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Sydney Low Carb Specialists submission to the parliamentary inquiry

Thank you for the opportunity to make a submission. Sydney Low Carb Specialists (SLCS), in Castle Hill is a sub-specialty clinic that is directed by Dr Deepa Mahananda (FRACGP) and Dr Alex Petrushevski (FRACGP). It incorporates their special interest areas of using evidence based nutritional approaches, including carbohydrate restriction & ketogenic therapies, as a therapeutic option or intervention. We work within a multidisciplinary team Accredited Practicing Dietitian Kate Oetsch, Health Coach Kathryn Mayne, Exercise Physio Christopher Kondos and GP Dr Amala Jayasuriya that is supported by a highly dedicated administrative team.

We have extensive clinical experience in the field of therapeutic carbohydrate restriction, having assisted over 1300 patients to date since our clinic opened in 2019. Our consultations involve identification of the root causes of disease through a comprehensive history & examination, providing further patient education and formulation of personalised management plans. Where appropriate we order investigations to identify & monitor insulin resistance, metabolic syndrome, nutritional deficiencies and assess cardiovascular risk. Of utmost importance is our understanding of how to deprescribe medications such as antihypertensives and oral hypoglycaemic agents. We assist patients to successfully traverse some of the real and perceived challenges of low carb eating patterns - 'keto flu' also known as sodium natriuresis, maintenance of muscle mass and how to maintain this pattern of eating long term. These consultations seek to translate the changing scientific information with regards to how to reverse metabolic syndrome.

We would like to focus on references 2, 4 and 5.

2. New evidence based advances in the prevention, diagnosis and management of diabetes, in Australia and internationally

Therapeutic Carbohydrate Restriction (TCR) has been shown to offer benefit to people with diabetes, particularly Type 2 diabetes (T2DM). Its' effect is more profound than pharmacotherapy.[1-2] We believe it should be offered as a first line option in prevention and treatment. Patients electing to use this therapeutic approach should be provided affordable access to monitoring and ongoing support to ensure that it is safely delivered and sustainable for the patient.

All food is composed of 3 macronutrients: proteins, fats and carbohydrates. When adequate fat and protein is consumed, the dietary requirement for carbohydrate is zero[3]. When there is no carbohydrate consumed, glucose dependent tissues are able to utilise glucose produced through gluconeogenesis and glycogenolysis.[4] Hence the idea of TCR which at its most fundamental level involves a restriction of starches, sugar and foods that may contain these such as some dairy. The body can only use carbohydrates as energy once they are broken into their component parts – glucose molecules. It is well known that the body can only hold 1-1.5 teaspoons of sugar in the bloodstream at any one time.[5] Any elevation past this level will provoke a compensatory rise in

insulin to ensure tight control of blood glucose. In the setting of prediabetes and diabetes this control is compromised with worsening levels of insulin resistance. Thus, carbohydrate restriction is the only dietary intervention that actually addresses the root cause of diabetes and recognises diabetes as a carbohydrate intolerance.

The problem with literature that discredits low carb diets is that their definition of low carb diets is often much higher than is required to achieve therapeutic benefits. In our clinic we have adopted the definition of a very low carbohydrate diet as 30g carbohydrate or less per day. [6] Moderate low carb diets sit between 30-50g carb/day and liberal low carb diets between 50-100g carb/day. Dietary carbohydrates may vary considerably in their ability to raise blood glucose.[7] However the density of carbohydrate in each food is also a factor.[8] When we ask patients to adopt a very low carb diet, we replace with protein and fats. Protein, being an essential macronutrient, is recommended at a minimum of 0.8g protein per kilogram of ideal body weight per day. However this is very likely a gross underestimate of what humans actually need[9]. We encourage intakes up to 2g protein /kilogram of ideal body weight. Fat is a primary source of energy when carbohydrates are restricted. It provides essential fatty acids and is needed for absorption of fat-soluble vitamins A, D, E and K. Saturated fat intake from whole foods has no association with increased risk of cardiovascular disease and outcomes.[10]

Examples of programs around the world currently delivering TCR treatment as either a first line treatment or in adjunct to pharmacotherapy are listed below:

1. NHS in the UK with the Low Carb Program App
2. Dr Davin Unwin and colleagues at Norwood surgery in the U.K have used a low-carbohydrate programme aiming to achieve remission since 2013.
3. Virta health
4. Defeat Diabetes

Guideline change elsewhere in the world:

1. American Diabetes Association 2019 consensus statement change – acknowledging low carb diets as an effective intervention [11] - Nutrition Therapy for Adults With Diabetes or Prediabetes: A Consensus Report

