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Diabetes Self-Management Education for Adolescent Patients: The Importance of a Developmentally Sensitive Approach

Laura Mumme

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> Tonia Kennedy, Ed.D., MSN, RN, CCRN Chair of Thesis

> > Lucinda A. Drohn, MSN, RN Committee Member

> > Michael R. Korn, Dr. rer. nat. Committee Member

Marilyn Gadomski, Ph.D. Assistant Honors Director

Date

#### Abstract

The attention of healthcare providers to the developmental level of adolescent patients with type one diabetes (T1D) maximizes effective patient education and glycemic control. Due to the dynamic changes that occur in the mind and body during adolescence, self-care for adolescent T1D patients is overwhelming as it envelopes activities of everyday life. The disease process and the unique aspects of adolescence in relation to T1D are important for caregivers to consider. Developmentally appropriate teaching during the initial onset of the disease must inform the patient and the patient's family of the basics of the disease process and the daily care necessary for survival. Continuous and gradually increasing education is accomplished through regular appointments with the healthcare provider as well as through the gradual handing over of self-care responsibility from the parent to the adolescent. The physical and social needs of the adolescent should be incorporated into the education for the best results in compliance and adherence to the diabetes regimen. Motivational interviewing (MI) is an effective intervention for patients who are entering puberty and who experience increased ambivalence toward diabetes self-management.

Diabetes Self-Management Education for Adolescent Patients:

The Importance of a Developmentally Sensitive Approach

Type 1 diabetes (T1D) is a disease that affects everyday life. Management of this disease is complex and is best accomplished when the adolescent is an active participant in his or her own care (Clement, 1995). Unfortunately, total self-management is not possible for persons at any developmental level during the initial diagnosis (Schiffrin, 2001). The goal of diabetes education is to motivate the patient to implement behavioral changes that promote tight glycemic control in the initial period and for the long stretch (Michel, 2011).

In adolescence, children transition into adulthood and experience dramatic physical, cognitive, social and emotional changes. Puberty refers to the initiation of reproductive organ functioning along with the accompanying developmental processes and secondary sex characteristics. The period of adolescence begins with the appearance of secondary sex characteristics and ends with the completion of physical maturation. People experience these phenomena at different times; however, secondary sex features generally appear at 11 or 12 years of age and physical growth is often completed by 18 to 20 years of age (Kollar, 2013).

An adolescent with T1D will experience barriers to a healthy lifestyle that relate to his or her developmental challenges. The adolescent period of life is characterized by a search for identity (Franklin & Prows, 2013). Puberty accompanies many physical and psychosocial challenges and the adolescent patient needs to continue to develop his or her growth despite the presence of chronic illness (Coffen & Dahlquist, 2009). However, these challenges tend to negatively affect blood glucose levels and glycemic control tends to diminish during the transition to adolescence (Christie et al., 2009; Coffen & Dahlquist, 2009; Lowes & Fernandes, 2008; Trast, 2014). Patient education that properly assesses the psychological needs of the adolescent will empower the patient to cope with diabetes and achieve normal developmental landmarks during this transitional period of life (Coffen & Dahlquist, 2009). Psychological therapy of adolescents with T1D has been shown to improve glycemic control (Serlachius et al., 2014), and by extension reduce long term complications (The Diabetes Control, 1993).

Adolescents are intensely social in their activities. They are growing more independent from parents and moving closer to friends. Peer groups strongly influence adolescent behavior; an adolescent desires to belong to a group and will act in such a way as to attain establishment within a group of peers (Kollar, 2013). Group education can enhance compliance and the desire to develop proficiency in their self-care skills. Educators can also facilitate normalization by gathering adolescent T1D patients in group settings and allowing them to share their experiences, skills, and information. This setting also provides the patients with a sense of belonging and gives them an avenue for trying different roles and developing their identities (Docherty, Barfield, Thaxton, & Brandon, 2013).

The initial education must provide the patient with the basic survival instructions so that the patient will avoid life-threatening situations. Discussing the pathogenesis of the disease helps the adolescent understand what is going on inside of his or her body. Having an understanding of the disease process also provides a knowledge base for understanding the necessity of treatment. The patient needs to understand the actions and purpose of the hormone insulin and the detrimental effects of the lack of insulin production by the pancreas. It is also important for the educator to provide psychological support by explaining to the adolescent that T1D is not necessarily caused by something that the patient did or did not do. The educator must relate the information in terms that the patient can understand so that the patient can develop an appropriate concept of the disease. The patient should understand the implications of preprandial and postprandial blood glucose levels so that the patient can effectively manage insulin therapy. Glycosylated hemoglobin (HgbA<sub>1c</sub>) readings are telling of the overall glucose levels in the past three months and will be emphasized in the patient's future education (Lange, Swift, Pankowska, & Danne, 2014).

