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**TITLE PAGE**

# ENDOCRINE AND METABOLIC CONSEQUENCES DUE TO RESTRICTIVE CARBOHYDRATE DIETS IN CHILDREN WITH TYPE 1 DIABETES: AN ILLUSTRATIVE CASE SERIES.

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ABSTRACT

Low carbohydrate diets for the management of type 1 diabetes have been popularised by social media. The promotion of a low carbohydrate diet in lay media is in contrast to published pediatric diabetes guidelines that endorse a balanced diet from a variety of foods for optimal growth and development in children with type 1 diabetes. This can be a source of conflict in clinical practice. We describe a series of six cases where adoption of a low carbohydrate diet in children impacted growth and cardiovascular risk factors with potential long term sequelae. These cases support current clinical guidelines for children with diabetes that promote a diet where total energy intake is derived from balanced macronutrient sources.

KEY WORDS: Type 1 Diabetes, Nutrition, Carbohydrate, Growth, Children

ABBREVIATIONS

ISPAD: International Society for Paediatric and Adolescent Diabetes

NICE: National Institute for Health and Care Excellence

## INTRODUCTION

Nutritional management is a core aspect of diabetes care. International clinical guidelines on the management of type 1 diabetes universally describe the requirement for a healthy diet based on a variety of nutritious foods. The ISPAD (1) and NICE (2) Paediatric guidelines recommend that approximately 50-55%, <35% and 15-20% of energy should be derived from carbohydrate, fat and protein respectively. (1), with an individualized assessment required. At the same time, alternative diets such as low carbohydrate (30-40% energy from carbohydrate) and very low carbohydrate diets (21-70g/day) (3) are promoted for the management of type 1 and type 2 diabetes in various media forums in order to optimise glycaemic control. While there is some evidence that low carbohydrate diet can be effective for weight loss in obese adults (4), and improve glycemia in adults with type 2 diabetes (5), there is no supportive scientific literature in children with type 1 diabetes, and there are concerns that any cardiovascular benefits of weight loss using a low carbohydrate diet in adults may be countered by an unfavourable lipid profile(4).

A nutrient-rich diet that meets individual energy, vitamin and mineral requirements is important for normal growth and development in children. Adherence to a low carbohydrate diet in a bid to reduce glycemic excursions and insulin requirements has the potential to result in a low total caloric intake, mineral deficiencies and lead to suboptimal growth. While clinical guidelines note the potential for poor growth in children adopting a low carbohydrate diet (1), there are no published data to support this. Furthermore, substitution of carbohydrate with other energy sources such as saturated fat, can lead to an increased risk of developing cardiovascular disease (6, 7).To address this gap in the literature, pediatric endocrinologists and dietitians

across Australia and New Zealand were invited to describe type 1 diabetes cases where adherence to a restricted carbohydrate diet was believed to result in endocrine and metabolic consequences. Publication of each case was approved by the local ethics committee from the contributing centre. Nutrient Reference Values for Australia and New Zealand (8) were used to determine recommended intakes for each individual. Anthropometry utilised the CDC reference growth charts in all cases(9).

**Case 1**

Patient “A” was diagnosed with Type 1 Diabetes aged 12 years 1 month. On initial presentation “A” had hyperglycemia and mild dehydration, with no history of polyuria, polydipsia or reported weight loss. At diagnosis, his HbA1c was 10.3% (89mmol/mol) and thyroid function tests were normal. Type 1 diabetes was subsequently confirmed with positive diabetes autoantibodies. Patient “A” commenced multiple daily injection therapy with meal-time insulin to carbohydrate ratios. Patient “A” lived with both parents and was involved in numerous sporting activities.