TCR is not a new intervention. There is evidence that prior to the discovery of insulin in the early 1920s, ketogenic diets (very low carb dietary interventions <30g of carbohydrate/day) were successfully used by multiple diabetic specialists.[12] Dr Frederick Banting's discovery on insulin in the early 1920s changed the field in terms of treatment. It allowed for people to consume higher carbohydrate foods and simply bandaid the blood sugar rise with increasing amounts of insulin. Today we have a plethora of glucose lowering drugs, including the latest and most expensive GLP1 agonists, as the most common initial therapy for T2DM. Even in the best-case scenarios with good medication compliance, long term management remains challenging and in this standard of care patients often become progressively more reliant on multiple medication agents, none of which completely mitigate the complications of diabetes. However, a very low carbohydrate diet (ketogenic diet) has been shown to halt and reverse Type 2 diabetes in the majority of people.[13]

We advocate for use of **continuous glucose monitors**. This innovation in the past decade has led to great improvement in the management of all forms of diabetes. [14] It has been great to see full subsidy and co-payment subsidies available to Australians with Type 1 diabetes as of July 2022. However, we feel this should be further extended to all those with T2DM so that improved glycemic control can be achieved with better patient self-education, awareness and instantaneous

biofeedback from the CGM. Additionally, it is a painless intervention comparative to requesting patients obtain fingerpick blood sugar levels 5-10 times a day depending on how often they eat.

4. Any interrelated health issues between diabetes and obesity in Australia, including the relationship between type 2 and gestational diabetes and obesity, the causes of obesity and the evidence base in the prevention, diagnosis and management of obesity

There is a significant problem with overweight and obesity in Australia, with the rates of morbid obesity on the rise. The pace of increase is most worrying amongst children. We know that children who are overweight or obese are likely to struggle with this into adulthood. Over half of Australians have T2DM or prediabetes. The numbers could be higher if we added the number of Australians who have insulin resistance. Not only is there an impact at an individual and family level but there is a financial impact that can no longer be absorbed by state and national governments.

The standard methodology for weight loss in Australian medical guidelines is to 'eat less, move more' This calorie centric methodology ignores the hormonal effects of protein, fat and carbohydrate calories. This is particularly of importance with the effect on the hormone insulin which is a key regulator of blood glucose. Calories from carbohydrates raise insulin the most, protein exerts a moderate rise if consumed in excess to what is needed to maintain lean muscle mass and fat does not prompt significant insulin rise. There may be some variations of a low carb diet where deliberate calorie restriction is incorporated such as in the setting of weight loss. However, patients often spontaneously reduce calorie intake with very low carb diets even though they are not specifically directed to do so. The mechanisms behind this aren't clearly understood but it is postulated in may be in part to circulating ketones, protein generating a potent satiety signal and lower insulin levels which decrease appetite.

Obesity is the result of chronic hyperinsulinaemia[15], rather than the cause of elevated blood sugars. Chronically elevated insulin and subsequent insulin resistance is the fundamental driver of modern chronic disease states. [16] Apart from storing glucose in fat, humans can store glycogen in the liver and skeletal muscle. It is not unusual for people to consume carbohydrate at a level that exceeds their oxidative and storage capacity, which necessitates surplus glucose being converted to fat through a process called de novo lipogenesis. T2DM is associated with significantly impaired oxidative metabolism and insulin stimulated glycogen synthesis stemming from defects in GLUT 4 transport activity, exemplifying why T2D is a disease of carbohydrate intolerance. The inability to dispose of dietary carbohydrate in a normal healthy manner leads to hyperglycemia, hyperinsulinaemia and enhanced rates of de novo lipogenesis, a process that leads to ectopic fat accumulation in tissues. [17] The accumulation of this fat is believed to be a primary mechanism that impairs the normal insulin signalling networks. Ectopic fat accumulation and altered adipokine secretion from adipose tissue contribute to the metabolic inflexibility and insulin resistant phenotype. Moderate intensity exercise can reduce glycogen stores and promote enhanced glucose uptake into skeletal muscle even in people with severe insulin resistance, but many people do not engage in enough physical activity to derive benefit from this method of improving insulin sensitivity.[18]