Insulin is available in multiple forms. Types of insulin vary and can be tailored to the patient's specific needs; adherence to the insulin regimen should be emphasized (Lange et al., 2014). Acute complications such as diabetic ketoacidosis may occur if regular insulin therapy is neglected (Jones, Brashers, & Huether, 2010). Chronic complications result from the effects of hyperglycemia when blood glucose levels are not controlled effectively (The Diabetes Control, 1993).

Initial patient education also includes information regarding hypoglycemia, exercise, and nutrition. The patient should be introduced to the signs, symptoms, and probable causes of hypoglycemia, which can quickly escalate if it is not treated swiftly (Lange et al., 2014). Exercise and nutrition need to be discussed as they relate to changes in the blood sugar. Although the patient should receive initial teaching on exercise and nutrition, the teaching should be built up and developed throughout the disease process, so the patient does not become overwhelmed and less likely to comply with the regimen (Coffen & Dahlquist, 2009). It is beneficial to emphasize the more pertinent self-care

activities with the adolescent and to discuss the nutrition plan and exercise schedule with the parent. The meal plan should be realistic enough for the patient to adhere to it. Meal planning should give attention to the glycemic index of foods and the patient's blood glucose levels in relation to insulin administration for each meal. When parents of adolescents with T1D govern the meal plan, these patients have better clinical outcomes (Swift, 2007). The parent or primary caregiver can then gradually transfer self-care activities over to the adolescent in accordance with the adolescent's readiness for increased responsibility and involvement (Roper et al., 2014). The goal of these efforts is the maintenance of tight blood glucose control as blood glucose levels that are uncontrolled are associated with increased morbidity in diabetic persons (The Diabetes Control, 1993).

There are numerous aspects to the management of diabetes. Covering all of these aspects during the first hospital admission is overwhelming for the adolescent and is not advised. Rather, it would be better for the educator to go into realistic depth in a few vital areas of self-care and then gradually build up the adolescent's holistic self-care abilities. Bulk information cannot be immediately absorbed, but life experience provides a medium of attachment and retention for further teaching (Coffen & Dahlquist, 2009).

Because diabetes education for adolescent patients is much more complex than transmission of facts, behavioral modification therapy is indicated (Lange et al., 2014). Motivational Interviewing (MI) is a behavioral change technique that is patient-focused and helps address the patient's ambivalence to change. MI affects change by increasing the patient's awareness of his or her own desire to change and be healthy (Rollnick, Miller, & Butler, 2007). Performing glucose checks, administering insulin, narrow

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dietary allowances, and monitored physical activity are only a few of the aspects to diabetes self-care that can cause the adolescent to feel different and isolated from his or her peers. The distaste for standing out can discourage an adolescent's self-care practices for the sake of achieving peer acceptance (Naar-King & Suarez, 2011). Addressing the patient's barriers to self-care must take place before solutions can be developed. MI takes the stance that people want to be healthy, but the behavioral changes that lead to health can seem daunting, which causes resistance to change. It is not the end goal of being healthy that is undesirable, but the journey to get there (Rollnick et al., 2007). MI focuses on building the adolescent's intrinsic desire to change and achieve a normal life (Naar-King & Ellis, 2011). This technique is sensitive to the patient's developmental level and autonomy and has been found to be an effective intervention for adolescents with chronic diseases, such as T1D (Naar-King & Ellis, 2011; Rollnick et al., 2007).

When the adolescent is an active participant in his or her own care and when selfcare responsibilities are transferred to the patient gradually, clinical outcomes are better (Clement, 1995; Coffen & Dahlquist, 2009). Healthcare providers should be alert for signs of diminished glycemic control in an adolescent because of the known psychosocial and physical predispositions of this age group (Christie et al., 2009). Although adolescents are capable of achieving competency in diabetes self-management, developmental issues often present barriers to self-care. For this reason, adolescents should receive care that addresses their developmental needs and helps them to overcome challenges and attain a healthy lifestyle (Lange et al., 2014). Simply prescribing medications and loading the patient with information are not enough to affect optimal health in the adolescent patient. Research has delineated the necessary self-care activities that diabetes patients should incorporate into their lifestyle for optimal outcomes, and so the challenge is to get the patient to put these activities into practice. MI is a developmentally appropriate and effective technique for addressing ambivalence to change and for promoting intrinsic motivation for improved diabetes self-care. Healthcare providers and adolescents with T1D alike will benefit from the incorporation of MI into patient care (Naar-King & Suarez, 2011). Because of this benefit, incorporation of MI into the plan of care for adolescents with T1D should be considered.