Three months after diagnosis his HbA1c had fallen to 6.1% (43mmol/mol). Eight months after diagnosis, aged 12 years 9 months, his HbA1c was 5.8% (40mmol/mol) with his height 149cm (-0.67 SDS) and weight 34.9kg (-1.21 SDS). At this time his parents expressed concern about patient “A”s blood glucose levels increasing above the normal range particularly following his afternoon recess at school. Patient “A” was advised to eat more at lunchtime in accordance with appetite and to cover this with additional insulin, as the family were reluctant to give an additional insulin injection at recess.

Three months later, at 13 years of age, patient “A” and his parents implemented a lower carbohydrate diet in an effort to control his blood glucose excursions. A three day food record showed an average daily intake of approximately 90grams of carbohydrate. His energy intake was an estimated 8,200 kilojoules/day with 20% energy from carbohydrate, 30% from protein and 50% from fat. Calcium intake was less than 30% recommended dietary intake (RDI). At the same time patient “A” joined a cycling team and began training sessions for one to two hours on four occasions per week. Between 13 years and 15 years, patient “A” attended clinic less

regularly than recommended. During this time his weight fell from -1.22 SDS to -1.88 SDS (Figure 1). Investigations for coeliac and thyroid disease were normal.

At 15 years 3 months, patient “A” requested a dietary review as he was hungry and becoming very fatigued during and after cycling. His family were also concerned about glucose excursions noted after high fat, high protein meals. A 24 hour dietary recall showed an average intake of 60g of carbohydrate per day with < 70% RDI of calcium and thiamin. He reported that he was finding the dietary restrictions difficult, and asked his family during the interview “*Can I please have milk after training?”* His usual post-exercise intake consisted of eggs, sausages, bacon and salad. Annual blood tests revealed an elevated fasting cholesterol level of 5.5mmol/l.

From this time onwards patient “A” increased his carbohydrate intake to 150g/day with carbohydrate pre- and post-training sessions. At 16 years his calcium intake met 80% RDI, weight had recovered to -1.31 SDS (Fig 1) and his HbA1c was 6.3% (45mmol/mol).

**Case 2**

Patient “B” was diagnosed with type 1 diabetes at age 8 years 7 months after a classical 3 week pro-drome of polyuria and polydipsia, and confirmatory diabetes autoantibodies. Born to Egyptian parents, her mother had a history of gestational diabetes, her father had type 2 diabetes (on insulin) and a paternal grandmother had type 2 diabetes. The mid-parental height, defined as the genetic height potential based on the parental height, was reported as 162.5 cm (-0.13 SDS). Patient “B” transitioned to insulin pump therapy at the age of 9 years. She was academically high achieving and required a high level of counselling support to manage diabetes related and social anxieties.

From age 10 years, patient “B” expressed concern about her body weight and a strong desire to lose weight. By age 12 years, patient “B” continued to identify her main concern as being her weight and had a firm goal to reach a desired weight of 50 kg and be more athletic. At this time, she was 59.2 kg (1.99 SDS) and had a BMI of 23.7 kg/$m^2$ (1.31 SDS) (figure 2). Menarche had occurred at age 11 years 10 months.

At the age of 12, patient “B” and her parents reported extensive lifestyle changes made within the family unit following her father’s own attempts to manage his Type 2 diabetes and weight loss following bariatric surgery. He encouraged patient “B” to join him in following a low carbohydrate diet. Her parents reported that she was exercising more regularly and insulin pump downloads revealed an average daily carbohydrate intake of approximately 50 grams per day. HbA1c at this time was 7.0% (53mmol/mol). Her dairy intake was noted to be minimal and calcium intake less than 50% of daily requirements. Concurrently, patient “B” reported poor concentration levels but was willing to pursue the low carbohydrate eating plan as she had started to achieve some weight loss (BMI 1.26 SDS) and her parents continued to encourage and provide this diet in the family home. At age 12 years 7 months, elevated fasting total cholesterol of 5.7 mmol/L and low vitamin D were noted. Systolic blood pressure was at the 95$^{th}$ percentile for age.