Whilst many individuals with metabolic syndrome or T2D are overweight or obese, many are also of 'normal weight.' The adipose centric model posits that each person has what can be considered as an individual threshold for fat storage in adipose tissue. Some people have a large capacity for storage and may double or triple adipose tissue mass without experiencing significant insulin resistance. This expansion acts as a sink for excess glucose, thereby protecting a person from hyperglycaemia or ectopic fat accumulation. This may explain in part the obesity paradox[19] – where an individual with higher body weight seems to have lower risks for certain health outcomes or

better prognoses than would be expected based on their size. In contrast, individuals who are thin on the outside but metabolically unwell (normal weight obese) have multiple markers for metabolic syndrome despite their normal body mass index (BMI). This could be explained by their adipose storage threshold being low, they do not store excess triglycerides in their adipose tissue but rather viscerally and ectopically such as liver, skeletal muscle and pancreas which further worsens dysregulated insulin signalling and consequent elevated blood glucose.[20]

TCR can assist in improving all facets of the metabolic syndrome – reducing blood glucose, reducing insulin levels, reducing blood pressure, waist circumference and triglycerides. This occurs when carbohydrates such as that from starch and whole grains, are restricted. As a results therapeutic levels of ketones are often produced. The appropriate level of carbohydrate restriction will vary among patients and should be health goal dependent. Generally at lower levels of carbohydrate restriction, most adults will enter into nutritional ketosis. In the state of nutritional ketosis, the body relies primarily on fatty acids for energy and a small amount of ketones are detected in blood, urine and the breath. Ketone levels induced by carb restriction don't approach the levels induced by frank insulin deficiency as is the case with diabetic ketoacidosis (DKA). This low level nutritional ketosis is not harmful and is actually therapeutic as it results in an anti-inflammatory effect . It is thought that the ketone body beta hydroxybutyrate blocks the NLRP3 inflammasome mediated inflammatory disease. Taken together, these findings suggest that the anti-inflammatory effects of caloric restriction or ketogenic diets may be mechanistically linked to BHB mediated inhibition of the NLRP3 inflammasome, and point to the potential use of interventions that elevate circulating BHB against NLRP3 mediated proinflammatory diseases.[21]

DKA is a rare risk in those with insulin deficiency such as in type 1 diabetes or sometimes insulin dependent Type 2 diabetes. Patients on SGLT2 inhibitors are at risk of euglycaemic DKA particularly in the context of illness or prolonged fasting, although this is thankfully rare.

GESTATIONAL DIABETES (GDM)

In Australia 1 in 7 women are affected by GDM, this is close to American statistics of 18% of pregnant women having gestational diabetes. We know that 2/3 women of reproductive age are overweight or obese. This increases the risk of developing type 2 diabetes.[22] Many women who are diagnosed with gestational diabetes actually had prediabetes that was undiagnosed prior to pregnancy. [23]

Research has made several things clear:

1. Blood sugar is naturally meant to run lower in pregnancy
2. Many women come into pregnancy with high blood sugar (prediabetes) without knowing
3. Even mildly elevated blood sugar carries risks for mother and baby

What influences increasing insulin resistance in pregnancy? Insulin resistance is required to send more nutrients to the baby. Normally this doesn't result in higher blood glucose levels (BGLs) as the pancreas secretes 2-3x more insulin in pregnancy[24] to account for this. But in GDM the body cannot keep up with insulin production and/or the insulin resistance may be so high that the body cannot maintain normal BGLs without significant changes to diet, exercise, supplement habits (and in some cases meds/insulin). Placental hormones and antenatal weight gain contribute to insulin resistance.

GDM is traditionally diagnosed using a 24-28 wk 75 OGTT test. Diagnostic criteria come from the HAPO Trial.[25] It has traditionally been managed with a low glycaemic index eating plan – high grain, low fat which asks women to have 45-75g carb at every meal and encourages physical activity if

possible. This equates to 9 servings of bread, rice, cereal or pasta a day and is setting women up for failure, nutrient deficiencies and excess energy intake in proportion to energy needs.[26]

The problems for the baby from uncontrolled maternal blood sugar
High blood sugar is a known cause of birth defects and can impact growth, development and metabolic health for life. Foetal blood sugar has a linear relationship with the mother's blood sugar. Higher insulin and sugars lead to higher percentage of body fat in the baby. Children of women with poorly controlled GDM face a 6x higher risk of obesity or T2DM in their lifetime. Some studies estimate up to 19x higher.[27]

The HAPO trial observed *o*ver 25000 women in 9 different countries, measuring their BGL and monitoring outcomes. It showed that high maternal blood sugar is the most common cause of excess foetal growth (macrosomia). Women with higher blood sugars had higher risks of caesarean section , neonatal hypoglycaemia, cord blood serum C-peptide – measure of foetal insulin production and higher risk of elevated blood pressure after delivery. [25]

