1DM: a randomized clinical trial. Diabet Med 2006: 23: 278–284.
114. Scheiner G. Think Like A Pancreas. New York: Marlowe & Company, 2004.
115. Walsh J, Roberts R. Pumping Insulin, 4th edn. San Diego: Torrey Pines Press, 2006.
116. Riddell M, Iscoe K. Physical activity, sport, and pediatric diabetes. Pediatr Diabetes 2006: 7: 60–70.
117. Siler SQ, Neese RA, Christiansen MP, Hellerstein MK. The inhibition of gluconeogenesis following alcohol in humans. Am J Physiol 1998: 275 (5 Pt 1): E897–E907.
118. Plougmann S, Hejlesen O, Turner B, Kerr D, Cavan D. The effect of alcohol on blood glucose in type 1 diabetes – metabolic modelling and integration in a decision support system. Int J Med Inform 2003: 70: 337–344.
119. Turner BC, Jenkins E, Kerr D, Sherwin RS, Cavan DA. The effect of evening alcohol consumption on next-morning glucose control in type 1 diabetes. Diabetes Care 2001: 24: 1888–1893.
120. Avogaro A, Beltramello P, Gnudi L et al. Alcohol intake impairs glucose counterregulation during acute insulin-induced hypoglycemia in IDDM patients. Evidence for a critical role of free fatty acids. Diabetes 1993: 42: 1626–1634.
121. Wilk B, Yuxia H, Bar-Or O. Effect of body hypohydration on aerobic performance of boys who exercise in the heat. Med Sci Sports Exerc 2002: 34 (Suppl. 1): XX–XX.
122. Petrie HJ, Stover EA, Horswill CA. Nutritional concerns for the child and adolescent competitor. Nutrition 2004: 20: 620–631.
123. Whincup G, Milner RD. Prediction and management of nocturnal hypoglycaemia in diabetes. Arch Dis Child 1987: 62: 333–337.
124. Adolfsson P, Nilsson S, Lindblad B. Continuous glucose monitoring system during physical exercise in adolescents with type 1 diabetes. Acta Paediatr 2011: 100: 1603–1609.

125. Oberg D, Ostenson C-G. Performance of glucose dehydrogenase-and glucose oxidase-based blood glucose meters at high altitude and low temperature. Diabetes Care 2005: 28: 1261.
126. Moore K, Vizzard N, Coleman C, McMahon J, Hayes R, Thompson CJ. Extreme altitude mountaineering and type 1 diabetes; the Diabetes Federation of Ireland Kilimanjaro Expedition. Diabet Med 2001: 18: 749–755.
127. Kalson NS, Davies AJ, Stokes S et al. Climbers with diabetes do well on Mount Kilimanjaro. Diabet Med 2007: 24: 1496.
128. Tudor-Locke CLS, Morgan CF, Beighle A, Pangrazi RP. Children's pedometer-determined physical activity during the segmented school day. Med Sci Sports Exerc 2006: 38: 1732–1738.
129. Guinhouya BC, Lemdani M, Vilhelm C, Hubert H, Apété GK, Durocher A. How school time physical activity is the "big one" for daily activity among schoolchildren: a semi-experimental approach. J Phys Act Health 2009: 6: 510–519.
130. Mota J, Silva P, Santos MP, Ribeiro JC, Oliveira J, Durate JA. Physical activity and school recess time: differences between the sexes and the relationship between children's playground physical activity and habitual physical activity. J Sports Sci 2005: 23: 269–275.
131. Long MW, Sobol AM, Cradock AL, Subramanian SV, Blendon RJ, Gortmaker SL. School-day and overall physical activity among youth. Am J Prev Med 2013: 45: 150–157.
132. Welch IM, Bruce C, Hill SE, Read NW. Duodenal and ileal lipid suppresses postprandial blood glucose and insulin responses in man: possible implications for the dietary management of diabetes mellitus. Clin Sci 1987: 72: 209–216.
133. Santiprabhob J, Likitmaskul S, Sriwijitkamol A et al. Improved glycemic control among Thai children and young adults with type 1 diabetes participating in the diabetes camp. J Med Assoc Thai 2005: 88 (Suppl. 8): S38–S43.
134. Post EM, Moore JD, Ihrke J, Aisenberg J. Fructosamine levels demonstrate improved glycemic control for some children attending a diabetes summer camp. Pediatr Diabetes 2000: 1: 204–208.
135. Strickland AL, McFarland KF, Murtiashaw MH, Thorpe SR, Baynes JW. Changes in blood protein glycosylation during a diabetes summer camp. Diabetes Care 1984: 7: 183–185.
136. Braatvedt GD, Mildenhall L, Patten C, Harris G. Insulin requirements and metabolic control in children with diabetes mellitus attending a summer camp. Diabet Med 1997: 14: 258–261.
137. Miller AR, Nebesio TD, DiMeglio LA. Insulin dose changes in children attending a residential diabetes camp. Diabet Med 2011: 28: 480–486.