### **Carbohydrate Metabolism**

T1D completely alters the metabolism of carbohydrates, the body's initial source of energy. Carbohydrates are broken down into monosaccharides during digestion and then released into the blood stream through the intestinal walls (Swinny, 2013). Glucose is a six-carbon monosaccharide that provides the body with an efficient source of energy (Guven, Matfin, & Kuenzi, 2009).

Glucose is taken into cells through the effects of insulin. In a well-oxygenated cell, glucose will undergo aerobic glycolysis and yield pyruvate. Upon entering the mitochondria of the cell, pyruvate can then be transformed by pyruvate dehydrogenase into acetyl coenzyme A (CoA), which fuels the tricarboxylic acid (TCA) cycle, also known as the Krebs cycle or the citric acid cycle. Through this cycle, pyruvate yields carbon dioxide (CO<sub>2</sub>), three nicotinamide adenine dinucleotide (NADH) molecules, and one flavin adenine dinucleotide (FADH<sub>2</sub>) molecule. NADH and FADH<sub>2</sub> are able to donate electrons to the electron transport chain located in the inner membrane of the mitochondria. As electrons are passed down the electron transport chain they lose energy. This energy is captured, in part, by adenosine triphosphate (ATP) when it

undergoes oxidative phosphorylation from adenosine diphosphate to ATP. ATP is a high-energy compound that releases approximately 7.3 kcal/mol (30.7 kJ) for each phosphate group hydrolyzed. This is the energy that makes glucose metabolism so valuable to the body (Harvey & Ferrier, 2011).

#### Insulin

Insulin is an anabolic hormone that lowers blood glucose levels. The precursor of insulin, called proinsulin, is a molecule composed of an A peptide, a B peptide, and a C peptide. The beta cells synthesize insulin from proinsulin by cleaving both ends of the C peptide from proinsulin, leaving the A and B peptide as the active form of insulin. Insulin synthesis in the beta cells is stimulated by parasympathetic activity or increased blood levels of glucose, amino acids, free fatty acids, or digestive hormones. Insulin production diminishes in the presence of hypoglycemia, sympathetic stimulation of pancreatic alpha cells, and hyperinsulinemia. Without insulin, glucose cannot permeate the cell membrane of the tissues. Insulin released from the pancreas travels to a tyrosine-kinase receptor subtype which then triggers phosphorylation of the insulin receptor substrate I and of other proteins. These actions result in the movement of glucose transporter (GLUT) proteins to the cell membrane where they act as catalysts of glucose intake. Without GLUT proteins, cellular glucose uptake would be significantly slower (Brashers & Jones, 2010; Guven et al., 2009)

Insulin also lowers blood glucose levels by promoting the storage of glucose as glycogen by the liver. Insulin inhibits proteolysis by inhibiting gluconeogenesis, the formation of new glucose molecules from amino acids and other compounds. On the contrary, protein synthesis is increased in the presence of insulin as insulin stimulates the

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active transport of amino acids into cells. Fat metabolism is also affected by insulin. Insulin draws glucose into fat cells, allows for triglyceride synthesis from glucose, and halts the breakdown of stored triglycerides in the cells (Guven et al., 2009).

# Pathophysiology of Type 1 Diabetes (T1D)

T1D is a disease in which the pancreas does not produces insulin due to autoimmune destruction of the insulin-secreting beta cells of the pancreas. There are rare cases of idiopathic T1D of which the cause cannot be identified as autoimmune (Guven et al., 2009). For the purposes of this discussion, T1D will be considered an autoimmune disease.

T1D is the result of the destruction of the beta cells by the body's own immune system. Therefore, the pathophysiology of T1D is marked by a stimulus that incites immune cells against pancreatic beta cells. The actual trigger varies from person to person; however, it involves interplay between genetic predisposition and environmental factors. Major histocompatibility complex (MHC) class II molecule genes in the DR and DQ loci are of particular importance in the pathogenesis of T1D. Human leukocyte antigen (HLA)-DQ and HLA-DR are correlated with a risk of T1D that is five to eight times higher than the general population. HLA-DR has been noted to relate to other autoimmune disorders as well. If a person has inherited the HLA-DR3 and the HLA-DR4 allele from both parents, this individual's risk of T1D is 20 to 40 times greater than average (Jones et al., 2010).

