# Diabetes Mellitus: Overview and Recommendations for Athletes and Coaches

**By: Emmett Campbell (87764149)**

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**School of Kinesiology, University of British Columbia**

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## Abstract

Diabetes is a common endocrine disorder that effects millions of North Americans and people all over the world. Type I diabetes is a specific form of diabetes that is related to dysfunctional insulin production and glucose regulation. Without proper management, there can be significant health effects, as well as impairments in the individual's ability to live their daily lives and participate in exercise and sport. While poorly managed diabetes can limit an individual's ability to participate and excel in sport, if properly managed the diabetic athlete will not be limited in their ability to both participate and excel. Consistency, diligence and a large amount of trial and error are necessary for an athlete to determine the steps they will need to take to gain a tight control over their blood glucose levels and to give themselves the best chance to succeed. This paper will outline the physiological basis of diabetes, its potential health effects, and will supply some recommendations for diet, insulin administration, training protocols, and other management strategies to help coaches and athletes develop management plans to best suit the athlete's specific needs. This will help to minimize the negative effects of their disease and allow for the easier participation in their respective sport.

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## Introduction

There are numerous athletes who suffer from various physiological impairments in the form of endocrine and metabolic disorders. These can create unique challenges in athletic training, sport performance, as well as numerous other aspects surrounding an athlete's lifestyle. The incidence of metabolic/endocrine disorders in adult populations in the United Stated is estimated be at least 5% of the population (Golden et al., 2009). It is also worth noting that the occurrence of these disorders are, as a whole, more common in ethnic minorities and communities of lower socio-economic status (Golden et al. 2009). The reason for this is not fully understood, but it is believed to be a result of poor diet, lack of education, sedentary lifestyle as well as potential genetic components (James et al., 1997). It is believed that the incidence of metabolic/endocrine disorders among the Canadian population, while less than the United States, is comparable (Riediger & Clara, 2011). The presence of certain physiological conditions/impairments can be detrimental to an athlete's training and performance and can complicate planning and preparation, affecting the duration of training, how long they can maintain high levels of intensity, how often they need to rest, when they must eat etc. While metabolic conditions certainly complicate athletic training and preparation there are often approaches and options that can negate or at least minimize the negative effects of their respective conditions.

Diabetes mellitus affects approximately 25.8 million Americans (Centre for Disease Control and Prevention 2011) and, according to Statistics Canada, 2.1 million Canadians over the age of 12 (2015). This makes diabetes the most common endocrine

disorder in North America. Diabetes is a life-altering disease and is one of the leading causes of death by disease. Assessing the specific connection between mortality and diabetes is difficult due to the occurrence of numerous vascular complications that can lead to stroke or heart disease (Hoffman, 2014). While Diabetes is a potentially damaging and life-threatening disease, proper monitoring, regulation and pharmaceutical/behavioural intervention can allow a person with the disease to live a long life without significant limitations. There are many athletes who, with proper disease management, have been able to compete at the highest levels of their respective sport, from Steve Redgrave who won his 5 Olympic gold medals in Rowing after he was diagnosed, to Jay Cutler who has played 11 of his 13 years in the NFL with diabetes. There are a host of successful athletes from a large spectrum of sport with different metabolic demands that have seen success and have successfully managed their diabetes. Having diabetes is no reason to avoid competing and participating in sport, from a recreational level to professional competition. In contrast, most of evidence, including data from the American Diabetes Association (2004) suggest that exercise and sport are not contraindicated in diabetic individuals but will promote positive health outcomes and potentially improve disease management with both type 1 and type 2 diabetes. While the positive health effects are more apparent in type 2 it has been shown to also improve type 1 (Wallberg-Henriksson, 1992)

The form of diabetes that this paper will predominantly investigate with regards to sport and performance is Diabetes mellitus type 1 or insulin dependent diabetes mellitus. Onset of type 1 often occurs in childhood and there is no cure. This presents the individual with the life-long challenge of management while maintaining a relatively

normal life. This is especially significant with regards to sport competition as the physiological implications of type 1 diabetes will require purposeful planning and consideration when it comes to sport involvement especially as the level of competition increases due to the inherent increases physical and physiological demands performance at a higher level will require. This paper will seek to differentiate type 1 diabetes from type 2, to outline the potential health risks associated with diabetes, and to give some recommendations and guidelines to help provide a beginning for the development of a management plan for the diabetic athlete in order to improve their sport participation outcomes.

**Basic Concepts**

To approach this subject, it is first important to describe and define certain physiologic components that are related to both type 1 and 2 diabetes and to provide a distinction to the two variations of the disease most common in the population.

Insulin is a peptide hormone that is produced by beta cells in the pancreas. It acts as the dominant anabolic hormone of the body. It functions by regulating the metabolism of fats, proteins, and carbohydrates – especially glucose - by regulating their absorption by the liver, fat cells and skeletal muscle. Once glucose is absorbed by the tissues it is used for energy production. This can be achieved through several metabolic pathways, such as the Krebs cycle or the Cori cycle, but the end goal is to restore the metabolic functional unit of energy at the cellular level: Adenosine Triphosphate (ATP). A healthy system at rest that does not require significant amounts

of readily-available sources of energy will produce increased levels of insulin in response to increased levels of blood glucose (from ingestion of carbohydrates, etc.). Consequently, the body's cells will uptake glucose and store it as either glycogen (via the process of glycogenesis) or fat (via the process of lipogenesis) (Griffin & Ojeda, 2000). Therefore, insulin has the action of an anabolic hormone, resulting in the conversion of smaller molecules in the blood into larger molecules within the cell. This glucose is stored to have relatively immediate access to molecules that can be converted into physiological energy substrates. Insulin also has the added effect of potentially promoting protein synthesis and preventing protein degradation in muscle (Gelfand & Barrett 87). Low levels of blood glucose result in low levels of insulin which promotes a catabolic state triggering the metabolism of glucagon and fat stores to meet the body's energetic demands that cannot be met by the present blood glucose levels.

Diabetes mellitus is a disease that is characterized by some dysfunction in the body's ability to regulate blood glucose levels as well as its inability to metabolize carbohydrates (Hoffman, 2004). There are predominantly two different forms of diabetes affecting the population, type 1 and type 2. Gestational diabetes, a third subtype of diabetes, results as a complication of pregnancy and is not within the scope of this paper.