High blood sugar in utero has a profound effect on foetal programming

- Maternal hyperglycaemia results in leptin dysregulation & impaired methylation (**higher neural tube defects**) and is likely contributing to long term programming of excessive adiposity later in life [28]
- High glycaemic index diet in pregnancy associated with metabolic syndrome in offspring at age 20 [29]
- Amniotic fluid insulin levels, which reflect foetal pancreatic insulin production, correlate with obesity during adolescence [30]
- Diets high in grains are linked to excess infant birth weight [31]

Put simply excess carbohydrate intake = higher Blood sugar = higher insulin levels = more fat storage

5. The effectiveness of current Australian Government policies and programs to prevent, diagnose and manage diabetes

The current standard Australian diet as set out by the **Australian Dietary Guidelines (ADG)** does little to change the trajectory of our patients with Type 2 diabetes. It makes very little sense to be feeding people more than 1-2 teaspoons of glucose per meal when Type 2 diabetes is a condition of carbohydrate intolerance. This diet promotes high carbohydrate intake primarily in form of grains – made up of simple and complex carbohydrates. Dietary carbohydrate is a primary driver of hyperinsulinemia, which is a hallmark of metabolic syndrome. Metabolic syndrome is a clear precursor & comorbidity of those with T2DM and as such any dietary strategy aimed at preventing T2DM needs to fundamentally address chronic hyperinsulinemia as a core driver of this disease.

The ADG low fat guidelines are asking people to eat in a way that works against their human physiology – higher carbs that trigger insulin which is a fat storing hormone. We now know from the totality of the evidence that saturated fats from whole foods do not worsen cardiovascular disease risk. [10] The dietary guidelines promote the use of seed oils (sunflower, safflower, margarine) in replacement of saturated fats from wholefood sources such as butter, ghee, tallow, suet and coconut oil. The emerging issues with the increased use of seed oils in the past 50 years has coincided with increased cardiovascular disease and Type 2 diabetes. There are strong mechanistic links showing that seed oils can change the ideal omega-3:omega-6 ratio from 1:1 to 1:30. This results in a high concentration of linoleic acid that causes oxidation to LDL and increases cardiovascular disease risk. [32] Furthermore, interventional trials incorporating high doses of seed oils

as a replacement for naturally occurring saturated fats actually showed an increased risk of death and coronary heart disease, despite their LDL cholesterol lowering properties. [33,34]

Our public institutions have programs in schools, hospitals and aged care facilities that follow the dietary guidelines – this has resulted in setting up our young, ill and elderly for worse health outcomes. The guidelines themselves are written for healthy populations as acknowledged on page 2 of the guidelines 'The Guidelines are intended for people of all ages and backgrounds in the general healthy population, including people with common diet-related risk factors such as being overweight. The Guidelines do not apply to people with medical conditions requiring specialised dietary advice, or to frail elderly people who are at risk of malnutrition.' Yet the accepted thinking amongst medical professionals is that the framework of caloric restriction is the only way to provide effective treatment.

None of the conventional **diagnostic criteria for Type 2 diabetes** include a measure of insulin, which explains in part why millions of people with significantly impaired insulin sensitivity remain undiagnosed. Often the diagnosis occurs when HBA1C IS >6.5% and fasting BGL is >7.0 mmol/L . We would be advising medical training programs to increase awareness and education on how to make earlier diagnoses of insulin resistance with the routine use of insulin levels as part of oral glucose tolerance tests and potentially the C-peptide blood test.

The guidelines ask people to reduce their glucose consumption without identifying or assisting people to **address sugar/food addiction and harmful use of ultra processed foods**. We work very hard in our clinic to support people through a health coaching program which aims to guide the patients from theoretical knowledge of TCR to the practicality of applying this in a consistent and sustainable manner. At present lifestyle related health coaching delivered by a certified health coach is not subsidised by the government or private health funds which makes access to this intensive service challenging for many individuals. Food addiction is yet to be recognised by the he Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision (DSM-5-TR) however there are effective programs that successfully address this to ensure sustainable dietary pattern change.[35]

Lastly doctors are not able to **prescribe diabetic medications with a PBS subsidy** when a patient has a HbA1c under 7%. Yet, it is known that tighter and earlier control has shown to result in continued reduction in diabetes microvascular complications.[36] This is an important omission in the criteria for medications to attract PBS subsidy for those patients committed to TCR as a first line option. For some patients to successfully maintain diabetes reversal, they may require the assistance of pharmacologic agents such as metformin, DPP4 inhibitors, GLP1 agonists and SGLT2 inhibitors at low dose. This will become prohibitive in cost if the PBS subsidy is not maintained for 2 specific groups; T2DM longstanding now achieving reversal OR a newly identified individual with insulin resistance who doesn't quite meet the Hba1c criteria for PBS subsidy.

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