Environmental triggers coupled with a genetic predisposition further heighten an individual's likelihood of developing T1D. Beta cell damage via the immune system has been associated with the presence of certain viruses, including congenital rubella,

cytomegaloviruses, mumps, and Epstein-Barr virus. Notably, 40% of individuals diagnosed with congenital rubella will develop T1D at some point. Bovine serum albumin which can be found in cow's milk is thought to be involved in stimulating beta cell autoantibodies. High dietary intake of nitrosamines and exposure to the rodenticide Vacor, the chemical alloxan, and the drugs Streptozotocin and Pentamidine are also environmental factors that increase the risk of developing T1D (Jones et al., 2010).

In the presence of the various genetic and environmental factors, beta cell destruction occurs through T-cell mediated inflammation of the islets and autoantibody production against islet cells, insulin, and glutamic acid decarboxylase (GAD). Pancreatic islet cells present autoantigens on the cell surface that circulate in the blood and lymphatics. Antigen-presenting cells (APCs) detect and ingest these autoantibodies and, in turn, stimulate CD4+ T helper lymphocytes to secrete interleukin 2 (IL-2). IL-2 is a cytokine that stimulates the proliferation of beta cell autoantigen-specific T cytotoxic CD8 lymphocytes. These cytotoxic cells secrete granzymes and toxic performs that further damage beta cells. CD4+ T helper cells also secrete interferon. Interferon is a cytokine that mediates the involvement of other proinflammatory cytokines and activates macrophages. The result is compounding activity against beta cell life (Jones et al., 2010).

The T helper cells that produce IL-2 and interferon to promote inflammation also produce interleukin 4 (IL-4), which relates to the development of autoantibodies. IL-4 triggers B cell proliferation and antibody production. Autoantibodies to islet cells, insulin,  $GAD_{65}$ , and tyrosine phosphatases are found in 85% to 90% of individuals with T1D when fasting. Diminished beta cells can be detected by positive islet cell autoantibodies in the blood years before a patient becomes symptomatic. This shows the slow autoimmune progression that typifies T1D. Insulin autoantibodies are more likely to develop during islet cell destruction. Thus, T1D develops due to inflammation and cell death of islet cells as well as the production of autoantibodies against beta cells, insulin, GAD, and other proteins. This results in the body's inability to cope with rising blood glucose levels (Jones et al., 2010).

# **Short-Term Complications**

Acute complications of T1D can be either hypoglycemic or hyperglycemic in nature. Hypoglycemia can occur if the patient receives too much insulin treatment, exercises without extra carbohydrate intake, ingests alcohol without food, has inadequate or delayed meals, or loses weight while continuing the same medication regimen. Hypoglycemia can lead to very serious outcomes and the patient needs to be able to recognize the above actions that can lead to hypoglycemia as well as the symptoms and treatment of hypoglycemia. Hypoglycemia constitutes a blood glucose level below 70 mg/dL where the patient may experience increased sympathetic stimulation, headache, hunger, unsteadiness, altered vision, altered speech, seizures or coma. Upon recognizing the symptoms of hypoglycemia, the patient should first sit down and ingest 15 to 20 grams of carbohydrates. Then after waiting at least 15 minutes to reassess blood sugar levels the patient should call a healthcare provider if these measures are inadequate to relieve symptoms (Michel, 2011).

Patients with T1D can experience a hyperglycemic condition called diabetic ketoacidosis (DKA). DKA is characterized by hyperglycemia, ketosis, and metabolic acidosis (Guven et al., 2009). In cases where there is a complete absence of insulin in the

blood, the body turns to fat tissue for energy because the cells are starved of glucose (Michel, 2011). Lipolysis generates ketone byproducts that increase the acidity of the blood while blood glucose levels raise unopposed (Michel, 2011). Insulin deficiency also leaves glucagon uninhibited which allows for the dysregulation of glycogenolysis and gluconeogenesis, further increasing blood glucose levels and protein degradation (Brashers & Jones, 2010).

DKA can develop rapidly and lead to life threatening fluid imbalances. Emergency medical treatment includes intravenous (IV) access with carefully monitored fluid and electrolyte replacement. Fluid administration must be slow and closely monitored due to the potential of rapid fluid shifts to cause cerebral edema (Michel, 2011).

## **Long-Term Complications**

Uncontrolled blood glucose levels lead to long-term complications in patients with T1D (The Diabetes Control, 1993). Patients who do not continue to have educational support for the management of their diabetes are at a higher risk of developing chronic co-morbidities (Lange et al., 2014). Even four to six years of diminished glycemic control during adolescence can permanently increase a patient's risk of developing co-morbidities (Silverstein et al., 2005). Poor glycemic control increases the risk of developing macrovascular damage, microvascular damage, and infection (Jones et al., 2010).

