DIET COMPOSITION AND PERCEPTIONS AROUND FOOD IN INDIVIDUALS WITH TYPE 2 DIABETES MELLITUS FOLLOWING A LONG-TERM LOW CARBOHYDRATE HIGH FAT DIET

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DECLARATION

I, Tamzyn Elizabeth Murphy, hereby declare that the work on which this dissertation/thesis is based is my original work (except where acknowledgements indicate otherwise) and that neither the whole work nor any part of it has been, is being, or is to be submitted for another degree in this or any other university.

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LIST OF ABBREVIATIONS

24HR	Twenty-Four Hour Recall
ADA	American Diabetes Association
AI	Adequate intake
AMDR	Acceptable Macronutrient Distribution Range
BF	Body fat
BMI	Body mass index
BMR	Basal metabolic rate
BP	Blood pressure
Bpm	beats per minute
BW	Body weight
CDE	Diabetes Care Programme
CHD	coronary heart disease
CI	Confidence interval
CVD	Cardiovascular disease
CW	Mr Christopher Webster (researcher, PhD candidate)
D	Body density
ESSM	Exercise Science and Sport Medicine
F	Female
FFQ	Food Frequency Questionnaire
FPG	Fasting plasma glucose
FPI	Fasting plasma insulin
HbA1c	glycosylated haemoglobin
HDL	High density lipoprotein cholesterol
HOMA-IR	Homeostatic model assessment of insulin resistance

HR	Heart rate
HRQoL	Health-related quality of life
IBW	Ideal body weight
ICD-10	International classification of diseases 10th revision
IF	Intermittent fasting
IQR	Interquartile range
ISAK	International standards for anthropometric assessment
JS	Dr James Smith (principal investigator and primary supervisor of TM)
KD	Ketogenic diet
KL	Dr Kate Larmuth (post-doctoral researcher)
LADA	Latent autoimmune diabetes of adults
LCHF	Low carbohydrate high fat diet
LDL	Low density lipoprotein cholesterol
M	Male
MetS	Metabolic Syndrome
MRC	Medical Research Council
MUFA	Monounsaturated fatty acids
NAFLD	Non-alcoholic fatty liver disease
NCD	Non-communicable disease
NRV	Nutrient reference value
PAF	Physical activity factor
PCOS	Polycystic ovarian syndrome
PUFA	Polyunsaturated fatty acids
RCT	Randomised controlled trial
RDA	Recommended dietary allowance
RDI	Recommended Dietary Intake

ROS	Reactive oxygen species
SAFOODS	South African Food Data System
SD	Standard deviation
SEMDSA	Society for Endocrinology, Metabolism and Diabetes of South Africa
SFA	Saturated fatty acids
SSISA	Sports Science Institute of South African
T2D	Type 2 Diabetes Mellitus
TD	Prof Timothy Noakes (secondary supervisor of TM)
TDEE	Total daily energy expenditure
TDEI	Total daily energy intake
TG	Triglycerides
TM	Ms Tamzyn Murphy (researcher, MSc candidate, author of this Thesis)
TNF	The Noakes Foundation
UCT	University of Cape Town
VLCHF	Very low carbohydrate high fat diet
WHO	World Health Organization
WHR	waist to hip ratio
WMD	Weighted mean difference
Y/O or y	Years old

DEFINITION OF TERMS

'Adequate intake' (AI), "The average daily nutrient intake level based on observed or experimentally-determined approximations or estimation of nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate."[1].

'Banting', a popular name for an LCHF diet in South Africa.

'Conventional diet', a moderate-to-high carbohydrate, low fat, calorie-restricted diet.

'Current' or 'post-LCHF', at the time of the study.

'Diagnosis', at the timepoint when T2D was diagnosed.

'Fatty vegetables', describe avocado pears and olives for the purpose of this dissertation. These food items were included in this category, rather than in the more technically correct category "fruit", in order to better describe their relatively fat-rich and carbohydrate-poor nutrient profile.

'High carbohydrate diet', carbohydrate content > 45 % of TDEI [2].

'Intermittent fasting' (IF), describes various recurring patterns of eating in which little or no energy is consumed for extended periods of time (e.g. 16 hours to 5 days), with normal intake resuming between these periods [3].

'Ketogenic diet' (KD), generally the same as 'very low carbohydrate high fat diet' (VLCHF), but in the context of treating refractory epilepsy carbohydrate intake is < 10 – 20 g. d^{-1}.

'Low carbohydrate high fat diet' (LCHF), carbohydrate < 130 g. d^{-1} or < 26 % TDEI [2].

'Moderate carbohydrate diet', carbohydrate content 26 – 45 % of TDEI [2].

'Nutrient reference value (NRV)', A daily nutrient level estimated to meet physiological requirements (however, NRV also takes social and emotional requirements into consideration when setting the NRV) [1].

'Pre-LCHF', at the closest timepoint before adopting an LCHF diet.

'Real food', unprocessed, whole food.

'Real-world', a free-living setting or experience, where diet is ad libitum and self-directed.

‘Recommended dietary intake (RDI) or allowance (RDA)’, “A daily nutrient level estimated to meet the requirements of half the healthy individuals in a particular life stage and gender group” [1].

‘Researchers’, refers to this study’s research team, and includes the author (TM) unless stated otherwise.

‘T2D pharmacotherapy’, includes medication and insulin prescribed to treat T2D.

‘The author’ refers to Tamzyn Murphy (myself).

‘The diet’, LCHF.

‘Very low carbohydrate high fat diet’ (VLCHF), carbohydrate 20–50 g. d^{-1} or <10% of TDEI (carbohydrate quantity required to induce ketosis in most people) [2].

ABSTRACT

Background: Type II diabetes mellitus (T2D) is described as a progressive metabolic disease, characterised by disrupted glycaemic regulation, and is associated with high rates of morbidity and mortality. Low carbohydrate high fat (LCHF) diets may be particularly effective and sustainable for the treatment of T2D and have become a popular 'self-therapy'. This study investigates the real-world dietary composition and potential effects of a long-term LCHF diet on T2D.

Materials and Methods: A multi-method descriptive study investigating the diet and related aspects in 28 adult T2D patients perceiving to follow an LCHF diet for ≥ 6 months. Data collection included a quantitative phase (general detailed health and a food frequency (FFQ) questionnaires, 24-hour diet recall (24HR), 3-day food record, anthropometry, blood pressure, blood sampling, past medical records) and a qualitative phase (semi-structured interviews).

Results: LCHF (duration 2.1 ± 1.5 y) consisted of 67% fat (143.2 ± 67.9 g, mainly saturated fat from added fat, eggs, meat, poultry, seafood and full cream dairy), 10% carbohydrate *(*64.0 ± 27.9 g, primarily from full cream dairy, nuts and seeds), 20 % protein (96.0 ± 37.4 g, primarily animal origin) and 3% alcohol (primarily wine). Cholesterol intake (616.3 (402.8–804.2) mg) was higher, and fibre (14.7 ± 7.5 g), calcium and folate lower than recommendations. Added sugar (0.5% of energy intake) and processed foods were seldom consumed. Daily energy intake was 1946.3 ± 807.2 kcal (436.7 ± 728.3 kcal. d^{-1} calculated energy deficit). Intermittent fasting (≥ 16 h. d^{-1}) was followed by 61% of the group. From pre-LCHF to the time of the study, reductions were seen in obesity prevalence (- 50 %), body weight (- 16.9 ± 11.7 %), HbA1c concentration (- 2.45 ± 2.59 %) and T2D prevalence (36 % were no longer classified as T2D based on HbA1c and T2D pharmacotherapy). Non-pharmacologically-induced changes in HbA1c concentrations showed HbA1c reductions in 75 % of the group during the time on LCHF. LCHF was generally reported as sustainable, linked to reduced hunger and cravings and improvements in overall health and health-related quality of life (HRQoL). Challenges included constipation, initial adaptation and social difficulties related to LCHF.

Discussion and conclusion: These findings are the first to describe the nature and composition of a self-selected *ad libitum* real-world LCHF diet and coexisting health parameters. LCHF can be sustainable in T2D patients and may be linked to improvements in glycaemic control, medication reduction, hunger, health and HRQoL.

1. INTRODUCTION

Type 2 Diabetes Mellitus (T2D) is described as a progressive metabolic disease, characterised by disrupted glycaemic regulation (chronic hyperglycaemia, insulin resistance and disrupted insulin secretion) and is associated with high rates of morbidity and mortality. Conventionally, a moderate-to-high carbohydrate, low fat, calorie-restricted diet has been recommended for improving body weight and glycaemic control in T2D [4]. However, a body of research is emerging which indicates that a low carbohydrate high fat (LCHF) diet may be superior in T2D for achieving glycaemic control [5, 6], allowing for decreases in medication [7, 8] and improving lipid profiles (specifically improvements in triglycerides (TGs) and high density lipoprotein (HDL) cholesterol, with no change in serum total and low density lipoprotein (LDL) cholesterol) [6]. LCHF may also surpass conventional diets for promoting weight loss [9] and lowering blood pressure (BP) [9, 10], but evidence is mixed [9-11]. However, concerns persist around the long-term safety of LCHF. This introduction is a narrative review of the research into T2D, an LCHF diet, conventional and LCHF dietary management of T2D, and concerns around LCHF.

1.1. Type 2 diabetes (T2D)

Global T2D prevalence amongst adults is 8.8 % (425 million people) and is projected to reach 14.3 % (693 million people) by 2045, with the greatest increase anticipated in the African region [12]. Estimated T2D prevalence (adults 20 – 79 years old (y/o)) in Africa and South Africa are 3.2 % (2.1 – 6.7 %) and 5.4 % (3.2 – 10.8 %) respectively, which is likely a gross underestimation due to high rates (66.7 %) of undiagnosed T2D in the African region [12]. T2D, together with the other top three non-communicable diseases (NCDs), cardiovascular disease (CVD), cancer and respiratory disease, is responsible for more than 80% of all premature deaths from NCD worldwide [12]. Diabetes accounted for 5 million adult (20 – 79 y/o) deaths in 2015, which is higher than the deaths in 2013 from HIV/AIDS, tuberculosis and malaria combined [13]. In 2015, Africa was the region with the highest death-rate from diabetes globally (79 % of which were in the economically active age group, 1.7 times higher in females (F) than males (M)). Despite Africa's high diabetes morbidity and mortality rates, diabetes-related health expenditure in Africa is the lowest worldwide (0.5% of global expenditure and 7.0 % of Africa's health budget). The economic burden T2D poses to patients,

their families and communities, and nations is very large, and further exacerbated within South Africa by low resources and the double burden of infectious and NCDs [13, 14].

Chronic T2D-associated hyperglycaemia damages vasculature, resulting in debilitating and life-threatening micro and macro vascular complications. These complications include diabetic retinopathy (leading to blindness), neuropathy (associated with pain and mobility problems), peripheral vascular disease (leading to ischaemic necrosis and amputations), and the primary cause of death in T2D: cerebro- and cardio- vascular diseases (e.g. coronary heart disease (CHD), myocardial infarction, stroke, and congestive cardia failure) [13]. Individuals with T2D are at 2 – 5 times greater risk for CVD, compared non-diabetic individuals [15]. Every 1 % increase in HbA1c (glycosylated haemoglobin) concentration is associated with an estimated 11 – 16 % rise in CVD events [15]. Atherosclerotic CVD or CHD is responsible for 70 % of T2D-associated deaths [13].

T2D is also associated with other crippling comorbidities including, obesity, hypertension, dyslipidaemia, sleep apnoea, non-alcoholic fatty liver disease (NAFLD), certain forms of cancer, poly cystic ovarian syndrome (PCOS), and metabolic syndrome (MetS) [13]. MetS represents a clustering of related metabolic abnormalities, namely hyperglycaemia, central adiposity, elevated blood TG or lowered HDL cholesterol concentrations, and hypertension [13]. Insulin resistance is the common and potentially driving factor underlying all of these conditions [16]. Some insulin resistant individuals reach a point when insulin (hyper)secretion can no longer compensate for impaired insulin action, which is when T2D hyperglycaemia develops [16]. Unsurprisingly then, having any one of these conditions is associated with increased risk for the others. MetS increases the risk of developing T2D fivefold and doubles the risk of developing CVD [13]. T2D-associated dyslipidaemia (specifically high blood TG and low HDL concentrations) and hypertension, are thought to be major contributors to atherosclerosis and the development of T2D-associated CVD [13, 16]. Obesity, present in 80 – 90 % of T2D patients [13], is strongly associated with increased risk for developing T2D [17], likely by promoting insulin resistance [18]. The T2D-obesity link is particularly concerning within the South African context with its high rate of obesity [19]. Weight loss of 5 – 10 % is consistently associated with T2D prevention [13] and is known to enhance insulin sensitivity [16]. One randomised trial including 1,079 participants found that every kilogram of weight loss was associated with a 16 % reduction in estimated risk of developing T2D [20]. T2D improvements, including glycaemic control, have been attributed to weight loss associated with reduced energy and carbohydrate intake [10, 16, 21]. Central

(visceral) adiposity is particularly deleterious to metabolic health, possibly due to its lipolytic and pro-inflammatory propensity, and is associated with insulin resistance and T2D, dyslipidaemia, hypertension, CVD and NAFLD [22, 23].

There is concern that T2D and its comorbidities are related to reduced health-related quality of life (HRQoL) (i.e. physical functioning (mobility), bodily pain, general health, vitality (energy), and emotional and mental health). Results from the Look AHEAD study indicate that T2D alone doesn't negatively impact on HRQoL [24]. However, lower HRQoL in T2D was related to increased body mass index (BMI), major comorbidities, physical complaints, insulin use, and reduce physical fitness [24]. Other studies corroborate these findings, relating T2D hyperglycaemia, progression, complications and comorbidities to lower HRQoL [25]. Obesity appears to mediate the relationship between T2D and HRQol, at least to some degree, as magnitude of overweight is related to degree of HRQoL-impairment [26].

1.2. T2D management guidelines

T2D management aims to reduce blood glucose concentrations, slow T2D progression and onset of complications, and reduce comorbidity risk. The Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA) provide T2D management guidelines consisting of a three-prong approach, including diet, exercise and pharmacological interventions: Physical activity recommendations entail 150 minutes of aerobic exercise per week, at 50 - 70% of maximal heart rate (HR), and resistance training three times per week, as well as flexibility and balance training 2-3 times per week in older adults, to reduce T2D morbidity and mortality [13]. T2D pharmacotherapy for glycaemic control includes medications (starting with Metformin) and insulin therapy (for patients not-achieving adequate glycaemic control with 2-3 oral glucose-lowing medications), and is generally combined with medications for prophylaxis or treatment of T2D comorbidities (e.g. statins for hypercholesterolaemia, and anti-hypertensives or aspirin for secondary prevention of CVD) [13]. T2D pharmacotherapy is associated with side effects, including weight gain [13]. A review of the evidence indicates that dietary intervention (detailed below) has comparable effects on glycaemic control compared to T2D pharmacotherapy, and can reduce glycosylated haemoglobin (HbA1c) by 0.5 – 2 % [27].

Previous (2012) dietary guidelines from SEMDSA recommended high carbohydrate intake (45 – 60 % of total daily energy intake (TDEI)), with <10 % of TDEI from sugar and focus

on low glycaemic index/load carbohydrates, evenly distributed between 5 – 6 daily meals [28]. SEMDSA (2012) guidelines also recommended low fat consumption (< 35 % total fat), with a focus on fats from plant rather than animal sources, < 7 % saturated fatty acids (SFA), < 10 % polyunsaturated fatty acids (PUFA), and the remainder from monounsaturated fatty acids (MUFA) [28]. Other SEMDSA (2012) guidelines included 5 – 20 % TDEI from protein, 25 – 50 g fibre daily, limited salt, added sugar, alcohol, cholesterol, fatty red meat and processed foods, and restricted energy for weight loss [28]. However, updated SEMDSA guidelines (2017) are moving away from high carbohydrate, low fat recommendations towards a more individualised dietitian-guided approach [13]. SEMDSA's stand, that there's no ideal percentage macronutrient prescription, is more in line with the 2014 American Diabetes Association (ADA) guidelines [27]. SEMDSA (2017) does not recommend one dietary strategy: "carbohydrate intake (both quality and quantity) should be individualised and guided by the patient's glycaemic control" [13]. However, for T2D prevention, SEMDSA does strongly recommend keeping total fat < 30 %, SFA < 10 % and fibre > 15 g. d^{-1}, and states that the safety and efficacy of long-term high-SFA and high-protein diets is not known [13]. Whole-food sourced carbohydrates (wholegrains, legumes, low fat milk, and 5 daily portions of fruit and vegetables) and fat are preferred over processed and refined foods [13]. Fat guidelines promote MUFAs and omega-3 (n-3) PUFAs from sources like nuts, seeds, fatty fish, and vegetable oils, over SFA-rich animal-sources and tropical palm and coconut oils [13]. T2D patients are told to limit sugar (< 5 % of TEDI), sodium (< 2300 mg. d^{-1}), added fat, fatty red meat, and alcohol (1 daily drink/unit for women and 2 for men) [13]. Energy restriction is recommended (350 – 500 kcal deficit when BMI is 30 – 34 kg. m^{-2}, or 500 – 1000 kcal deficit when BMI ≥40 kg. m^{-2}) to promote 10% weight loss over 6 months [13].

1.3. LCHF diet for T2D management

Despite these guidelines, there is a rapidly emerging scientific and public interest in managing T2D and obesity using carbohydrate restriction. Carbohydrate restriction reduces insulin resistant individuals' requisite compensatory hyperinsulinaemia in response to dietary carbohydrate; and in T2D, associated hyperglycaemia (when additional insulin secretion is no longer possible) [16] . Two recent systematic reviews and meta-analyses of randomised controlled trials (RCTs) comparing LCHF diets to conventional normal or high carbohydrate dietary interventions (carbohydrate > 45 %) for the long-term (6 – 24 months) management

of T2D, indicate LCHF's benefits in T2D [10, 11]: Huntriss et al. (2017) included RCTs with LCHF diet arms as defined by the study-author (106 g. d^{-1} mean carbohydrate intake) [10]. Meng et al. (2017) defined an LCHF diet as prescribed carbohydrate < 130g. d^{-1} or < 26 % TDEI [11]. These systematic reviews and meta-analyses found statistically significant improvements in favour of LCHF diets for HbA1c (estimated effect at one year = −0.28%, 95% confidence interval (CI) −0.53, −0.02, P = 0.03 [10]; weighted mean difference (WMD) 6-24 months: -0.44; 95% CI: -0.61, -0.26; P = 0.00 [11]), HDL cholesterol (estimated effect at one year = 0.06 mmol/L, 95% CI 0.04, 0.09, p < 0.00001 [10]; WMD 6-24 months: 0.07; 95% CI: 0.03, 0.11; P = 0.00 [11]), TG (estimated effect at one year = −0.24 mmol/L, 95% CI −0.35 , −0.13, p < 0.0001 [10]; WMD 6-24 months: -0.33; 95% CI: -0.45, -0.21; p = 0.00 [11]) and systolic BP (estimated effect at one year = −2.74 mmHg, 95% CI, p = 0.03[10]). However, meta-analyses for serum total cholesterol, LDL cholesterol and diastolic BP did not show statistically significant differences between LCHF and conventional dietary interventions [10, 11].

Hallberg et al (2018) conducted an open-label, non-randomised, controlled study investigating the effects of a novel technology (app)-administered LCHF diet-based intervention compared to usual care, in 218 T2D participants at 1 year [29]. In line with findings from the systematic reviews and meta-analyses discussed above [10, 11], Hallberg's LCHF group experienced significant improvements in HbA1c (7.6 ± 0.09 % to 6.3 ± 0.07 %, p < 1.0×10^{-16}), TG (- 24%, p < 1.09×10^{-16}), and HDL cholesterol (+ 18%, < 1.09×10^{-16}) [29]. Other significant improvements were observed in biomarkers of insulin resistance, namely: homeostatic model assessment of insulin resistance (HOMA-IR), liver function, and inflammation. However, unlike the systematic reviews and meta analyses, Hallberg et al. (2018) reports a small but significant increase in serum LDL cholesterol concentrations in the LCHF group (+ 10 %, p = 5.1×10^{-5}). Contrastingly, the group receiving usual care from their medical providers and diabetes education program had no significant changes in biomarkers at 1 year.

The systematic review and meta-analysis by Huntriss et al. (2017) reported decreased T2D pharmacotherapy in the LCHF intervention group of 14 RCTs (9 of which were statistically significant) [10]. Hallberg et al. (2018) also reported reductions in T2D pharmacotherapy in the LCHF app arm: a significant reduction in non-metformin pharmacotherapy (56.9 ± 3.1% to 29.7 ± 3.0%, p < 1.09×10^{-16}) and 94 % stopping or reducing insulin therapy [29]. These reductions in T2D pharmacotherapy likely decreased the observed improvement in HbA1c in these studies.

RCTs indicate that LCHF consistently surpasses energy restricted conventional diets for early weight loss (3 – 6 months), however weight regain thereafter appears to offset this initial weight loss benefits [10, 11, 30]. Huntriss et al (2017) reported no significant difference in weight loss or energy intake at one year between intervention arms [10]. Contrastingly, Hallberg et al. (2018) found that the LCHF-app arm lost significantly more weight than the usual care intervention ($p < 10\text{-}16$).

Therefore, an LCHF diet may be a feasible, and possibly superior, alternative to conventional moderate-to-high carbohydrate, low fat, energy-restricted diets for T2D patients, particularly for improving glycaemic control, reducing medication usage, improving cardiovascular risk (by improving risk factors including lipid profiles, body weight and BP), improving liver health and reducing inflammation.

1.4. LCHF: describing the diet

The definition of an LCHF diet varies between studies, making it difficult to compare study results and investigate relationships between the diet and its components and their clinical effects [10]. However, the definition proposed by Feinman et al. (2015) is being increasingly accepted in subsequent publications, namely that an LCHF diet contains < 130 g. d^{-1} or < 26 % carbohydrate [2]. Carbohydrate intake of 20 – 50 g. d^{-1} (or < 10 %) is a very LCHF (VLCHF) or ketogenic diet (KD), 26 – 45 % carbohydrate is moderate carbohydrate, and > 45 % is considered high carbohydrate [2]. An LCHF diet generally contains > 60 % fat; and 15 – 35 % protein (1.3-2.2 g or approx. 1.5 g. kg^{-1} body weight) [10, 11, 29, 31]. Defining LCHF's macronutrient content according to absolute (g) rather than relative (%) intake for carbohydrate and protein is preferable, particularly in the context of energy restriction. The reason for this is that during an energy restricted diet, absolute carbohydrate content may remain below the 130 g cut-off but exceed relative (26 %) carbohydrate limits [10]. Similarly, during times of low energy intake, the lower range of protein requirements, expressed as percent of TDEI (e.g. 15 %), may be insufficient to meet protein requirements, which are proportional to body weight not energy intake [32].

Popular LCHF information sources (books, websites, social media) provide guidelines regarding LCHF diet quality and food choices. Arguably, the most popular LCHF information-source within South Africa is The Real Meal Revolution, co-authored by well-known University of Cape Town (UCT)-emeritus Professor, Timothy Noakes [33]. The Real Meal Revolution

includes lists of foods to choose (green), limit (orange) and avoid (red), on an LCHF diet [33]. It recommends focusing on whole foods (i.e. avoiding processed, refined foods). It encourages avoiding sugar and grains (which have a high carbohydrate load), and high PUFA (particularly omega-6 (n-6) rich) seed oils (to achieve an optimal n-3: n-6 ratio, which is associated with reduced inflammation and health benefits [34]) [33]. Also limited are starchy vegetables and fruit (due to their high carbohydrate content), artificial sweeteners (due to their insulin-stimulating effects [35] and potential for sparking cravings for sweet taste [36]) and alcohol (which is nutrient-poor, energy-dense, and associated with adverse metabolic effects [37]) [33]. Moderate limitations are placed on nuts, seeds and high-carbohydrate dairy (e.g. milk and yoghurt), which are relatively high in carbohydrate, protein and fat [33]. Low carbohydrate, nutrient-rich whole-foods are encouraged, including animal-sourced foods high in protein and fat (i.e. meat, seafood, poultry, organ meats, eggs and lower-carbohydrate dairy, including cheese and cream), certain fats (high in SFA and MUFAs, e.g. butter, lard, olive and coconut oils), and non-starchy vegetables [33].

Individuals who follow LCHF diets are generally encouraged to eat *ad libitum*, to satiety and to follow their hunger cues [33]. LCHF advocates believe this strategy to be effective and sustainable, due to LCHF's association with reduced hunger and spontaneous reductions in energy intake [38]. RCTs in T2D and non-diabetic participants, comparing weight loss effects of *ad libitum* LCHF diets matched with conventional weight loss control diets (with a usual prescribed 500 kcal deficit), show similar weight loss effects [10, 39]. Therefore, a spontaneous mean energy-deficit in the vicinity of 500 kcal may be anticipated for ad libitum LCHF diets, as most [10, 39], but not all [40], evidence indicates that weight loss experienced on LCHF diets results from reduced energy intake. However, actual energy intake and energy deficits associated with free-living LCHF remains unconfirmed due to the nature of these interventions [39], and more research is required in this regard.

Intermittent fasting (IF) is popularly used in conjunction with LCHF diets, but the research investigating the combination of these two dietary interventions is lacking. IF describes various recurring patterns of eating in which little or no energy is consumed for extended periods of time (e.g. 16 hours to 5 days), with normal intake resuming between these periods [3]. There is compelling evidence supporting IF's comparable or superior improvements in T2D-related metabolic risk factors in normal and overweight humans, including: weight and fat mass loss with relative maintenance of lean body mass [41-46], improved fasting insulin concentrations and insulin sensitivity [47, 48], and reduced risk

factors for cardio- and cerebral- vascular disease (blood lipid concentrations [45] and BP [47]). IF appears to prevent the usual compensatory mechanisms that counter weight loss during continuous energy restriction [41], and is associated with improved dietary adherence [44]. In addition to the overall reduction in energy intake during fasted periods, IF may result in spontaneous reduction in energy intake on non-fasted days as well, without a compensatory increase in hunger (in the context of *ad libitum* protein and fat intake) [44]. Some evidence indicates that IF may be particularly effective in T2D, offering superior fat mass reductions in these patients compared to healthy controls [49]. IF in T2D patients is associated with equivalent or superior reductions in body weight [50-52], HbA_{1c} [50-52], T2D pharmacotherapy [51, 52], and fasting glucose concentrations [53], compared to continuous energy restricted diets. Patients with T2D also experience improvements in HRQoL when adopting IF [54]. The only RCT in T2D comparing long-term IF (2 days energy restriction followed by 5 days of usual diet) with continuous energy restriction, found equivalent benefits for glycaemic control (HbA1c reductions), and a trend towards better weight loss in the IF arms over 3 months [51]. These effects were maintained over the 12-month trial. While HbA1c improvement in this long-term RCT appeared weight-dependent, other shorter-term T2D studies have found IF-associated HbA1c improvements independent of weight loss [50]. A recent 5-week randomised crossover-design controlled-feeding trial found that prediabetic men following an 18-hour IF schedule experienced increased insulin sensitivity, beta cell function and lower BP, oxidative stress and appetite, even in the absence of weight loss [47].