At age 13 years, carbohydrate intake was restricted further, to an average intake of 22 grams carbohydrate per day. Patient “B” reported low energy levels and lack of enjoyment when playing sport. Fear of hypoglycemia was expressed, impacting upon her sleep, engagement in sport and normal daily activities, fear of entering

carbohydrate into the insulin pump, frequently decreasing the recommended bolus dose for meals, and disconnection of the insulin pump from 1am – 3am. There was an increase in her HbA1c from 6.0% (42mmol/mol) to 8.1% (65mmol/mol).

Patient “B” then developed secondary amenorrhoea. She underwent numerous investigations, including MRI of the brain and pituitary, laboratory tests for pubertal hormones, coeliac and thyroid serology, folate and iron studies, all of which were normal. Her BMI continued to decrease over this time (Figure 2), despite frequent dietetic interventions that aimed to support the family in achieving a balanced, nutritious diet. Concerns regarding the development of disordered eating were raised when she was 13 years of age, primarily due to self-reported fear of including additional carbohydrate in her diet because it may lead to weight gain. This was coupled with obsessively weighing herself every day. Extensive psychological counselling continued to help patient “B” manage these persistent feelings of high anxiety.

From age 14 years onwards, her diet started to become less restrictive, as she herself identified that *‘it was not the right diet for me’* and concern over the disruption to her menstrual cycle was high. She increased her carbohydrate intake to 120 – 140 grams per day, her menstrual cycle resumed and energy levels improved enough to engage in regular physical activities. At this time, her BMI was 24.3 kg/m$^2$ (0.84 SDS).

**Case 3**

Patient “C” was diagnosed with Type 1 Diabetes aged 6 years 3 months. He presented with hyperglycaemia, polyuria, polydipsia, ketosis and bed wetting. His weight on diagnosis was 20.9 kg (-0.22 SDS) and height was 124.8 cm (1.32 SDS).

Positive autoantibodies for type 1 diabetes were confirmed. He started multiple daily injection therapy using insulin-to-carbohydrate ratios for meals and long acting insulin before bed. A 24 hour diet recall based on pre-diagnosis intake showed a wide variety of foods which met Nutrient Reference Value requirements (1) for age. His mother demonstrated a good dietary knowledge; however his father was unable to attend education sessions despite efforts made by the diabetes team.

Two weeks following diagnosis patient “C” was commenced by his family on a low carbohydrate, high fat diet in an attempt to avoid mealtime insulin injections. Carbohydrate intake was limited to 75g daily, with tinned fish and green salad replacing his usual sandwich at school. The mid-morning fruit break was restricted to low carbohydrate fruit only. At this time, it was noted that patient “C” had only gained 500g in weight since diagnosis (see Fig 3) so a carbohydrate intake of at least 40g was recommended at school in accordance with requirements and food preferences prior to diagnosis.

At his second review appointment  2 months after diagnosis, patient “C” had lost further 0.6kg (weight 20.3kg, -0.55 SDS) and was still eating only salad at school with lean meat or fish despite the previous recommendation. The lunch-time injection and often the dinner injection were omitted. A diet history showed his carbohydrate intake was reduced further to 60g/day, providing an estimated 20% of his daily energy intake. His diet met approximately 70% of his expected energy needs, with calcium intake providing only 200mg/day, 28% of his requirement for age. The multi-disciplinary team recommended that a minimum of 30g carbohydrate were included at both lunch and dinner to meet nutritional needs and also assist with patient “C”s self-reported hunger.

On review three months later, patient “C” had lost further weight (400g). Thyroid function tests were normal and a coeliac screen negative. He was continuing to follow a low carbohydrate meal plan with approximately 50g carbohydrate/day, with less than 7g carbohydrate at lunch and dinner. Insulin injections were not being given at school. His HbA1c at this time was 7.9% (63 mmol/mol). Patient “C”s mother expressed the opinion that a very low carbohydrate diet would achieve the best results for her son’s overall health and diabetes. This was related to information provided by social media. A hospital admission for re-stabilisation of his diabetes was recommended but refused by his mother.