Macrovascular complications are more common in patients with type 2 diabetes but are still a risk for those with T1D. Damage to the large and medium-sized vessels can lead to conditions such as coronary artery disease, stroke, and peripheral artery disease (Jones et al., 2010). Obesity, smoking, hypertension, a high-fat diet, and a sedentary lifestyle are factors that increase a patient's risk for macrovascular complications and should be addressed by the healthcare provider (Michel, 2011).

The most common forms of microvascular damage include retinopathy, nephropathy, and neuropathy. The increased risk of infection in diabetic patients is multifactorial: 1) impaired vision due to retinopathy diminishes a first-line defense mechanism to skin breaks, 2) oxygen release from hemoglobin in red blood cells is more difficult when HgbA<sub>1c</sub> levels increase, 3) microvascular and macrovascular changes diminish oxygen delivery and white blood cell availability, 4) the white blood cells that are available are functionally altered by ischemia and hyperglycemia, 5) pathogen proliferation may be enhanced due to the increased availability of glucose as a fuel source, 6) the inflammatory response to pathogens is lessened and signs and symptoms of infection may be unnoticed, and 7) the neuropathy of diabetes decreases the ability to sense injury and infection (Jones et al., 2010).

Vessel damage is theorized to proceed through several mechanisms. Glucose metabolism for some tissues occurs via the polyol pathway. Glucose is shunted to the polyol pathway when the blood is hyperglycemic and is converted to sorbitol. Slowly, the sorbitol is converted to fructose, and the increased concentrations of both molecules pulls water into the vessels and out of the cells, predisposing them to injury. Sorbitol affects nerves by altering ion pumps, Schwann cells, and nerve conduction. Sorbitol also leads to swelling red blood cells and eye lenses. Hyperglycemia causes untimely activation of protein kinase C (PKC) in various tissues. PKC activation causes insulin

resistance, cytokine production, rapid reproduction of vessel cells, intensified contractility, and enhanced permeability. The effect of inappropriately activated PKC is linked to macrovascular, microvascular and neurologic complications. Chronic hyperglycemia also shunts excess intracellular glucose into the hexosamine pathway which results in complications of insulin resistance and cardiovascular damage (Jones et al., 2010).

Persistent hyperglycemia further damages vessels when glucose becomes attached to proteins, lipids, and nucleic acids in a phenomenon called nonenzymatic glycosylation. Specifically, glucose binds to vessel walls, interstitial tissue, and red blood cell proteins. It is the byproducts of glycosylation that are harmful. Based on the properties of these byproducts, injury may be caused by the following mechanisms: 1) thickening of the basement membrane of vessels and protein trapping in the membrane; 2) increased permeability of blood vessels and nerves; 3) release of cytokines and growth factors in the glomeruli and vascular smooth muscle, 4) triggering of lipid oxidation, oxidative stress, and inflammation; 5) nitric oxide inactivation; and 6) increased coagulation in the endothelium. Reactive oxygen species are more prevalent when blood sugar is chronically elevated; oxidative stress contributes to cellular injury as well as the development of gene products that secondarily induce cellular harm (Jones et al., 2010).

### **Patient Education**

T1D affects daily life and entails complex management. Management of this disease is best accomplished when the adolescent takes responsibility for his or her own care (Clement, 1995; Michel, 2011). According to Michel (2011), "Patients who actively manage their diabetes care have better outcomes than those who do not" (p. 1240).

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Clement (1995) defined diabetes self-management education as "the process of providing the person with diabetes with the knowledge and skills needed to perform self-care, manage crises, and make lifestyle changes required to successfully manage the disease" (p. 1204). The goal of diabetes education is for the patient to be empowered to be highly knowledgeable and the most active participant in his or her own healthcare (Clement, 1995).

## **Emergent Education Needs**

At diagnosis, the patient will need to have an understanding of the basic survival skills until more comprehensive understanding can be developed (Lange et al., 2014). It is important to have a parent or legal guardian present because much of the self-care activities and information will have to be taught to the patient at home, requiring the parent to ensure adherence to the diabetes regimen during the early stages of the disease (Jaacks et al., 2014). Knowledge of adolescent development and learning abilities will enhance the provider's communication with the patient as well as contribute to better patient understanding and adherence to the teaching (Coffen & Dahlquist, 2009). It is important to explain the diagnosis to the adolescent and to assuage any feelings of guilt or regret by affirming that the patient did not do anything to cause T1D. The patient will need to integrate the use of insulin into daily life immediately. Explaining the digestion of carbohydrates and functions of insulin will help the patient understand why insulin is needed at so frequently throughout the day. Thus, education regarding the disease process of T1D is beneficial and provides a basis for understanding of the treatments and complications related to diabetes (Lange et al., 2014).