1.5. Concerns regarding LCHF diets

Despite the clear benefits of LCHF for T2D management, various concerns exist around potential risks associated with the diet. One such concern is regarding the nutritional sufficiency of this diet, particularly potential micronutrient deficiencies and insufficient fibre. The concern over micronutrient insufficiency originates in the knowledge that the KDs used to treat paediatric refractory epilepsy are deficient in various micronutrients (calcium, vitamin D, and selenium in particular), necessitating nutritional supplementation [55-57]. Nutritional deficiencies associated with paediatric KDs are largely due to the stringent restriction of protein and calcium-rich foods (e.g. dairy, meat and seafood) and vegetables, necessary to meet the very low carbohydrate and limited protein intake required for clinical effectiveness (<10 g. d^{-1} carbohydrate in the classic KD, and < 20 g. d^{-1} in the modified Atkins KD) [55]. VLCHF

or KDs, as used in non-epileptic adults, limit carbohydrate to < 50 g. d^{-1} or 10 % TDEI [2], which allows for less stringent restrictions on these carbohydrate and protein -containing micronutrient-rich foods compared to KDs used for paediatric epilepsy, allowing for improved nutritional intake [40]. Analysis of two 7-day weighted food records from a recent 6-week *ad libitum* KD (containing 40.4 ± 13.1 g carbohydrate), showed sufficient micronutrient intake for most micronutrients [40]. However, mean intakes for folate, vitamin B5, and iron were marginally low. This indicates that the majority of the group consumed sufficient quantities of these micronutrients, and minor dietary refinements would allow the remainder of the group to meet recommendations. However, vitamin D and selenium intake was very low (for both habitual and KDs diet), indicating that free-living KDs probably provide inadequate quantities of these micronutrients. The vitamin D content of conventional diets is also known to be deficient [58] as vitamin D is not contained in meaningful quantities in most foods (apart from oily fish and fish liver) [59]. However, sufficient sunlight exposure can make up for any dietary deficit [59]. The selenium deficiency associated with KDs is more concerning, and has been linked to cardiomyopathy [57, 60] and sudden cardiac death [61] in children following KDs for epilepsy. Selenium is an essential component of the body's antioxidant system [57], which is particularly important in the context of T2D, as reactive oxygen species (ROS) are implicated in insulin resistance and T2D pathophysiology [62]. Additionally, selenium deficiency is associated with CVD and cancer [57], conditions for which individuals with T2D are already at increased risk [13]. Dietary selenium deficiency would therefore be of particular concern in T2D. However, VLCHF and LCHF diets with non-epilepsy-therapeutic aims, appear to be sufficient in selenium (as well as other micronutrients) [31, 63], as they do not limit the selenium-rich foods (meat and seafood) that are restricted on the strict protein-restricted KDs that are prescribed for epilepsy. Zinn et al. (2018) recently published a descriptive study evaluating the nutritional content of two dietitian-designed non-ketogenic LCHF diets for hypothetical case-studies based on parameters for the average Australian adult male and female [31]. Results showed, "all of the meal plans exceeded the minimum nutrient reference value (NRV) thresholds, apart from iron in the female meal plans, which achieved 86%–98% of the threshold"[31]. This demonstrates that in theory, LCHF can meet nutritional requirements (possibly even female's iron requirements within the context of an LCHF diet that is rich in bioavailable iron sources and low in iron-absorption inhibitors, like phytates found in grains) [31]. However, whether individuals following a real-world *ad libitum* LCHF

diet, would get sufficient quantities of all these nutrients is not known and requires investigation.

The concern over the dietary fibre content of an LCHF diet emanates from exclusion or limitation of certain high fibre foods (specifically whole grains, legumes, starchy vegetables and fruit) from the diet. These foods are major contributors to dietary fibre in conventional diets [64, 65]. Evidence indicates that LCHF diets are not necessarily lower in fibre than conventional diets [40], and when properly formulated to include sufficient non-starchy vegetables and small amounts of fruit and starchy vegetables, can meet recommendations for fibre intake [31, 63]. Epidemiological evidence indicates that dietary fibre is associated with a dose-response reduced risk of mortality from CVD, CHD, cancer, and all causes [66-68]. Prospective cohort studies show an association between diets low in fibre and high in rapidly absorbable carbohydrates and T2D incidence [69-73], in Caucasians, but not African-American individuals [74]. However, in epidemiological studies, diets lower in dietary fibre are also higher in NCD-risk-promoting nutrients, including added sugar, trans-fats, and processed foods [75]. So, extricating fibre from the other nutrients, in order to determine the cause of the increased NCD-risk, is difficult. As such, low dietary fibre intake may be related to health concerns particularly relevant in T2D, but more research is required. Concern also exists regarding low dietary fibre consumption and associated changes in gut microbiome, which may be deleterious to metabolic, immune and gastrointestinal health [76]. However, fibre intervention study results have been inconsistent [76]. Additional research is required to determine the role that different microbiomes and fibre types play, as well as the mechanisms responsible, for the suspected downstream health effects [77].

There is also major concern regarding an LCHF diet's high fat (particularly SFA) content, which is theorised to further increase the CVD (specifically CHD) risk associated with T2D [78]. The diet-heart hypothesis purports that dietary SFA results in elevated serum total and LDL cholesterol concentrations, which in turn promotes endothelial cholesterol deposition, resulting in atherosclerosis and CHD [79]. This hypothesis was advocated by Keys and based on his landmark epidemiological Seven Countries Study, which found associations between SFA intake and serum cholesterol concentrations, and associations between serum cholesterol concentrations with CHD prevalence [78]. Despite, the inability of epidemiological evidence to prove causation, and the lack of supporting evidence from RCTs at the time, the United State of America (USA) and United Kingdom (UK) introduced dietary guidelines (in 1977 and 1983 respectively) recommending reducing total fat intake ≤ 30 % and SFA to ≤ 10

% of TDEI [80]. The implication of these recommendations remains relevant, as they were adopted globally (including within South Africa [81]) and have remained relatively unchanged [82]. While SFA intake is indeed associated with increased serum total and LDL cholesterol concentrations, this may not translate to increased CVD risk [83, 84]. More recent epidemiological studies contradict Keys' conclusions associating total and SFA intake with CHD [85, 86]. The PURE study, in 135,335 individuals from 18 countries, found that total fat and the various types of fat (including SFA) were not associated with CVD, CVD mortality, or all-cause mortality, and that SFA was inversely associated with stroke [85]. Systematic reviews and meta-analyses of observational studies corroborate the lack of association between SFA intake and CVD, CHD, ischaemic stroke, and all-cause mortality [87, 88]. Eating less of one macronutrient means eating more of another in order to keep TDEI constant. Therefore, when investigating the relationship between dietary macronutrient content and health risk, the health implications of increasing the intake of one macronutrient (generally n-6 PUFA or carbohydrate) in order to reduce intake of another (SFA) must be considered. The PURE study showed that unlike SFA intake, carbohydrate intake was associated with increased all-cause mortality [85]. Results from the Nurse's Health Study found that replacement of SFA with refined, high glycaemic load carbohydrate was associated with increased CHD risk [89]. Generally it appears that replacing SFA with MUFAs or PUFAs is associated with reduced CVD risk [90]. Some RCTs investigating reducing SFA intake in favour of n-6 PUFAs indicate no reduction of CHD events, CHD mortality or all-cause mortality [83, 84], while others indicate a slight risk reduction [91-93]. In line with this epidemiological and RCT evidence, recent reviews indicate that biomarkers other than LDL-cholesterol concentration are better predictors of atherosclerotic CVD risk [94]. These include genetic risk scores (GRS) based on a growing number of risk alleles [94], inflammatory and thrombotic markers [94], lipid components and functional measures (specifically triglycerides, apo-B particle number rather than cholesterol concentration, HDL particle number and concentration rather than HDL cholesterol concentration, LDL particle density rather than concentration, cholesterol efflux capacity, lipid metabolites [94]), protein metabolites [94], microRNAs [94], and a variety of subclinical atherosclerosis imaging measures (most notably, the coronary artery calcium (CAC) scan) [94]. The CAC scan surpasses all biomarkers for its capacity to predict atherosclerotic CVD risk across various populations and risks [94].

Concerns also exist regarding LCHF's high fat content potentially promoting insulin resistance [95, 96] and mitochondrial dysfunction [97], both of which have been implicated

in T2D pathogenesis [98, 99]. However human research in these areas is sparse and studies are small [95-97]. Interestingly, there's an incongruency between LCHF diet-associated insulin resistance and the lack of anticipated associated elevations in blood glucose and insulin concentrations [29]. LCHF may induce an adaptive response to reduced dietary glucose and increased fat availability. Potentially, downregulation of pathways involved in glucose metabolism and upregulation of those involved in fat metabolism in response to fuel availability may be responsible [100]. In other words, LCHF may induce a physiological insulin resistance (associated with reduced endogenous glucose supply) in which insulin is elevated appropriately in response to hyperglycaemia. This form of insulin resistance may be distinct from pathological insulin resistance (associated with T2D), in which the increase in insulin resistance is out of proportion to the degree of hyperglycaemia. Additional concerns arise around high dietary fat-induced alterations in gut microflora [77, 101]. Further research is required to investigate the implications, mechanisms and nature of LCHF-induced changes to insulin sensitivity, mitochondrial function and the microbiome.

Safety concerns have been raised over LCHF diets' relatively high protein content, for renal function and bone health [102]. While protein intake on LCHF diets may be higher than on conventional diets, it seldom exceeds 30 % TDEI [10, 103]. Critics theorise that high protein intake (> 25% of TDEI or > 2 g. kg body weight $(BW)^{-1}$ [104]) may chronically increase glomerular pressure and hyperfiltration, thereby damaging the kidneys [105] – particularly concerning in T2D with its high nephropathy risk. Indeed protein restriction helps preserve renal function in pre-existing kidney disease [106]. However, evidence doesn't support the theory that high protein diets impair renal function in the absence of kidney disease [102, 104, 105, 107], even in obese individuals with T2D [108]. In fact, VLCHF diets may improve renal function [109], as indicated by results showing reversal of diabetic nephropathy in rodents [110] and reduced creatinine concentrations in an intervention study in overweight and obese humans with and without T2D [5]. Concerns about bone demineralisation stem from the high protein diet-induced hypercalciuria observed in humans [111]. It is hypothesised that this is indicative of skeletal resorption caused by bone buffering the higher acid-load associated with high protein consumption [112, 113], which must be offset by increased consumption of alkalinising foods (i.e. vegetables and fruit) [102, 114]. While some epidemiological evidence has associated higher animal protein (95 g vs 68 g. d^{-1} in 85,900 women from the Nurse's Health Study) with increased fracture risk [115], the majority indicate that higher intake of protein in general and animal protein in particular, is associated

with better bone mineral density, particularly in the context of adequate calcium intake [116-118]. Short term intervention studies indicate increased calcium absorption, reduced calcium from bone in hypercalciuria, and no differences in bone turn-over on higher protein diets compared to controls [111, 119, 120]. Recent reviews agree that higher protein intake (1.2 – 1.8 (possibly up to 2.4) g. kg BW^{-1}. d^{-1}) in the context of sufficient calcium intake, does not disrupt calcium homeostasis and bone mineral density during weight maintenance or loss, and may benefit bone health and reduce fracture incidence [121-124].

As adherence predicts the success of a dietary intervention [38], there is concern over the poor long-term adherence to LCHF diets that contain < 50 g. d^{-1} carbohydrate [125, 126]. However, adherence to and sustainability of higher carbohydrate LCHF diets (containing 50 – 130 g . d^{-1} carbohydrate), may be no difference from other diets [127, 128]. LCHF [39] and energy restricted (< 800 kcal) [44, 51, 129] diets have unique hunger-suppressing and satiating effects, which may assist with adherence. However, an LCHF diet's less restrictive energy content may make it more suitable than very energy restricted diet for long-term use. More research is required regarding long term LCHF adherence and sustainability.

A recent prospective cohort study and meta-analysis by Seidelmann et al. (2018) has raised concern that low carbohydrate diets may increase mortality. The study examined the dietary records of 15 428 adults (45–64 y) from four US communities and found that low (< 40 % of TDEI) and high carbohydrate (> 70 % of TDEI) diets were associated with increased all-cause mortality risk, particularly when fat and protein were animal- rather than plant- sourced [130]. Moderate carbohydrate intake (50 – 55 % of TDEI) was associated with the lowest mortality risk. Seidelmann's study, has been criticised due to its weak hazard ratios, insufficient control for confounding factors and subjective carbohydrate-intake categories which may affect the accuracy of results [131]. As discussed previously, it is impossible to prove causation from epidemiological studies. Ioannidis (2018), recently wrote in *JAMA*, "In recent updated meta-analyses of prospective cohort studies, almost all foods revealed statistically significant associations with mortality risk"[132]. Therefore, caution must be exercised when interpreting results and drawing conclusions from epidemiological studies regarding particular nutrients' health effects.

1.6. Summary

While it is accepted that the greatest contributor to postprandial blood glucose concentration is dietary carbohydrate [133], the optimal diet and macronutrient composition for the sustainable management for T2D is still debated [10, 134] . Evidence is accumulating in favour of managing T2D with an LCHF diet, over traditional high carbohydrate, low fat, energy-controlled dietary approaches. RCTs indicate that a long-term (6 – 24 months) LCHF diet may provide superior glycaemic control, reductions in medication, and improvement in CVD markers (TG, HDL cholesterol, and BP), with comparable improvements in body weight, and serum total and LDL cholesterol concentrations, compared to conventional diets [10, 11]. However, there are still concerns regarding potential risks associated with an LCHF diet (e.g. implications of high dietary fat for CVD, possible micronutrient and fibre insufficiency, sustainability and adherence). Potential risks may be considered particularly relevant in individuals with health conditions, such as T2D, who follow a self-selected and self-administered LCHF diet, without the approval or supervision of a dietitian or healthcare practitioner. However, health (including T2D status) and related quality of life, actual diet composition, dietary patterns and behaviours, and perceptions around diet (including sustainability), have not been investigated in these high-risk individuals.

Through an online survey, this research group became aware of a number of anecdotal reports of individuals who believed that they'd achieved T2D remission (i.e. they no longer medicate for T2D or meet T2D diagnostic criteria) using an LCHF diet. However, these reports required confirmation, and the health status and risk in these individuals and nature of their dietary intervention, required further investigation. Their diet was adopted and adapted based on self-identified actual life requirements, challenges, and changes in health and T2D status, to create, what to them was a workable and sustainable real-world version of an LCHF diet. This study investigates a group of these individuals, their T2D status, health and HRQoL, diet composition and perceptions around diet. Knowledge about this group's health, diet and experiences will provide insight into the optimal dietary management for T2D in a real-world context and can assist in developing sustainable dietary interventions.

1.7. Aims and objectives

1.7.1. Aims

The primary aim of this pilot study was to investigate the composition of a real-world LCHF

diet and perceptions around diet and health in individuals with T2D who perceive to have followed an LCHF diet for more than 6 months.

1.7.2. Objectives

1.7.2.1. To assess any changes in participants' diabetes status while following their LCHF diets, using quantitative and qualitative tools.

1.7.2.2. To assess participants' personal characteristics and current metabolic health.

1.7.2.3. To assess the nutrient composition of participants' habitual LCHF diets using quantitative tools

1.7.2.4. To assess participants' perceptions around diet, eating behaviours, health and diabetes using qualitative tools.

2. METHODS

2.3. Ethical considerations

This study was approved by the UCT, Faculty of Health Sciences, Human research Ethics Committee (REF: 608/2016, approved on 1 September 2016). The study was performed in accordance with the principles of the Declaration of Helsinki (2013, Fortaleza, Brazil), the International Conference on Harmonisation and the South African Good Clinical Practice guidelines. Participants were informed that their participation in the study was entirely voluntary and that they had the right to withdraw from the study at any time without giving any reasons. All participants received a unique identification code to hide their identity and ensure anonymity.

2.4. Study design overview

This was a multi-method descriptive study investigating the diet (nutrient composition and foods consumed) and aspects relating to the diet (including diet history, behaviours and patterns, and perceptions around diet) of 28 adult T2D patients who believed they had been following an LCHF diet for at least the previous 6 months.

Participants completed two online questionnaires: a general detailed health questionnaire and a food frequency questionnaire (FFQ). The general detailed health questionnaire captured information about participants' current and past health, medication, exercise and diet, as well as family health history, and socioeconomic status. The FFQ requested that participants indicate how often they consumed specified portions of listed foods and beverages over the past 6 months. After completion of the online questionnaires, eligible participants visited UCT Exercise Science and Sports Medicine (ESSM) laboratories located at the Sports Science Institute of South Africa (SSISA) in Cape Town. During the visit, 24-hour diet recall (24HR), blood sampling, and semi-structured interviews were conducted. Anthropometry, BP, and breath and blood ketones were also assessed, and participants were instructed on how to complete a 3-day food record. For participants who were unable to visit this laboratory due to living outside of Cape Town, interviews and diet records were collected and diet record-taking instructions were given via telephone or Skype; but anthropometric

data, BP and blood were not collected. All participants completed a 3-day food record, detailing the type and quantity of foods and beverages consumed over 3 consecutive days. Past medical records were collected (from participants, their doctors and/or pathologists) to confirm diabetes diagnosis and medical history.

2.5. Participants

Inclusion and exclusion criteria. Participants were eligible if they were over the age of 18, had proof of T2D diagnosis from a medical doctor, and perceived that they'd been eating an LCHF diet for the previous 6 months or longer. Participants were not eligible if they had significantly changed their diet within the past 6 months, or could not understand or speak English.

Recruitment. Participants were recruited through electronic and poster advertisements (Appendices A - C) and personal communication (individuals who had contacted us enquiring about participating in LCHF studies). Electronic adverts were distributed through ESSM, SSISA, UCT and The Noakes Foundation (TNF) websites and emailing lists; Twitter and Facebook accounts; and on Facebook LCHF and Banting groups. Individuals who had enquired about participating in research studies on TNF website received recruitment advertisements via a TNF emailing list. The ESSM mailing lists comprise ESSM staff and students. The SSISA mailing lists comprise SSISA staff, SSISA members, and press (for SSISA press releases). Posters were displayed on medical clinic and shopping mall notice boards throughout Cape Town.

Sampling and sample size determination. Convenient sampling was used and sample size was not pre-determined, due to the exploratory and descriptive nature of this pilot study.

Normal practice for determining interview sample size in qualitative research is to continue interviewing until theoretical saturation is reached, which is when the research questions have been adequately answered [133]. This it thought to have occurred when [133]:

1. No new or relevant data emerges
2. The areas of interest are well enough developed and show sufficient variation
3. The relationships between areas of interest are clear

All three of these criteria had been fulfilled by the time all 28 interviews had been

analysed.

2.6. Details of analytical procedures

2.6.1. General health assessment

The general detailed health questionnaire (Appendix D) was developed for this study by Christopher Webster (CW), and edited by Tamzyn Murphy (TM), to capture information about participants' current and past health, medication, exercise and diet, as well as family health history, and socioeconomic status. The questionnaire was hosted online by LimeSurvey, and a link to the questionnaire was emailed to participants.

2.6.2. Diet

2.6.2.1. Dietary Assessment

Habitual diet was assessed using an online FFQ, researcher-administered 24HR, and self-reported estimated 3-day food record. Combined data from the 24HR and 3-day food record, as well as data derived from the FFQ, were used for the various dietary analyses presented in the Results section.

FFQ. The FFQ was developed for this study by TM and CW, to estimate past macronutrient intake ratios (i.e. percentage of energy intake from fat, protein and carbohydrate) in an LCHF population. Participants were emailed a link to the online FFQ (hosted by LimeSurvey) and asked to indicate how often they consumed specified portions of 110 foods and beverages over the past 6 months (Appendix E). The FFQ was co-developed by a registered dietitian (TM) according to guidelines proposed by Cade et al. (2002)[134], by adapting the South African Medical Research Council's (MRC) FFQ [135] to include food items frequently eaten by people following an LCHF diet. It underwent a period of pilot testing in volunteers that habitually followed an LCHF diet, and was modified accordingly. The FFQ will be validated as part of another postgraduate project, incorporating data from this study.

24HR. 24HRs were conducted face-to-face during the laboratory visit, or remotely via telephonic or Skype interviews. Participants were asked to recall all the foods and beverages

that they had eaten for twenty-four-hours, starting from the time that they woke up the previous day. The standard open-ended response structure (requesting additional detail) was employed to prompt comprehensive and detailed reporting of all foods and beverages consumed. Detailed dietary descriptors were captured, including time of day; source, brand or type; and portion size, of each food and beverage consumed. Visual aids (images, food models and eating utensils) were used when required, to help participants more accurately determine portion size [135].

3-day food record. The 24HR was used as an educational platform to instruct participants on how to complete a 3-day food record. All 28 participants completed a consecutive 3-day food record, detailing the type and quantity of foods and beverages consumed over 3 days using a standardised template Excel spreadsheet (Appendix F). If anything was unclear after record submission, participants were asked to clarify, and their record was amended accordingly.

2.6.2.2. Dietary Analysis

Diet records were analysed to describe the nature and nutrient content of the diet. FFQ data was used to estimate ratios of macronutrient intake (i.e. percentage of TDEI from fat, protein and carbohydrate). Diet logbooks and 24HR were analysed using the MRC South African Food Data System (SAFOODS) database combined with a missing food list (Appendix J). The Nutrient Reference Values (NRV) for Australian and New Zealand [1] were used for micronutrient threshold comparison, in the absence of South Africa-specific reference values.

FFQ. Macronutrient ratios were determined from FFQ data by multiplying the frequency of food items eaten per month, by their respective assigned macronutrient contents (g). Macronutrient content was derived from the FatSecret database [136]. FatSecret was used because the MRC's SAFOODS database omits many LCHF food choices. CW co-developed the code which generated an automated output for each completed FFQ (pie-chart and percentage contribution of macronutrients to TDEI). This output was used as data for this project, as well as diet feedback which was sent to participants.

24HR and 3-day food record. When mass quantities were not stipulated by the participant, researchers used the MRC food photo manual or, failing that, Google, to convert all quantities of captured foods and beverages to mass (g). Foods and beverages were also assigned their codes in order to be analysed using the MRC SAFOODS. When consumed foods and beverages did not have an associated SAFOODS code, they were added to a missing food list (Appendix J). Nutritional information (macro and micro nutrient content) for each missing food item was extracted from online databases, FatSecret [136] and NutritionDataSelf [137]. The energy, macronutrient (total protein, carbohydrate and fat, SFA, MUFA, PUFA, cholesterol and fibre) and sodium content of the missing food items were available for most missing foods and included in the list. However, for the majority of these foods the non-sodium micronutrient content was unavailable, and therefore missing from the list, and thus from the final MRC nutrient analysis.

The following macronutrient mass-to-energy conversions were used:

1 g glycaemic carbohydrate = 4 kcal

1 g fibre = 2 kcal [138]

1 g protein = 4 kcal

1g fat = 9 kcal

1 g alcohol = 7 kcal

In order to provide context to energy intake, estimate total daily energy expenditure (TDEE) (or energy requirement for weight maintenance) was calculated by multiplying the Basal metabolic rate (BMR, calculated using the Harris-Benedict equation [139]) by a physical activity factor (PAF) [140, 141]:

BMR (men) = 66.4730 + 13.7516 w + 5.0033 h - 6.7550 a

BMR (women) = 655.0955 + 9.5634 w + 1.8496 h - 4.6756 a

Where w = weight (kg), h = height (cm), and a = age (years)

PAF for this group = 1.4 (group mean: light to moderate exercise)

2.6.3. Anthropometry

Anthropometric measurements were taken for the 18 participant who attended the laboratory visit at SSISA.

Weight (kg). Participants were instructed to remove shoes, heavy clothing and items in their pockets, before stepping onto the scale (Detecto, UWE BW-150, digital bench scale). The stable reading was recorded.

Height (cm). Height was measured using a wooden stadiometer. Participants were instructed to stand with heels as far back as possible, feet together, head in the Frankfurt plane, and to breathe in. Height reading was taken on inhalation. Participants were asked to step away from the stadiometer before the reading was taken.

BMI (kg. m^{-2}). BMI was calculated from weight and height measurements, and classified according to WHO criteria [142]:

Normal weight: 18.5 to < 25 kg. m^{-2}

Overweight: 25 to < 30 kg. m^{-2}

Obese Class I: 30 to < 35 kg. m^{-2}

Obese Class II: 35 to < 40 kg. m^{-2}

Obese Class III: ≥ 40 kg. m^{-2}

Waist and hip circumferences (cm). Circumferences were measured in duplicate using a flexible steel anthropometric measuring tape. Waist circumference was taken against the skin, after exhalation, at the narrowest circumference around the waist. If it was unclear where the narrowest point was, the measurement was taken 2 cm above the navel. Hip circumference was measured with feet together, over a thin layer of clothing, at the greatest circumference in the hip area.

Waist circumference was classified as normal if it was < 80 cm for females (F) and if it was < 94 cm for males [143].

Waist to hip ratio was calculated and classified as normal if it was ≤ 0.85 for females (F) and ≤ 0.90 for males (M) [143].

Body composition. Skinfold thickness (mm) was measured using Innovare 3 Creskorff calipers at the following sites: tricep, bicep, subscapular, iliac crest, according to the procedures as described in the International standards for anthropometric assessment (ISAK) manual [144].

Body density (D) was determined from these four skinfold measurements, using the Durnin-Womersley formula [145]:

$$D \text{ (men)} = 1.1610 - 0.0632 \text{Log} \sum 4 \text{ kg. m}^{-3}$$

$$D \text{ (women)} = 1.1.581 - 0.0720 \text{Log} \sum 4 \text{ kg. m}^{-3}$$

where D = body density and ∑4 = sum of 4 skinfolds (mm)

Body Fat percentage (BF %) was calculated using the body density (D) and the Siri Equation [146]:

$$BF (\%) = (4.95/D - 4.5) \text{ X } 100$$

2.6.4. Blood pressure and heart rate

Participants remained seated at least 10 minutes before resting BP and HR measurements (mmHg) were taken using a sphygmomanometer (Omron 711, Automatic IS blood pressure monitor with comfort cuff, Typ 40S). Readings were taken in duplicate, consecutively, on the same arm.

2.6.5. Blood sampling

Blood samples for the determination of glucose, insulin, HbA1c, and lipid fraction concentrations were collected in appropriate vacutainers (as specified by the testing pathologist, Lancet laboratories). The tubes were inverted at least five times to ensure thorough mixing of blood and tube-additives, and bagged for collection and analysis by Lancet.

HbA1c (mmol. mol^{-1}) is classified, according to World Health Organisation (WHO) diagnostic guidelines, as normal 20 – 41.99 mmol. mol^{-1} (4 – 5.9%), prediabetic 42 – 47 mmol. mol^{-1} (6 – 6.4%), and diabetic > 48 mmol. mol^{-1} (>6.5%), with elevated diabetes risk from 5.7 % [147].

Insulin resistance was calculated using HOMA-IR [148]

HOMA-IR = (FPG X FPI) / 22.5

where FPG = fasting plasma glucose (mmol. L^{-1}) and FPI = fasting plasma insulin (µU. ml^{-1}).

2.6.6. Qualitative phase: interviews

A qualitative component was included in the study to provide context, meaning and relevance to the quantitative data, as stated in the aims and objectives (section 1.7). Diet is more than the nutrients it consists of, and is influenced by physiological, social, psychological and economic factors, amongst others. These factors are pivotal to the success of failure of a diet, and play a large role in the health effects and sustainability of the chosen diet. Perceptions around diet, health, lifestyle, and behaviour change were gathered from interviews and analysed using qualitative data analysis and coding techniques, and represented according to emergent themes. Researchers were trained in qualitative research techniques, including conducting and analysing interviews. The quality criteria for qualitative, mixed and multi-method research, as presented in the review by Fàbregues and Molina-Azorín et al. (2017) was used to assess the planning, interpreting, undertaking and disseminating phases of this study in order to improve design, implementation and reporting quality [149].