**Type 1 Diabetes**

Type 1 diabetes is characterized as a multisystem disease. It is a chronic disease in which the metabolism of carbohydrates, fats and proteins is impaired due to a lack of, or

insufficient production of insulin by beta cells found in the islets of Langerhans in the pancreas (Hoffman, 2004). This diminished insulin production occurs as a result of lymphocytic infiltration and destruction of said beta cells. These cells make up about 1-2% of the total mass of the pancreas and exist in small groupings scattered throughout the pancreas. The beta cells produce insulin and the alpha cells of the islets of Langerhans produce glucagon and together allow for very tight control of blood glucose levels

With prolonged destruction, the beta-cell mass will continue to decline resulting in a decrease in insulin production to the point that there are no longer sufficient blood insulin levels to maintain proper levels of blood glucose. With the destruction of 80-90% of the pancreatic beta cells hyperglycemia begins to develop and it is at this stage that type 1 diabetes is generally diagnosable (Alberti & Zimmet, 1998). It is only with exogenous insulin that a person will be able to re-establish their normal blood glucose levels, prevent ketosis and the subsequent ketoacidosis, as well as normalizing their metabolic use of protein and fat (Perry et al., 2009). Ketoacidosis will result from the accumulation of ketones as a by-product of non-glucose-based metabolism. If Ketoacidosis persists it can result organ failure, coma, and death. The destruction of the beta cells is permanent and as of yet irreparable. This requires that people with type 1 diabetes take lifelong insulin therapy, and usually requires multidisciplinary care with the aim to establish glycemic control with the purpose of preventing or limiting the development of the damaging physiologic complications that can occur such as peripheral nerve damage, renal neuropathy etc.

Type 1 diabetes most often has a dramatic and sudden onset, usually occurring in children and adolescents (American Diabetes Association 2010), which is why it is often referred to as juvenile diabetes. This form of Diabetes mellitus accounts for between 5% and 10% of all diabetes cases. (IVY, Zderic, & Fogt, 1999) As a result of having low insulin levels blood glucose levels tend to remain high. The individuals with type 1 are more likely to become ketotic and require exogenous insulin. (Hoffman, 2004). This leads to several physiological abnormalities that require specific treatment to maintain health as well as specific consideration and preparation to allow for any physical activity especially training and competition. If the athlete becomes ketotic they will be at a metabolically unable to produce the energy that they will require to train and compete in sport.

Insulin deficiency can have significant effects on the metabolism of carbohydrates, lipids and proteins. (Saltiel & Kahn, 2001) With reduced insulin concentrations there will be a reduction of glucose uptake by skeletal muscle and the liver (Saltiel & Kahn, 2001). With a reduction in glucose uptake there will be an insufficient level of glucose available in the cell, and a reduction in glycogen synthesis will occur. This in turn results in the liver and muscle being driven to produce more glucose. This is achieved by the process of glycogenolysis, by which glucose molecules are produced via the catabolism of glycogen branches by phosphorylation. Another metabolic pathway that is activated in the absence of sufficient glucose is gluconeogenesis occurring predominantly in the liver. This process is characterized by the production of glucose molecules from non-carbohydrate carbon substrates, including proteins, lipids, and other metabolic by-products such as pyruvate and lactate. These processes will result in a hyperglycaemic

(high glucose concentration) state. Once this blood glucose concentration exceeds the kidneys abilities to reabsorb it, then glucose will appear in urine, a state known as glucourea. With the addition of glucose to the urine a state known as osmotic diuresis can occur as a result of the abnormal filtrate concentrations. This leads to drawing more water into the filtrate (down its osmotic gradient) resulting in a need to frequently urinate (polyuria). This frequent urination will cause greater levels of thirst and may even cause dehydration if water consumption is insufficient (polydipsia). This increased thirst and frequent urination are often the first noticeable signs of diabetes and often leads to its diagnosis. If this state of dehydration progresses too far it can lead to circulatory failure.

Type 1 Diabetes is officially diagnosed by meeting one of 4 testing parameters:

- Having symptomology and a blood sugar level of 200 mg/dL of higher
- Having a fasting (no food for more than 8 hrs) blood sugar level equal to or greater than 126 mgl/dL
- Have a 2hr oral glucose tolerance test (using 75g anhydrous glucose dissolved in water) result of 200 mg/dL of greater blood glucose level.
- Having a hemoglobin A1c that is 6.5% or higher

(ADA 2010)

With a decrease in insulin production and blood insulin concentration a decrease in triglyceride synthesis will also result. This occurs because of the systemic insufficient cellular glucose to meet metabolic demands driving the system to rely on other means of energy production. This in turn leads to an increase in circulating free fatty acids. With an increase in free fatty acids the liver will tend to use them as an energy substrate

resulting in the production of ketone bodies which will subsequently increase the risk of ketosis and metabolic acidosis. While acute, low-level ketosis is normal and not usually a risk, a prolonged state of ketosis and unusually high concentration along with the presence of unusually high concentrations of blood glucose can lead to a state of diabetic ketoacidosis. This state can occur rapidly (in a 24hr period) and is a leading cause of death in diabetes patients (American Diabetes Association, 2010). It can be identified by the symptoms of excessive thirst, frequent urination, nausea and vomiting, abdominal pain, weakness/fatigue, shortness of breath, fruity-scented breath, and confusion. Everyone involved with a diabetic athlete should be acutely aware of these symptoms and take immediate steps to prevent the progression of ketoacidosis. If the diabetic person has a blood sugar level >300mg/dl, has ketones present in their urine, or is suffering from multiple signs and symptoms, they should seek immediate emergency care (Flood & Constance, 2002).

**Potential Causes of Type 1**

It is currently suspected that autoimmunity is a primary factor that results in the pathophysiology of type 1 diabetes in the population at large (American Diabetes Association 2010) In specific people who are genetically susceptible it is suspected that a viral infection may stimulate the production of antibodies for a specific viral protein which may be antigenically similar to the individual's specific beta cell molecules resulting in an autoimmune response to them (Pociot & Lernmark, 2016). The specific cause of type 1 diabetes is currently unknown, but the prevailing thought is that it is caused by a combination of genetic and environmental factors. There are clear signs that indicate the heritability of type 1. A child's risk of developing type 1 is approximately

5% if the father has it, 8% if a sibling has it, and 3% if the mother has it. (Taplin & Barker, 2008).