138. Bryson P, Edge C, Lindsay D, Willshurst P. The case for diving diabetics. SPUMS J 1994: 24: 11–13.
139. Davies D. SPUMS statement on diabetes. SPUMS J 1992: 22: 31–32.
140. Pollock N, Uguccioni D, Dear G, eds. Diabetes and recreational diving: guidelines for the future. Proceedings of the Undersea and Hyperbaric Medical Society/Divers Alert Network. Durham, NC: Divers Alert Network, 2005.
141. Dear GdL, Pollock NW, Uguccioni DM, Dovenbarger J, Feinglos MN, Moon RE. Plasma glucose responses in recreational divers with insulin-requiring diabetes. Undersea Hyperb Med 2004: 31: 291–301.
142. Pollock NW, Uguccioni DM, Dear G, Bates S, Albushies TM, Prosterman SA. Plasma glucose response to recreational diving in novice teenage divers with insulin-requiring diabetes mellitus. Undersea Hyperb Med 2006: 33: 125–133.
143. Edge CJ, St Leger Dowse M, Bryson P. Scuba diving with diabetes mellitus – the UK experience 1991-2001. Undersea Hyperb Med 2005: 32: 27–37.
144. Pollock NW. Annual Diving Report - 2007 Edition (Based on 2005 data). Divers Alert Network: Durham NC, 2007.
145. Lerch MLC, Thurm U. Diabetes and diving: can the risk of hypoglycemia be banned? SPUMS J 1996: 26: 62–66.
146. Adolfsson POH, Jendle J. The benefits of continuous glucose monitoring and a glucose monitoring schedule in individuals with type 1 diabetes during recreational diving. J Diabetes Sci Technol 2008: 2: 778–784.
147. Hu FB, Stampfer MJ, Solomon C et al. Physical activity and risk for cardiovascular events in diabetic women. Ann Intern Med 2001: 134: 96–105.
148. Pinhas-Hamiel O, Standiford D, Hamiel D, Dolan LM, Cohen R, Zeitler PS. The type 2 family: a setting for development and treatment of adolescent type 2 diabetes mellitus. Arch Pediatr Adolesc Med 1999: 153: 1063–1067.
149. Faulkner MS. Cardiovascular fitness and quality of life in adolescents with type 1 or type 2 diabetes. J Spec Pediatr Nurs 2010: 15: 307–316.
150. Shaibi GQ, Cruz ML, Ball GDC et al. Effects of resistance training on insulin sensitivity in overweight Latino adolescent males. Med Sci Sports Exerc 2006: 38: 1208–1215.
151. Lindstrom J, Louheranta A, Mannelin M et al. The Finnish Diabetes Prevention Study (DPS): lifestyle intervention and 3-year results of diet and physical activity. Diabetes Care 2003: 26: 3230–3236.
152. Boule NG, Haddad E, Kenny GP, Wells GA, Sigal RJ. Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. JAMA 2001: 286: 1218–1227.
153. Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C, White RD. Physical activity/exercise and type 2 diabetes: a consensus statement from the American Diabetes Association. Diabetes Care 2006: 29: 1433–1438.
154. Zammitt NN, Frier BM. Hypoglycemia in type 2 diabetes: pathophysiology, frequency, and effects of different treatment modalities. Diabetes Care 2005: 28: 2948–2961.
155. Zinman B, Ruderman N, Campaigne BN, Devlin JT, Schneider SH, American Diabetes Association. Physical activity/exercise and diabetes. Diabetes Care 2004: 27 (Suppl. 1): S58–S62.
156. Wasserman DH, Zinman B. Exercise in individuals with IDDM. Diabetes Care 1994: 17: 924–937.

157. Colberg S. The Diabetic Athlete: Prescriptions for Exercise and Sport. Champaign, IL: Human Kinetics, 2001.
158. Lettgen B, Hauffa B, Mohlmann C, Jeken C, Reiners C. Bone mineral density in children and adolescents with juvenile diabetes: selective measurement of bone mineral density of trabecular and cortical bone using peripheral quantitative computed tomography. Horm Res 1995: 43: 173–175.
159. Salvatoni A, Mancassola G, Biasoli R et al. Bone mineral density in diabetic children and adolescents: a follow-up study. Bone 2004: 34: 900–904.
160. Heilman K, Zilmer M, Zilmer K, Tillmann V. Lower bone mineral density in children with type 1 diabetes is associated with poor glycemic control and higher serum ICAM-1 and urinary isoprostane levels. J Bone Miner Metab 2009: 27: 598–604.
161. Maggio ABR, Ferrari S, Kraenzlin M et al. Decreased bone turnover in children and adolescents with well controlled type 1 diabetes. J Pediatr Endocrinol Metab 2010: 23: 697–707.
162. Hamed EA, Abu Faddan NH, Adb Elhafeez HA, Sayed D. Parathormone – 25(OH)-vitamin D axis and bone status in children and adolescents with type 1 diabetes mellitus. Pediatr Diabetes 2011: 12: 536–546.
163. Maggio ABR, Rizzoli RR, Marchand LM, Ferrari S, Beghetti M, Farpour-Lambert NJ. Physical activity increases bone mineral density in children with type 1 diabetes. Med Sci Sports Exerc 2012: 44: 1206–1211.