The initial diabetes education must also serve to build up a basic skill set in the patient because the patient is the most effective regulator of his or her body. Blood glucose monitoring and insulin injection techniques are the most important skills involved in diabetes self-care. Knowledge regarding the relationship of blood glucose levels and insulin administration should be addressed during this portion of the educational process. The patient should be educated on sources of carbohydrates and carbohydrate counting to quantify insulin administration (Lange et al., 2014).

After the basics of self-care are addressed, the healthcare provider should explain the detriments of uncontrolled blood sugar. Patients with T1D need to understand their physiological needs and be able to recognize the signs of hypoglycemia and hyperglycemia. This understanding will develop over time; however, in the beginning the provider should help the patient think through the triggers of hypoglycemia and hyperglycemia. Dietary choices, exercise, hormonal influences, insulin administration levels, peer pressure, and feelings of shame or embarrassment can all lead to a deviation in a patient's normal blood glucose levels. The patient should be taught to consider the circumstances in which these adverse effects occur, along with the accompanying signs and symptoms, treatment, management, and prevention of abnormal blood glucose levels. The goal is for the patient to be informed and empowered to respond to his or her own body and to maintain a healthy lifestyle (Lange et al., 2014).

The initial treatment concludes by addressing the use of medical alert identification accessories, available diabetes support services, home and school diabetes management, psychological adjustment, emergency contacts, and integration of the selfcare activities into the patient's everyday life. The provider should stress the importance

of continuing care and form a plan for regular appointments with the patient. Diabetes self-management is an enormous task for any person, and not all the information and teaching can be effectively received by an adolescent during the initial visit. Ongoing education is necessary for successful outcomes as the patient will encounter new experiences and individual problems with this disease (Lange et al., 2014).

Initial and continuing education involve life-changing tasks that the pediatric patient must put into practice to be healthy. Success is not likely to be obtained if the adolescent patient is responsible for the totality of self-care and glycemic control. Coffen and Dahlquist (2009) have proposed four implications of diabetes education with young patients. First, diabetes education for adolescents requires a collaborative team approach to diabetes education, involving input from physical health professionals, mental health professionals, and diabetes educators in the effort to bring developmentally appropriate teaching to the patient. These professionals must also be able to discern when the teaching is more appropriate for parents to receive if the adolescent is developmentally unable to handle the teaching (Coffen & Dahlquist, 2009).

Second, in recognition of the magnitude of the diabetes regimen, promoting adherence to a specific aspect of self-care is better than stressing compliance to the grand scheme of diabetes self-care. Inviting in a whole new lifestyle is seemingly impossible to an adolescent patient. An overwhelming feeling may lead the patient to lose hope and give up on trying to cope. Instead of presenting an overall picture of diabetes selfmanagement "it may be best to maintain a realistic depth of detail in assessment but reduce the breadth of what one strives to evaluate at any one point in time" (Coffen & Dahlquist, 2009, p. 306).

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Third, interventions can be more focused and concise with implementation of detailed task analyses rather than analyses of every single aspect of patient adherence. Intervention programs often cover all facets of diabetes care without targeting the patient's particular needs for intervention. In this light, interventional efforts are more efficient when guided by knowledge of the patient's specific behavior problems (Coffen & Dahlquist, 2009).

Finally, "Although the ultimate goal is for all youth with diabetes to be competent self-managers of their illness, the process of transferring control of the myriad selfmanagement tasks to the youth must be gradual (Coffen & Dahlquist, 2009, p. 306). Every young person can do something to care for his or her diabetes, however, educators should promote patient involvement in self-care rather than responsibility for self-care. The parent-adolescent team is the best approach to diabetes self-management when the parents gradually transfer responsibility for self-care tasks over to the adolescent at appropriate benchmarks (Coffen & Dahlquist, 2009).

# **Continuing Education**

After the initial diagnosis, the patient with T1D needs to continue to meet with healthcare providers on a regular basis in order to progress in self-care. In these regular meetings, the patient should develop a deeper understanding of diabetes and metabolism. HgbA<sub>1c</sub> should be introduced and explained to the patient so that the patient understands how regimen adherence and long-term goals are assessed. HgbA<sub>1c</sub> goals should be discussed during the primary care appointments that follow the initial diagnosis. Nutrition planning can be geared more toward the patient's personal preferences and realistic nutritional goals can be made as the patient discusses the regimen with the provider regularly (Lange et al., 2014).