While type 1 has very clear genetic links, there is also strong evidence to suggest that environmental factors play a key role in its expression. When looking at identical twins, when one child develops the condition, the child's twin, who has an identical genome, will only have a 30-50% likelihood of also developing diabetes (Owen, 2014). There is also evidence that would suggest that the occurrence of type 1 diabetes us dependent upon geographic region, as populations with very similar genetic origins may have upwards of a 10x difference in disease presentation depending on where they live (Owen, 2014)

**Type 2 Diabetes**

When speaking about diabetes it is important to provide distinction between type 1 diabetes (independent diabetes mellitus or IDDM) and type 2 diabetes (non-independent diabetes mellitus or NIDDM). While type 1 diabetes is generally characterized by an inability to produce or dysfunction in the production of insulin, type 2 diabetes is more associated with insulin resistance/insensitivity at the liver and skeletal muscle. Type 2 diabetics may have abnormally high or low blood insulin levels because of some dysfunction or dysregulation in the pancreas, but in most cases their insulin levels will be comparable to that of the normal population (Hoffman, 2014). Type 2 diabetes is primarily seen in adults and the risk for developing it increases with age (Hoffman, 2014). The precise cause of type 2 is unknown, but its occurrence has been

highly related to lifestyle as both obesity and a sedentary lifestyle have been shown to be strongly correlated with its occurrence (Ivy, Zderic, & Fogt, 1999). Type 2 diabetes accounts for approximately 90%-95% of all cases of diabetes (Ivy, Zderic, & Fogt, 1999). While it is possible for a person who has type 2 diabetes to be a competitive athlete, depending on their sport requirements, it is more often than not that the disease occurs alongside a lifestyle that would be counter to that of a competitive athlete. Exercise is also used as a primary form of treatment to type 2 diabetes and for a person to reach a level of fitness that would be required for competitive athletic pursuit it is likely that their diabetes would be treated/controlled by their athletic participation (Boule et al., 2001).

Insulin resistance refers to the body's inability to respond adequately to insulin levels in the blood. This can occur in the form of dysfunctional insulin responsiveness or sensitivity (Hoffman, 2014). Insulin responsiveness is related to the biological response to a maximally-stimulating insulin concentration, said response being potentially reduced in diabetics due to a post receptor defect, that being a dysfunction in signal transduction or phosphorylation pathway, or a defect or decrease in the glucose transporter (Ivy, Zderic, & Fogt, 1999). Insulin sensitivity is related to a biological response at a given submaximal concentration, with the response again being potentially reduced in diabetic populations due to an insulin receptor defect that being a decrease in the number of receptors or a decrease in the sensitivity of said receptors (Ivy, Zderic, & Fogt, 1999). In obese populations both hyperinsulinemia, a condition in which there is an excess in levels of circulating insulin relative to the levels of circulating glucose, as well as downregulation of insulin receptors which results in a decrease in

the number of insulin receptors and thereby increasing an individual's resistance to insulin by decreasing sensitivity is often observed (Kahn, Hull, & Ultzschneider, 2006). In many cases the individual's pancreas can produce and secrete higher levels of insulin to counter the deficiencies, but given prolonged stress and a predisposition towards diabetes, the capacity for compensation by the Beta cells may be overcome, or the cells may no longer be able to continue production at the heightened level (Griffin, & Ojeda, 2000). This will result in a glucose intolerance which means that the body is no longer able to physiologically cope with fluctuating levels of blood glucose (Griffin, & Ojeda, 2000).

**Potential Causes of Type 2**

While the specific cause of type 2 diabetes remains unknown, there are some mechanisms that have been proposed that might supply a potential cause for the disease. One mechanism relates to how a sedentary lifestyle might result in an excessive caloric balance causing fat storage in the form of adipocyte hypertrophy (Ivy, 1997). With the enlargement of adipocytes and a not equivalent increase in the number of insulin receptors, insulin receptor density may be reduced resulting in insulin resistance (Ivy, 1997). Insulin resistance may also be related to specific insulin receptor substrates being degraded or inhibited. It has also been shown that in type 2 diabetics there is a three-fold increase in lipid concentration in areas just deep to the sarcolemma, the outer cell membrane of a striated muscle fibre (Nielsen et al., 2010).

This has in turn been proposed to potentially result in dysregulation of skeletal muscle insulin sensitivity (Nielson et al., 2010). An increase in blood lipid concentrations will also be seen with the accumulation of free fatty acids in the blood they may cause a stimulation in gluconeogenesis as well as hepatic glucose output and inhibiting muscle glucose clearance that would normally be stimulated by the presence of insulin (Boden et al, 2001). If the free fatty acids also begin to accumulate within the muscle tissue itself, insulin resistance may occur along with a compensatory increase in beta-cell production (Ivy, 1997). If this cycle is continued then the Beta cells will eventually be overburdened resulting in the impairment of insulin production (Ivy, 1997) This may also lead to an increase in insulin-resistance and reduced free fatty acid clearance, accelerating hepatic glucose output and leading to the physiological condition we know as type 2 diabetes.

Another proposed mechanism is based on a genetic predisposition that can be triggered by a sedentary lifestyle. This proposed mechanism suggests that a genetic defect may exist in skeletal muscle which, in response to elevated blood glucose levels, will develop insulin resistance (Ivy, Zderic, & Fogt, 1999). It is thought that the hyperinsulemia that results due to elevated blood glucose levels will act to suppress free fatty acid oxidation, and lead to an increase in triglyceride storage and adipocyte hypertrophy (Ivy, Zderic, & Fogt, 1999). Similar to the previous proposed mechanism, this will result in further insulin resistance, the up-regulation of pancreatic beta cells and with this prolonged upregulation the eventual impairment of said Beta cells and an overall decrease in insulin production. This again in turn results in increased hepatic

glucose output, reduced free fatty acid clearance, and increased insulin resistance resulting in the diabetic state.

**Potential Health Risks**

While the diabetic athlete can perform, and participate at all levels of sport it is important to recognize the potential harm that can occur because of poor management, and erratic blood glucose levels. These risks are always present as the greater awareness of them there is the greater the likelihood that they can be prevented of caught before irreparable damage occurs.

Several complications that are often seen that occur concurrently or as a direct result of the physiologic state that is caused by type 1 diabetes. There is an increased risk of heart disease due to the increased deposition of atherosclerotic plaques that occur because of prolonged elevated blood glucose concentrations which is in turn associated with an increase in lipid synthesis and deposition on arterial walls (Hoffman, 2014). Atherosclerotic changes are also observed in cerebral and vascular beds and may lead to microvascular lesions in the kidneys and retinas, with diabetics often suffering from kidney disease as well as visual impairment or blindness. Other neuropathies may also develop in the peripheral nerves, the spinal cord and the brain (Nathan, 1993). This can result in loss of feeling in the hands and feet. Normal wound healing is also impaired in the extremities, resulting in minor damage becoming more permanent injuries as well as increasing the individual's susceptibility for infection (Nathan, 1993). These long-term risks to the type 1 diabetic can have lifelong

consequences and is thus very important to establish a tight control of blood glucose levels. Athletes, coaches and parents should be very aware of these potential complications and early signs, and must weigh the benefits and harm that sport participation might have, especially if said participation has a negative result on the diabetic individual's ability to maintain consistent and predictable blood glucose levels.