2.6.6.1. Conducting the interviews

Semi-structured interviews (n 28) were conducted by two researchers (TM and CW) during the lab visit (n 18) or remotely using Skype (n 10). At the start of each interview the participants were informed that the interview would be recorded, using the researchers' smart phone software or a Dictaphone. The researchers were trained in interviewing skills, and employed best practice techniques such as active listening, probing but not leading questioning, remaining neutral, verifying unclear responses, interpreting body language, and

avoiding double-barrelled questions [150]. All audio files were saved in a secure folder on Google Drive, accessible only by the study researchers, and then deleted from the researchers' smart phones. The interview questions probed the following topics:

- Sustainability of the LCHF diet
- Negatives and challenges of the LCHF diet
- Perceived LCHF-related changes to health and HRQoL
- Motivations, reactions, emotions
- Social impact
- Dietary behaviours (control, frequency)
- LCHF diet description (food choices, diet evolution)
- Other diets
- Perceptions of medications
- Perceptions of and interactions with medical professionals

See Appendix G for structured interview instructions and questions.

2.6.6.2. Transcribing the interviews

Audio recordings of interviews were commercially transcribed word for word by Ontimetranscribers (Cape Town, South Africa). Researchers' formatted the transcripts, checked for and re-transcribed portions that were incorrectly transcribed, and removed or replaced any mention of participant names or identifiers. The final transcriptions were saved as protected documents in a shared research study folder on Google Drive.

2.6.6.3. Developing the codebook

A codebook is an interview-analysis guide containing qualitative data-derived categories or themes, called 'nodes'. 'Coding' interviews into nodes, is how qualitative data (interviews) are analysed. A codebook includes definitions and details about each node, to improve inter-coder and inter-interview reliability, by promoting consistent interpretation or understanding of each node. See Appendix I for our codebook.

Two researchers (TM and CW) co-developed the first draft codebook by reading interview transcripts and noting general impressions, as well as looking for major opinions, attitudes, and themes expressed by the group. Themes were combined into node categories associated with areas of interest (e.g. research questions and objectives) to create the initial codebook, which was used separately by TM and CW to code one transcript. Thereafter, TM and CW met to discuss the coding process and discrepancies, and to code an interview together, using and refining the first draft codebook and rules of coding as they went, to develop the second draft of the codebook. This meeting, coding and refining process was repeated four times, adapting and modifying the codebook as new themes and codes emerged, until the fifth and final codebook had been developed. The final codebook was used for the final analysis of all transcripts. The codebook development process was also used to improve agreement and consistency regarding interpretation and rules of coding between researchers, thereby improving intercoder reliability.

2.6.6.4. Establishing intercoder reliability

Multiple coders ensure a consistent, standardised and unified coding process, and a more reliable (less subjective) interpretation [151]. Inter-coder reliability represents the extent to which coders assign the same words or transcript sections (data) to the same node (variable). Thus, it's an indication of how well the data represents the variables or areas of interest [152].

Intercoder reliability is established when two conditions are met: Firstly, agreement between coders must be eighty percent or higher (meaning that less than twenty percent of analysed data is inaccurate) [152]. However, percent agreement doesn't account for chance. So, a kappa value, which controls for random agreement, of ≥ 0.6 must also be achieved [152]. Kappa can range from -1 to +1, with ≤ 0 being chance agreement, and +1 being perfect agreement [152]. Twenty percent of transcripts were dual coded by both TM and CW independently, using the final code book to ensure intra- and inter- coder reliability [152] to assess inter-coder reliability. An agreement of 97.2 ± 2.49 % and a kappa of 0.74 ± 0.24 was achieved.

2.6.6.5. Analysis (coding)

Once intercoder reliability had been established the remaining transcripts were randomly

divided between TM and CW and coded by only one analyst. NVivo 11 Pro software was used for all interview analysis, including codebook development, coding, and establishing intercoder reliability.

2.6.6.6. Representing quotes in results

Adding text to a quote. Square brackets indicate text added by the author to explain local terms and expressions, or to provide clarity or context to a quote. The author referred back to the original transcripts to check the context, intended meaning, or accuracy of statements, whenever areas were unclear.

Removing text from a quote. At times, text has been removed from quotes (indicated by an ellipsis) to make confusing or lengthy quotes easier to read or understand, or to remove repetition, provided that meaning or emphasis did not change significantly.

Participant information accompanying quotes. Each quote is accompanied by a participant code. In Appendix H, each code is accompanied by basic information regarding the participant's age, sex and weight and diabetes status (i.e. changes HbA1c and T2D pharmacotherapy from pre-LCHF to the time of the study). 'y/o' means 'year old'. 'M' or 'F' means male or female respectively. Changes in HbA1c, pharmacotherapy and weight represent the difference between pre-LCHF and current status (at the time of the study).

2.7. T2D pharmacotherapy

T2D pharmacotherapy (Figure 9 and Table 10), is defined as treatment with medications (biguanides (e.g. metformin), suphonylureas (e.g. Gliclazide), peroxisome proliferator-activated receptor-gamma (PPARγ) (e.g. Pioglitazone), DPP-4 inhibitors (gliptins) GLP-1 receptor agonists, SGLT2 inhibitors (gliflozins), alpha-glucoside inhibitors (aracarbose) [13]) and/or insulin. An overall reduction in T2D pharmacotherapy was defined as reduction in medication and/ or insulin dosage. In determining overall change in T2D pharmacotherapy, insulin trumps medication (i.e. if insulin increased and medication decreased, it was seen as an overall increase in T2D pharmacotherapy).

2.8. Statistical analyses

Quantitative data regarding diet, participants health and T2D status (captured from the general detailed health questionnaire), measurements taken during the lab visit, and medical records and blood test results, were analysed using descriptive statistics. D'Agostino and Pearson omnibus normality test was used to determine data distribution. Normally distributed data is represented as mean +- standard deviation (SD). Non-parametric data is reported as median (interquartile range (IQR)). Insufficient sample size to test distribution normality is indicated with a "#". Descriptive statistics were performed using Graphpad Prism 6 software.

2.9. Research team responsibilities

The project's research team included three researchers (investigators), under the supervision of Dr James Smith (JS) and guidance of Prof Timothy Noakes (TN). The researchers comprised the author of this thesis (TM, myself, towards fulfilment of a Masters degree), Christopher Webster (CW, towards a PhD degree), and Dr Kate Larmuth (KL, a postdoctoral fellow).

JS wrote the ethics application, which was edited by TM and CW. The recruitment material was developed by TM and edited by CW and JS. Informed consent and medical records were obtained by TM and CW. The online questionnaires, laboratory visit protocol and standard operating procedures, interview questions and dietary assessments, were developed, standardised, and conducted by TM and CW. The FFQ analysis and output was prepared and coded for by CW. Interviews were co-operatively analysed (coded) by TM and CW. 24HR and diet logbook data was coded and prepared for SAFOODS analysis by TM and KL. Diet record coding and analysis was checked by TM. All raw data was exported to spreadsheets by CW and locked to prevent later edits. CW managed the project's online data management, and coded and compiled participant feedback reports. Standard operating procedures were developed and followed for each procedure and outcome measured, by all involved researchers, to ensure consistency in data collection and analysis.

Project data was divided between TM and CW, for degree purposes, as follows:

- TM focused on the diet data and the relationships between diet and other parameters, as well as aspects related to HRQoL

- CW focused on medical data (including blood results, medication and co-morbidities).

3. RESULTS

The results presented in this section describe the basic characteristics of the group, their health risk parameters at the time of the study, and how their T2D and weight status changed over time (particularly after adopting an LCHF diet). The changes are evaluated from the time of diagnosis to the point when they adopted an LCHF diet (pre-LCHF), and from pre-LCHF to the time of this study (current). The results also include a description of the groups' self-selected LCHF dietary intervention, including: nutritional composition, diet patterns, food choices, modifications to the diet over time, and perceptions around the diet (particularly relating to factors involved in dietary adherence and sustainability). Finally, perceived changes in aspects related to HRQoL after adopting an LCHF diet are presented.

3.1. Participant basic characteristics

Forty-five individuals responded to the recruitment advertisements. Prior to enrolment, nine became unresponsive and three were too busy to participate. Four were not eligible (no T2D diagnosis or < 6 months on LCHF) and were excluded at the beginning. One was diagnosed with latent autoimmune diabetes of adults (LADA) after completion of the study and was consequently excluded. Twenty-eight eligible participants completed the study: 14 females and 14 males (Table 1). The group was predominantly white, relatively affluent, tertiary-educated, South Africans from the Western Cape (Table 1). At the time of our study, all participants, except for one 33-year-old female, were 40 - 70 years old (mean age 57.4 ± 9.6) (Table 3).

Table 1: Socio-demographic profile (n 28)

Variable	Value	Percent (n)
Sex	Female	50.0 (14)
	Male	50.0 (14)
Ethnicity	Black	0.0 (0)
	Mixed / coloured	7.1 (2)
	White	92.9 (26)
Current residential location	Western Cape, South Africa	64.3 (18)
	Other areas of South Africa	14.3 (4)
	International	21.4 (6)
Highest level of education	High school	25.0 (7)
	Tertiary undergraduate or diploma	35.7 (10)
	Tertiary postgraduate	39.3 (11)
Monthly income	< R10,000	14.3 (4)
	R 10,000 - R 25,000	21.4 (6)
	> R 25,000	64.3 (18)
Number of people residing in the house	1-2 people	71.4 (20)
	3-4 people	28.6 (8)
Rooms per person per house	3 or less rooms per person	42.9 (12)
	4-6 rooms per person	53.6 (15)
	7 or more rooms per person	3.6 (1)

3.2. T2D status

3.2.1. T2D status over time

For participants with HbA1c data available for two consecutive time-points (i.e. n 12 for diagnosis to pre-LCHF, and n 22 for pre-LCHF to current), where pre-LCHF is at least 90 days after diagnosis, reduction in HbA1c from diagnosis to pre-LCHF was - 1.65 ± 2.86 %. HbA1c reduced by a further 2.45 ± 2.59 % from pre-LCHF to the time of this study.

At diagnosis, all but one of the participants who had HbA1c available for all time points (n 16) were classified as having T2D based on HbA1c (> 6.5 %) (Figure 1). Pre-LCHF, T2D classifications remained unaltered for all of these participants, apart from two who changed from a T2D to an intermediate hyperglycaemia classification (Figure 1). At the time of this study, overall improvement in classification was seen for the group, with over a third of the

group having achieved a normal, non-diabetic HbA1c (Figure 1). The entire sample (n 28) had Hba1c measured at the time of the study. Of these, 79 % (n 22) had HbA1c concentrations below the cut-point for diabetes diagnosis (< 6.5 %), and 54% (n 15) had normal HbA1c concentrations (< 5.7 %).

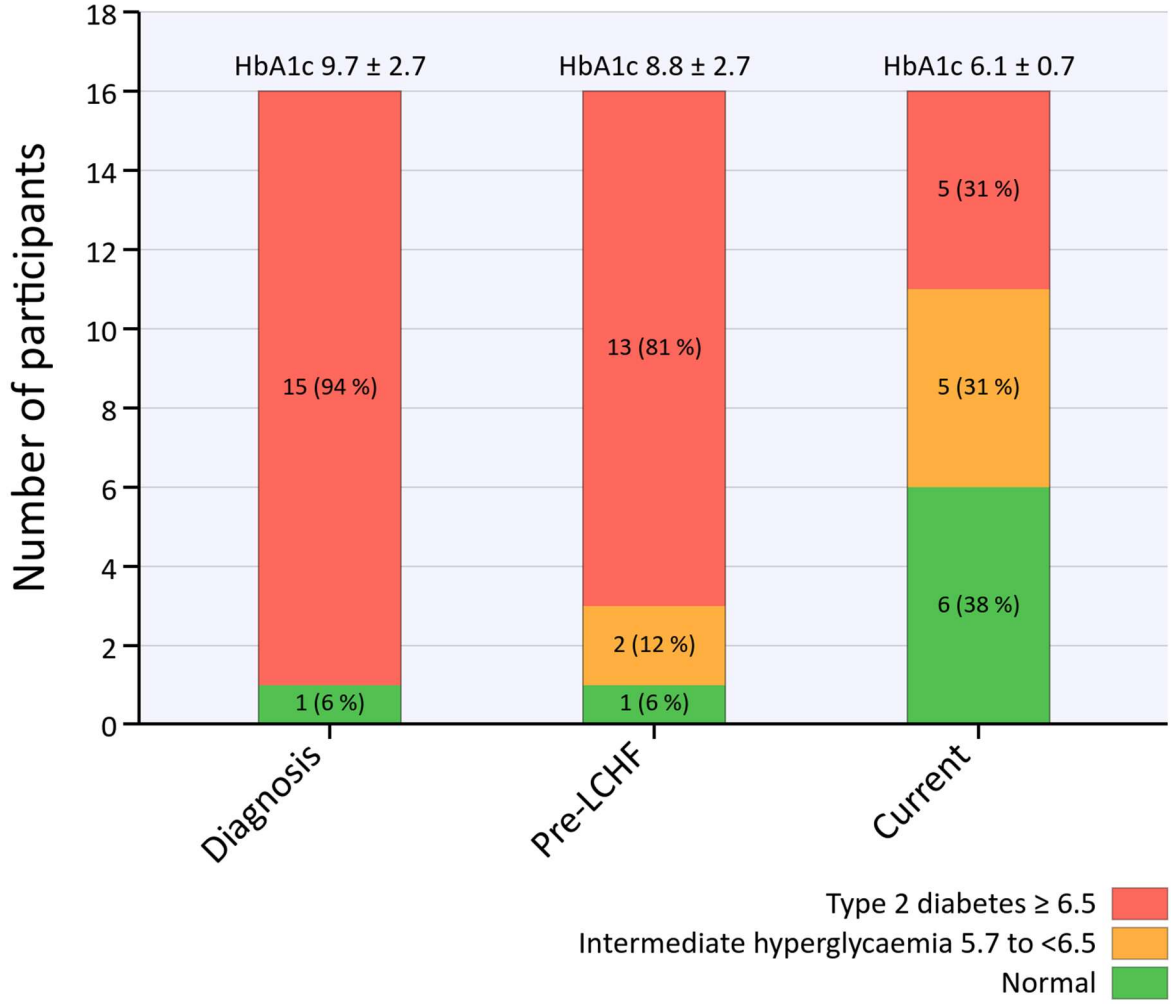

Figure 1: Type 2 Diabetes Classification [147] based on HbA1c at diagnosis, pre-LCHF, and current time points (n 16).

Twenty-four participants' (86 %) were either not on T2D pharmacotherapy (medication or insulin) or their T2D pharmacotherapy remained unchanged, decreased or ceased, during the period that they followed an LCHF diet (Table 2). These participants' HbA1c blood results (Figure 2) therefore represent HbA1c concentrations changes independent of the hypoglycaemic influence of T2D pharmacotherapy. Twenty-one (75 %) participants experienced a reduction or maintenance of HbA1c concentration, despite reduction or maintenance of T2D pharmacotherapy (or lack thereof), during their time on LCHF (Figure 2). At the time of this study, eleven participants were not on any T2D pharmacotherapy. Ten of

these had non-diabetic (< 6.5) HbA1c concentrations. Seven participants, not on T2D pharmacotherapy at the time of the study, had healthy HbA1c concentrations.

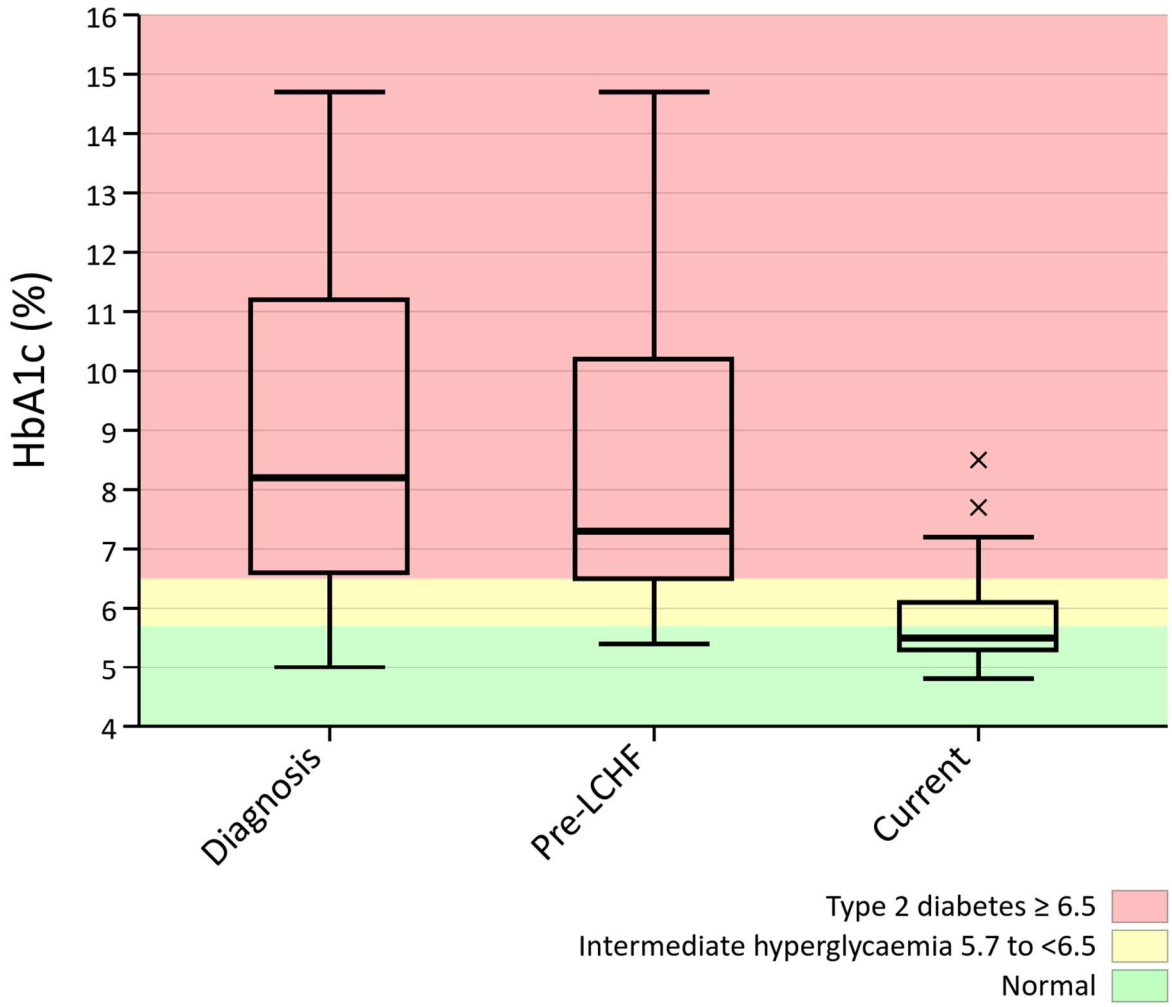

Figure 2: HbA1c blood results at diagnosis, pre-LCHF, and current timepoints, for the participants whose T2D pharmacotherapy did not increase (i.e. it was maintained, decreased, or not applicable) from pre-LCHF to the (current) time of the study (n 24). The effect of non-T2D pharmacological factors (including diet) on glycaemic control is isolated by excluding the four participants whose overall T2D pharmacotherapy increased from pre-LCHF to current. Represented as Tukey boxplot.

Table 2: Change in T2D pharmacotherapy from pre-LCHF to the time of the study (n 28)

Change in T2D pharmacotherapy	T2D medication percent (n)	Insulin percent (n)
Increase	14 (4)	7 (2)
No change	46 (13)	4 (1)
Decrease	0 (0)	0 (0)
Cessation	25 (7)	32 (9)
No pharmacological T2D treatment	14 (4)	57 (16)

Data comes from reported participant medication and insulin use (via the detailed online questionnaire) at two timepoints: before adopting LCHF (pre-LCHF), and at the time of the study (current).

3.2.2. Current diabetes markers

Mean fasting blood glucose, HbA1c, and insulin concentrations were normal (non-diabetic) at the time of the study (Table 3). At the time of the study, HbA1c was normal in over half the group (n 15), indicative of IGT in a fifth (n 6), and consistent with T2D in a quarter (n 7). Median TG to HDL ratio (TG/HDL), a surrogate marker of insulin resistance [153], was normal for the group, for males and females separately, and for 19 of 20 participants measured. Conversely, another surrogate marker of insulin resistance, HOMA-IR [148], was indicative of insulin resistance for the group, and for males and females respectively. Fifty-seven percent of participants had a higher than normal HOMA-IR.

Table 3: Current cross-sectional measurable health risk characteristics

Variable	Females	Males	Combined	N: >N (n)	N reference range
Age (years)	56.1 ± 11.7 (n 14)	58.7 ± 8.5 (n 14)	57.4 ± 9.6 (n 28)		
HR (bpm)	67.7 ± 10.1 (n 10)	65.0 ± 13.4 (n 7)	66.6 ± 11.3 (n 17)	17: 0	< 100
Blood pressure (mm Hg)				1: 16	< 120 / 80
systolic	140.9 ± 17.0 (n 10)	129.8 ± 11.0 (n 7)	136.3 ± 15.5 (n 17)	2: 17	< 120
diastolic	86.4 ± 12.5 (n 10)	77.4 ± 8.2 (n 7)	82.6 ± 11.56 (n 17)	5: 12	< 80
Anthropometry					
Weight (kg)	88.9 ± 21.6 (n 11)	92.7 ± 9.8 (n 7)	90.4 ± 17.7 (n 18)		
BMI (kg. m^{-2})	31.6[a] ± 6.2 (n 11)	28.1[b] ± 2.6 (n 7)	30.3[a] ± 5.3 (n 18)	3: 15	18.5 - 25
Waist circumference (cm)	101.2[c] ± 16.8 (n 11)	103.2[c] ± 13.0 (n 7)	102.0[c] ± 15.0 (n 18)	3: 15	< 80 (F); < 94 (M) [143]
Waist to hip ratio	0.9[c] ± 0.1 (n 11)	1.0[c] ± 0.1 (n 7)	0.9 ± 0.1 (n 18)	6: 12	≤ 0.85 (F); ≤ 0.90 (M) [143]
Body fat (%)	34.0[d] ± 9.4 (n 11)	38.0[d] ± 8.0 (n 7)	35.6 ± 8.9 (n 18)	5: 13	Age and sex - dependent [154]
Plasma markers					
HbA1c (%)	5.4 (5.3 – 5.7) (n 11)	6.0 ± 0.7 (n 17)	5.5 (5.3 - 6.2) (n 28)	15: 13 or 15 N: 6 I: 7 D	N: 4.1 – 5.6 I: 5.7 – 6.4 D: ≥ 6.5 [147]
Fasting blood glucose (mmol. mol^{-1})	5.7 ± 1.2 (n 11)	6.2 (5.3 – 7.6) (n 12)	6.0 ± (5.1 – 7.4) (n 23)	5: 18 or 5 N: 4 I: 14 D	N: < 5.6 I: 5.6 – 6.9 D: ≥ 7 [155]
Insulin (µU. ml^{-1})	11.1 ± 5.6 (n 11)	12.2 ± 8.5 (n 10)	11.6 ± 7.0 (n 21)	20: 1	2.6 – 24.9
Total cholesterol (mmol. l^{-1})	5.5 (4.6 – 7.0) (n 5)[#]	4.8 (3.9 – 6.3) (n 14)	5.2 (3.9 – 6.1) (n 19)	8: 11	< 5
LDL (mmol. l^{-1})	3.2 (2.6 – 5) (n 4)[#]	3.6 ± 2.0 (n 15)	3.6 ± 1.9 (n 19)	9: 10	< 3
HDL (mmol. l^{-1})	1.3 (1.1 – 2.3)[#] (n 5)	1.4 (1.1 – 2.1) (n 15)	1.4 (1.1 – 2.1) (n 20)	17: 3	>1
Triglycerides (mmol. l^{-1})	0.9 (0.8 – 1.5)[#] (n 5)	1.2 (0.8 – 1.9) (n 15)	1.1 (0.8 – 1.6) (n 20)	15: 5	< 1.7
Surrogate markers of insulin resistance [156]					
TG/ HDL	0.8 (0.3 – 1.3)[#] (n 5)	0.9 (0.5 – 1.4) (n 15)	0.8 (0.4 – 1.3) (n 20)	19: 1	< 3.5 [157]
HOMA-IR	3.0 ± 1.8 (n 10)	4.1 ± 3.2 (n 11)	3.5 ± 2.6 (n 21)	9: 12	< 2.29 [158]

Data collected during the lab visit. Data expressed as mean +- standard deviation where normally distributed and median (interquartile range) where not normally distributed. [#] indicates insufficient sample size to test distribution normality. N, normal. >N, higher than normal. I, High risk / intermediate hyperglycaemia or pre-diabetes. D, diabetes. HR, resting heart rate (bpm, beats per minute). BMI, body mass index. LDL, low density lipoprotein cholesterol. HDL, homa-ir lipoprotein cholesterol. TG/ HDL, triglyceride to HDL ratio. HOMA-IR, homeostasis model of insulin resistance.

3.3. Metabolic disease risk factors

3.3.1. Cardiovascular risk

Current CVD risk markers are presented in Table 3. Resting HR (n 17), ranged from 52.5 to 90.2 beats per minute (bpm), and averaged a 66.6 bpm. Mean systolic and diastolic BP (n 17) were 136.3 ± 15.5 mmHg and 82.6 ± 11.56 mmHg respectively (prehypertensive) [159]. One participant (5.9%) had normal BP, 7 (41.2 %) were prehypertensive, 7 (41.2 %) had stage 1 hypertension, and 2 (11.8 %) were classified as stage 2 hypertensive [159]. Systolic BP ranged from 120.5 (normal) to 173.0 (stage 2 hypertension) [159]. Diastolic BP ranged from 57.5 (normal) to 98.5 diastolic (stage 1 hypertension) [159]. Mean serum total and LDL cholesterol concentrations were slightly higher than the recommended ranges (males mean serum total cholesterol concentration < 5 mmol/l). Mean serum HDL cholesterol and TG concentrations were in the healthy reference ranges, for the entire sample, and for males and females respectively.

3.3.2. Anthropometry

3.3.2.1. Current anthropometric parameters

Current anthropometric parameters are listed in Table 3. Mean weight and BMI was 90.4 ± 17.7 kg and 30.3 ± 5.3 kg. m^{-2} respectively, placing the group average in the obese class I category [142]. BMI ranged from 19.1 (normal weight) to 48.4 kg/m^2 (obese class III). Mean BMI category for females was obese class I and for males was overweight [142]. Mean waist circumference and waist to hip ratio (WHR) was high for the group and for males and females respectively. Eighty-three percent (n 15) had high waist circumferences and BMIs, and 67 % (n 12) had elevated WHRs. Mean body fat percentage for females was 34.0 ± 9.4 % (within 20^{th} and 30^{th} percentile) and for males was 38.0 ± 8.0 (below the 10^{th} percentile) [154]. Seventy-two percent (n 13) of the group had body fat percentages exceeding recommendations for health and fitness.

3.3.2.2. Weight status over time

At diagnosis and pre-LCHF, 20 of 28 participants were classified as obese according to their BMI (based on reported weight and height) (Figure 3 - 4). Mean body weight at diagnosis and pre-LCHF were 106 ± 25.7 and 111.0 ± 30.1 kg (n 28) respectively. Over the 2.0 ± 1.5 years (Figure 6) from pre-LCHF to the time of this study, the number of obese participants had dropped from 20 to 10, representing a 50 % reduction in obesity prevalence (Figure 3 - 4).

There is a corresponding improvement in mean group BMI classification from obese class II to overweight after adopting LCHF (Figure 3 - 4).