The psychological issues that a patient with T1D may experience cannot be sufficiently addressed during the initial diagnosis. As the patient continues to live with the disease new emotions and experiences will arise in the patient's life and need to be discussed as they come. Issues such as diabetic crises, hypoglycemia and glucagon treatment, and diabetes management during illness are all factors for which the patient will need to develop competencies. Problem-solving in these situations is enhanced by previous education (Lange et al., 2014).

Adherence to the diabetes regimen is a difficult task for a person at any developmental level (Schiffrin, 2001). Discussing the patient's ambivalence and barriers to adherence is a pertinent intervention for adolescent patients (Trast, 2014). Meeting with a trusted healthcare provider on a regular basis allows for identification of these barriers and provides support for patient-specific interventions. Educating the patient about the long-term vascular complications of uncontrolled diabetes is an important motivational factor, but should be communicated in such a way that is not overwhelming for the patient (Lange et al., 2014).

Finally, continuing education serves as a platform for discussing areas of life that are affected by the patient's diabetes. Holidays and periods with less structure require the same nutritional vigilance and care as the structured times. These times call for increased planning and foresight of the physical needs and psychosocial pressures that the adolescent will face. The strict dietary needs can make traveling difficult for diabetics, and the healthcare provider should address the issues by gathering and assessing ideas while the patient is in a safe environment. Substance abuse, academic achievement, employment, driving privileges, sexuality and new research findings should also be brought into the ongoing patient-provider conversation as these issues will present unique challenges for the patient with T1D (Lange et al., 2014).

### **Adolescent Development**

In adolescence, a person begins to move from concrete operational thinking to formal operational thinking. Their ideas are more adaptable and flexible and they are able to assimilate new information into their framework of life more easily than could a school-age child. They can think about long-term scenarios more effectively and are able to consider paradoxes and inconsistencies and can make logical deductions based on facts. Adolescents can think about abstract concepts such as love and hope, they can think about their own thinking and about the thoughts of others. In fact, they are generally very concerned about the thoughts other people might have about them (Kollar, 2013).

Adolescence is characterized psychosocially by a search for identity. Insecurity is rampant among adolescents as their bodies change on the inside and their appearance is transformed. The roles the adolescent played as a child no longer fit the perceived demands of his peer-group. An adolescent is intensely concerned with social acceptance. He or she will do seemingly anything to achieve acceptance into a group. In this endeavor, an adolescent seeks to find how his or her role fits with the fashions and values of his or her peers. Developing personal values and integrating them with those of the society are important processes of adolescence (Franklin & Prows, 2013). Developmental changes during adolescence include profound physiologic changes that affect diabetes care. The rapid growth and development that occur during puberty is linked to many changes, including an increase in growth hormone (GH) and insulin-like growth factor-1 (IGF-1) levels (Trast, 2014). GH works to promote growth and development, increases blood glucose levels, and diminishes glucose utilization as a fuel source. GH secretion is doubled during puberty (Matfin, 2009). GH and IGF-1 both lead to insulin resistance and, therefore, adolescent patients will need to administer more insulin than their younger and older counterparts (Trast, 2014).

The psychosocial aspects of adolescence predispose this age group to decreased glycemic control. Adolescent patients with T1D are different from their peers at a time when they feel increased pressure to conform to social standards. Body image stressors are often difficult for diabetic patients. They must consciously and continuously manage the life sustaining actions of insulin, which their peers never have to consider. At times, the intense desire for social acceptance may trump the motivation to care for basic physiologic needs because self-care activities may feel embarrassing and cause the adolescent to stand out in an undesirable way. These psychosocial factors combined with a physiologic increase in counter-regulatory hormones require a developmentally appropriate intervention by healthcare professionals (Trast, 2014).

#### **Motivational Interviewing (MI)**

Motivating an adolescent to prioritize diabetes self-care despite the strong tendencies to give in to competing social pressures must go beyond a simple transferring of knowledge from the healthcare provider to the patient. According to Trast (2014), adolescents live healthier lives when their parents are supportive, care is individualized,

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care includes psychosocial and psychological support, and their illnesses is not a major source of family strife. MI is a behavioral change counseling method that is focused on helping patients surface their own desires to change rather than using pedagogical means to promote change. In various clinical trials, patients who were exposed to MI were more likely than their counterparts to participate in their care, participate in follow-up visits, comply with glucose monitoring, maintain better glycemic control, and experience fewer adverse effects and hospitalizations (Rollnick et al., 2007).