**Exercise Metabolism as it relates to Diabetes**

When talking about type 1 diabetes and the challenges it presents in sport performance it can be helpful to first identify and separate sport demands based the performance and training variables of intensity and duration of physical activity required. Without going into the complex energetic and metabolic pathways related to both the duration and intensity of physical activity, there are some basic concepts pertinent to how the dysfunctions of type 1 diabetes can affect exercise/performance and how these effects might be accounted for, avoided or mitigated all together.

When speaking about the metabolic demands of exercise/sport it is generally referring to how much energy that is required and which energetic pathways will be employed to best meet those demands. These pathways can be related to either requiring oxygen (aerobic) or not (anaerobic). While the pathways are distinct they all function to produce/utilize the fundamental unit of energy in the body: adenosine triphosphate (ATP). Most forms of exercise/sport will be a combination of both aerobic and anaerobic pathways and the extent to which each is involved is directly related to the intensity and the duration of the activity. Sport that requires maximal exertion for a

very brief time (8-10 seconds) will rely on the ATP-PC energy system (Hoffman 2014). This system utilizes ATP and PC (phosphocreatine) that is already present in the tissue and therefore immediately available for use (Hoffman, 2014). Any activity that requires continued near maximal exertion lasting upwards of 2 minutes will rely on anaerobic glycolysis. This pathway is characterized by the production of ATP by breaking down glucose into lactate. This energy pathway is limited as while it acts quickly to produce energy, it can only produce about 5% of glucose potential energy (Gastin, 2001). Once activity extends past this point the process of aerobic glycolysis becomes the primary source of energy production. This process fully metabolizes glucose molecules and is only functionally limited by the presence of glucose molecules and oxygen (Gastin, 2001).

With regards to type 1 diabetes, certain challenges arise in fuel regulation. In activities that are characterized by extended duration while exerting moderate intensity, often referred to as aerobic endurance, muscle glycogen stores will be depleted followed by the release of glucose from the liver which is triggered by a fall in insulin levels as well as a rise in glucagon levels (Wasserman & Zinman, 1994). This shifting in hormonal levels is necessary to achieve optimal glycogenolysis and gluconeogenesis (Wasserman et al., 1989). While this is the case in normal individuals, diabetics have a different hormonal response. In the diabetic person, injected insulin does not reduce with the onset of exercise and may even show a rise due to increased circulation and absorption of deposits in subcutaneous tissue that can result as exercise progresses (Grimm, 2005). Without the decrease in insulin levels relative over-insulation can result. This can then block hepatic glucose production which in turn increases the risk of

hypoglycaemia, characterized by a blood glucose concentration of < 70mg/dL (Horton et al. 2016) occurring after 20-60 minutes aerobic endurance exercise (Schiffren & Parikh, 1985). In Diabetics there is also a general predisposition to hypoglycaemia following exercise. This results from the increased transport of glucose into muscle and the liver after exercise has stopped to restore glycogen stores to pre-exercise levels. Insulin levels will persist and if there are inadequate blood glucose levels (Riddell & Iscoe, 2006). Hyperinsulination can result potentially leading to hypoglycaemia (Riddell & Iscoe, 2006). As the intensity of exercise approaches the upper aerobic threshold (%hr 70) the highest rate of glucose use occurs, further resulting in a higher risk of hypoglycaemia (Francescate et al., 2004). This will require the athlete to increase their glucose supplementation before during and after exercise in order to avoid or minimise the hypoglycaemic state.

With aerobic exercise lasting several hours the risk of hypoglycaemia is decreased due to a shift in primary fuel utilization from glucose to free fatty acids (Perry & Gallen, 2009). There is also a decreased risk of hypoglycaemia occurring in the insulin dependent diabetic due to the length of time since the last injection will place that person outside of the period of maximal insulin activity (Perry & Gallen, 2009). With the decrease in insulin activity there is the potential risk of hyperglycaemia occurring if the individual continues any form of oral glucose supplementation at the same level as previous. The athlete should shift the glucose intake, decreasing it as the time since injection increases. This can only be accurately determined with trial and error.

In short duration, high intensity exercise, the athlete will be primarily functioning above the lactate threshold with a large reliance upon anaerobic metabolism (Gallen,

2001). At high intensity, it is believed that catecholamines become primarily responsible for hepatic glucose production, instead of insulin and glucagon (Wassermann & Zinman, 1994). This increased catecholamine is thought to results in a relatively higher level of glucose production/release from the liver potentially resulting in hyperglycaemia, even in individuals who are not diabetic. In individuals with normal pancreatic function this relative increase in glucose can be compensated by an increase in endogenous insulin production/secretion. This however is not possible in individuals with type 1 diabetes and can easily lead to a state of hyperglycaemia.

In activities and training that involve aerobic exercise dispersed with intermittent high intensity exercise some interesting interactions occur. While moderate intensity exercise lasting longer than 20-60 minutes can cause a state of hypoglycaemia, especially in diabetic individuals, but with the addition of intermittent high-intensity bouts there is an increase in counter-regulatory hormones that will counter the general predisposition the diabetic person would have towards hypoglycaemia and might even result in a hyperglycemic state (Perry et al., 2009). This unique exercise induced metabolic interaction will likely alter the diabetic athlete's management approach. The athlete might need to reduce their insulin administration as well as reduce the amounts of carbohydrate supplementation that would usually be necessary in prolonged aerobic exercise alone (Guelfi et al., 2005). Any combination of these various training intensities and durations will require the athlete to experiment with their glycemic reactions, with the largest influence on their approach being the overall amount of glucose used to accomplish their exercise needs. With smaller amounts metabolised there will be a

tendency to hyperglycemia and with larger amounts there will be a tendency towards hypoglycemia.

**Counter-regulatory Hormones**

Counter-regulatory hormones, such as glucagon, noradrenaline, adrenaline and cortisol, are produced from the stress-response that occurs during exercise. These hormones act to increase the production of glucose while simultaneously interfering with insulin activity (Riddell & Iscoe, 2006). In a healthy individual, this relationship between insulin and counter-regulatory hormones act in a complex balance, but in the diabetic person this balance is disrupted by the person's inability to produce insulin, as well as their self-administered insulin treatments. With high levels of these counter-regulatory hormones, the diabetic person is at a higher risk of becoming hyperglycaemic and with low levels they are more prone towards hypoglycaemia (Riddell & Iscoe, 2006). This occurs because the counter regulatory hormones act to increase glucose utilization from the liver and tissues. The individuality of a person's specific counter-regulatory hormone response is believed to be one of the reasons it is so difficult to predict a diabetic individual's specific response to training and competition. The exact mechanisms that drive and result from the counter-regulatory hormones and not well understood. There are also several factors that can influence the counter-regulatory response. Competition stress (Riddell & Iscoe, 2006), heat stress, humidity (Riddell & Iscoe, 2006) and hydration status (Ahlborg et al., 1986) have all been shown to influence the counter-regulatory hormone response, promoting hyperglycaemia, especially in the diabetic person. High blood glucose prior to exercise will also likely lend to the risk of

hyperglycaemia occurring. Inversely, repeated bouts of exercise (Sandoval et al., 2004) or the being in a hypoglycaemic state (particularly antecedent hypoglycaemia) prior to the onset of exercise (Galassetti et al., 2003) have been shown to reduce/impair this response encouraging a hypoglycaemic state. This will require the athlete to be more aware of their potentially low blood glucose and have readily available glucose on hand.