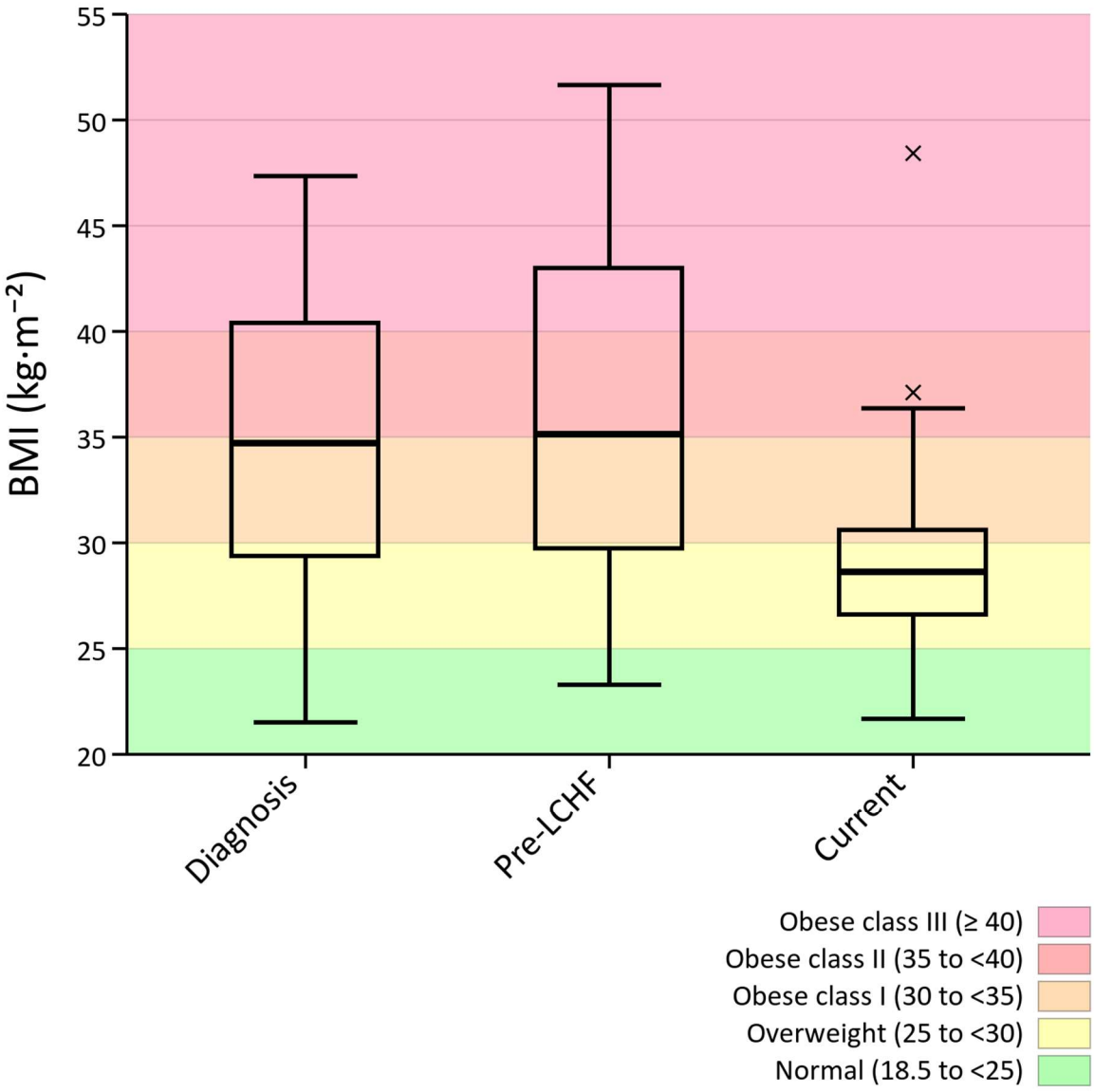

Figure 3: Body mass index (BMI) and weight classifications [142] at diagnosis, pre-LCHF and current time points (n 28). Represented as a Tukey boxplot.

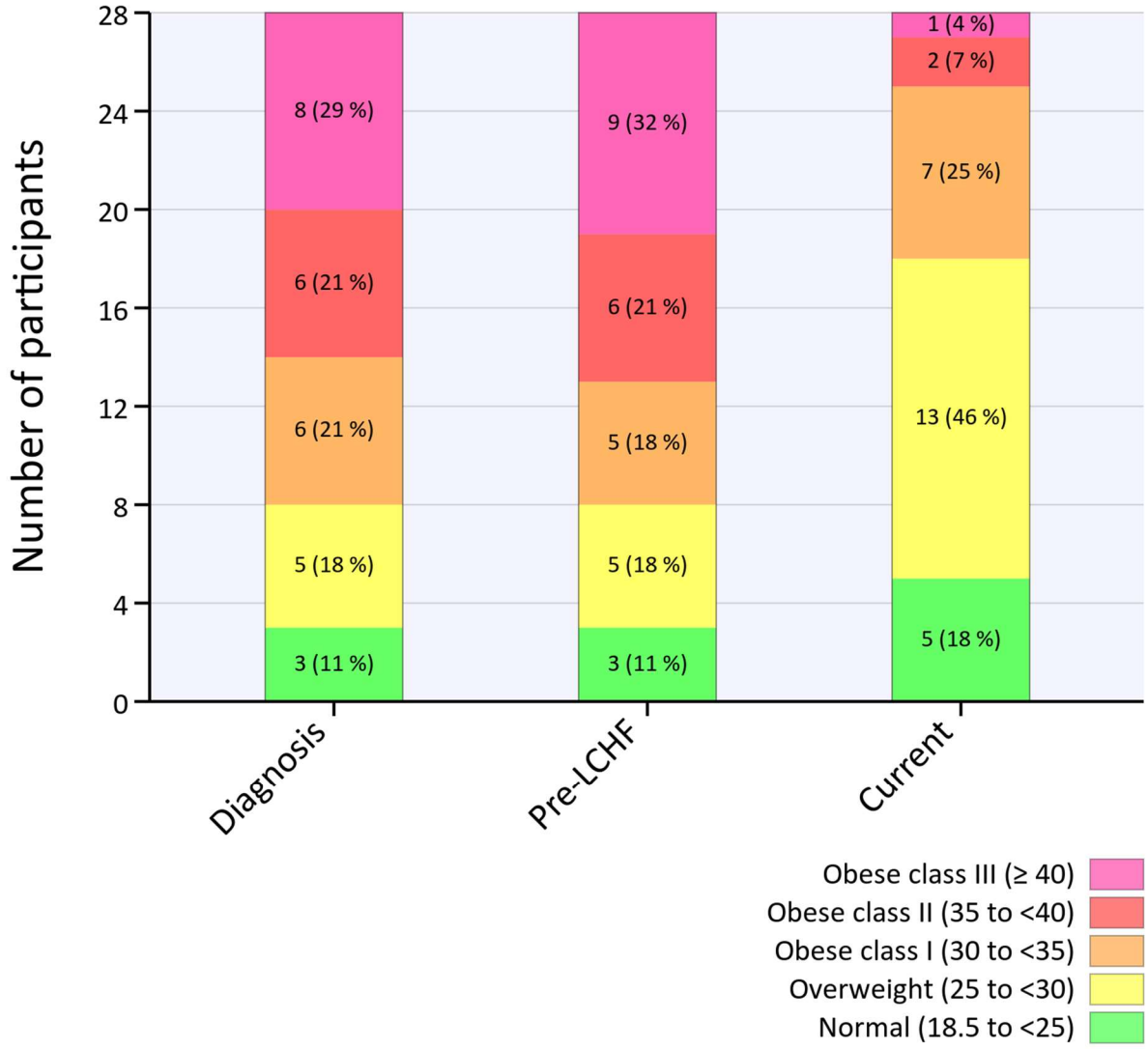

Figure 4: Participants' body mass index (BMI)-based weight classifications [142] at diagnosis, pre-LCHF, and current time points (n 28).

Every participant reported weight loss after following LCHF. The mean loss of body weight from pre-LCHF (111.0 ± 30.1 kg) to the time of this study (89.8 ± 18.6 kg), was 21.2 ± 19.1 kg (n 28), representing a 16.9 ± 11.7 % reduction in body weight (Figure 5). Percentage weight loss in females was - 6.5 (5.9 - 22.8) % and in males was - 20.0 ± 10.8 % (Figure 5).

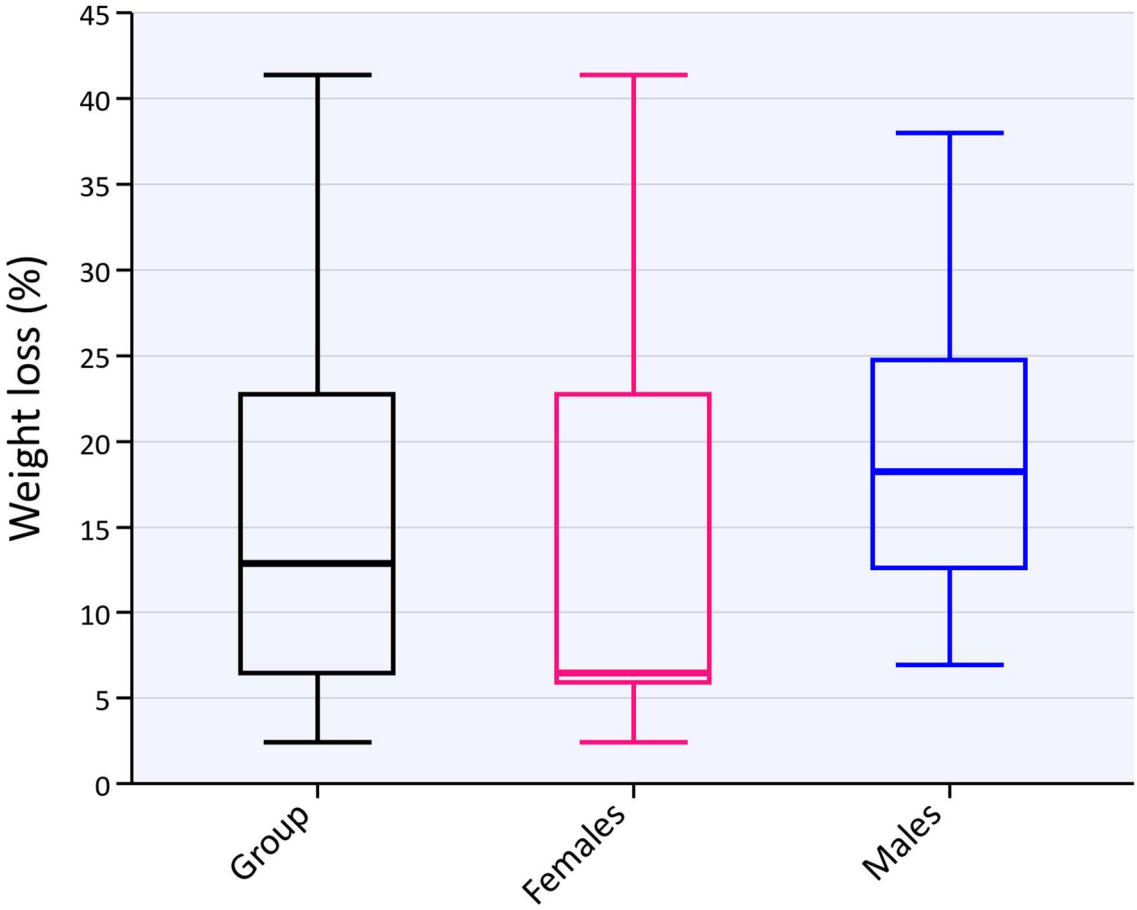

Figure 5: Percentage weight loss from before adopting LCHF to the current time of this study (n 28). Represented as Tukey boxplot.

3.3.3. Other health risks

Modifiable health risk (Table 4). Over half of our sample had never smoked, and only one participant was a smoker at the time of the study. Seventy-five percent of participants were incorporating exercise into their life at the time of the study. The American College of Sports Medicine recommends at least 150 minutes of moderate intensity cardiorespiratory exercise per week (30 minutes or more, 5 times per week) or at least 75 minutes of vigorous intensity cardiorespiratory exercise per week (20 minutes or more, 3 times per week) [160]. Forty-three percent of participants were meeting cardiorespiratory exercise guidelines. A quarter of our sample did not exercise at all beyond activities of daily living.

Familial health risks (Table 4). The majority of our sample reported having close family members (parents, siblings or grandparents) with overweight or obesity, prediabetes and hypertension. A third of our sample reported high cholesterol, and a quarter reported T2D in

a close family member. Just over half of our sample reported having parents or siblings with CVD.

Table 4: Reported health risk characteristics (n 28)

Variable		Percent (n)
Cigarette smoking	Never smoked	53.6 (15)
	Previously smoked	42.9 (12)
	Currently smoke	3.6 (1)
Exercise	Currently performing exercise	75.0 (21)
	Meeting guidelines [158]	42.9 (12)
Family History	T2D[b]	25.0 (7)
	Prediabetes[b]	67.9 (19)
	CVD[a]	53.6 (15)
	Hypertension[b]	89.3 (25)
	Cholesterol[b]	32.1 (9)
	Overweight / obese[b]	82.1 (23)

Self-reported data from online health questionnaire. [a] parents or siblings. [b] parents, siblings or grandparents. T2D, Type 2 Diabetes. CVD, cardiovascular disease.

3.4. Diet characteristics

3.4.1. LCHF diet patterns

Mean duration from pre-LCHF to the (current) time of this study was 2.0 ± 1.5 years (Figure 6). Sixty-eight percent had followed LCHF for over a year (Table 5). Mean time since T2D diagnosis was 9.2 ± 7.8 years (Figure 6).

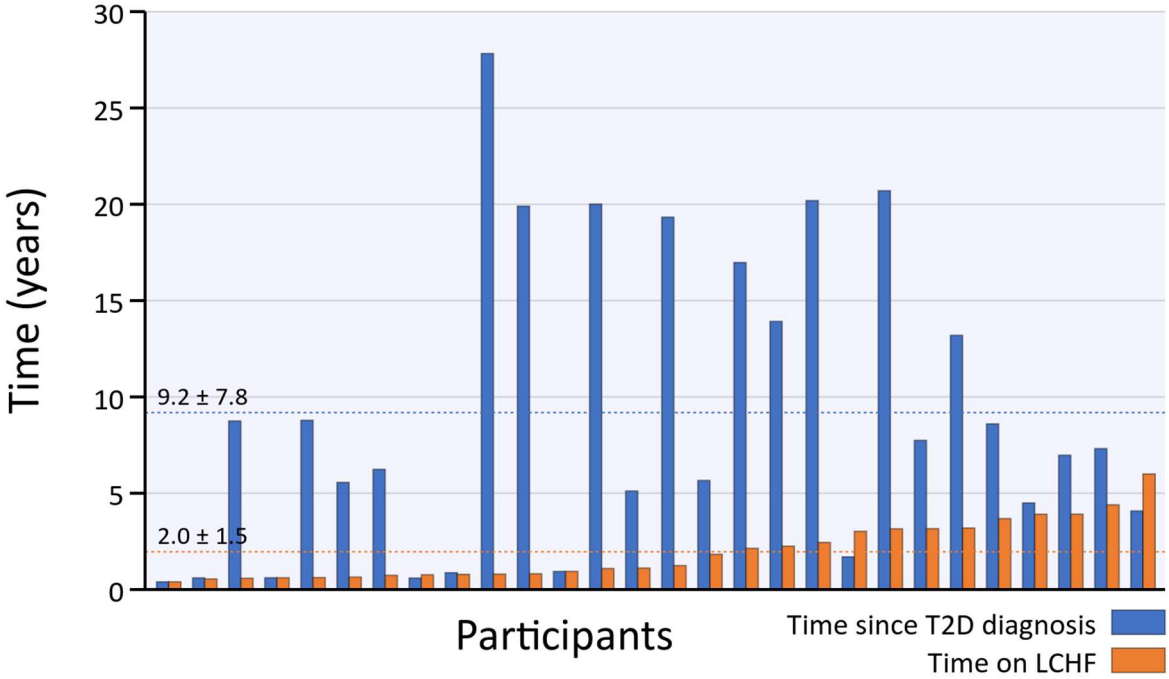

Figure 6: Reported time since T2D diagnosis and duration of following an LCHF diet for each participant (n 28). Dashed lines indicate mean and SD for the group.

Sixty-one percent ate 2 or less meals or snacks per day (excluding snacks or drinks that contributed calories exclusively from fat, e.g. bullet proof coffee). The minimum longest daily fast between meals in these participants was 16 hours (Table 5). Most of the participants who ate 2 or less times per day reported delaying breakfast or skipping it completely, and favouring evening meals, sometimes in combination with a small fat and protein rich snack or lunch.

Table 5: Diet patterns (n 28)

Variable	Value	Percent (n)
Years on LCHF diet	0.5 to < 1	32.1 (9)
	1-3	39.3 (11)
	> 3	28.6 (8)
Meals per day	1 - 2	60.7 (17)
	2 - 3	32.1 (9)
	≥ 4	7.1 (2)
Longest fast	< 12 hours per day	10.3 (3)
	12-15 hours per day	17.2 (5)
	16-18 hours per day	41.4 (12)
	19-24 hours per day	31.0 (9)
	40 – 48 hours per week	3.4 (1)

Eating frequency per day includes the number of meals and snacks eaten in 24 hours but excludes foods containing fat as the only macronutrient (e.g. bullet proof coffee). Fasting is the longest duration between eating.

3.4.2. Energy and macronutrient distribution

3.4.2.1. Total energy intake

Mean group TDEI was 1946.3 ± 807.2 kcal, or 22.8 ± 11.2 kcal . kg^{-1}. day^{-1} (Table 6). Mean calculated basal or total daily energy expenditure (BEE or TDEE) was 2383.0 ± 375 kcal, or 27.4 ± 6.0 kcal. kg^{-1}. day^{-1}, reflecting the group's average estimated energy requirement for weight maintenance. Mean energy deficit was 436.7 ± 728.3 kcal (Figure 7). Males consumed 2224.0 ± 829.7 kcal daily, which was 429.3 kcal less than their 2653.3 kcal ± 318.3 kcal calculated TDEE (Figure 7). Females consumed 1668.0 ± 705.9 kcal, which was 444.8 kcal less than their 2112.8 ± 183.4 kcal calculated TDEE (Figure 7).

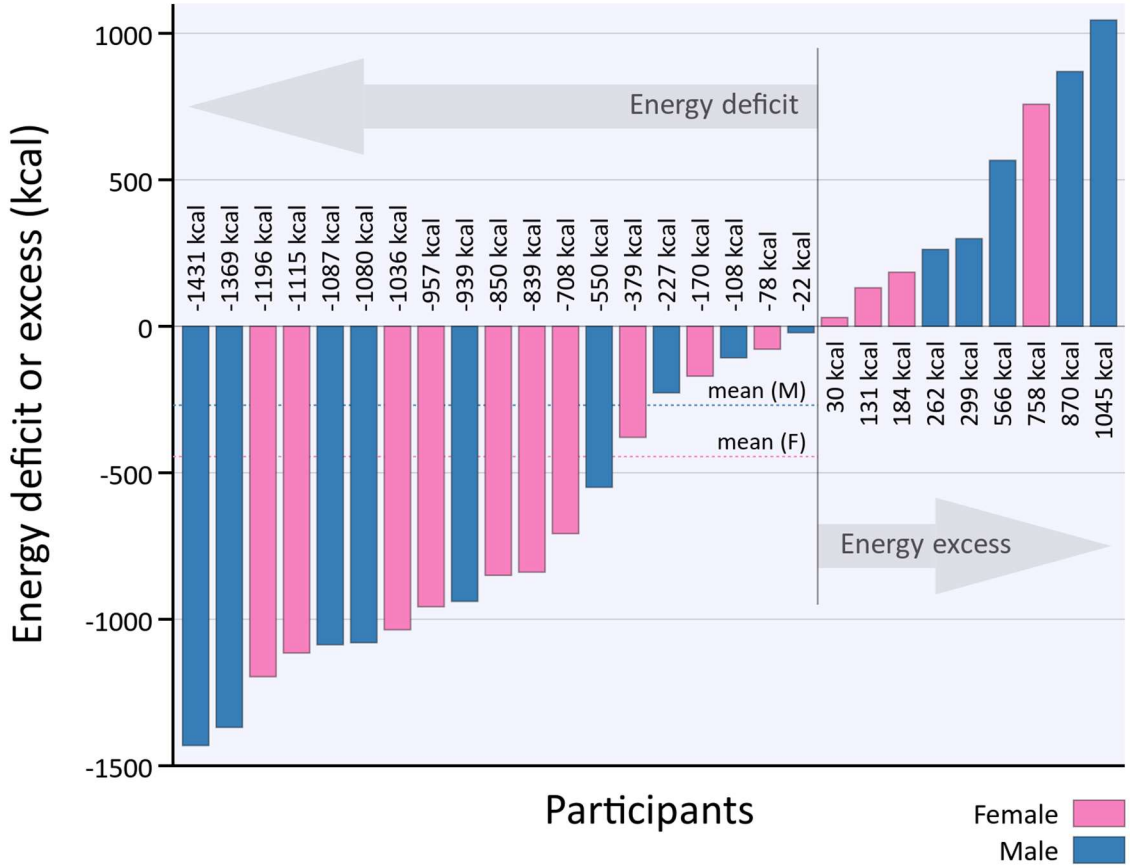

Figure 7: Habitual energy intake deficit or excess relative to requirement for weight maintenance (n 28). Bars represents the difference between total daily energy intake (TDEI) (from SAFOODS analysis of 3-day diet record and 24-hour recall) and calculated total daily energy expenditure (TDEE) requirement for weight maintenance (Harris-Benedict equation multiplied by physical activity factor).

3.4.2.2. Macronutrient intake

Mean macronutrient intake (protein, fat, carbohydrate and alcohol's relative contribution to TDEI) is represented in Figure 8 and summarised in Table 6. Individual variation in habitual macronutrient intake is represented in Figure 9.

Protein. Mean daily protein intake was 96.0 ± 37.4 g (1.1 ± 0.5 g. kg BW $^{-1}$. day $^{-1}$ or 1.3 ± 0.5 to 1.7 ± 0.6 g . kg ideal body weight (IBW)$^{-1}$. day^{-1}), which represents approximately 20 % of TDEI (Table 6, Figure 8).

Carbohydrate. Mean total daily carbohydrate consumption was 64.0 ± 27.9 g, contributing 14 % of TDEI (Table 6, Figure 8). Two thirds of total carbohydrate intake comprised available or glycaemic carbohydrate – most of which was naturally-occurring sugar (Figure 10). Sugar

(sucrose) (5 % added sugar and 36 % naturally-occurring sugar) comprised 41 % of total dietary carbohydrate (Figure 10). Daily added sugar intake was 0.1 (0 – 5.6) g, comprising 0.7 % of TDEI (Table 6). Our sample consumed 14.7 ± 7.5 g of fibre, which represents one quarter of the total carbohydrate intake (g) (Table 6).

Fat. Mean daily fat intake was 143.2 ± 67.9 g, contributing 66% of TDEI (Table 6, Figure 8). Dietary fat was mostly SFA (41%), followed closely by MUFA (36%) (Table 6, Figure 11). Daily PUFA intake was 15.4 ± 8.8 g, making up 11% of total dietary fat (Table 6, Figure 11).

Cholesterol and alcohol. Median cholesterol and alcohol consumption was 616.3 (402.8 – 804.2) and 0.0 (0.0 – 11.5) g respectively (Table 6).

Table 6: Dietary macronutrient content (n 28)

	Quantity per day	Quantity per kilogram body weight per day	Quantity per kilogram ideal body weight per day[a]
Energy (kcal)	**1946.3 ± 807.2**	**22.8 ± 11.2**	**28.9 ± 4.7 – 33.1 ± 5.1**
Protein (g)	**96.0 ± 37.4**	**1.1 ± 0.5**	**1.3 ± 0.5 – 1.7 ± 0.6**
Carbohydrate (g)	**64.0 ± 27.9**	**0.7 (0.5 – 0.9)**	**0.8 (0.6 – 1.1) – 1.1 (0.8 – 1.5)**
available	38.2 (27.5 – 55.4)		
total sugar	27.6 (15.7 – 35.1)		
added sugar	0.1 (0 – 5.6)		
fibre	14.7 ± 7.5		
other	5.1 (1.5 – 10.5)		
Fat (g)	**143.2 ± 67.9**	**1.4 (1.0 – 2.0)**	**1.8 (1.2 – 2.5) – 2.4 (1.6 – 2.4)**
Saturated fat	49.2 (39.2 – 80.0)		
MUFA	45.1 (30.2 – 62.7)		
PUFA	15.4 ± 8.8		
Unknown	15.5 (9.2 – 19.7)		
Cholesterol (mg)	**616.3 (402.8 – 804.2)**		
Alcohol (g)	**0.0 (0.0 – 11.5)**		

Quantity represents our sample's daily intake from four days of diet records (3-day diet record and 24-hour recall). MRC SAFOODS was used for dietary analysis. Data is expressed as mean +- standard deviation where normally distributed, and median (interquartile range) where not normally distributed. [a] – Ideal body weight is based on normal BMI range 18.5 – 24.9 kg. m^{-2}. The SAFOODS dietary analysis results for carbohydrate and fat components summed to less than the total. Therefore "Other carbohydrate" and "unknown fat" categories were added to compensate for this deficit. Available carbohydrate, non-fibre glycaemic carbohydrate component.

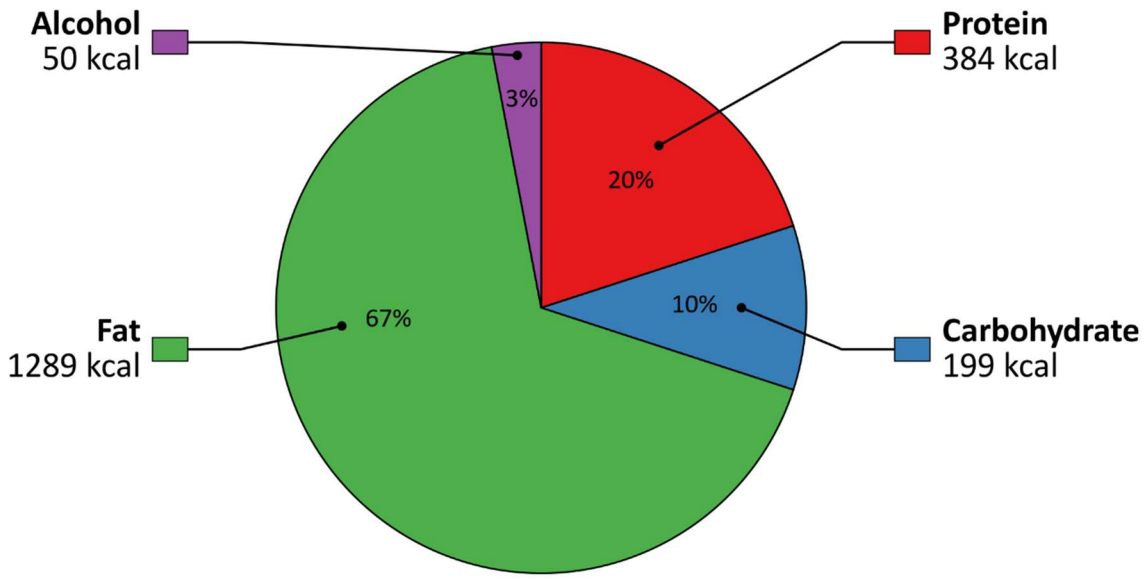

Figure 8: Daily dietary macronutrient composition and relative contribution to total energy intake (n 28). Data is expressed as mean ± standard deviation where normally distributed, and median (interquartile range) where not normally distributed. SAFOODS was used for dietary analysis of nutritional information acquired from four days of diet records (comprising a 3-day diet record and 24-hour recall).