MI is based on the premises that motivation for change can be developed within a therapeutic relationship and that people want to change but are often hindered by their own ambivalence. For example, a 14-year old diabetic boy may have a habit of eating large amounts of candy with his friends when they are together. He knows that this practice negatively effects blood sugar control, but to abstain would separate him from his peers and cause him to stand out in an undesirable way. Unfortunately, these warring thoughts often leave a patient feeling bewildered and no change occurs (Rollnick et al., 2007). The MI intervention is fueled by the idea that "motivation for change is actually quite malleable and is particularly formed in the context of relationships" (Rollnick et al., 2007, p. 6).

The technique of MI is described as being collaborative, evocative, and protective of patient autonomy. These three characteristics make MI an appealing and effective technique in dealing with adolescent patients. First, MI is a collaborative process in that it is conversational, a dialogue in which both sides influence the end result. This collaboration allows for specificity and individualization of care. It also allows the adolescent to feel respected and essential to the healthcare process because his or her

ideas are integral to the process. The ability of the adolescent to exhibit formal thinking patterns enables the adolescent to recognize and discuss ambivalence to change. Secondly, MI is evocative. Instead of telling the patient what to do, the goal is to elicit the healthy desires existing within the patient. Drawing these out of the patient is more powerful than trying to persuade the patient to do what the provider wants. The MI technique takes into account the idea that human nature intrinsically resists coercion. People want to be healthy, but their desires and efforts toward health are often buried under loads of fears and misconceptions about the demands of healthy living. Thirdly, MI honors patient autonomy. The technique hinges on the patient's choice to change. A healthcare provider cannot really make the patient do anything to promote health. Despite the risk of the patient making unhealthy choices, a technique free of coercion is an important factor in behavioral change. These three MI characteristics are to permeate the environment of the conversation with the patient in need of behavioral change (Rollnick et al., 2007).

When MI is put into practice, the provider will turn away from reflexive correcting, seek out the patient's intrinsic motivation factors, listen to the patient empathetically, and empower the patient to accomplish healthy change. MI is accomplished through establishment of a therapeutic provider-patient relationship in which the provider resists the tendency to persuade the patient to do what the provider desires. When a provider learns of an unhealthy pattern in the patient, it is natural for the provider to tell the patient that they should put a stop to the behavior. Yet this "righting reflex" puts the patient on the defensive and does not allow the patient to explore his or her own desires to change (Rollnick et al., 2007, p. 7). Rather, it brings forth the surge of fears and ambivalence within the patient which leads the patient to resist change. Instead, the provider will search out the patient's motivational sources and desires. In this exploration, the provider must practice good listening skills and exhibit empathy. MI is built on the provider first being a learner and then guiding the patient to explore the findings (Rollnick et al., 2007). In the words of Pascal (n.d.), "People are generally better persuaded by the reasons which they have themselves discovered than by those which have come into the mind of others" (para. 10). Finally, the provider empowers the patient to implement the change that he or she already desires. Therefore, the practice of MI is guided by these four principles of resisting, understanding, listening, and empowering (Rollnick et al., 2007).

It has been established that diabetics experience better glycemic control when they are the main proponents of their own healthcare (Michel, 2011). MI is an excellent behavior change technique to use in the care of T1D patients transitioning into adolescence because it calls for an environment of patient empowerment and collaboration, which foster a positive identity formation (Rollnick et al., 2007). As children enter into adolescence, they become more and more responsible for their own diabetes care and parental supervision tends to diminish. Because of this, care should focus on building internal motivation rather than an external reward system (Naar-King & Ellis, 2011). Clinical trials have found that exposure to MI positively correlates with increased utilization of healthcare, stronger commitment to blood glucose monitoring, and better glycemic control (Rollnick et al., 2007).

# Conclusion

T1D is a life-threatening disease that necessitates drastic lifestyle demands. Adherence to the diabetes self-management regimen is important for both emergent and long-term health outcomes. Adolescent patients face significantly different psychological and physical challenges to regimen adherence than do children and adults; therefore, patient education for diabetic adolescents must be tailored to the specific aspects of their development. The healthcare professional's role is to provide the patient with the necessary tools for managing care, both physical and psychological. Describing the selfcare activities and lifestyle changes required for diabetes self-management is the first step in pursuing optimal clinical outcomes. Helping the patient to walk through his or her ambivalence to change must then be addressed continuously. MI is an effective behavioral change technique that empowers the patient to change by addressing developmental and personal barriers to change and promoting his or her intrinsic desire to achieve optimal health. Because of the positive outcomes associated with utilizing MI in chronic diseases, MI should play a role in every adolescent's T1D care.

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