**Antecedent Hypoglycaemia**

If a diabetic experiences a state of hypoglycaemia up to 24 hours prior to beginning exercise, the normal counter-regulatory hormone response can be impaired (Galassettie et al., 2006). This impairment has been shown to be dose dependent starting at a blood glucose level of 70mg/dL (Galassettie et al., 2006), and the risk of and severity of hypoglycaemia is predicated upon the extent and duration of the antecedent hypoglycaemic episode. Women have also been shown to be more resistant to this effect, having a relatively preserved counter-regulatory hormone response after experiencing antecedent hypoglycaemia and thus having a lessened deviation from their normal blood glucose response to exercise (Galassetti et al., 2002). With this is mind it is important for any athlete to be aware of any instances of experienced hypoglycaemia, as it may have an impact upon later bouts of exercise and impair the person's ability to predict and maintain their blood glucose levels. This is achieved by testing often to catch any instances of hypoglycaemia as well as knowing how a hypoglycaemic episode feels and to keep that in mind when engaging in later exercise.

**Recommendations for Insulin Administration in Athletes**

When speaking about general management goals for the individual with type 1 diabetes, it generally comes down to achieving the greatest level of stability and tight glycaemic control. With that in mind it is pertinent to factor in the individuals age, with different recommendations from the ADA (2005) for those aged 0-6 years old, 6-12 years old and 13-19 years old and adults. Individuals within these age groups are given different glycaemic targets. Refer to Tables 1 and 2 in the appendix for glycemic targets.

**Guidelines for Insulin Use**

The general goal of the treatment and management of an athlete with type 1 diabetes is to maintain the persons glycemic homeostasis while simultaneously enabling optimal metabolic function related to the athlete's individual physiological and sport needs. One approach to achieving this goal is to try and replicate the control of insulin levels by the pancreas that is present in a healthy individual (Perry et al., 2009). To achieve these ends most individuals will likely need multiple injections each day, using short acting or analogue insulins (Perry et al., 2009). The use of an insulin pump will offer will very effectively reproduce normal insulin function and can rapidly adjust to changes in insulin levels; unfortunately, these pumps are not an option for every athlete due to their large price tag as well as the specific nature of the sport and training the in which the athlete participates.

When injecting insulin, it is important to note that there may be differences between different types of insulin and how each will affect the person. With that being, there are some factors that have been shown to have an effect on the absorption rates of insulin.

The site of injection (Grimm, 2005) injecting into an exercising limb (Koivosto & Felig, 1978) and intramuscular injection (Frid, Ostman, & Linde, 1990). Changing the injection site or injecting into an exercising limb will cause increased variability in insulin action so extra care should be taken when changing injection sites and carefully monitor the different action of insulin at said different sites to better predict and administer their insulin and injecting into an exercising limb should be avoided all together (Perry & Gallen, 2009). Intramuscular injection should be avoided because of its tendency to cause hypoglycaemia, especially if it is followed by or done during exercise (Frid, Ostman, & Linde, 1990).

For the majority of exercising diabetics, a basal bolus regimen is recommended (Grimm, 2005). It is also recommended that a new analogue insulin be used as they have a more rapid onset and shorter duration which helps to prevent post-prandial hyperglycaemia as well as any delayed hypoglycaemia that often occurred with the older insulins (Rabasa-Lhoret et al., 1998).

**Insulin Therapy**

With the reduced or halted production of insulin those with type 1 diabetes will require lifelong insulin therapy. This will involve multiple daily insulin injections. The specific number of injections, and volume will depend on the individuals self-monitored blood glucose levels (which will fluctuate depending on numerous factors: for example, activity level, caloric composition and volume, and the varying physiologic state of the

individual). Insulin will usually be administered as basal insulin usually in the form of either long-acting (glargine or detemir) or intermediate acting (NPH) and premeal insulin, that is either rapid-acting (lispro, aspart, insulin inhaled, or glulisine) or short acting (regular) (Perry & Gallen, 2009).

Since Insulin is an anabolic hormone, and if used by healthy individuals will act as a metabolic modulator giving unfair advantage in sport it is on the banned substance list from WADA. In order for the diabetic person to participate in WADA regulated competition they will need to get a therapeutic use exemption. This will require the athlete to have medical documentation and to have their case reviewed by a committee. Once this is done the athlete is free to participate in their sport while using insulin.

**Common Insulin Regimens**

- Split or mixed: NPH with rapid-acting (e.g., lispro, aspart, or glulisine) or regular insulin before breakfast and supper
- Split or mixed variant: NPH with rapid-acting or regular insulin before breakfast, rapid-acting or regular insulin before supper, and NPH before bedtime (the idea is to reduce fasting hypoglycemia by giving the NPH later in the evening)

- Multiple daily injections (MDI): A long-acting insulin (e.g., glargine or detemir) once a day in the morning or evening (or twice a day in about 20% of patients) and a rapid-acting insulin before meals or snacks (with the dose adjusted according to the carbohydrate intake and the blood glucose level)
- Continuous subcutaneous insulin infusion (CSII): Rapid-acting insulin infused continuously 24 hours a day through an insulin pump at 1 or more basal rates, with additional boluses given before each meal and correction doses administered if blood glucose levels exceed target levels (Khardori, 2018)

**Dietary Recommendations**

It is important for every individual to have a comprehensive diet plan that outlines a daily caloric intake recommendation, advised amounts of dietary carbohydrates, fats and protein and instructions on how to best divide calories over meals and snacks

The general nutritional recommendations for the type 1 diabetic athlete is very similar to that of the normal population. It is not specifically what the diabetic athlete consumes that is most important, but rather when, their consistency and how the balance and time their consumption with their insulin injections.

It is generally held that the diabetic athlete should follow the dietary recommendations of the ACSM, the American Dietetic Association, and the dietitians of Canada for the nutritional requirements of the non-diabetic competitive athlete (2000). These recommendations are as follows:

-Carbohydrate consumption ranging from 6-10g/kg of body weight /day. This being the amount necessary to maintain blood glucose and replenish glycogen stores. The specific amount will depend upon the athlete's daily energy expenditure, sex, age, sport type, and environment.