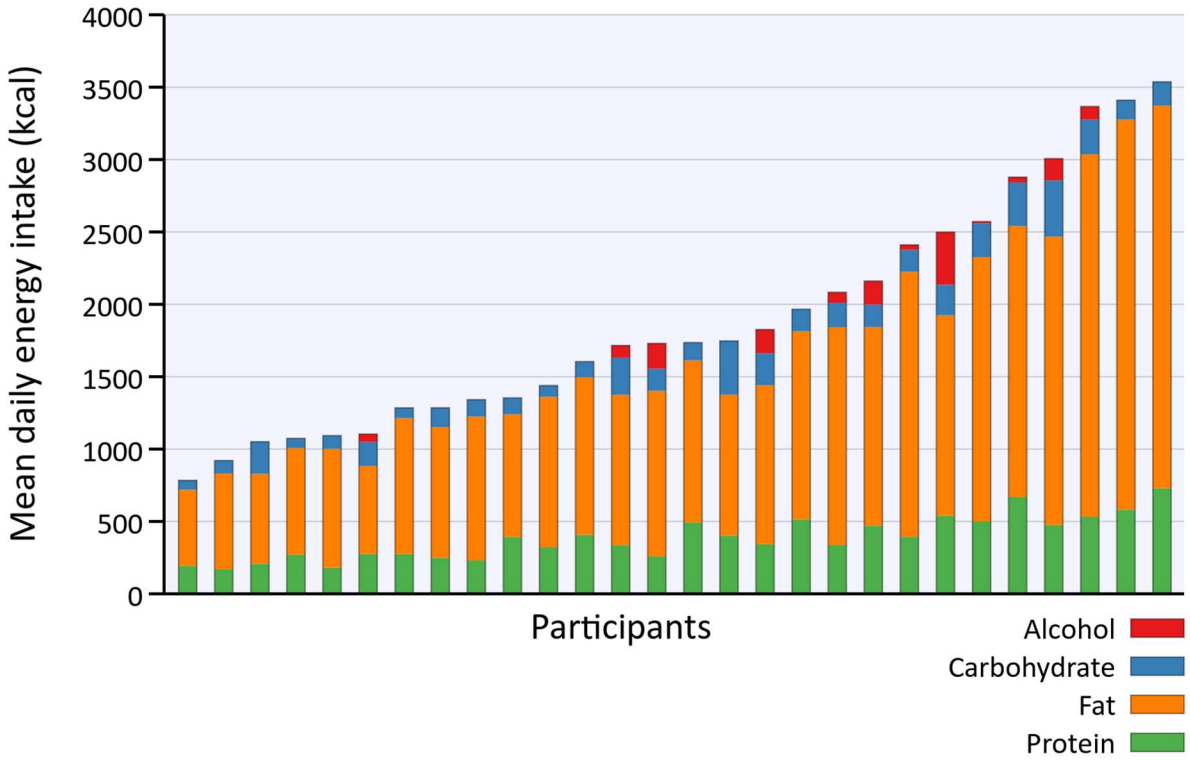

Figure 9: Habitual daily energy and macronutrient intake (kcal) (n 28). Represents variation in individual participants' protein, fat, carbohydrate and alcohol percentage contribution to total daily energy intake (TDEI).

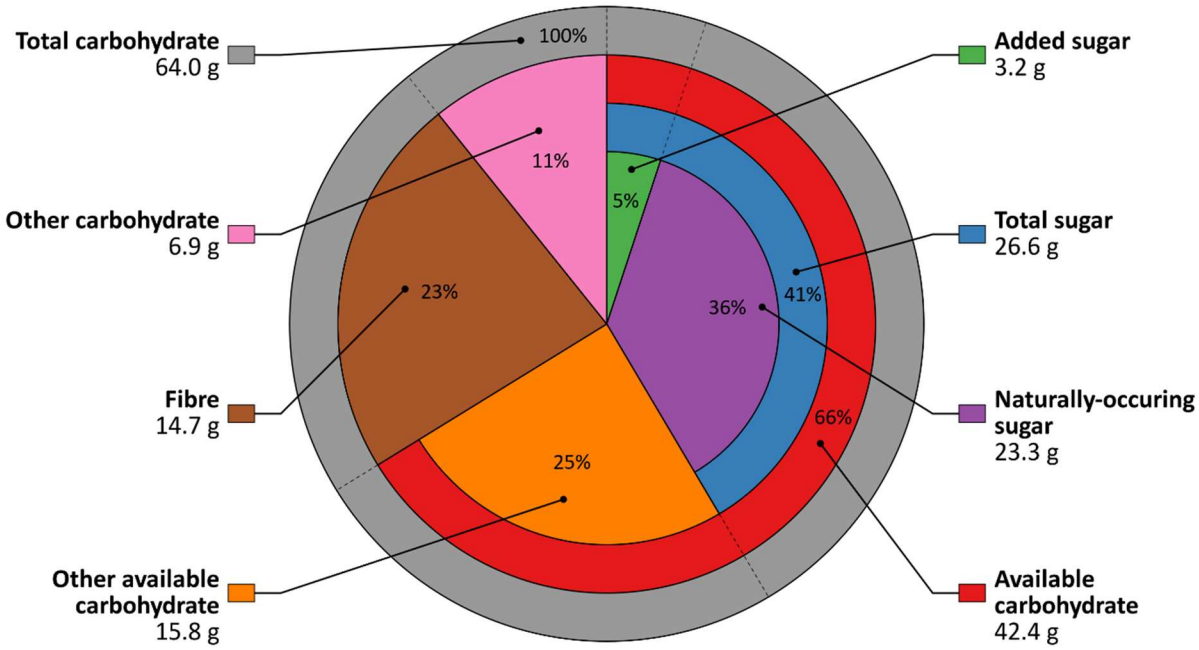

Figure 10: Dietary carbohydrate composition and daily intake (n 28). Percentages are represented as part of total daily carbohydrate intake. The total daily carbohydrate intake and carbohydrate types consumed represent participants' mean daily carbohydrate consumption over four days of diet records (comprising a 3-day diet record and 24-hour recall). MRC SAFOODS was used for dietary analysis. The SAFOODS dietary analysis results for the carbohydrate components summed to less than the total. Therefore, the "other carbohydrate" category was added to compensate for this deficit.

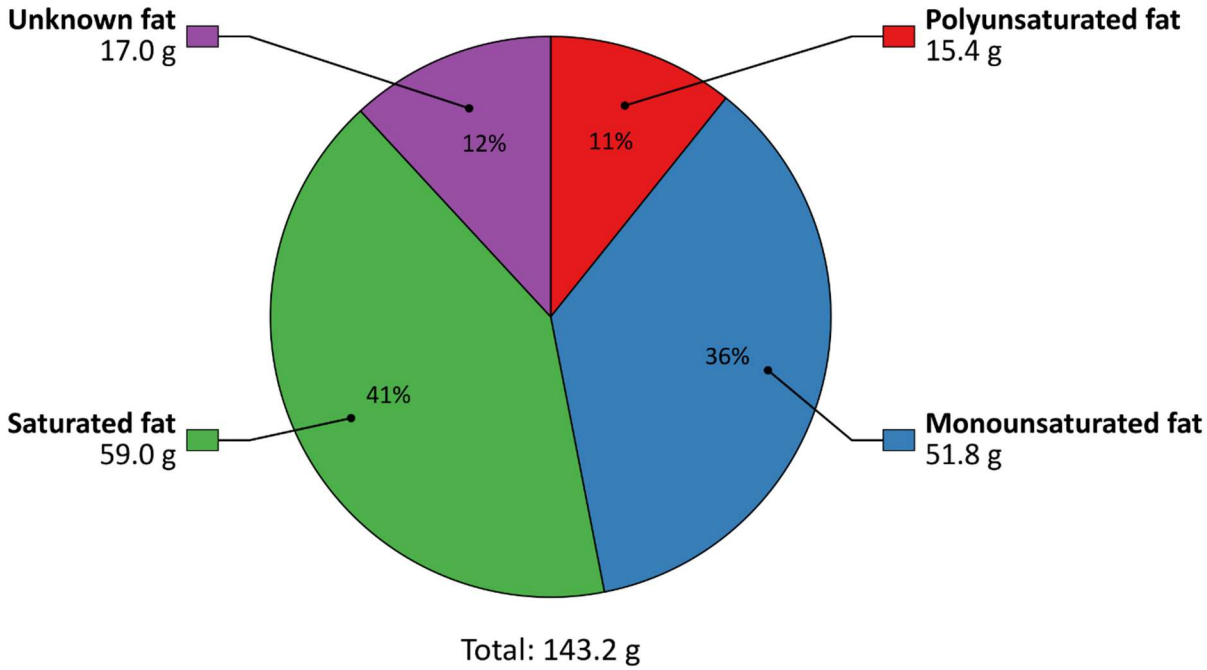

Figure 11: Dietary fat composition and daily intake (n 28). Percentages are represented as part of total fat intake. The total daily fat intake and fat types consumed represent participants' mean daily fat consumption over four days of diet records (comprising a 3-day diet record and 24-hour recall). MRC SAFOODS was used for dietary analysis. The SAFOODS dietary analysis results for the fat components summed to less than the total. Therefore, the "unknown fat" category was added to compensate for this deficit.

3.4.3. Micronutrient intake

The South African MRC SAFOODS database was used for dietary analysis of nutritional information, including micronutrient intake, acquired from four days of diet records (comprising a 3-day diet record and 24-hour recall). As reported in the methods section, this data (Table 7) is an under-representation of micronutrient intake, due to not capturing micronutrients contained in those consumed foods that were missing from SAFOODS, and in nutritional supplements. Despite this, the group met the recommended dietary intake (RDI) or Adequate Intake (AI) levels for many micronutrients [1]. However, the group fell more than 55% short of the RDI or AI for folate (25%), manganese (35%), calcium (48%) and magnesium (52%). Their vitamin D (62%), potassium (78%), thiamine (87%), vitamin B6 (94%) also fell short of the RDI or AI for those micronutrients.

Table 7: Minimum habitual daily micronutrient intake (n 28)

Micronutrient	Quantity	Average RDI / AI for 51 – 70 y/o	Percent of average RDI / AI
Calcium (mg. day^{-1})	502.3 (373.1 – 688.3)	1050	47.8
Iron (mg. day^{-1})	11.4 ± 5.8	8	142.5
Magnesium (mg. day^{-1})	244.7 ± 122.1	470	52.0
Phosphorus (mg. day^{-1})	1072.8 (860.5 – 1593.2)	1000	107.3
Potassium (mg.day^{-1})	2571.7 ± 151.9	3300	77.9
Sodium (mg. day^{-1})	1837.6 (1055.2 – 2648.7)	710	258.8
Zinc (mg. day^{-1})	11.0 (8.1 – 16.9)	11	100.0
Copper (mg. day^{-1})	1.5 (0.8 – 2.1)	1.45	103.4
Manganese (mcg. day^{-1})	1837.9 (1181.7 – 2524.0)	5250	35.0
Vitamin A (re or mcg. day^{-1})	1598.4 (574.4 – 2468.0)	800	200.0
Thiamin (mg. day^{-1})	1.0 (0.5 – 1.4)	1.15	86.9
Riboflavin (mg. day^{-1})	1.6 (1.2 – 2.2)	1.2	133.0
Niacin (mg. day^{-1})	20.7 (15.9 – 28.6)	15	138.0
Pantothenic acid (mg. day^{-1})	5.8 (5.1 – 8.8)	5	116.0
Vitamin B6 (mg. day^{-1})	1.5 ± 0.7	1.6	93.7
Folate (mcg. day^{-1})	98.1 (75 – 148.8)	400	24.5
Biotin (mcg. day^{-1})	35.5 (27.8 – 52.0)	27.5	129.1
Vitamin B12 (mcg. day^{-1})	6.3 (3.8 – 10.2)	2.4	262.5
Vitamin C (mg. day^{-1})	96.3 ± 63.0^{N}	45	210.0
Vitamin D (mcg. day^{-1})	6.2 (3.7 – 9.2)	10	62.0
Vitamin E (mg. day^{-1})	9.2 ± 5.7	8.5	108.2

Data from SAFOODS analysis of 3-day diet record and 24-hour recall, excluding supplements and added table salt. Data is expressed as mean ± standard deviation where normally distributed and median (interquartile range) where not normally distributed. RDI, recommended daily intake [1]. AI, adequate intake [1].

3.5. Diet description

3.5.1. Foods commonly consumed

Participants obtained most of their daily energy from animal-derived protein- and fat- rich sources, such as eggs, full-fat dairy, meat, poultry and seafood, as well as from added fat (e.g. butter, olive and coconut oils) (Table 8, Figure 12). Nuts and seeds were also substantial

contributors to TDEI (Table 8, Figure 12). These foods were also the major contributors to our sample's dietary fat (Table 9). Full cream dairy and nuts and seeds were also the primary contributors to participant's carbohydrate intake (Table 9).

Estimates from our sample's FFQ indicate that our participants' daily consumption consisted mainly of approximately 3 palm-sized portions of protein-rich foods (i.e. meat, poultry, seafood, organ meats, processed meat, or eggs), 4 table spoons of added fat (e.g. butter) or oil, 1 portion of non-starchy (usually above-ground-growing) vegetables, half a portion of fatty vegetables (i.e. avocado pears or olives), one-and-a-half portions (handfuls) of nuts and seeds, and one-and-a-half cups of full-cream milk/yoghurt or about 60 g cheese (Table 8). Our participants ate a portion of fruit twice per week (Table 8). Approximately one portion of starches, starchy vegetables, fast foods, alcohol, and dark chocolate were eaten approximately once per week (Table 8). Legumes, dried fruit, added sugar, sugar sweetened beverages and fruit juice, confectionary (excluding dark chocolate), and beer were seldom consumed (Table 8, Figure 12).

Table 8: Frequency of food groups eaten (n 28)

Food	Portion size	Portions number eaten weekly
Starches	½ cup cooked starch or 1 slice bread or 3 crackers (approx. 75 kcal).	0.9 (0.4 – 2.2)
Protein-rich foods (animal source)	Palm-sized protein-rich portion (approx. 225 kcal). Meat, poultry, seafood, eggs, organ meat, or processed meat.	21.4 ± 10.0
Vegetables		
- Non-starchy	½ cup cooked or 1 cup raw non-starchy vegetables (approx. 25 kcal).	5.6 (3.6 – 16.7)
- Starchy	½ cup cooked starchy vegetables (approx. 75 kcal).	0.7 (0.4 – 1.6)
- Fatty (avocados / olives)	¼ avocado, or 10 small or 5 large olives (approx. 45 kcal).	3.3 (1.6 – 7.3)
Nuts / seeds	1 handful or 30 g of nuts or seeds or unsweetened nut or seed butters (approx. 160 kcal).	9.7 (2.1 – 16.8)
Legumes	Half cup cooked legumes (approx. 75 kcal).	0.2 (0.0 – 0.8)
Fruit		
- Fresh fruit	1 tennis-ball sized, 2 golf-ball sized, or half cup chopped fruit (approx. 65 kcal)	2.0 (0.7 – 4.2)
- Dried fruit	3 pieces	0.0 (0.0 – 0.0)
Dairy	(Approx. 75 kcal)	
- Full cream	Half cup milk/yoghurt, 30 g hard cheese, or 2 Tbsp soft cheese or cream.	19.8 (13.2 – 33.2)
- Low fat	Half cup	0.0 (0.0 – 1.8)
Fat	1 Tbsp oil, butter, lard, ghee, margarine, salad dressing, or mayonnaise; 5 Tbsp coconut cream; or 1 small handful coconut flakes (approx. 135 kcal)	26.4 (17.3 – 39.1)
Sugar		
- Added sugar	1 tsp or 4-5 g table sugar, honey or syrup (approx. 16 kcal).	0.0 (0.0 – 0.0)
- Sugar-sweetened beverages or fruit juice	300 ml fruit juice or 340 ml soft drink or 375 ml energy drink or 1 cup hot chocolate/milo (approx. 130 kcal)	0.0 (0.0 – 0.0)
- Confectionary: Dark chocolate	½ slab (41 g) dark chocolate (approx. 250 kcal)	1.3 (0.4 – 2.9)
- Confectionary: sugar-sweetened	1 pastry, rusk, biscuit, waffle, brownie, muffin, or piece of cake; ½ slab chocolate or 1 chocolate bar (41 g)	0.0 (0.0 – 0.7)
Fast food	1/4 medium pizza or 1 cup fries or 1 pie (approx. 250 kcal)	0.2 (0.0 – 0.4)
Alcohol (wine, beer, lite beer, coolers, excludes spirits)	200 ml glass wine or 340 ml beer or lite beer or 300 ml cider (approx. 130 kcal)	0.4 (0.0 – 3.6)
- wine	200 ml glass wine	0.4 (0.0 – 3.6)
- beer / cider	340 ml beer or lite beer, or 300 ml cider	0.0 (0.0 - 1.0)

Data from food frequency questionnaire (FFQ). Data expressed as mean ± standard deviation where normally distributed, and median (interquartile range) where not normally distributed.

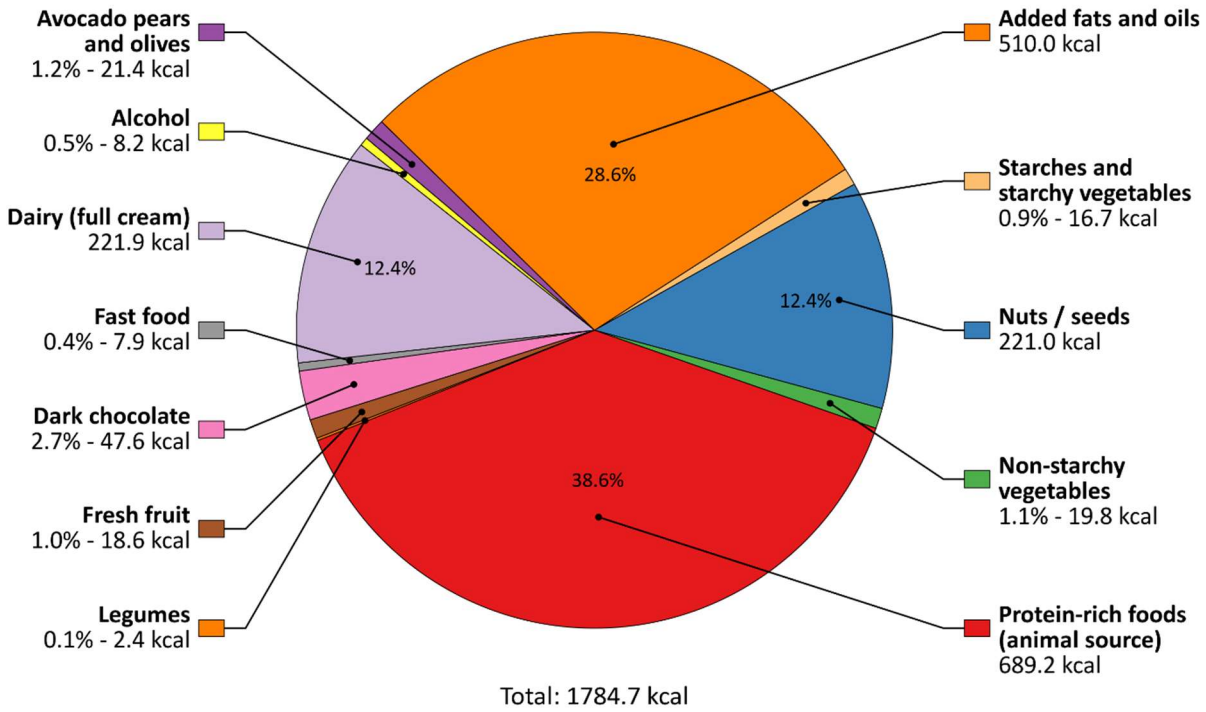

Figure 12: Food frequency questionnaire (FFQ) food groups' estimated relative contribution to habitual daily energy intake (n 28)

Table 9: Main sources of carbohydrates and fats

Main sources of carbohydrates	**Main sources of fats**
Full cream dairy (whole milk and yoghurt)	Added fat (butter, coconut oil, olive oil)
Nuts and seeds	Eggs
Fruit	Meat, poultry, seafood
Dark chocolate	Full cream dairy (cheese, cream, whole milk and yoghurt) and Nuts and seeds
Non-starchy vegetables and fatty vegetables (avocado pears and olives)	Fatty vegetables (avocado pears and olives)
Starches and grains (e.g. wheat bread, crackers, oats, rice, rye bread, pasta)	Dark chocolate
Starchy vegetables	

Food frequency questionnaire (FFQ)-reported food items' contribution towards dietary carbohydrate and fat intake. Main sources of carbohydrate and fat are listed in descending order of quantity of contribution towards total dietary carbohydrate and fat respectively (based on the fat and carbohydrate content (g) assigned to each FFQ food item).

The foods mentioned by participants during their interviews, as those chosen, limited or avoided (Table 10) are in line with the FFQ data in Table 8 and 9, and Figure 12 above. Participants said that they chose to eat protein and fat-rich animal-source foods such as eggs,

full-cream dairy, meat, poultry and seafood, which were also the primary contributors to their energy and fat intake (Table 8 - 10, Figure 12). Similarly, participants said that they limited foods that were reported in the FFQ as seldom being consumed (Table 8, Figure 12), such as fast or processed foods, confectionary, food and beverages containing added sugar, alcohol, and starchy food (including grains) (Table 10). LCHF or “Banting” processed food items and sugar-substitutes were also mentioned as food to avoid or limit (Table 10). Other food frequently limited by participants included dairy, fruit and starchy vegetables (Table 10). Food lists in RMR [33], were often mentioned as guidelines used by our participants when making food choices. Many participants attempted to stick to the green list, with occasional food inclusions from the orange list, while avoiding foods on the red list [33].

Table 10: Foods that participants report they currently 'choose', 'limit' or 'avoid' when asked during a semi-structured interview.

MORE FREQUENTLY TO LESS FREQUENTLY CONSUMED

Choose	Limit	Avoid
- Eggs - Dairy (specifically cheese - hard, cheddar, soft, Melrose, camembert, brie; and cream) - Bacon - Green vegetables (specific mentions: spinach, kale, broccoli) - Non-starchy vegetables (mushrooms, tomatoes, cauliflower, spinach and leafy greens, brinjals, marrow, pumpkin, salad, onions, zucchini, egg-plant, radish, garlic) - Avocado - Fats (butter, coconut oil) - Nuts and seeds (macadamia nuts, almonds, Brasil nuts, seed crackers) - Coffee (bullet proof coffee) - Chicken (drumstick, wing, carcass) - Seafood (fish - fatty fish, sardines; prawns;) - Organ meats (liver, kidney) - Meat (pork, fatty cuts, beef, lamb) – many people choose pork over "red meat" - Herbs and spices (cinnamon) - Fats (butter, coconut oil, olive oil) - Lard (e.g. goose fat) - Greek / full cream yoghurt - Berries - Blood (black pudding) - Bone broth (made out of chicken carcasses) - Apple cider vinegar - Banting / LCHF products (bread, biscuits, crackers, granola, pizza, Heba pap) - Tea (green, black, French) - Alcohol in the form of limited red wine - Dark chocolate - RMR green list [33]	- Dairy (cream, high fat plain or Greek yoghurts, sour cream; full cream milk; in some cases, cheese) - Fruit (berries, apple, pear, nectarines) - Grains (specific mentions include rice and oats, less frequently bread) - Starchy vegetables (sweet potato, butternut, carrots) - Alcohol (wine – particularly red wine, whiskey, lite beer) - Nuts (all, almonds, cashews), seeds - Chocolate (dark chocolate, one mention of nut chocolate) - Banting products (seed crackers, Banting bread, Banting pizza bases, Banting mayonnaise, LCHF porridge, LCHF granola) - Sweeteners (xylitol, erythritol, sucralose) - Red meat - RMR orange list [33]	- Processed food (some mentions include: crisps, sauces, canned food, processed meats, pastries, bread, "anything in a packet", "anything that comes from a box or a tin", additives, diet drinks) - Sugar and sugar-sweetened foods and beverages ("anything with sugar in it", coke, cake, biscuits, pastries, custard slice, sweets, chocolates, table sugar, condensed milk, diet drinks, sugar substitutes) - Confectionary (chocolates, sweets, cake, biscuits, dessert, pastries, baked foods, ice-cream) - Wheat and wheat-based foods (bread, pasta, Weetbix, pizza) - Potatoes and potato-based foods (French fries, crisps) - Sweeteners and sweet-tasting food (e.g. Banting products, sugar-substitutes, diet drinks) - Certain seed oils (soya, sunflower, corn, canola, margarine) - Grains (wheat and wheat-based products including bread and pasta; breakfast cereals; rice, oats) - Starchy or root vegetables (particularly potatoes, sweet potato, butternut, carrots, parsnips) - Alcohol (any alcohol in some cases, but particularly beer) - Dairy, except butter (milk, yoghurt, sometimes cheese) - Fruit (grapes, bananas, paw-paw) - Processed meats (sausage with added starch) - Fast foods or take-aways (pizza) - Cashew nuts and peanuts - RMR red list (some people also avoid foods on the orange list) [33]

3.5.2. LCHF diet evolution and progression

During the interview, we asked participants if and how their LCHF diets had evolved. A few participants said that their current LCHF diet is "*very much the same*" (T163) as it was when they initially adopted it. However, most of the participants' LCHF diets evolved over time – sometimes spontaneously, but more often, deliberately. Some participants mentioned modifying their food choices and diet based on postprandial blood glucose readings (i.e. to eliminate food items that appeared to be associated with higher blood glucose readings). For more example quotes see Appendix I.

3.5.2.1. Modifications to food choices and macronutrients over time

Fat. Some participants modified the fat content of their LCHF diet over time. While a few reduced the fat content of their diet, most increased it (Appendix I). Generally, participants said that as their weight loss increased, so did dietary fat content. In some cases, this change occurred spontaneously. However, some participants intentionally increased their dietary fat intake, as they lost body weight, in order to compensate for diminishing endogenous fat fuel stores, explained by the participant quote below.

- *Whilst I was losing weight, initially on the low carb approach I guess I didn't really need the fat because I was burning my body fat. Now that I'm like ten percent body fat I actually need the fats and there is a difference between low carb for weight loss, low carb high fat for maintenance and I guess instinctively, I came to realise that If I wanted to stabilise my weight I needed to start eating more calories because of all the training I was doing.* (T103)

Some participants directly preceded their current LCHF diet with a conventional very energy-restricted weight loss diet. Usually, the energy restricted diet did cause weight loss, however, participants also reported that it resulted in hunger. Interestingly, when these participants added fat to their existing restricted intake - thereby increasing their TDEI and relative contribution of fat to their TDEI – they reported, not only reduced hunger, but also that weight loss was easier and more pronounced (illustrated below).

- *I changed from having oats for breakfast to eggs for breakfast and increased my olive oil, I think I was on one teaspoon of olive oil per meal and just started to have that more liberally and I remember noting that that's when my weight loss increased.* (T109)

Data regarding food choices and modifications over time were in line with increase fat intake over time, and consistent across interviews and FFQ (Table 8 - 10). Many participants mentioned deliberately introducing or increasing their intake of certain high-fat foods. Specific food items that were mentioned included avocado pears, organ meats, cream, cheese, butter and certain oils (avocado, coconut and olive oils). One participant illustrates:

- *I have increased my intake of butter, I have increased my intake of cream, I have increased avocado oil, avocados and olive oil.* (T151)

Protein. Certain participants were concerned about their protein intake. Some became stricter about limiting protein as they modified their diet:

- *…what I'm trying to do currently, is to keep my carbs at 30 grams per day or under, or my carbs plus proteins at less than 100.* (C97)

Other participants, like the one quoted below, become more relaxed over time about limiting their protein consumption:

- *I am aware that I am having too much protein but I am having it anyway and that is kind of what I am living with* (T109)

Also see Appendix I.