At the time of his next appointment, the diabetes team were contacted by staff at patient “C”s school. They reported patient “C” was hungry and asking for food from other children and staff. His teachers noted he had only salad and tins of fish, with protein balls in his lunchbox. During a visit to the school by the Diabetes team, there was a discussion of the importance of a healthy diet at school inclusive of fruit, wholegrains and dairy foods and patient “C”s mother seemed prepared to begin to include carbohydrate foods at school. However, later it emerged that patient “C”s mother was angry with the advice given and expressed the desire to have a different multi-disciplinary team.

Nevertheless, at his subsequent appointment a diet history showed a much less restrictive dietary intake of 150g carbohydrate with 45-60g eaten at school. Patient “C” had gained 1.8kg in weight (22.3kg, -0.35 SDS). His carbohydrate intake was an estimated 35% of his current energy intake with an excessive fat intake >40% energy. Laboratory results revealed an elevated fasting cholesterol 6.3mmol/L (reference range <5.5mmol/L); LDL cholesterol 3.04mmol/L (reference range <2mmol/L) and Triglycerides 1.66mmol/L (reference range <1.59mmol/L). Lower fat

alternatives to achieve <10% energy from saturated fat and fish oil supplementation were recommended.

In subsequent visits, with a more liberalized carbohydrate intake patient “C”s growth improved with weight 24kg (-0.1 SDS) and height 131.8cm (1.32 SDS) centile (Figure 3). Food seeking behaviour ceased at the school.

**Case 4**

Patient “D” was diagnosed with type 1 diabetes aged 4 years. She presented with classical symptoms of polyuria, polydipsia and a 1-2 kg weight loss over a 4 week period. She was not in diabetic ketoacidosis. Her HbA1c at diagnosis was >14% (>130mmol/mol). Her father also had type 1 diabetes, but died 9 months after patient “D” was diagnosed. Her grandparents were the primary caregivers thereafter.

Four weeks after diagnosis her height was 93.5cm (-2.0 SDS) and weight 13.5kg (-1.49 SDS). The mid-parental height was 154cm (-1.6 SDS). Within 4 months of diagnosis a reduced carbohydrate diet had been implemented (less than 40% energy from carbohydrate), and there was weight loss to 12.85kg. For the next 8 years while followed at the clinic, her growth continued to be poor (Figure 4). Patient “D” continued a diet low in carbohydrate with a proportionally high amount of energy derived from fat. For example, based on a 3 day food record at the age of 11yrs, she was reaching 76% of expected energy requirement, with 39% coming from carbohydrate, 42% from fat (48% saturated), and 19% from protein. This food record reached 47% of the calcium, 70% of the phosphorous, and 74% of the magnesium recommended daily intake respectively. She had elevated fasting total cholesterol of 5.2mmol/L while adhering to this diet.

Patient “D” was last seen aged 11.3yrs; height 123.9cm (-3.28 SDS), weight 27kg (-1.77 SDS). At this point her HbA1c was 8.6% (71mmol/mol), on 0.7U/kg/day of insulin, using a twice daily injection insulin regimen. Subsequently, the family refused to attend clinic due to conflict between the clinical team and her grandparents regarding the diet implemented in the household. She was lost to follow up despite efforts from social services. Her final adult height is not available.

Patient “D” had a mean HbA1c over her entire paediatric follow up of 8.1% (65mmol/mol) (range 6.4 – 10.4%, 46 – 90mmol/mol). She had no admissions for diabetic ketoacidosis, and while she experienced frequent mild and moderate hypoglycaemia, there were no documented severe hypoglycaemic events resulting in coma or seizure. Her poor growth was extensively investigated; she had normal thyroid function, coeliac screen, growth hormone testing and normal female karyotype. Her bone age was delayed by 2 years. She was screened for an eating disorder and this was discounted.