-Protein consumption should range from 1.2-1.4 g/kg body weight/day for the endurance athlete and 1.6-1.7g/kg body weight/day foe the strength-trained athlete to develop and maintain muscle mass. It should be noted that large amounts of protein consumption, that being more than 2.4g/kg body weight/day may place additional strain on the kidneys, and those that have pre-existing renal conditions may be at heightened risk. This is especially important to note as approximately 30% of individuals with type 1 diabetes will develop kidney disease (Hornsby & Chetlin, 2005). As such it is important to mitigate any unnecessary stress on the kidneys in the diabetic athlete.

-Fat consumption should range from 20-25% of daily caloric intake, best in the form of unsaturated fat. This is expected to be about 5-10g/kg of body weight/day depending on training intensity (Sherman et al., 1993). This is to provide energy and allow for absorption/use of fat soluble vitamins

-Total energy consumption is recommended from 37-41 kcal/kg body weight/day for the endurance athlete and 44-50+ kcal.kg body weight/day for resistance trained athlete. IF the athlete is working to gain mass then sufficient consumption to support training as well as muscle growth is necessary. It is also important to note that these estimations are simply a guideline, and not terribly accurate. It is important that the energy intake allow the athlete to participate in training, maintain or gain musculature as needed and maintain the necessary body composition to perform optimally.

While these recommendations are the same for the non-diabetic competitive athlete it is important to note that there is a greater importance for the diabetic athlete to closely adhere to these guidelines. If they fail to adhere the diabetic athlete is more likely to suffer from metabolic states detrimental to their performance and training as well as potentially developing body compositions not ideal to their sport.

Some athletes may seek to employ specific non-traditional diets such as diets that may restrict carbohydrate consumption with the aim of mobilizing ketones for metabolism potentially causing ketoacidosis. Ketoacidosis in a diabetic athlete is known to be quite detrimental to health and therefore carbohydrate restricted diets should be avoided.

With athletes that participate in sports where weight and aesthetics are a factor it is common to see certain risky behaviours that coaches and athletes should be aware of. For athletes that compete in weight categories they may employ some of the common weight loss practices such as dehydration, the use of laxatives, diet pills, and diuretics (Kiningham & Gorenflo, 2001). With the diabetic person who is already metabolically unstable this can be riskier than it is in normal, healthy populations. Some diabetic athletes may also withhold or reduce their insulin injections as this will allow them to rapidly lose weight. While this might be a tempting approach to making weight, it may lead to a dysregulation in metabolic control and can lead to ketoacidosis (Hornsby, 2005) especially if other unhealthy weight loss strategies are used concurrently.

Another athletic population that may be at specific risk of unhealthy weight control behaviours is adolescent females, especially those that are involved in sport that emphasize low body weight such as distance running, gymnastics, figure skating etc. A

study of weight control practice in adolescent females with type 1 diabetes showed 37.9% held to unhealthy weight control methods that including altering their normal insulin injections (Neumark-Sztainer et al., 2002).

**Metabolism and Exercise in the Type 1 diabetic**

A significant increase in glucose uptake occurs during exercise (Kanj 1988). With an increase in glucose requirement an increase in hepatic glucose production occurs. A shifting in hormone blood concentrations is a major driving force in the overall maintenance and balancing of blood glucose levels. A decrease in insulin concentrations triggers an increase in the activity of glycogen phosphorylase activity in the liver. This, along with increased catecholamines levels, effectively increases the breakdown of stored glycogen in the liver and an overall increase in glycogenolysis, which is the conversion of glycogen into metabolically ready glucose. Once exercise is halted the opposite of this process will occur, that being insulin levels will increase, glucagon levels will decrease leading to a decrease in glycogenolysis and an increase in glycogen synthesis both in the liver and in affected musculature. After exercise there is also a period in which insulin sensitivity is increased allowing the liver and muscles to more readily replenish depleted glucose stores. This physiologic response to exercise can last for several hours after exercise has halted and as such it is important for the diabetic athlete to continue supplemental glucose ingestion post exercise. In the diabetic athlete blood insulin levels will not decrease and as such sufficient blood glucose levels need to be present.

In the type 1 diabetic person the issue of glucose metabolism occurs as a result of the individual's inability to respond to and maintain glycemic homeostasis. This is an issue during exercise due to the increase in energy demands that will send the system into flux. This can cause the diabetic individual to become either hyperglycemic or hypoglycemic depending on their insulin intake, the nature of the exercise, their glucose intake along with specific factors that will pertain to the individual. During exercise hypoglycaemia is the more common. This can occur if insulin levels are too high at the onset of exercise. This will usually occur because of a too large insulin injection or due to accelerated absorption at the site of injection (Koivisto et al. 1980). If insulin levels don't decrease in a normal fashion then the liver will be unable to produce sufficient glucose to meet the peripheral demands, leading to a drop in blood glucose concentrations. The risk of hypoglycaemia also increases with an increase in exercise duration and intensity (Wahllberg-Henriksson 1992). This has considerable implications for an athlete's specific sport demands as well as the training approach they take. While the greatest risk of hypoglycaemia occurs during exercise there will still be a risk of it occurring while the metabolic demands of the musculature are raised post exercise a state of hypoglycaemia may occur up to 4-6 hours after exercise has halted (Wahllberg-Henriksson 1992).

While hyperglycaemia in individuals with type 1 diabetes is rare, it can occur if blood glucose levels are abnormally high prior to the onset of exercise (Wahrenm Hegenfeldt, & Felig, 1975). If there are insufficient levels of insulin in the diabetic person's system, then glucose transport into the working muscle will be impaired during exercise. This will result in an increased reliance upon free-fatty acids for energy. This

increased reliance and the process of free fatty acid metabolism through beta oxidation and the glucose -fatty acid cycle, may result in an increase in ketone levels in the system (Stojanovic& Ihle, 2011). An increase in the concentrations of the counter regulatory hormones: glucagon, catecholamines and growth hormone may also accelerate this process (Galaestti et al., 2002). These factors may exacerbate the hyperglycemic state and possibly lead to the development of a ketotic state. As such it is important for an athlete or any diabetic individual to have their glycemic levels under sufficient control before beginning exercise or training.

Other metabolic dysfunction may either be affected or have an effect upon the diabetic condition. Two such examples of this are overtraining syndrome and Relative Energy Deficiency in Sport (RED-S). Overtraining is characterized by a maladaptive response to excessive training and inadequate rest that can result in perturbances in neurologic, endocrinologic, immunologic function. This can also alter catecholamine response and may have an effect on blunting action (Kreher & Schwartz, 2012) and thereby further complicating and unbalancing the diabetic athletes ability to predict their blood glucose response to exercise and their insulin administration. RED-S refers to impaired metabolic dysfunction including metabolic rate, menstrual function, bone health, immunity, protein synthesis, cardiovascular health as a result of an relative energy deficiency (Mountjoy et al., 2014) This condition may be a great risk to the diabetic athlete as energy deficiency may result not only from inadequate intake but also as a result in metabolic imbalance and improper insulin administration.