Carbohydrate. A few participants reduced the carbohydrate content of their diet over time (Appendix I).

- *I started to go lower on my diet, as with regards to lower carb and higher fat, and then eventually moving towards ketosis*. (T139)

In line with this trend, participants frequently modified their LCHF diets over time to exclude foods thought to increase their blood glucose concentrations. Eliminated or limited foods mentioned included fruit, dairy, sugar substitutes and hidden sources of glucose, pumpkin, nuts and seeds, and starchy vegetables. One such modification is represented in the participant quote below (other can be seen in Appendix I)

- *I used to love fruit, so it was a norm eating two or three fruit a day. The moment I cut that out of my diet the sugar dropped to where it is now, averaged between 4 – 5 now, never really getting up to 5.5.* (C19)

Sweet taste. Most participants felt that sweet taste, whether nutritive or non-nutritive, sparked sugar cravings. So, as their LCHF diet progressed they began to avoid sugar substitutes and sweet treats.

- *Initially I thought, oh well this was wonderful, that I was being able to have the erythritol, but then I realised, actually, I need to cut down on that sweetness, because my body is still craving sweetness. So, I have gone down on that as well, but there are occasions that I need to have something sweet. Celebrations or whatever. More and more I have learnt to adapt… Before diagnosis, it was cakes and all sorts of things. I went from that to xylitol and then from xylitol to erythritol. So, when we do it, I try to have cake, but made with erythritol and not eat a lot.* (T139)

Food quality. Another trend with some participants was an increased attentiveness to food quality, and food choices that focused on better perceived food quality, more whole food and less processed food (also see Table 10):

- *Well when I first started low carb I didn't understand the distinction between, or the benefit of removing processed food. I was still eating some packet goods. Some ready meals that I thought well they're relatively low carb. But I hadn't understood the benefit of that. So that certainly changed.* (T103)

Dietary modifications in response to a weight loss plateau. Often weight is reported to plateau after a while on LCHF. A couple of participants noted that during this weight loss plateau, their clothing size continued to drop despite no change in weight. Many participants found that tweaking their LCHF diet allowed them to initiate weight loss again. Common tweaks include increasing dietary fat, and reduction in energy intake by reducing eating frequency (i.e. introducing IF or eliminating snacking), and reducing portion size (where previously there was only carbohydrate reduction, but no portion control), as discussed during the interview (see example quotes in Appendix I. Some participants reported that including even a small amount of carbohydrate or sugar in their diet (e.g. from 'healthy' sources, such as fruit or starchy vegetables, or 'unhealthy' sources, such as sweet treats like cake) halted their weight loss and interfered with blood sugar control.

3.6. Perceptions around the diet

3.6.1. Dietary control

3.6.1.1. Hunger

A strong and recurring theme throughout most interviews was a distinct and pronounced reduction in hunger after adopting an LCHF diet.

Participants often contrasted the hunger that they experienced while following traditional 'balanced' energy-restricted diets to their lack of hunger on an LCHF diet, as illustrated by this participant's story.

- *I got so frightened about this idea of bariatric surgery that I really starved myself so I even reduced the diet that* [the dietitian] *gave me and I did lose weight. I lost about twenty, twenty-five kilo's with that process. But I remember being so hungry that I used to wear ear plugs to sleep, which I don't do anymore. That is also an interesting thing, I sleep more soundly. I remember waking up chewing on my earplug that I was like so hungry that in a dream I thought like this is food. So, I was like really, really starving myself. The biggest difference that I remember when introducing the fat was that the hunger was sated.* (T109)

However, even when participants were not restricting their energy intake (before following an LCHF diet), they complained of hunger, which improved or resolved after adopting an LCHF diet:

- *one of the things I noticed before I was diagnosed was for example, when I got hungry, it wasn't kind of like, 'oh, I think I would like to eat something now', it was, 'if you don't eat something now, you'll die!' kind-of-hunger. It was a really sharp pain in the stomach, like 'I just have to eat something!'. I haven't experienced any of those hunger pangs really ever since I started this diet. And there have been a couple of days where I just decided when I woke up, and I've not been hungry, I decided not to bother. And I got through most of the day and not bothered and then maybe had an early meal in the evening… And obviously one of the other things I have measured, is my ketones*

and clearly as soon as you're in a proper ketone state, you stop feeling hungry. It's not hard then to not eat. (C97)

In line with most participants' feeling that an LCHF diet is responsible for the reduction in and control over their hunger, a few participants describe a resurgence of hunger after increasing their carbohydrate intake, or 'cheating' on their LCHF diet:

- *The only time I feel hungry is after a weekend when I've sort of been a bit bad. When I've eaten maybe more carbs than I normally do. It's funny because often, if ever I feel hungry, I would think, "what have I eaten?", and it will always be carbohydrates.* (C343)

When participants strictly adhere to an LCHF diet, with or without intentionally fasting, and follow their satiety cues, generally they experience little hunger and improved control over their appetite, as illustrated by the interview quotes below:

- *the beauty of the whole Banting scenario in terms of the lifestyle situation is you don't get the hunger cravings, so it's not that you have to go somewhere and you feel well I have to eat. You don't have to eat.* (C89)

However, a few participants still complained of hunger, or a boredom-associated perception of hunger, as described below:

- *So, if I stick to the low carb thing properly, I'm never ever hungry. I am more bored, or need food because of boredom, than I am actually feeling hungry…* (C343)

3.6.1.2. Portion and calorie control

Participants were asked how they decide how much to eat and whether they restrict calories or portion size, on their LCHF diet. Most participants reported an awareness of the energy content and portion size of their food, but that they didn't count calories or stick to precise daily energy targets. The quotes below illustrate:

- *I never counted calories I never weighed, measured or counted anything.*(C13)

Some participants use the maintenance of their body weight as an indicator of appropriate energy intake:

- *I'm conscious of calories, but I generally don't restrict them. I generally eat until I've had enough… My weight is fairly stable. It is not going up, it is not going down terribly fast, so I presume I am basically eating as much as I'm using.* (C17)

A few participants mentioned using diet-tracking apps initially or occasionally to get a better idea of their macronutrient distribution and TDEI:

- *I tried the Macro Calculator and the My Fitness Pal … at the beginning it helped me size my meal, and since then I know pretty much what size the meal should be, so I go with that. But sometimes … I am hungrier at night, I do eat something, I'm not trying to go against my hunger.* (C19)

Most participants eat *ad libitum*, allowing their satiety cues to determine their energy intake. Below is a participant's description of how they rely on their satiety cues to determine when and how much to eat.

- *I go with the principle of eat when you're hungry and when you're full, stop and that's it.* (C79)

Some participants use IF to help control or restrict their energy intake.

- *I don't restrict my calories but I do do Intermittent fasting.* (T157)

While few participants counted their calories or stuck to specific daily energy targets, many of them were cognisant of their macronutrient intake or ratios - all of them restricted their carbohydrate intake, and some consciously limited their protein intake (many specifically mentioned not eating protein more than the size of their hand palm or fist), such as the participant quoted below.

- *I suppose in a way I do restrict, it is not like weighed or whatever but it's like this sort of size meat* [palm sized], *I stick to probably a hundred and fifty and two hundred grams sort of meat size. You know that sort of size meat. But the vegetables I mean are on a normal plate.* (T101)

Some mentioned limiting their portion size, often by eating off a smaller plate.

- *… I always use a small plate. I don't use a large plate. I don't weigh anything and I am not over aware of the portion size.* (T139)

3.6.1.3. Food addiction

Many participants experienced their relationship with certain foods (usually those high in sugar, with a sweet taste, or carbohydrate-rich) as addictive. This participant's description of his sugar -seeking and -consuming behaviour exhibits various addictive manifestations that were commonly described by participants, including a strong desire or compulsion to eat sweet or sugar-sweetened food, impaired control over consumption of this food (particularly in terms of level of use or the amount of sugar-sweetened food or beverage consumed), and continued use despite negative consequences (in this case, weight gain) [161]:

- *I went through a phase in this winter where I broke down and my sugar addiction kicked back in and I started raiding the biscuit cupboard at work, and if I have 1 biscuit it becomes 3 biscuits, it becomes the rest of the packet… And then also, on my way home, I would stop and buy chocolate and then 1 was not enough, buy 2, and with that, my weight went back from …77, it jumped up to 88, and fortunately I've been able to get control of that, although it's a little bit difficult. The sugar addiction is extremely real and I have managed to get all that back into control again and it's* [the weight is] *basically coming off slowly.* (C13)

Also, in line with addiction or substance abuse, participants often relayed the experience of developing tolerance – needing progressively more sugar-containing foods, with increasing or continued consumption of those foods [161]. More than one participant compared their sugar addiction and tolerance to cigarette smoking.

- *If I smoke I will smoke, kind of thing, and I used to control it, a lot of the time I would have 1 or 2 smokes in the evening, but I would always land up going back to smoking about 20 a day kind of thing. And that is what I realised from Banting actually, is carbs are probably something that I'm probably never going to be able to eat for the rest of my life, certainly not on a regular basis. Maybe I will have a cheat once a month, have a cheat like go out and have a really nice pasta, something like that. But I guess my days of carbing are over, and my days of smoking were over in the year 2000.* (C37)

As described by the participant above, the theme of having to completely cut out sweet taste, irrespective of whether it was from real sugar or sugar-substitutes, in order to prevent 'relapse' and gain control over food addiction and cravings, was common. Once sugary foods or taste was sufficiently reduced or eliminated, participants usually experienced reduced desire or 'craving' for sugar-sweetened foods and beverages, as mentioned below.

- *my husband eats cookies and chocolates and things in our home… I thought it would drive me, but it doesn't bother me at all. You know I am so surprised to be able to walk passed all of them in the shopping centre and it is no trigger. Whereas before* [LCHF] *I would never be able to do that.* (C311)

While craving for sweet foods generally disappeared after adopting LCHF, a few participants occasionally still experienced a strong desire or 'craving' for high carbohydrate foods.

- *I mean sometimes I still crave, I feel I crave carbohydrates sometimes. So sometimes I feel like it's not as easy as other times.* (C319)

Interestingly, a reduced desire for and consumption of sugar-sweetened or sweet-tasting food was frequently accompanied by an increased sensitivity to sweet taste:

- *I'll eat a lemon now and coffee and 90% chocolate tastes sweet. So, my sweet taste has completely changed.* (C73)

Many manifestations of food addiction appear to be present amongst participants. However, incongruous with substance dependence, participants usually managed to successfully cut sugar-sweetened foods and beverages from their diet when they were diagnosed with diabetes (conversely, substance dependence is often associated with persistent substance use despite clear evidence and awareness of harmful consequences), as illustrated by these participants:

- *At that time* [when diagnosed with T2D]*, I dropped the sugar, everything that contained sugar, I just wouldn't eat anymore, but I wasn't aware about bread and potatoes and rice, that was something I didn't know at the time, so I continued to eat potatoes rice and carbs, but the added sugar was out of the question, I stopped it. I didn't drink any sodas anymore.* (C31)

Participants in this sample frequently described themselves as 'extreme' or 'all-or-nothing' personalities, while a minority felt that they could eat addictive foods occasionally or in moderation.

- *I'm an all or nothing person, either you're having no carbs at all, consciously, or if you're having some, it creeps back.* (C89)

3.6.2. Psychology and dietary motivations

3.6.2.1. Motivation

Almost half of the study participants said that they adopted an LCHF diet to control T2D and its sequalae (Table 11). Almost a third said that they did it to lose weight. Fourteen percent said that they adopted an LCHF diet to reduce their insulin and medication use. Another 14 % said that they did it in response to advice or information received from books or medical professionals.

Table 11: Factors that motivated participants to follow an LCHF diet (n 28)

Motivation to start LCHF	Percent (n)
Blood glucose control, diabetes control and avoidance of complications	46.4 (13)
Weight	28.6 (8)
Insulin and medication	14.3 (4)
Books	10.7 (3)
Health and nutrition	10.7 (3)
Advice from doctor	3.6 (1)
Avoid gastric bypass	3.6 (1)
Prevent premature death	3.6 (1)

Self-reported data from online health questionnaire. Some participants gave more than one reason as motivation for adopting an LCHF diet.

3.6.2.2. Psychological obstacles during an LCHF diet

Psychological motivations behind eating behaviours and choices emerged from the interviews.

Some participants mentioned difficulty breaking long-held eating habits, such as adding sweetener to hot beverages:

- *I was still having sweeteners in my coffee and tea at the time, I found that habit very hard to break. I eventually broke through that…* (C319)

Quite a few participants struggled with eating out of boredom (see Results section 3.6.1.1 Hunger).

However, the largest psychological hurdle that our participants mentioned having to overcome was 'comfort-eating', or eating in response to stress or sadness. One participant explains:

- *But I have always been somebody who has taken comfort in food and I can remember whenever it got stressed, I would go and of course carbs are the cheapest things to buy, so I would eat. So, I certainly had a very unhealthy lifestyle… I don't any longer get tempted by cake and I am not one of those who has to have masses of ice-cream and things like that. That doesn't bug me anymore, at all... And when I do feel that right now I am stressed, all I have got to do, is to do what I would do with any 5-year old child, say, "Here is a new toy, let us play with it", and 2 minutes later, the tantrum is gone. And I have got to realise, I am like that too. So, if I am sitting down panicking, I am going to want to eat. If I could break that cycle for 60 seconds, the desire to feed my face will go away.*

  …I grew up … [in] *an Afrikaans family, and food was a sign of love. So, your mother would overcook and you would have to eat, because if the plate was not empty, she thought you didn't love her… So, you had to start breaking the link between love and food and that was not all that easy. It was tougher than I thought. But now it is a matter of you eating until you're full and then you stop.* (T137)

3.6.3. Sustainability

During the interview, when asked how sustainable an LCHF diet is for them or how long they plan on eating an LCHF diet, all except one participant, said that an LCHF diet is sustainable and that they will stay on the diet indefinitely. Many participants expressed that for them an LCHF diet was a lifestyle that they planned to follow for the rest of their lives:

- *Knowing how I feel and how good I feel health wise and weight wise, I would say, I would never want to go back to the other way, because I wouldn't want the battles that I had with the excess weight and with the diabetes particularly, and all the other health problems, which have gone and it is being like a miracle. And so, I would say, for me it would be totally sustainable.* (T127)

Participants often said that they find LCHF easy to follow:

- *It's very easy. I've been doing it essentially for 6 years now*. (C73)

A few took it a step further, explaining that for them LCHF is not only easy to follow and sustainable, but also enjoyable:

- *It's really sustainable and really filling and tasty and great food. I love it.* (T103)

Only one person doubted its sustainability

- *I think it takes out much of the joy for me at this stage. Maybe it's just early days. …* [Since eat LCHF] *I couldn't freely partake in everything anymore.* (C311)

3.6.4. Challenges

The challenges and negatives of LCHF, as experienced by this study's participants are discussed below and include, social, economic, negative-symptom and potentially-deleterious health effects.

3.6.4.1. No negative aspects

Approximately half of the study participants said that there were no negative aspects associated with an LCHF diet.

- *Do you know what? I can't think of one thing that has not improved.* (T103)

3.6.4.2. Social consequences

During interviews, the most commonly cited challenge of an LCHF diet was related to social situations and stigma. Some participants felt negatively judged, misunderstood, not supported, and pressured by the general public and their wider social circles, for following an LCHF diet.

- *I think it is difficult for me at times, in a social atmosphere. Whereas other people don't understand that I am trying to do it without medication and I am trying to lower my sugars with diet and you know. They will say things like, "oh, just have a piece of cake. It won't kill you," and inside I am saying, "well actually it may just"* [laughing]*. Because they don't understand. So that is really the only sort of the negative side of it.* (T139)

Other participants expressed concern about fitting in, saying that an LCHF diet makes them different:

- *I am trying to fit in with everybody. Socially it's been difficult.* (T101)

Some participants said that they struggle to deal with eating an LCHF diet at friends' houses or restaurants, no longer fitting in with the eating habits of loved ones, with offending or upsetting anyone or appearing fussy, or withstanding the temptation of non-LCHF foods, as described below:

- *The only time it becomes a little awkward, is if you get invited around to somebody's house for a dinner party or something or other like that, and now you're kind of caught between the world where they're dishing up food that you don't want to eat, or you actually might want to eat, but you shouldn't eat. You don't want to offend them and you don't want to be picky. I mean we don't eat out every night it's something that happens once every 3 months or something, so when it happens, deal with it.* (C37)

3.6.4.3. Inconvenience

In their interviews, some participants said that LCHF is challenging or inconvenient when looking for a quick snack, take-away food, suitable options when eating out or when travelling. Some found that pre-making suitable non-perishable LCHF foods helped them to overcome this hurdle.

- *Eating out is not as easy. You've got to sort of think where you are going, what they serve and what's on the menu and things like that.* (C343)
- *The only problem is that, if you are on the road traveling as we do, if you feel hungry, you can't just walk into a take-aways and find something Banting-friendly, because it is not yet everywhere.* (C319)

However, others disagreed.

- *I don't find it at all difficult to eat out. The difficulty is cheap, you know you can't eat out fast foods, but you can eat out.* (C97)

3.6.4.4. Expense

There was some concern over the perceived expense of following an LCHF diet. This concern was particularly around the pre-made LCHF starch-replacement food items (e.g. shop-bought seed crackers, pizzas, bread, etc.), which a few participants enjoyed including in their diet, but most accepted were not integral to following an LCHF diet:

> *…the only negative that I can find, especially with your breads and things, it is a bit expensive.* (T131)

However, one participant said that while the diet started out expensive, once he started to experience the LCHF-associated reduction in appetite, it became much cheaper.

> *So, in the beginning it was very expensive. Now it is much cheaper, because I can do one week with half of the things I bought earlier on.* (C31)

Conversely, one participant said that he finds eating an LCHF diet cheaper than more conventional diets:

> *What I found, for example, is because fat is so vilified, practically all the meat that you get in a supermarket is priced inversely proportional to how good it is for you. So, for example, my meat bill has gone down dramatically, because now what I do is I buy the cheapest cuts, because those are the ones that have got fat in them.* (C97)

Multiple participants mockingly said that the only negative or challenge of an LCHF diet is the expense associated with buying a new wardrobe after losing so much weight:

> *It's just expensive, that is all… to buy a new wardrobe* [Laughing]. (T163)

3.6.4.5. Adjusting and adapting

Participants reported that the initial adjustment to LCHF food choices was often challenging:

> *the first two weeks is not so nice, but thereafter, just amazing and I will never go back.* (T127)

Some participants reported that they initially missed high-carbohydrate-content food items from their previous diet. However, in most cases, after a while they no longer missed these foods.

- *It hurt for a couple of weeks, I missed my spuds, the potatoes, rice, pasta etcetera, but after a couple of weeks, I settled into it and was fine with it.* (C17)

Others found adjusting to the high fat content of the diet difficult.

- *I don't particularly like rich or fatty food… it took me a while to get used to… But, you know, after a week or two it's fine.* (C19)

There were also a few reports of 'keto-flu' (a combination of flu-like symptoms, thought to mostly result from natriuresis [162, 163]), which is associated with the early stages (particularly the first week) of following an LCHF diet:

- *…it's very difficult to, if I can use the word, cold turkey. It's just impossible. Interestingly enough, only in retrospect did I realise … what happened to me when I went onto the LCHF… About 2 or 3 weeks after we started the LCHF, I got extreme vertigo and it lasted for about 2 weeks, but on 2 of the days, I couldn't even lift my head. And I think that was a carb flu type symptom. I think it was a withdrawal that took place.* (C89)

3.6.4.6. Gastrointestinal complaints

As discussed previously, in their surveys, a quarter of participants reported worsening gastrointestinal issues after adopting LCHF (Table 12). In line with this, in interviews, twenty percent of participants complained of varying degrees of constipation since adopting an LCHF diet:

- *I think the only thing that I struggle with time to time is constipation.* (T107)

3.6.4.7. Other

The only medical related challenges mentioned during interviews were raised cholesterol, and gout:

- *The only thing…that I am having problems with is my cholesterol.* (C319)
- *I have developed some gout and that could be diet.* (T127)

One participant cited a case of bad breath, when first adopting an LCHF diet, which disappeared over time:

> *Well the ketosis, I think I used to have more ketosis, so my wife would complain about my breath. Which is interesting actually now because I have to run to get ketosis. It doesn't matter what I eat.* (C73)

3.7. Perceived changes in aspects related to health-related-quality-of-life (HRQoL) before and after following an LCHF diet

Before adopting LCHF, participants reported deteriorations in various aspects of HRQoL, as illustrated by this participant's description:

> [after T2D diagnosis and before adopting an LCHF diet] *my blood glucose went up, my pain in my feet went up and my weight went up, everything went up.* (C31)

Conversely, after following an LCHF diet many participants reported, often dramatic, improvements in these same areas, namely:

- Diabetes progression, signs and symptoms
- Body weight
- Energy and Exercise capacity and performance
- Pain
- Mobility and activities of daily living
- Mental or psychological (mood, concentration, memory, cognition, self-confidence)
- Sleep
- Gastrointestinal
- General health

- Hunger (discussed in Results section 3.6.1.1 Dietary control: Hunger)

Table 12: Perceived changes to conditions or symptoms after adopting an LCHF diet

Condition or symptom	Drastic improvement % (n)	Some improvement % (n)	No change % (n)	Some deterioration % (n)	Drastic deterioration % (n)	Not applicable % (n)
Diabetes symptoms	75 (21)	18 (5)	0 (0)	4 (1)	4 (1)	0 (0)
Allergies	4 (1)	11 (3)	21 (6)	0 (0)	7 (2)	57 (16)
Eyesight	14 (4)	4 (1)	11 (3)	0 (0)	4 (1)	68 (19)
Skin conditions	18 (5)	25 (7)	25 (7)	4 (1)	0 (0)	29 (8)
Gastrointestinal issues	14 (4)	11 (3)	11 (3)	25 (7)	0 (0)	39 (11)
Post nasal drip	4 (1)	18 (5)	11 (3)	7 (2)	0 (0)	61 (17)
Concentration	11 (3)	39 (11)	18 (5)	4 (1)	0 (0)	29 (8)
Sleep	10 (3)	43 (12)	18 (5)	7 (2)	0 (0)	21 (6)
PMS	0 (0)	4 (1)	7 (2)	0 (0)	0 (0)	89 (25)
Other	18 (5)	0 (0)	0 (0)	0 (0)	0 (0)	82 (23)

Data comes from participant answers to the detailed online questionnaire question: "Since you've been following an LCHF diet, have you experienced any of the following?" Participants selected a Likert-scale option for each "condition or symptom", listed in the first column of this table. "Other" conditions, which participants listed as changing after adopting an LCHF diet, included pain, mobility, exercise capacity, energy and mood.

3.7.1. Diabetes progression, signs and symptoms

In surveys, 93 % of participants (26 out of 28) reported an improvement in their diabetes progression and symptoms after adopting LCHF (Table 12). Seven percent (2 out of 28) participants reported a deterioration, despite an improvement in HbA1c (from 14.7 to 5.3 and from 7.1 to 5.3 respectively), and maintenance or slight increase in T2D pharmacotherapy, after adopting LCHF (Figure 2). This is in line with the overall reductions in HbA1c and diabetic medication and insulin usage since adopting an LCHF diet (Figure 1 - 2). One participant exemplifies the common theme of perceived improvement in T2D using an LCHF diet:

> *It's just the frustration that people don't understand how easy it is to manage diabetes on this diet.* (C73)

Frequently mentioned diabetic complications that resolved or improved after following an LCHF diet included eyesight and the pain associated with neuropathy. Here, one participant explains the drastic improvement in pain experienced after adopting an LCHF diet.

- *I have no sense of the neuropathy anymore* [after LCHF]*. It was so incredibly painful it was just like hurting in my legs. Even having the air-conditioning on in our bedroom, the wind current from the air conditioner would cause me pain. It's bizarre to think that. In any sense, it was awful. Horrible horrible horrible. So, that is completely gone.* (T109)

3.7.2. Perceptions regarding body weight

3.7.2.1. Body weight and an LCHF diet: Perceptions and experiences

In contrast to most other diets participants tried in the past (see "other diets" below), participants found LCHF to be the one diet that allowed them to lose weight and keep it off, thereby overcoming their lifelong battle with overweight. In interviews, most participants reported weight loss after following an LCHF diet (Table 12), with the most rapid loss occurring in the beginning (first 3 months) of following LCHF.

> *All of my adult life, I was too heavy and I always wanted to find a way to fix it, to change it to make myself better, but I couldn't find a way until I found about LCHF. And since then, like I said, before I've lost about 130 pounds which is about 60 kilos of weight loss…I've never been able to lose weight for more than a few months and I've been at it for almost a year now, so I would say I'm pretty much going to eat that way for the rest of my life.* (C29)

However, not all participants reported drastic reductions in weight on adopting an LCHF diet:

> *I was a size 16, I am a size 14 now. So, even if it is in a year, I only got down one size, quite frankly, I don't care.* (T131)

Perceived reasons for weight loss after adopting an LCHF diet included reduced hunger, increasing the fat- and/or lowering the carbohydrate- content of their diet, and stopping insulin (See example quotes in Appendix I).

3.7.2.2. Body weight and mental health

In addition to the well-known deleterious physical health effects of overweight and obesity [9], our qualitative data indicates that excessive body weight can also have a negative impact on mental health. Overweight and obesity was often reported to be linked to negative

feelings, including shame and lack of confidence and pride, as is evident from this participant's description of themselves prior to their weight loss and adopting LCHF:

> *I was so overweight, I was just a fat slug.* (T163)

When reflecting back on her pregnancy, one participant said that despite not eating excessively or unhealthily she picked up huge amounts of weight:

> *I was like a beached whale. The first time I saw myself in a mirror after 5 or 6 months and I said to my husband you can divorce me if you want.* (C53)

So, unsurprisingly, initial rapid weight loss frequently experienced when adopting LCHF (Table 12) was reported by some to be a very motivating factor to stick with the diet.

3.7.2.3. Body weight and diet history

A long, ofttimes life-long, battle with overweight was an emerging theme. Many participants reported being "heavy" children and adolescents. Those that didn't, reported a time in early or late adulthood after which they began battling to control excessive body weight. Participants also reported progressively gaining weight over time. One participant explains:

> *I was always an overweight child and no matter what sport I did I would firm up but I will never lose weight. You know I'd be one of these real like muscular, chunky type sports people, you know?* (T101)

The extended struggles with overweight correspond with reports of attempting various diets to lose weight. The most common diets were low GI, low fat, calorie restricted diets; diets prescribed by healthcare practitioners (e.g. dietitians and the CDE diabetes programme); Weigh Less; Weight Watchers; and Sure Slim. However, these diets, while sometimes resulting in weight loss, were experienced by our participants as unsustainable. This explains the reports that any weight that may have been lost while following these diets was usually regained, as mention in this participant's quote and the quote examples in Appendix I.

> *I was trying, but I couldn't* [keep the weight off]*, every year or every other year, I would start a diet, lose some weight and gain it back.* (C29)

Reasons cited for these diets' lack of sustainability included hunger, difficulty with weighing food or counting calories, feeling deprived, or lack of palatability, as described in quotes in Appendix I.