**Case 5**

Patient “E” was diagnosed with type 1 diabetes aged 2 years, after a classical history of polyuria and polydipsia. He did not present with diabetic ketoacidosis. His HbA1c at diagnosis was 12.6% (114mmol/mol). He was the first born child of his parents, and there was no family history of type 1 diabetes. Two weeks after diagnosis his height was 84cm (-1.36 SDS) and his weight was 14.2kg (1.31 SDS). The mid parental height was recorded 182cm (1.15 SDS).

18 months after diagnosis, at the age of 3.5years, a low carbohydrate diet was introduced by the parents. Two months after commencing this diet, HbA1c had improved from 6.1% (43mmol/mol) to 5.3% (34mmol/mol) with no severe

hypoglycaemia. Patient “E” was not growing well, with height now 92.4cm (-2.16 SDS) and weight 15.3kg (-0.54 SDS) (Figure 5). A strict low carbohydrate and high fat diet continued to be adhered to, and 6 months later patient “E” had not gained significant weight (15.5kg, -0.86SD). Dietetic evaluation from a 3 day food record revealed that total energy intake reached 86% of estimated energy requirements. Energy derived from carbohydrate was 6%, protein 27%, fat 67% (saturated fat 36%). He had 406% of recommended daily sodium intake. Calcium was 50% of recommended daily intake.

The poor weight gain and short stature were investigated. Thyroid function and coeliac screen were normal. IGF1 was very low (<25mcg/L). Other laboratory investigations demonstrated an elevated total cholesterol of 4.7mmol/L, with normal triglycerides and normal HDL (1.9mmol/L). Serum magnesium, Vitamin B1 and folate were normal.

He proceeded to growth hormone testing, initially with an arginine stimulation test which failed. Peak growth hormone was 4.3 mU/L (normal >19 mU/L). At this stage after negotiation with the family, more carbohydrate was introduced into the diet.

Two months later, average daily carbohydrate intake was 45g/day, total insulin dose was 0.31U/kg/day (on insulin pump therapy), and the HbA1c was 4.9% (30mmol/mol). The patient demonstrated significant hypoglycemia with 2 weeks of continuous glucose sensor data showing 20% of sensor glucose values less than 3.9mmol/L and 3% of time spent with a sensor glucose below 2.9mmol/L. 47% of hypoglycemic events occurred between 11pm and 5am. There were no episodes of hypoglycemic seizures. Poor height velocity was continuing with height now 95.9cm (-2.36 SDS), and weight 16.6kg (-0.55 SDS). Further laboratory investigations

demonstrated an improvement in IGF-1 (28mcg/L). Pituitary growth hormone secretion was tested again with an overnight growth hormone profile, which showed only one overnight peak of 20mU/L. Other pituitary hormones, and a brain MRI, were normal.

**Case 6**

Patient “F” was diagnosed with Type 1 diabetes at the age of 22 months after presenting with a one month history of polyuria, polydipsia, and a 2.0kg weight loss. Four months after diagnosis she was 95.1cm tall (-0.18 SDS) and weighed 14.7kg (0.07 SDS). The mid-parental height was recorded as 170cm (1.1 SDS). Patient “F” transitioned to insulin pump therapy at age 3.5 years and at this time, her parents decided to commence Patient “F” on a low carbohydrate diet in order to achieve less excursions in the blood glucose readings. They cited popular literature and used other internet based blog sites to support this change, including low carbohydrate recipes. However, they were only able to maintain their daughter on a restricted carbohydrate intake of 40 grams per day for one month as they could not find an adequate variety of low carbohydrate foods that their child would accept.

At aged 6 years 9months of age, patient “F” was placed on a low carbohydrate diet again by her parents. The carbohydrate content of her diet was reduced to approximately 40 grams per day for 3 months by following a plan that provided 12 grams of carbohydrate for each main meal and 6 grams of carbohydrate for one midmeal, with other carbohydrate free foods also offered. The goal in doing this was to help improve glycaemic stability, as although she continued to have an average HbA1C of 7.6% (60mmol/mol) (range 7.3 – 7.8% (56-62mmol/mol)) her parents

hoped to alleviate the spikes in her blood glucose levels. At this point patient “F” weighed 21kg (-0.31 SDS) and measured 120cm (-0.31 SDS).