**Guidelines and Considerations for Blood Glucose Management**

There are several guidelines that the Insulin-dependent diabetic individual should follow to reduce the risk of experiencing unwanted and potentially problematic shifts in their metabolic state. The American College of Sports medicine and The American Diabetes Association have supplied recommendations for the diabetic engaging in sport/exercise:

- Measure blood glucose levels before, during and after exercise
- Avoid exercise during periods of peak insulin activity (2-4hrs post injection)
- Unplanned exercise should be preceded by increased carbohydrate consumption (e.g. 20-30 g per 30 min of exercise) insulin may have to be decreased post exercise
- If exercise is planned, insulin dosages should be decreased before and after exercise according to the intensity and duration of said exercise as well as the individual's fitness level. This reduction in insulin may amount to up to 50-90% of the individual's daily insulin requirements
- Easily absorbable carbohydrates may need to be consumed during exercise
- Post-exercise carbohydrates may be necessary (e.g. 1.5g/kg body weight within 30 of cessation of exercise and a further 1.5g/kg body weight 1-2 hours later)
- The individual and the people around them should be aware and on the look out for the different signs and symptoms of hypoglycaemia
- It may be pertinent to make use of a personal fitness trainer with experience with diabetic individuals, especially at the beginning of an exercise program or with a person who is newly diagnosed.

Exercise can act to have an insulin-like effect. While much of the research is conflicting acute exercise has been shown to reduce blood glucose concentrations (Wallberg-Henriksson et al., 1992) While diabetes does not need to limit an individual's ability to participate in sport in any level, the diabetic athlete as well as those surrounding them, from coaches to trainers as well as fellow athletes should be cognisant of the condition, the necessary precautions that should be taken in order to avoid unstable glycemic states, being hyper or hypoglycaemia and ketoacidosis, as well as specific signs or risks that the individual experiences before the onset of these physiological states.

The timing of insulin injections and insulin absorption should also be carefully considered for each athlete, taking into consideration the individuals specific needs as well as the specific sport requirements that are present.

When insulin is injected prior to exercise, it is common for a state of hypoglycaemia to occur between 2 to 3 hours following injection. If a rapid-acting insulin analogue is used hypoglycaemia will likely occur between 40 and 90 min following injection (Robertson et al., 2009).

Athletes, coaches and parents should also be aware of the effect specific injection sites may have upon performance and glycemic equilibrium during exercise. When choosing a specific injection site any muscle groups and areas that will be largely active during athletic performance should be avoided, as the increased blood flow to these muscles and areas will cause an increased rate of absorption and metabolic action (Hornsby et al., 2005). Rather than injecting functioning muscle groups it would be better to inject areas that are not of primary use in the sport specific context or to inject at sites in which the musculature, while active, is less metabolically demanding such as

musculature that is more slow-twitch in nature, which has a slower energetic turn around and this does not experience the same increase in blood flow that primary muscle groups will experience (Hornsby et al. 2005). Using the abdomen as an injection site over the limbs is generally preferable, or if the sport is upper limb or lower limb dominant then using the non-active limb as the site of injections. There is also some evidence to suggest that changing the injection site may alter the time course of insulin absorption (Hornsby, 2005). Changing the site of injection should be done carefully and if the site is changed then blood glucose levels should be carefully monitored during training to determine the individual's specific response to the change. The site should not be changed near competition due to the unpredictability that may result.

The ambient temperature may also influence increasing the absorption rates of insulin while also placing greater strain on the cardiovascular system. It is proposed that in cold conditions there will be an increase in blood flow to maintain homeostatic body temperature (Robertson, 2009). This increase in blood flow may cause an increase in absorption and an overall reduction in blood glucose concentrations potentially leading to hypoglycaemia. (Robertson 2009) There will also be an increase in muscular absorption of glucose to produce heat, adding further risk of hypoglycaemia (Kenny et al. 2016). Both hyperglycaemia and hypoglycaemia are a risk in hot temperatures. With high temperatures, a concern is that of dehydration. With the increase in water loss dehydration becomes more likely and if the athlete already has relatively high blood glucose levels then this adds to the risk. There is also a noted increase in metabolism in warmer conditions (Kenny et al. 2016). This implies an increase in utilization of glucose potentially dropping blood glucose levels faster than normal. The vasodilation that

occurs at higher temperatures can also have issues as this may increase insulin absorption and also potentially increase blood flow to the sites of insulin injection, which will increase the insulin's action (Sindelka et al., 1994).

As with cold temperatures there is also an increased risk of hypoglycaemia at altitude. This occurs because of an increased reliance on carbohydrate fuel sources that is observed at altitude (Hoyt & Honig, 2011). The athlete will also be more likely rely upon anaerobic metabolism due to the reduction in oxygen tension at altitude (Hoyt & Honig, 2011). This heightens the risk of the diabetic individual to suffer from hypoglycaemia and ketoacidosis, further complicated by the potential decreased meter accuracy at altitude (Moore et al., 2001). The hydration status of the athlete at altitude will also be of concern as altitude has been shown to increase the risk of dehydration (Hoyt & Honig, 2011). This is an increased risk to the diabetic athlete who is already predisposed to dehydration (Riddell & Iscoe, 2006). It is also possible that the use of acetazolamide to treat and mitigate the effects of altitude may have an effect in increasing the risk of ketoacidosis (Moore et al., 2001).

**Further Recommendations/Management**

With the complexity and individual characteristics that individuals with type 1 diabetes show, it is difficult to supply a definitive management plan for the diabetic athlete independent of the nature of their sport. The best that can be done is to supply some recommendations and guidelines that may help the athlete to manage their

condition while training for and participating in their sport. With these recommendations, the athlete will need to learn by trial and error to determine which approaches will help in their specific circumstances. It is also very common in the diabetic community to share the approaches that more experienced individuals have developed. Using this anecdotal knowledge can be very useful and may help to give diabetic athletes a starting point or some different approaches. (There are a number of online sources and support groups that seek to accomplish this, connecting diabetic individuals with the aim of sharing knowledge and helping each other in living with and managing this disease).