However, in cases where a conventional energy-restricted weight loss diet directly preceded an LCHF diet, the weight that was lost and health benefits achieved on that diet were maintained, and in some cases furthered, after adopting an LCHF diet, as described by this participant:

> *The weight that I lost at the beginning of this year, during the very low calorie* [diet shake] *2 months,* [and] *my HbA1c, remained normal at normal levels since then.* (C17)

3.7.3. Energy and Exercise capacity / performance

Improvements in energy after adopting an LCHF diet was a very common theme which emerged from most participants during their interviews. When asked how an LCHF diet influenced aspects of their lives, participants mentioned that they experienced increased energy, reduced fatigue and lethargy, and an improved desire to move and perform daily activities at work and at home, as mentioned below and in quotes listed in Appendix I:

- *…before* [LCHF]*, I was always sleepy…and tired and just had no energy, and now I am sort of up and clearing the house and spring cleaning and my husband is very amused. Because I am always active, like I used to be in my twenties. Okay, so I have got a lot more energy. And before, you know, going for a walk was a chore. Now I really enjoy it.* (T127)

Many participants reported, not just an increased desire to exercise, but also ergogenic changes including improved fitness, exercise capacity and performance, and recovery after exercise, after adopting and following an LCHF diet (see below and Appendix I).

- *I've got more energy, I'm fitter than I've ever been. My health's great, my energy is great. I sleep well. My athletic performance has improved. The other thing I've noticed is after exercise, where ordinarily you'd feel sore for three or four days, I recover very quickly now... It's quite remarkable.* (T103)

However, one participant said that they did not experience ergogenic benefits after adopting an LCHF diet:

And interestingly enough, having lost the 10 or 15 kilos in the last 2 years, since I went on the LCHF, I'm sad to say that my cycling speed has not increased. (C89)

The cause of improvements in exercise was not always clear. While most felt that their increased desire to exercise was a direct result of an LCHF diet, some mentioned that they had increased their exercise since adopting an LCHF diet, but did not say that it was a consequence of the diet. Some felt that it was unclear whether their increased desire and capacity to exercise, and their improved exercise performance, was a function of improved energy levels, weight loss, or reduced pain. This participant explains:

I am not able to distinguish [whether weight loss or diet is responsible for my improved exercise capacity and performance]. *The function of the diet is the weight loss and the function of the weight loss is that I am not carrying seventy-three extra kilo's… because of* [the weight loss] *I am able to exercise without the pain that came before and that is why I am fitter.* (T109)

3.7.4. Pain

Pain prior to an LCHF diet was mentioned as a common complaint amongst our participants. While some participants still experience some degree of pain, most reported an improvement and sometimes a complete resolution of pain after following an LCHF diet, as illustrated by the interview quotes below and in Appendix I.

I am now able to walk bare-feet in the house, which I could never before, it was too painful. I really couldn't have done that. I used to have to wear Mohair socks, 24 hours a day, I have just changed them three times a day, because my feet were permanently cold and that is not the case, anymore. (T137)

3.7.5. Mobility and activities of daily living

Impaired mobility was commonly cited in participant interviews as having a substantial negative impact on quality of life, and was usually related to obesity. Participants with impaired mobility generally said that an LCHF diet very positively impacted on their movement and consequently HRQoL. Notably after adopting an LCHF diet, these participants also experienced significant weight loss and also often reported reduction in pain, which likely

contributed to the improved mobility and HRQoL. This participant quote and those in Appendix I illustrate:

> *I feel a lot better and I'm 100% convinced it's because I'm a lot lighter. My knees are not sore. I'm up and about.* (C19)

Prior to adopting an LCHF diet some participants were unable to drive due to poor health (e.g. risk of falling asleep behind the wheel or hypo or hyper glycaemic incidents) or occasionally had to use a wheelchair to get around. After following an LCHF diet these participants were able to drive again and no longer had to use wheel chairs.

> *…in 2012, the doctor said, "you are not allowed to drive a car, it's too dangerous you can have hypos or hypers". So, I wasn't allowed to drive. And now, a few weeks ago, I renewed my driver's license.* (C31)

There were also other less expected ways in which participants felt that an LCHF diet, and its associated weight loss, impacted on HRQoL-related to activities of daily living. One participant explains the effect that being able to choose from a large selection of clothes again had on him:

> *The first time I was able to go to Woolies* [clothing shop] *and buy a shirt, I was completely, I was so traumatised, I couldn't do it. Because for how much of your life, you would go to a shop and you would say, "this is my size," and they say, "these are the three shirts, that will fit you". That is it. You choose from three. Now you are going to Woolies and suddenly there are 30 or 40 shirts that could fit you. And I didn't know how to make a decision. I actually had to get help, I had to get my friends to come with me and say, "it is too traumatic, I can't, I can't choose. I don't know how to do it anymore, I haven't learned how to choose clothes"…and even now, this past weekend was the first time I bought a shirt, without help. I am finally now able to do it. So, in that sense the quality of my life… has changed on so many levels, ja* [Afrikaans for "yes"]. (T137)

3.7.6. Mental and psychological

During interviews participants cited improvements in mood (happier, more stable), memory, concentration, cognition, stress-coping ability, alertness, and self-confidence, after adopting and following an LCHF diet. No participants reported a deterioration in mental or psychological HRQoL during the interviews. Fifty percent of participants surveyed cited an improvement in concentration, with only one saying that their concentration got worse after adopting an LCHF diet (Table 12). This participant quote and those in Appendix I describe the perceived impact an LCHF diet and the associated health benefits have had on their psychological and mental wellbeing:

> *I am aware of being able to cope with stress better having lost weight. Things that used to stress me don't so much. Not that I am immune from stress but I am better able to cope with it. I kind of think my cognition is better, I think it's improved. I haven't tested it and I don't know, but I feel smarter. I also feel braver. Yes, having succeeded to lose so much weight it's given me a real confidence in myself.* (T109)

3.7.7. Sleep

More than half of participants surveyed reported improvements in sleep after adopting an LCHF diet (Table 12). Interviews indicated that these improvements often coincided with weight loss and resolution of sleep apnoea. Two participants said that their sleep has become worse since following an LCHF diet (Table 12). Here and in Appendix I, participants describe some changes in sleep experienced since following an LCHF diet:

> [sleep] *started to improve, probably within 3 weeks of starting the lower carbs seriously. And the trigger was, I had to rush off to PE* [Port Elizabeth, a town in the Eastern Cape of South Africa] *and I left my machine behind. The machine that kept me breathing at night. Because I would fall asleep, even in meetings, I would fall asleep. And at night, I would wake up and realise that I had stopped breathing. And it was really very difficult to start breathing again, because I knew what I had to do: in my case, I had to jump out the bed, I had to run towards the cupboard and then I had to try and cough, so I could start the breathing process. But by then you know, you have wet the floor and, you know, your body has just done what it is supposed to do. And then you have got to go and shower. And this happened every night. So, later you become afraid of going to bed, because you know what is going to happen. And this*

machine, then stopped that process… Every single night you had to plug it in and when there was a power failure, you couldn't sleep... But when I got to PE, I suddenly realised that I had left my machine behind and I didn't choke and that would have been a couple of weeks [of sleeping unassisted by the apnoea machine]. *I have kept the machine, just as a souvenir.* (T137)

3.7.8. Skin

Forty-three percent of participants surveyed said that their skin improved after adopting an LCHF diet (Table 12):

… look I always had a good skin, but my skin is getting better. (T131)

One participant listed a worsening in skin condition since following an LCHF diet (Table 12).

3.7.9. Gastrointestinal

A quarter of participants surveyed said that they experienced an improvement in gastrointestinal issues, while another quarter experienced a worsening of gastrointestinal issues since adopting an LCHF diet (Table 12). Participant interviews indicate that improvements in gastrointestinal issues generally encompassed a reduction in gas, bloating and flatulence.

What's that!?… flatulence. [Laughing] *I so seldom do that anymore and I used to do it all day, every day.* (T163)

However, constipation was a common complaint during interviews (illustrated in section Results, Psychology and dietary motivations, 3.6.4 Challenges, subsection *Gastrointestinal complaints*).

3.7.10. Fertility

Three women spoke about a struggle with infertility at some point during their lifetime. None reported being diagnosed with PCOS. Two of these women commenced an LCHF diet after menopause. The one who adopted an LCHF diet before menopause described how she fell pregnant while following an LCHF diet and after losing weight:

I've never actually been tested for polycystic ovarian syndrome. My husband and I have been married since I was 34, all of that weight and various other things meant that we never had the child we wanted. I lost all of the weight in 2014. September of 2015, I went to my GP and said, "okay, I've gone into menopause. I'm 40…" and this and this and this. So, she said well it could be that, or it could be that, or you're pregnant. I said don't be ridiculous, we've been waiting for like 6 years for a child and it's not going to happen… and my daughter is now a month old. (C79)

3.7.11. General health

During interviews, many of the participants enthusiastically described the improvements in general health and overall HRQoL that they've experienced since following an LCHF diet. A common theme that emerged was that participants felt that an LCHF diet helped "fix" or "heal" them, bringing their bad health and associated impaired HRQoL back to "normal" or "natural" and illustrated by the quote below and those in Appendix I.

I then got the benefit and spin of weight loss and all my blood numbers improving and normalising and I'm saying but hang on, my life has normalised. (C13)

Participants often expressed amazement and awe at the health benefits they felt an LCHF diet has brought about in their lives:

So really my health has improved so much. If you knew me then and you know me now, you'd be amazed. (C319)

3.8. Summary of results

This study represents a group of individuals with T2D who independently adopted and implemented their own personalised versions of an LCHF diet. Their LCHF diets consisted of 67 % fat (143.2 ± 67.9 g, mainly SFA from added fat, eggs, meat, poultry, seafood and full cream dairy), 10 % carbohydrate *(*64.0 ± 27.9 g, primarily full cream dairy, nuts and seeds), 20 % protein (96.0 ± 37.4 g, primarily animal origin) and 3% alcohol (primarily wine). Cholesterol intake (616.3 (402.8–804.2) mg) was higher, and fibre (14.7 ± 7.5 g), calcium and folate lower than recommendations. Added sugar (0.5% of energy intake) and processed foods were seldom consumed. Daily energy intake was 1946.3 ± 807.2 kcal (436.7 ± 728.3 kcal. d^{-1} calculated energy deficit). IF (≥ 16 h. d^{-1}) was followed by 61% of the group. This real-world version of an LCHF diet adopted by this group was generally reported as sustainable, linked to reduced hunger and cravings

From pre-LCHF to the time of the study (duration 2.0 ± 1.5 y), reductions were seen in HbA1c concentration (- 2.45 ± 2.59 %) and T2D prevalence (36 % were no longer classified with T2D, based on HbA1c and T2D pharmacotherapy). Twenty-one (75 %) participants experienced a reduction or maintenance of HbA1c concentration, despite reduction or maintenance of T2D pharmacotherapy (or lack thereof), during their time on LCHF (Figure 2). Reductions in body weight (- 16.9 ± 11.7 %) and obesity prevalence (- 50 %) and improvements in overall health and HRQoL were also reported during this time. However, there were challenges, primarily, constipation, initial adaptation and social difficulties related to LCHF. The health improvements and real-world LCHF diet used to achieve them, as well as the associated challenges and perceptions, are discussed below.

4. DISCUSSION

4.1. T2D status

The primary finding of this study was that there was a general trend towards normalisation of HbA1c after adopting LCHF, often while maintaining or decreasing T2D pharmacotherapy. HbA1c reduced by 2.45 ± 2.59 % from pre-LCHF to the time of this study, which is well above the 0.5 % reduction that is thought to be clinically and statistically significant for reducing long-term microvascular complications and mortality [164, 165]. The majority (89 %) of the group met SEMDSA (2017) [13] and ADA [164] glycaemic targets (≤ 7 % HbA1c for most individuals with T2D) to reduce micro- and macro- vascular disease and mortality (with current HbA1c of 6.1 ± 0.7 %). The ADA reports that only 51 - 67 % of T2D patients meet this target on conventional T2D management [166]. Approximately half of the group had normal HbA1c, and HOMA-IR at the time of the study (95 % also had normal a TG/HDL ratio), which is indicative of insulin sensitivity and glycaemic control.

Reduced requirements for T2D pharmacotherapy pre-LCHF to the time of this study, may have attenuated the observed benefit of non-pharmacological factors (including LCHF) on HbA1c. HbA1c changes from pre-LCHF to the time of this study, independent of T2D pharmacotherapy (Figure 2), indicate a reduction or maintenance of HbA1c in 75 % (n 21) of participants; 36 % (n 10) of which achieved T2D remission (defined as non-diabetic HbA1c concentrations (< 6.5 %) achieved without any T2D pharmacotherapy). At the time of the study, three of the remissions had intermediate hyperglycaemia (HbA1c 5.7 – 6.4 %), and the remaining 7 had normal HbA1c. Adoption of LCHF is the most consistent and pronounced common change amongst our participants and is therefore likely a major contributor to the observed improvements HbA1c and T2D status (though it is likely that an LCHF diet's contribution may be mediated by downstream health benefits, such as weight loss, as discussed later).

4.2. Health and perceived health and HRQoL changes after adopting LCHF

From the time of T2D diagnosis to adopting an LCHF diet, participants reported deteriorations in various aspects of health (including general health) and HRQoL, and a subsequent improvement in these after following an LCHF diet. The most consistent and dramatic

reported improvement post-LCHF was in T2D progression and symptoms (Table 12), which is in line with the measurable reductions in HbA1c and T2D pharmacotherapy (Figure 1 Figure 2).

CVD and metabolic disease risk at the time of the study. In addition to meeting glycaemic targets, the ADA recommends keeping BP ≤ 140 mmHg systolic BP (which is associated with comparable CVD risk reductions as seen with BP ≤ 120 mmHg in T2D patients [167, 168]) and serum LDL cholesterol < 2.6 mmol. L^{-1} [166]. Seventy one percent of the group met ADA BP targets, compared to the 51 – 67 % of T2D patients in the United States of America (U.S.A.), nationally, meeting BP targets using conventional management [166]. Serum LDL cholesterol outcomes were similar to those reported by the ADA, with 37 % of the group reaching the target [169]. These comparisons must be seen within the relative contexts of this study's group (which is biased towards T2D-management success) and the general U.S.A. T2D population. As discussed later in this Discussion (section 4.3.1 Energy and macronutrients, subsections *Fats* and *Cholesterol*), the group's high serum HDL cholesterol and low TG concentrations, even in the presence of slightly raised total and LDL cholesterol concentrations, doesn't indicate raised CVD risk. Additionally, all participants had healthy resting HRs. Thus, based on HbA1c and CVD risk markers, the CVD risk for the group as a whole, was low.

Body weight and adiposity. Body measurements (of general obesity: BMI and BF %; and central adiposity: waist circumference and WHR) for over two thirds of the group, as well as means for males, females, and the group as a whole, exceeded recommendations - indicating high adiposity [170] in the majority of the group. Excess adiposity, particularly central depots (indicated by increased waist circumference and WHR) are associated with hypertension, dyslipidaemia, hyperglycaemia, CVD, T2D, and all-cause mortality) [143]. The females fared better than the males for body composition, which was below average in females and below the 10th percentile in males for health and fitness [154].

Despite excess body weight and adiposity in the majority of the group at the time of the study, there was a 100 % improvement in weight status (21.2 ± 19.1 kg) and a more than 50 % reduction in obesity prevalence in the 2-year mean period following LCHF. Many participants explained that they'd struggled with excess weight for most of their lives, and

that they'd only achieved long-term weight loss success on this LCHF diet. It's recommended that overweight and obese individuals should lose at least 5 – 10 % BW to improve their health risk [169]. Magnitude of weight loss is significantly associated with improvements in CVD and metabolic disease risk factors (hyperglycaemia, hypertension, hypertriglyceridemia and low HDL cholesterol) in T2D. So, while any weight loss in overweight or obese T2D patients improves health and reduces CVD risk, body weight losses of >10 % are associated with greater health benefits [171]). Of the 26 overweight or obese participants in this group pre-LCHF, 2 (8 %) lost < 5 %, 8 (31 %) lost 5 – 10 %, and 16 (61 %) lost ≥ 10 % of body weight (Figure 5), indicating a significant reduction in risk and improvement in health after following LCHF [171]. Eighteen (95 %) of the 19 participants in this study, who had been following LCHF for 1 year or longer, lost and maintained > 10% body weight loss, with a mean body weight loss of 16.9 ± 11.7 %. This self-directed and administered LCHF diet-associated weight loss surpassed the weight loss experienced by the hands-on LCHF-app intervention group in the Hallberg et al. (2018) study (– 12 % ($p < 1.0 \times 10^{-16}$) or – 13.8 ± 0.71 kg ($p < 1.09 \times 10^{-16}$) after 1 year) [29]. This indicates that it's possible to experience similar weight loss results without supervision and contact sessions. Both this group and Hallberg's LCHF-app intervention arm surpassed the < 10 % mean weight loss achieved after 1 year of adhering to energy-restricted diets [170]. Interestingly, participants with higher pre-LCHF weights, experienced greater absolute weight loss, as would be expected. However, pre-LCHF weight didn't predict relative (%) weight loss, indicating that percentage weight loss is unrelated to pre-diet body weight in this group.

Systematic reviews and meta-analyses indicate that an LCHF diet is associated with rapid initial weight loss (in the first 3 – 6 months), which surpasses that seen on energy-restricted high carbohydrate low fat dietary interventions. However, this is followed by weight regain over the long-term, which offsets any initial LCHF diet weight loss benefits [10, 11, 30]. This study did not capture data regarding the change in rate of weight loss over the LCHF diet period. However, the fact that 100 % of participants lost weight after adopting an LCHF diet, yet the TDEI in 32 % exceeded TDEE at the time of this study (Figure 7), which could imply that 32 % of the group may have increased their energy intake overtime and been regaining weight at the time of the study. However, this is speculative due to insufficient data and concerns regarding the accuracy of the energy balance theory (i.e. energy intake must equal energy output for weight maintenance), which offers an incomplete explanation of weight changes and status [173]. Indeed, research often indicates discrepancies between

energy balance and expected weight loss [174], indicating that other factors influence energy metabolism and its relationship with body weight. Possible alternate explanations for discrepancies between current energy intake and expected weight loss since adopting LCHF, may include changes in hormonal milieu (females) [175] or increased carbohydrate intake over time.

HRQoL. Participants attributed many HRQoL improvements to their weight loss, which is in line with existing research indicating that weight loss improves HRQoL [26]. Reported improvements included mobility and activities of daily living, pain, sleep (often due to resolved apnoea), mental health (e.g. confidence, improved self-regard, bravery), and fertility. Improvements to the latter aspects of HRQoL after adopting LCHF were not always ascribed to weight loss however, implying that other factors related to LCHF may also play a role in improving HRQoL. Other pervasive reported improvements after adopting LCHF included control over hunger, higher energy levels and exercise capacity and performance, improved mental health (mood (happier, more stable), memory, concentration, cognition, stress-coping ability, alertness, and self-confidence) and skin conditions, and certain gastrointestinal improvements (reduction in gas, bloating and flatulence, likely due to the reduced fermentable carbohydrates ingested when following LCHF). A minority of participants reported not experiencing improvements, or experiencing deteriorations in these aspects. Deleterious Health and HRQoL aspects experienced post-LCHF were worsening of gastrointestinal issues (particularly constipation) (see Discussion section 4.3.1 Energy and macronutrients, subsection *Fibre*) in 25 % of participants, a few participants mentioned concern about high cholesterol (the relevance of which is discussed Discussion section 4.3.1 Energy and macronutrients, subsections *Fats* and *Cholesterol*), one mentioned worsening of gout and another complained of bad breath. Concern exists regarding potential flare-ups of pre-existing gout during the initial adaptation stages of an LCHF diet due to competitive excretion of uric and ketone acids at the level of the kidneys. However, intervention studies show no evidence for raised uric acid concentrations in healthy [176], obese [177] and T2D individuals [178], nor higher incidence of gout attacks [179] or gout incidence [29] in LCHF diet intervention groups with T2D. This is in line with observations that hyperuricaemia is a feature of insulin resistance and MetS, and is associated with fasting insulin, serum LDL cholesterol and TG concentrations, total to HDL cholesterol ratios, and diastolic BP [179].

Clearly, for the vast majority of this group, the number and magnitude of health and HRQoL benefits experienced after adopting LCHF, outweighed the few reported negatives. Improvements in T2D symptoms and pharmacotherapy, as well as weight loss were amongst the greatest measurable and perceived LCHF-associated health benefits. These improvements also had the greatest knock-on benefits to aspects intrinsic to HRQoL and activities of daily living. Pain, impaired mobility, sleeping difficulties and low energy levels pre-LCHF were debilitating and linked to severe functional impairments in some participants. Post-LCHF improvements in these were life-changing. The pervasive theme of health going from bad to a normal or "natural" state, illustrates the overarching feeling in this group.

4.3. LCHF diet composition

4.3.1. Energy and macronutrients

This group's perception that they were following an LCHF diet was in line with an LCHF diet as described in the literature (see Table 6 and Figures 8-9) [2].

TDEI. At the time of the study (after 2.0 ± 1.5 on LCHF)*, ad libitum* energy intake was 28.9 ± 4.7 to 33.1 ± 5.1 kcal. IBW^{-1}. d^{-1} (Table 6), which is within to slightly above the usual 25 – 30 kcal. IBW^{-1}. d^{-1} prescribed in energy restricted diets for promoting weight loss in obesity [180]. The group (males and females alike) achieved a mean energy deficit (calculated difference between TDEE and TDEI) in line with the best clinical practice recommendations for weight loss (- 500 kcal) (Figure 7) [13]. However, the individual energy differences between TDEI and calculated TDEE varied widely, with a range of -1,431 to +1,045 kcal daily. Post-LCHF TDEI was below calculated TDEE in 68% (n 19) of participants. There was a frequent perception amongst participants that their energy intake reduced from pre- to post- LCHF. This perception, together with the 100 % weight loss rate post-LCHF, and TDEI data corroborates that an energy deficit was likely introduced after adopting LCHF. However, the cross-sectional nature of this study's energy intake data prevents drawing conclusions regarding relationships between TDEI and weight. The role of energy balance and other physiological factors' in energy metabolism and distribution, lipogenesis and body weight, is unclear [173]. So, it is impossible to make retrospective projections regarding changes in energy intake over time,

based on body weight.

Protein. Twenty percent of our sample's energy comes from protein, which is within the 10-35 % Acceptable Macronutrient Distribution Range (AMDR) for protein [181]. However, this relative intake is less relevant than the absolute intake, as protein requirements are a function of lean body mass, rather than TDEI [32]. The group's mean daily protein intake was sufficient (1.1 ± 0.5 g · kg BW $^{-1}$ · day $^{-1}$ (Table 6) or 1.3 ± 0.5 – 1.7 ± 0.6 g . kg IBW^{-1} . day^{-1}) and above the recommended dietary allowance (RDA) (0.8 g · kg BW $^{-1}$ · day $^{-1}$) [182]. The RDA for protein is the estimated lowest dietary protein intake level that meets the protein requirement (i.e. the intake required to prevent urinary nitrogen loss) of nearly all (97–98%) healthy individuals [182]; which is insufficient to optimise health [181]. Protein intake above RDA is required to optimise cell signalling, satiety, and glycaemic control, amongst other functions, and is beneficial for prevention and treatment of T2D, obesity, MetS, and CVD [32]. Interestingly, this group's protein intake was below the 1.5 g · kg BW $^{-1}$ · day $^{-1}$ in the Hallberg et al. (2015) LCHF-app intervention in T2D, which found various significant health benefits in the intervention group [29]. Additionally, higher protein intakes (≥ 1.5 g · kg IBW $^{-1}$ · day $^{-1}$) may improve weight and fat loss, maintenance of lean body mass during rapid weight loss, and promote satiety and thermogenesis [183-185]. Evidence indicates that higher protein consumption also improves glycaemic control (insulin sensitivity, HbA1c and 24h and fasting blood glucose concentrations) in T2D [178, 186, 187] (particularly when increased protein takes the place of dietary carbohydrate [188]). Some participants reported deliberately limiting protein intake (to the size of a fist or hand-palm per meal), due to their belief that protein can elevate blood glucose. Some research indicates that protein may indeed increase insulin, glucagon and gluconeogenesis [189, 190]. Conversely though, other studies show that increasing dietary protein, particularly in the context of a hypocaloric diet, may improve fasting and postprandial glucose control and reduce postprandial insulin response (in overweight and T2D) [186, 187]. Therefore, this groups' protein consumption is moderate, and in line with the protein recommendations for promoting weight loss and optimal health.

Ninety percent of protein consumed was complete, and of high biological value; intentionally chosen by participants to be from animal sources (meat, poultry, seafood, organ meats, processed meat, eggs, full-fat dairy). The complement of amino acids contained in complete high biological value protein is essential for health [188], and much easier to attain

in this form than from plant sources. Yet, epidemiological studies have associated increased total and animal protein intake (from sources including processed meat) with T2D prevalence, and higher vegetable protein with lower risk [191-193]. When unprocessed meat is extricated from processed meat, the increased T2D risk is reduced [194, 195]. The relative risk and hazard ratios of eating an additional 85 – 100 g daily serving of unprocessed meat is 1.19 and 1.12 respectively, compared to 1.29 and 1.51 respectively for the smaller 28 - 50 g additional daily portion of processed meat [195]. Though a systematic review and meta-analysis of 12 cohort studies conclude that it's impossible to completely exclude the effects of "residual confounding factors and temporal bias" [194]. Nuts and seeds contributed most of the remainder of the group's protein intake.

In the context of the group's moderate protein consumption, protein's beneficial effects on relevant risk factors (including glycaemic control and body weight) outweigh any potential risks associated with (unprocessed) meat consumption.

Glycaemic Carbohydrate. Total daily carbohydrate consumption was 64.0 ± 27.9 g (10 % of TDEI) (Table 6, Figure 8), which is in line with definitions of an LCHF diet (< 130 g^{-1}. d^{-1}, or < 26 % TDEI), and only slightly higher than a VLCHF KD (20-50 g $^{-1}$. d $^{-1}$ or < 10 % TDI) [2]. LCHF dietary recommendations generally go hand-in-hand with recommendations to eat "real food" (unprocessed foods) [33]. Unsurprisingly then, processed, refined and sugar-laden foods (with the exception of dark chocolate) and beverages were seldom consumed (Table 8). SEMDSA (2017) also recommends avoiding these carbohydrate sources in favour of carbohydrates from whole grains, legumes, low fat milk, vegetables and fruit [13]. However, in contrast to the SEMDSA (2017) guidelines, this group favoured carbohydrates from full cream dairy, nuts and seeds, fruit, dark chocolate, non-starchy and fatty vegetables, over starches and grains and starchy vegetables (Table 9). Added sugar intake was 0.7 % of TDEI, which surpasses SEMDSA recommendations (added sugar < 5 % of TDEI) to improve overall health [13]. Considering the deleterious health effects of processed, refined and sugary foods and beverages [13], at least some of the groups' health improvements are likely attributable to the avoidance of these items, and the focus on 'real food' carbohydrate sources.