Adherence to the low carbohydrate diet was challenging. Her mother described the daily challenge of finding an adequate variety of acceptable foods for her daughter to eat as annoying and relentless, made more difficult by the fact that her daughter’s school was egg and nut-free. Patient “F” quickly tired of the limited number and repetitive offering of low carbohydrate foods and would often demand more food at bedtime or wake during the night, complaining of hunger. She did continue dance and swimming classes despite her reports that she was hungry all the time.

After 3 months, patient “F”s parents decided to cease the diet again due to their concerns that she looked unwell and tired most of the time, describing her as “deflated” and lacking in energy. She was reportedly very irritable, was always hungry and never satiated by the foods that were offered. She had lost weight, now weighing 20kg (-0.89 SDS) but her linear growth was tracking (121.5cm (-0.08 SDS)) (Figure 6). Laboratory investigations showed normal thyroid function and a negative coeliac screen.

Patient “F” returned to a normal family diet that contained on average 130 – 140 grams of carbohydrate per day. The family continue to offer predominantly foods of a low glycemic index. After three months she regained 3.7kg to return to her pre-low carbohydrate diet weight percentile. This trend was sustained at her subsequent follow-up visit six months after returning to normal carbohydrate intake (Figure 6).

## Discussion

This case series illustrates that carbohydrate restriction in growing children can lead to anthropometrical deficits and a higher cardiovascular risk metabolic profile. Further, fatigue and low enjoyment of sports was reported. To our knowledge, this is the first time that cases have been collated which illustrate the published guidelines that warn to this effect (1, 10). The likely mechanism is that carbohydrate restriction, without compensatory energy intake through other macronutrients (fat and protein), leads to a deficit in total energy intake. This occurs more easily in children than adults as children have additional energy needs for growth. A similar observation for growth has been observed when a very low carbohydrate (ketogenic) diet has been applied to children with intractable epilepsy (11, 12). Furthermore, when dietary fat becomes the principle source of energy, this can result in a high proportion of saturated fat intake, and lead to a lipid profile that raises cardiovascular disease risk (13) as seen in cases 1,2, 4 and 5. High fat diets have also been shown to blunt pituitary growth hormone release (14), which may explain the poor growth hormone response shown in case 5.

A number of studies have examined the association of carbohydrate intake with glycemic control in people with type 1 diabetes (13, 15, 16, 17). Delahanty et al (15) explored the dietary intakes of 532 intensively treated participants in the Diabetes Control and Complications Trial and reported that diets lower in carbohydrate and higher in fat were associated with worse glycemic outcomes. This finding has been supported by studies in children and adolescents using intensive insulin therapy that found higher total fat (16) and lower carbohydrate (17) intakes were associated with higher hemoglobin A1c levels. These studies concluded that improving dietary

quality by increasing consumption of fruit and wholegrain bread and cereals may enhance metabolic profiles in young people with type 1 diabetes.

The effects of restrictive diets in type 1 diabetes are not confined to the physical domain. Common themes in these illustrative cases are anxiety, fatigue, subjection to unnecessary medical investigations and in some cases, clinical conflict and loss to follow up which can have long term implications. The families who adopt low carbohydrate diets, as in this case series, are often well educated, yet rely on personal blogs from social media as evidence that such a diet is in the best interest of their child. Furthermore, while under-reporting of food intake should be considered in any assessment of the nutritional intake of children and adolescents with diabetes (18), families who follow restricted carbohydrate diets often fastidiously monitor carbohydrate intake to ensure they adhere to strictly imposed limits. Health professionals face a predicament between trying to maintain a positive patient relationship, while trying to convince the family that the diet may be detrimental to their child's health.