For sport/training that has a longer duration at moderate intensity, primarily aerobic there is a predisposition for hypoglycaemia to occur after 20-30 minutes of continuous exertion (Wasserman & Zinman, 1994). This is especially true for athletes exercising at the upper aerobic threshold (around 70% of maximal heart rate) (Wasserman & Zinman, 1994). This hypoglycaemia occurs because of injected insulin staying static after commencing exercise, when in normal populations blood insulin levels would decrease. Without a decrease in insulin levels, the diabetic person might experience over-insulination, increasing the risk of hypoglycaemia (Wasserman et al., 1991). The best approach to counter this is to reduce the insulin dosage prior to planned exercise. This should reduce the risk of hypoglycaemia occurring. If the exercise is unplanned or the duration or intensity of said exercise is higher or longer than planned then additional carbohydrate should be consumed prior to, during and post exercise. Moderate-intensity exercise (about 50% max oxygen uptake) will increase glucose use by 2-3 mg/kg/min (Wasserman & Zinman, 1994) therefore a 70kg person would require 10-15 g of additional carbohydrate per hour of exercise. As the intensity increases so will the

required carbohydrate intake. High intensity exercise (80-100 max oxygen uptake) the rate of glucose uptake may increase 5-6mg/kg/min (Wasserman & Zinman, 1994). While the amount of glucose uptake is increased, the risk of hypoglycaemia occurring is reduced because the athlete will not be able to maintain this level of intensity for a sufficient duration to become hypoglycaemic. On the other hand, if the athlete is participating in a sport that requires intermittent bursts of high intensity output over a long period of time (such as a soccer) the risk of hypoglycaemia is again present (Wasserman & Zinman 1994). In such cases athletes should test often, even running to the sideline during short breaks, having support staff test and determine if glucose supplementation is necessary. While hypoglycaemia is a risk during and after intermittent exercise it does not increase the athletes risk of early post-exercise hypoglycaemia (Guelfi et al., 2005). Sport that are very short in duration are generally the least difficult for the diabetic person to engage in. This is generally the case as, during very brief bouts of exercise, glucose is not actively metabolized and there is less flux in blood glucose levels and corresponding blood insulin levels. While there is little risk of the athlete experiencing hyperinsulemia while participating in short duration sport/training there is an added risk of hyperglycaemia. This is thought to result from catecholamines becoming the primary controllers of hepatic glucose action rather than insulin and glucagon. This can lead to an increased mobilization of hepatic glucose causing hyperglycaemia (Christensen, 1979).

The goal of every diabetic person especially diabetic athletes is to maintain a tight control of their blood glucose levels and to avoid states of either hyperglycaemia or hypoglycaemia. The best way to achieve this is by constantly monitoring blood glucose

levels and adjusting insulin and carbohydrate intake accordingly. A common blood glucose goal is between 150 and 250 mg/dl (Gregory et al., 1994). If the athlete tests and has a blood glucose level around 80mg/dl their risk of experiencing hypoglycaemia is high and should ingest carbohydrates prior to engaging in exercise. (Horton & Subauste, 2016) It is also recommended that if the athletes blood glucose is >250mg/dl and ketone bodies are present in urine, or if the blood glucose is >300mg/dl then more insulin should be administered, and exercise should be delayed (Horton & Subauste, 2016). The rate of change in glucose levels should also be noted. If the athletes blood glucose sits a bit low but is stable, this is better than if the athletes blood glucose is in an acceptable range but is dropping significantly between tests: for example, from 150mg/dl to 100 mg/dl (Horton & Subauste, 2016). It also seems common that athletes (especially endurance athletes) will often maintain high blood glucose levels prior to sport and training participation in order to prevent experiencing a hypoglycaemic state (Sane et al., 1988). While this may help the athlete avoid hypoglycaemia during competition, there are inherent health risks of prolonged hyperglycemia and it should be avoided whenever possible.

The risk of experiencing hypoglycaemia is also related to the time of day exercise is done. If exercise is done in the morning before breakfast and the pre-meal insulin dose hypoglycaemia is less likely as at this time blood insulin levels are at their lowest (Toni et al., 2006). Exercise in the late afternoon and evening have an increased risk of leading to hypoglycaemia during sleep if the athlete does not consume sufficient glucose as blood glucose levels will continue to be depleted after exercise has halted (MacDonald, 1987).

If the athlete is required to complete all day in tournaments or has multiple events dispersed throughout the day the athlete should employ long acting insulin the evening before competition day (Toni et al., 2006). This when included with regular testing will help the athlete maintain their blood glucose levels. Due the changing nature of competition and training, changes in the environment, intensity, duration and the emotional state of the athlete may all influence the individual's insulin absorption and their glucose response. With the unpredictable and fluctuating nature of sport training and competition, the diabetic athlete should keep rapidly absorbable carbohydrates on hand, and the coaching staff, the athlete themselves as well as their teammates should be aware of what hypoglycaemia looks like.

## Conclusion

The overall take away is that planning, preparation and consistency are the keys to avoiding undesirable blood glucose levels. Insulin injections, food consumption, and training/competition need to be balanced and timed with each other to achieve the best results. The best way to mitigate and control the diabetic condition is knowledge and discipline. There are many approaches, insulin regiments, training protocols etc. that can prove helpful but in the end the only way to be sure of how a specific person's blood-glucose levels will react is through trial and error. This will require the athlete and their coaches to be very mindful and systematic. Once a viable and effective management approach has been found it is then important foe the athlete and their coaches to diligently stick to the plans so as to mitigate any disadvantages. While the diabetic athlete will face challenges that those in the general population will never have

to deal with, they are fully capable of competing and succeeding at any level of sport that they aspire to achieve.

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## Appendix

**Table 1 Children's and youth glycemic goals**

| Values by age | Plasma blood glucose goal range (mg/dl) | | Bedtime/overnight | A1C |
|---|---|---|---|---|
| | | Before meals | | |
| Toddlers and preschoolers (<6 years) | 100–180 | 110–200 | <8.5 (but >7.5) % | • High risk and vulnerability to hypoglycemia |
| School age (6–12 years) | 90–180 | 100–180 | <8% | • Risks of hypoglycemia and relatively low risk of complications prior to puberty |
| Adolescents and young adults (13–19 years) | 90–130 | 90–150 | <7.5%* | • Risk of hypoglycemia |
| | | | | • Developmental and psychological issues |

**Note: Taken from** Care of children and adolescents with type 1 diabetes: a statement of the American Diabetes Association (Silverstein et al., 2005)

**Table 2 Adult's glycemic goals**

| | |
|---|---|
| A1C | <7.0% (53 mmol/mol)* |
| Preprandial capillary plasma glucose | 80–130 mg/dL* (4.4–7.2 mmol/L) |
| Peak postprandial capillary plasma glucose† | <180 mg/dL* (10.0 mmol/L) |

**Note: Taken From Standards of Medical Care in Diabetes (2014)**