It is well documented that children and adolescents with type 1 diabetes are at a greater risk for psychological disorders, including a two-fold risk of experiencing a psychiatric disorder and 2.4 times more likely to develop an eating disorder (19). Adoption of restricted eating behaviour can further contribute to the social isolation that patients with type 1 diabetes already experience, and feasibly add to psychosocial burden. Further, the diet may become an additional source of conflict within the family as the child approaches adolescence.

The decision made by families to adopt a restrictive diet is likely to be multi-factorial. Increasing use of newer technologies such as continuous glucose monitoring has

enabled parents of children with type 1 diabetes to observe excursions resulting from food contributing to anxiety. Behaviours such as giving insulin during or after the meal (20) or inaccurate insulin to carbohydrate ratios (21) will cause marked deviations in blood glucose levels following carbohydrate containing meals. Low carbohydrate diets are also fashionable for weight control and are promoted as healthy in lay media. These issues should be carefully addressed by the clinical team. Contributing to this is the variety of media, including popular television, books, magazines articles, and personal internet blogs, that have popularised restrictive diets in type 1 and type 2 diabetes. Such media exploit the intuition that if carbohydrate is the cause of glycemic excursion, then the reduction of carbohydrate intake provides a solution for families dealing with the frustration of daily glycemic variability. Further, the notion that “less insulin is better” is a commonly expressed theme by families and necessary for the multi-disciplinary team to respectfully discuss.

Healthcare professionals working in a multi-disciplinary team may use a variety of strategies to encourage maintenance of a balanced diet when families express the desire to adopt a low carbohydrate diet to control glycemic excursions. For example, postprandial glycemic excursions due to delayed administration of insulin boluses should be checked for, and reassurance given that postprandial glycemic targets can be met without carbohydrate restriction when insulin to carbohydrate ratios are optimized. Further, substitution of lower glycemic index (GI) for higher GI carbohydrates (22), and enhancing dietary quality by decreasing foods high in saturated fat and increasing fibre intake (16, 17), can assist in improving glycemic control. Encouraging meal-time routines, whilst minimising snacking episodes, is also important (23). Utilizing continuous glucose monitoring can be an effective tool

to reinforce that pre-prandial insulin dosing and improved dietary quality can achieve better glycemic control, as well as facilitating insulin dose optimization.

Currently, there is a lack of balanced lay information warning against the dangers of restricted carbohydrate diets in growing children with type 1 diabetes. Published diabetes guidelines, and the societies that commission and endorse them are not visible in the popular media with respect to their dietary recommendations, nor do they react to popular media that promotes potentially dangerous advice. Hence, it is vital that the diabetes team educate families about behaviours to lower postprandial glucose excursions within the context of a balanced diet and the potentially adverse consequences of low carbohydrate diets in childhood.

In conclusion, diets that restrict carbohydrates in children with type 1 diabetes can lead to growth failure, low energy intakes, and a higher risk lipid profile. They are also likely to contribute to psychological co-morbidity and social isolation. Health care professionals caring for children with type 1 diabetes need to carefully monitor families who adopt restrictive nutritional plans to improve glycemic control, and should counsel about the possible physical and psychosocial implications that this may incur. The purpose of this manuscript is not to comment on the adoption of low carbohydrate diets in adults with type 1 diabetes, where optimal growth and development is no longer an issue. Clinical guidelines should continue to caution against such diets in children with type 1 diabetes.

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CONFLICT OF INTEREST

The authors have no relevant conflicts of interest to disclose.

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FIGURE LEGENDS

Figure 1: Height and weight percentiles for Patient “A”

Figure 2: Height and weight percentiles for Patient “B”

Figure 3: Height and weight percentiles for Patient “C”

Figure 4: Height and weight percentiles for Patient “D”

Figure 5: Height and weight percentiles for Patient “E”

Figure 6: Height and weight percentiles for Patient “F”