Fibre. Daily fibre consumption was 14.7 ± 7.5 g (Table 6), which was similar between the sexes. While this quantity is only slightly below the average 17 g per day intake in Western diet-

eating USA [64], it does not meet the AI of 21 - 25 and 30 - 38 g for women and men respectively [64, 182]. The AI for fibre is based on the median intake associated with lowest CHD risk (and T2D risk as a secondary endpoint) in epidemiological studies [64]. Causation cannot be inferred from these studies, in which fibre covaries with other nutrients that are expected to affect energy homeostasis (e.g. lower fibre diets are often higher in processed foods) [75]. Epidemiological and intervention studies indicates that fibre may have various health benefits [196, 197], many particularly relevant to T2D, including improving satiety [198] and weight status [199], reducing glycaemic impact of carbohydrate-containing foods [131, 200], and cholesterol reduction [201]. However, while effect sizes are often statistically significant, fibre's benefits for body weight, satiety and cholesterol reduction generally lack clinical significance [75, 197, 201]. In the context of this group's reduced HbA1c (Figure 1 - 2), body weight (Figure 3) and hunger (section 3.6.1.1), and healthy HDL and TG concentrations at the time of the study (Table 3), the possible contribution that additional dietary fibre may make to these factors is likely insignificant. Additionally, increasing our group's fibre intake from 15 g. d^{-1} to 30 g . d^{-1}, to meet recommendations would be expected to result in reductions of 0.05 – 0.1 mmol. l^{-1} serum total and LDL cholesterol respectively [201]. Even if serum total and LDL cholesterol concentrations were good predictors of CVD risk [202], this reduction wouldn't be enough to result in normal serum total and LDL cholesterol levels in our group, and thus doesn't represent a clinically significant reduction in serum total and LDL cholesterol concentrations. The single most common LCHF-related adverse effect that participants reported, was constipation. While it is possible that increasing dietary fibre may improve this symptom (increasing stool frequency and softness) [203], it may instead worsen idiopathic constipation [203, 204]. Evidence regarding fibre's benefits for constipation is inconclusive and insufficient to make recommendations [205]. Reports of lower flatulence while following LCHF is likely due to the reduction in fibre and corresponding reduction in fermentation within the colon [203, 206]. More research and improved methodologies for assessing fibre's direct effect on health measures are necessary. The greatest contributor to our group's fibre intake is non-starchy vegetables (Table 8). Considering the group consumed less than 1 portion of vegetables per day, there is room for increasing fibre intake (Table 8). In light of the constipation reports, the possibility of other beneficial health effects associated with fibre and low risk associated with higher fibre intake, practitioners should consider advising T2D patients following LCHF to try increasing fibre from non-starchy vegetables, and alter dietary fibre intake thereafter based on individual response.

Fat The group's total fat intake (143.2 ± 67.9 g, 66% of TDEI) was over double SEMDSA guidelines of < 30 % of TDEI from fat [13]. Therefore, it is hardly surprising that SFA consumption was also well above SEMDSA recommendations [13]. This group consumed 41% of TDEI as SFA: four times SEMDSA's recommendation for SFA (< 10 % TDEI). There's concern that high SFA intake increases CVD morbidity and mortality. However, as reviewed in the introduction (section 1.5 Concerns regarding LCHF diets), the evidence does not support this. An LCHF diet consistently results in superior reductions in TG and elevations in HDL concentrations compared to conventional dietary interventions [10, 11, 207], indicating a protective CVD effect. Contrastingly, replacing dietary SFA with carbohydrate, as seen in conventional low-fat diets, raises TG and atherogenic small dense LDL cholesterol, while lowering HDL cholesterol concentrations – a profile associated with increased CVD risk [208]. This group's serum HDL and TG concentrations (better indicators of CVD risk than serum total and LDL cholesterol [209-211] were within recommended ranges at the time of this study. Other CVD risk factors, including diabetes status (Figure 1) and overweight and obesity (Figure 3 - 4) clearly improved after the adoption of an LCHF diet (and the concurrent increase in dietary SFA). Thus, there is no evidence that the increase in SFA intake increased markers of CVD risk in this group.

Within the context of the group's high total fat consumption, it is interesting that the PUFA intake (11%) was only slightly higher than the < 10% SEMDSA (2012) guideline [28]. Unfortunately, this study did not capture the n-3 and n-6 intake. Considering the central role that the EFAs have in immune system modulation (inflammation in particular) and many other aspects related to health [34], as well as the paucity of EFA-intake data in the LCHF population, future research should capture EFA intake. In line with SEMDSA [13, 28] recommendations, MUFAs provided the remainder of energy from total fat.

Many participants mentioned modifying their LCHF diet (some deliberately, others spontaneously) by introducing or increasing their intake of certain high-fat foods (e.g. those listed in Table 9). Fat was generally believed to help control hunger and increase satiety, accelerate weight loss, and improve certain blood markers (e.g. HDL). Participants who had followed low fat, calorie restricted diets in the past, found them unsustainable, mainly due to hunger and feelings of deprivation.

Cholesterol. Median cholesterol intake was 616.3 (402.8 – 804.2) mg (double the maximum recommendation for health and reduction of CVD risk [79]). This high dietary cholesterol is hardly surprising, considering the group's high intake of cholesterol-containing foods, such as eggs, organ meats and animal products. The recommendation to limit dietary cholesterol to <300mg [79] is based on the assumption that dietary cholesterol raises serum cholesterol concentrations, which in turn is thought to raise CVD risk [88]. While epidemiological evidence does indicate that diets high in cholesterol (which usually co-occur in the same foods with high SFA) are associated with increased serum total, LDL and HDL cholesterol concentrations, this does not translate to increased CVD or CHD risk [212]. Similarly, lowering serum total and LDL cholesterol concentrations using statins [213, 214] or dietary intervention [215] does not reduce adverse CVD event or mortality, indicating that these serum cholesterol concentrations are not related to CVD risk [202]. Despite this group's high dietary cholesterol intake, their mean serum total and LDL cholesterol concentrations were only slightly higher than the recommended ranges, with males' mean serum total cholesterol concentration falling within the normal range (Table 3). Additionally, mean serum HDL cholesterol and TG concentrations (better predictors of CVD risk) than serum total and LDL cholesterol, were in the healthy reference ranges, for the entire sample, and for women and men respectively (Table 3).

Alcohol. Median alcohol consumption was 0.0 (0.0 – 11.5) g or 3% of TDEI. Practically, this equated to half a glass of wine (or one unit of alcohol) per week (according to FFQ results), which is well within recommendations of < 1 unit of alcohol daily for women and < 2 for men [79]. Some participants were happy to include red wine in their LCHF diet, while others deliberately limited or avoided alcohol, particularly beer (Table 10). An RCT, investigating long term daily moderate alcohol intake (150 ml water, white wine or red wine daily) in T2D, combined with a Mediterranean diet, indicates that red wine has unique health benefits: improved serum HDL cholesterol (by 0.05 mmol.L-1; $p < 0.001$), total to HDL cholesterol ratio (by 0.27; $p = 0.039$) and components of MetS (by 0.34; $p = 0.049$) [216]. This groups' reasons for limiting or eliminating alcohol included finding it easier to exclude something completely than moderate intake, and perceptions that alcohol increased hunger and blood glucose concentrations. Indeed, acute high alcohol intake impairs postprandial glucose uptake, promoting hyperglycaemia, and promotes hypoglycaemia in the fasted state [217, 218]. On the other hand, moderate frequent alcohol intake (12-13 g. d^{-1} or 1 u. d^{-1}) actually

inhibits gluconeogenesis [216] and improves insulin sensitivity [219]. Generally, RCTs agree that moderate frequent alcohol intake is beneficial rather than detrimental for blood glucose control [125, 216], improving fasting plasma glucose ($p = 0.015$ [125]). However, a 2-year RCT found that these improvements only occurred in slow ethanol metabolisers [216]. Interestingly, RCTs found no effect of moderate frequent alcohol intake on T2D pharmacotherapy [216] or postprandial glucose [125] in T2D. Moderate alcohol consumption also improves sleep (ability to fall asleep ($p < 0.001$) [125] and sleep quality ($p = 0.040$) [214]) but appears to have no effect on liver function [125, 216], or on BP, adiposity, or HRQoL [216]. However, a few participants in this study reported missing the social side of drinking. Moderate alcohol consumption (approximately ≤ 48 g or 3 units per day), particularly when consumed more frequently (≥ 5 days per week), is also consistently associated with reduced risk of developing T2D in prospective cohort studies [220, 221]. However, a 2015 systematic review of and dose-response meta-analysis of over 1.9 million individuals from 38 observational studies concluded that moderate alcohol consumption's protective effects again T2D risk may be over-estimated and only confined to certain populations (e.g. women and non-Asians) [222]. Overall, contrary to the groups' perceptions, evidence indicates that moderate frequent alcohol consumption is beneficial for T2D prevention and management.

4.3.2. Micronutrients

The reported dietary micronutrient content (Table 7) is underestimated (see 2 METHODS section above and 4.8 Limitations sub-section below). Therefore, although it fell short of the RDI or AI for some micronutrients, this is largely due to exclusion of micronutrient data for various food items (see Appendix J – missing food items list), and may be sufficient in reality, particularly for the micronutrients that fall only slightly short of recommendations (e.g., vitamin B6, thiamin, vitamin D and potassium). Despite this, it is somewhat surprising that the sufficiency of dietary vitamin B6, thiamine and potassium would even be borderline, due to the abundance of these vitamins in foods that are major contributors to this groups' diet (e.g. animal foods like meat, poultry, seafood, organ meats, and eggs; and nuts and seeds). The reported deficiency of dietary vitamin D is likely accurate though, as dietary sources of this vitamin are not plentiful, and it is known to be deficient in most diets [58]. However, sunlight is the most significant source of vitamin D, calling into question the importance of diet to

vitamin D status in those living in the Southern hemisphere and being exposed to sufficient sunlight [59]. It is more likely that those micronutrients that fell more drastically short (around 50 % or less) of recommendations (folate (25%), manganese (35%), calcium (48%) and magnesium (52%)), reflect a genuine insufficiency.

Before proceeding with this micronutrient discussion though, it is important to note that the bioavailability of micronutrients from animal foods, which were staples in this group, is generally far superior compared to plant sources [223]. Additionally, diets (e.g. an LCHF diet) lower in plant foods, particularly grains and legumes, contain fewer micronutrient-binding anti-nutrients known to inhibit micronutrient absorption [223]. Therefore, lower micronutrient requirements may be expected from a diet rich in animal foods and low in plant foods (particularly grains and legumes) [31, 224].

Folate. Recorded folate (25 % of RDI / AI) is a relatively accurate reflection of dietary intake, as the only major contributor to folate intake that was excluded from the analysis was supplementation. Folate is pivotal to mental and cardiovascular health as well as cell division (and cancer prevention) and prevention of neural tube defects in utero. Thus, the recorded substantially-deficient intake is concerning.

High-folate foods include liver (approx. 200 mcg per 90 g), and non-starchy green vegetables (e.g. kale, broccoli, Brussels sprouts, lettuce and avocado), legumes and starches or grains (e.g. fortified cereals, pasta and rice) - the latter with half of liver's folate content (i.e. approx. 100 mcg) [225]. As liver and non-starchy vegetables are LCHF foods, an LCHF diet which includes sufficient quantities of these foods will provide adequate folate, even in the absence of legumes and starches (Appendix J). Individuals following an LCHF diet should be advised to include sufficient quantities of these foods in their diet.

Major clinical signs of folate deficiency are due to megaloblastic anaemia, and include fatigue, weakness, headaches, dizziness, concentration difficulties, heart palpitations and shortness of breath [225]. Healthcare practitioners need to be aware that these symptoms are similar to 'keto flu' and the associated natriuresis of fasting (or low carbohydrate consumption). So, if patients report symptoms of 'keto flu' lasting longer than the first 10 days of LCHF, differential diagnoses should be investigated. The NIH report that other signs of folate deficiency include, "soreness and shallow ulcerations in the tongue and oral mucosa; changes in skin, hair, or fingernail pigmentation; and elevated blood concentrations of

homocysteine" [225]. Of note, none of the clinical signs of folate deficiency appeared to be present in this group (however, researchers weren't specifically looking for them). On the contrary, participants usually reported improvements in symptoms such as fatigue and concentration (Table 12). If there is any indication of folate deficiency (e.g. clinical signs) and/or insufficient folate intake (based on dietary record analysis), healthcare practitioners should test for megaloblastic anaemia and elevated homocysteine, and recommend supplementation and dietary changes accordingly. Future research should investigate the possibility of folate deficiency in this population.

Manganese. Manganese is central to essential processes involved in T2D, including redox reactions (manganese superoxide dismutase), and glucose metabolism (specifically gluconeogenesis) [226]. So, a deficiency in this mineral would be particularly concerning in T2D. High carbohydrate foods, not consumed by this group, such as wheat-based products, rice, oats, and beans are rich in manganese. However, certain foods eaten by this group are also high in manganese, including shellfish, nuts, coconut, spinach. Manganese-rich food sources that were excluded from the analysis included Banting products (e.g. seed crackers LCHF bread), which are usually high in manganese-rich nut and seed flours, almond milk, and black and green teas. So, recorded manganese intake is likely an underestimation. However, the missing foods' (Appendix J) contribution to manganese intake is unlikely to increase intake by the 65 % shortfall required to meet New Zealand and Australian recommendations (used as reference intakes in this thesis) [1]. However, this group does meet the U.S. AI for manganese [182]. So, this group's manganese intake is unlikely to be of concern. Future research should test for manganese depletion or deficiency, using clinical signs of deficiency (scaly dermatitis and hypocholesterolaemia), and/or plasma and serum manganese concentration and leucocyte manganese superoxide dismutase activity [182].

Calcium. Major contributors to calcium intake, missing from the micronutrient analysis (Table 7, Appendix J), were double and full cream yoghurts. On average, participants ate half a cup daily (providing 225 mg calcium), which, when added to the recorded calcium intake (of 502 g, which also mainly came from dairy – see Table 7), brings total daily calcium intake up to 727 g - 69% of RDI/AI. The other missing foods items are not major sources of dietary calcium and are therefore unlikely to contribute much more to calcium intake. The group's median

daily dairy intake was 3 portions (in this work, a dairy portion is defined as half a cup of milk/yoghurt or 30 g cheese), which is half of recommendations [79]. Actual calcium intake underestimation was further compounded by not including calcium from the frequently consumed bone broth (stock, made from boiled bones, with or without meat and vegetables and added salt). For the dietary analysis, reported bone broth intake (diet records) was recorded and analysed as the SAFOODS' item: "Gravy, meat (50 % fat, unthickened". As this food item isn't made from high-calcium bones, it contributed to further underestimation of true calcium intake. Had the calcium content of yoghurt, bone broth and supplements been included in the dietary analysis, many participants' may have met requirements. Therefore, future research into the micronutrient content of an LCHF diet should more accurately capture true micronutrient intake from all sources.

Despite the possibility that actual calcium intake may have been sufficient, it is probably prudent to advise individuals following an LCHF diet on how to ensure that they are meeting calcium requirements. This group could improve calcium intake by doubling their dairy consumption - possibly by increasing cheese rather than milk or yoghurt intake, in order not to concurrently increase carbohydrate consumption too much. Considering the group's low intake of non-starchy vegetables (1 portion daily), folate and fibre, and the high-calcium and fibre content of dark green leafy vegetables, this group should be advised to increase consumption of dark leafy greens. Other LCHF high-calcium content food sources that could be added to this group's diet includes small soft-boned fish (e.g. pilchards, sardines, and tinned salmon).

Magnesium. The exclusion of whole grains (high in magnesium) and starches (moderate in magnesium), could be responsible for the apparent lower magnesium in this LCHF group. Other magnesium-rich foods were eaten by this group and captured in the nutritional analysis, including green leafy vegetables and nuts, as well as intermediate sources such as meat and milk. However, various frequently-consumed magnesium-rich food items (included in Appendix J) were excluded from the analysis, including dark chocolate, full and double cream yoghurt, and bone broth. So recorded magnesium intake represents a substantial underestimation of actual intake, and the apparent dietary deficiency of this mineral needs to be repeated and confirmed or dispelled by future studies. Clinicians and researchers should look out for signs of magnesium deficiency such as symptomatic hypocalcaemia and muscle

cramps and spasms [182], as blood magnesium concentrations are not good indicators of intracellular magnesium [227].

In summary, the nutrients of concern generally come from bone broth, liver and brightly coloured (particularly dark green leafy) non-starchy vegetables, nuts, and dairy. Therefore, individuals following LCHF should ensure sufficient intake of these foods. They should continue to include (and possibly increase) dairy, as well as nuts and seeds, and increase bone broth, liver and green leafy vegetable intake. An increased vegetable consumption simultaneously increases intake of fibre and other health-promoting nutrients (e.g. phytonutrients and antioxidants), which may offer additional health benefits. Most of this group independently adopted LCHF, without the help of a dietician or doctor. So, there is probably room for improvement and there is a need for professional support to ensure individuals meet their nutrient requirements for optimal health. Healthcare providers have a responsibility to support and guide their patients following an LCHF diet.

4.4. LCHF dietary patterns and behaviours

The reason for the observed reduction in TDEI is likely multifaceted, involving reduction in hunger, IF, and portion and craving control.

Hunger. The vast majority of the group experienced a distinct and pronounced reduction in hunger after adopting LCHF. Participants felt that decreased hunger and improved appetite control was intrinsically linked to higher fat and lower carbohydrate intake. Many reported that this reduction in hunger resulted in the observed reduction in energy consumption. The literature corroborates that an LCHF diet's hunger-suppressing effects result in spontaneous reductions in energy intake [38]. The mechanism behind an LCHF diet's appetite suppressant effect isn't fully understood. However, modulation of appetite control hormones (e.g. ghrelin, leptin and cholecystokinin), a possible direct appetite-suppressant action of ketones [38], and reduction of circulating (anabolic) insulin concentrations [173] may contribute.

Generally, this group didn't explicitly control portions or energy intake, corroborating that they were indeed following an *ad libitum* diet. They allowed themselves to eat to satiety, which some reported to be less than they ate pre-LCHF. However, a few participants

transiently tried recording dietary intake using diet-tracking apps (usually when acquainting themselves with the diet), and many explained that they were aware of their portions and tried not to overeat (often in response to a weight loss plateau).

IF. Most participants (61 %) ate 2 or fewer times per day (≥ 16 h. d^{-1} fast), usually skipping breakfast completely (Table 5). This implies that reduction in frequency of eating may be a major contributor to the calorie deficit seen 68% of participants (Figure 7). This is in line with research indicating that T2D patients following 16 – 20 hour IF preferentially skip breakfast and start eating mid-afternoon, and spontaneously reduce overall energy intake [53]. Mostly IF occurred unintentionally, due to reduced hunger on an LCHF diet combined with following satiety cues. The hunger-suppressing effects of LCHF diets could result in a spontaneous reduction in eating frequency, resulting in short-duration IF (e.g. 16 – 18 hours). However, some participants introduced IF intentionally, in response to a weight loss plateau or to reduce TDEI. This is in line with the popular combination of IF with LCHF diets to "turbocharge" weight loss [228].

It's conceivable that the frequent intentional combining of LCHF with IF may be due to the similar metabolic states induced by these dietary interventions, namely, reduced circulating insulin concentrations and improved insulin sensitivity, reliance on fat and ketones as primary fuels over glucose, and (in the case of VLCHF diets or KDs) ketosis [29, 44]. It may be easier to combine IF with intervening periods of LCHF rather than conventional higher-carbohydrate eating, as the latter would require constant metabolic cycling between two very different states and associated pathways (i.e. those required for preferentially metabolising fat vs carbohydrate). This difference in fuel availability between IF and non-fasted days may be particularly problematic in T2D, especially when hypoglycaemic agents (medication or insulin) are involved. Indeed, research indicates that close monitoring and medication adjustments are required in patients on hypoglycaemic agents during IF days, due to increased risk of hypoglycaemia when fasting [51, 54]. The vastly different hypoglycaemic-medication requirements for period of IF compared to intervening periods of conventional high carbohydrate dietary intake may increase hypoglycaemia during IF, which speculatively could be offset by following an LCHF diet on non-fasted days.

Cravings and food addiction. Dietary carbohydrate content of participants' VLCHF diet was

often intentionally reduced over time (by exclusion of high carbohydrate foods, e.g. fruit, dairy, nuts seeds and starchy vegetables), in order to promote weight loss and/or improve blood glucose control. Sugar (including the occasional sweet treat) and sweet taste (including from sugar substitutes) were frequently perceived to illicit cravings and reduce control over dietary intake, and were generally limited or excluded completely. In fact, many participants experienced their relationship with certain foods (usually those high in sugar, with a sweet taste, or carbohydrate-rich) as addictive. Some participants in this sample described themselves as 'extreme' or 'all-or-nothing' personalities (frequently also known as addictive personalities, associated with binging, craving and overeating) [229]). These participants felt it necessary to completely eliminate added dietary sugar and sweet taste in order to prevent a 'relapse', and reduce sweet cravings. Cravings and 'food addiction' appears to be present in around 15 - 20 % of overweight and obese individuals [230, 231]. Food addiction or addictive food behaviours are linked to foods high sugar or both fat and sugar (particularly processed foods) [230]. These foods are excluded from an LCHF diet, but generally included in limited quantities in conventional diets. Therefore, in individuals prone to food addiction, LCHF may improve adherence compared to conventional diets.

In keeping with the perception of sugar as addictive (see Results 3.6.1.3 Food Addiction), qualitative data relating to food addiction is described in the context of the WHO's international classification of diseases 10th revision (ICD-10) classification for substance dependence or addiction [161]. Perceived manifestations of food addiction ("dependence syndrome"), in-line with WHO classification included, 1.) a strong desire or compulsion to take a substance; 2.) an impaired capacity to control substance-taking behaviour (onset, termination, level of use); 3.) tolerance with increasing use; and 4.) continued use despite obvious deleterious health effects. However, other manifestations of addiction or substance dependence that were not mentioned in interviews were: 5.) signs of physiological withdrawal (participants did mention feelings of psychological withdrawal, however physiological withdrawal symptoms were not present, possibly because sugar manifests as a more psychological than physiological addiction), and 6.) preoccupation with or abandonment of alternative pursuits due to substance use (possibly due to the ubiquitous availability and acceptability of sugar-sweetened and carbohydrate rich foods, (making it easy to attain these foods and socially acceptable to consume them). Thus, four out of six dependence syndrome manifestations were reported by participants for sugary and sweet food, implying that sugar may have addictive potential for susceptible individuals. In order to

classify dependence syndrome "three or more of the manifestations [listed above] should have occurred together for at least one month or if persisting for periods of less than one month then they have occurred together repeatedly within a twelve month period" [161]. This study was not designed to classify dependence syndrome, and as "sugar addiction" was an emerging theme, rather than a predetermined research question, future research study designs need to allow for better investigation and classification of perceived sugar addiction, possibly using the standardised Yale Food Addiction Scale [232, 233].

4.5. Adherence and sustainability

The success of a diet depends largely on adherence [234]. Results from this study show that long term (mean 2 years, range 6 months – 6 years) adherence to an LCHF diet is possible at least in certain individuals with T2D. Also, adherence to LCHF can be effective for glycaemic and T2D control (Figures 1 - 2), weight loss (Figures 3 - 5), a reduction in T2D symptoms, and improved parameters related to HRQoL (Table 12). In this group, observed health benefits persisted with long term (> 6 months) adherence.

Perceived sustainability. From the interviews it is clear that the vast majority of participants find LCHF to be sustainable and generally beneficial to various aspects of HRQoL, with a few minor exceptions. However, this study's eligibility criteria ensured that only those who had followed LCHF for 6 months or longer were included. Therefore, there is a selection bias in favour of participants who find LCHF sustainable and experience desirable results; and against those for whom LCHF may not have been sustainable or effective. The strategies employed by this group could be responsible for the difference between the diet "working" for some and being unsustainable for others. Strategies cited for improving adherence included IF, increasing fat and reducing carbohydrate intake, and not cheating (i.e. being strict, so as not to illicit cravings). Recommending these tips for improving adherence may assist in advising other patients regarding strategies to improve LCHF adherence and therefore success and associated health benefits.

Most participants said that the main and overarching reason for LCHF's perceived sustainability and adherence is the diet's associated reduction in hunger. Reduced hunger persisted despite the 500 kcal mean energy deficit. Participants often contrasted this to the persistent hunger they experience when previously following calorie-restricted low-fat

diets. They felt that hunger made these diets difficult to adhere to and unsustainable and resulted in them gaining back any weight lost. The peer-reviewed evidence supports this sentiment: feeding studies demonstrate that changes in energy balance result in antagonistic biological adaptations (decreased energy expenditure and increased hunger), which thwart weight loss attempts [235].

Therefore, an LCHF diet may be an effective alternative weight loss and T2D management-strategy for overweight and obese T2D patients who find other diets hard to adhere to, or struggle with weight regain and hunger when following calorie-restricted low-fat diets.

4.6. Psychology: Dietary motivations and challenges

The reasons participants adopted and adhered to an LCHF diet could be utilised by healthcare practitioners, community workers, health advocates and government to motivate others to do the same to improve their health. The main reason for adopting an LCHF diet was T2D glycaemic control (and associated prevention of complications and comorbidities, and reduction of pharmacotherapy), followed by weight loss (Table 11). Fourteen percent were convinced to do it by books or medical professionals, so these conduits can be used and expanded upon to motivate change for health.

An understanding of the potential pitfalls and challenges associated with LCHF is necessary to pre-empt difficulties, enhance sustainability and adherence, and maximise health benefits and weight loss effects. This knowledge would be useful for health practitioners prescribing an LCHF diet for the management of T2D and for people who put themselves onto an LCHF diet independently and could be pivotal to the success or failure of the diet.

The group cited difficulty eating an LCHF diet within social contexts (e.g. dinner parties) due to judgement and ostracism. Patients adopting this diet should be prepared for these difficulties and given coping strategies. Another common challenge was inconvenience, inaccessibility or difficulty finding appropriate foods in some environments (e.g. restaurants, travelling, quick snacks). Education regarding nutrient content and appropriateness of certain foods for LCHF can help patients make better choices with more ease. Planning ahead (e.g. appropriate food stops or restaurants) or packing travel snacks can also help patients

overcome these hurdles.

Interestingly, it was assumed by participants, researchers, and media that expense would be a concern, but in practice it wasn't (so long as pre-prepared Banting foods didn't form a major part of the diet). Expense may be mitigated by a reduction in TDEI and food intake on an LCHF diet. Additionally, many participants emphasised the trivialness of food-expense as a concern (particularly when offset against the perceived benefit of weight loss and associated health benefits, which often included reduction in healthcare and medication costs) by expressing humour ("*It's just expensive, that is all… to buy a new wardrobe [Laughing]")*. However, this study's participants were middle to high socio-economic status, who were likely already eating more expensive, higher quality and lower carbohydrate protein sources (e.g. meat), rather than cheaper, lower quality, higher carbohydrate protein sources (e.g. legumes). Had our participants been poorer, it is possible that the replacement of high carbohydrate food staples (e.g. maize and legumes) with lower carbohydrate options may have incurred additional expense, which could be a major hurdle to adopting an LCHF diet. Additional research is required to determine feasibility, challenges and sustainability of an LCHF diet in lower socioeconomic status groups.

Initial adjustments to an LCHF diet were also challenging for some. These included breaking long-held eating habits and beliefs (e.g. regarding SFA and CVD) and psychologically adjusting to a drastic change in diet. Coping strategies should be given to assist with these. The initial physiological adaptation ('keto flu') was also challenging for some. 'Keto flu' is the popular name for the symptoms associated with the natriuresis of fasting which may accompany the early adaptation to (V)LCHF [162]. The natriuresis of fasting results from low insulin associated urinary losses of sodium and water (resulting in dehydration) and, failing replenishment of the former, consequent magnesium and potassium losses [163]. Symptoms may include headache, fatigue, weakness, light-headedness, muscle aches or cramps, and constipation [162]. Practical management strategies need to be given to combat 'keto flu' symptoms and the most commonly cited adverse effect, constipation (which can also result from dehydration [236]). These strategies should include replenishment of fluid, sodium and, based on clinical symptoms, magnesium and potassium (when indicated).

4.7. Participant characteristics

Our sample was older, potentially due to T2D incidence increasing with age. Also, younger people with T2D likely have more work- and family-related time-constraints which makes participating in research more challenging. As many of our participants were retired, the monthly income reflected in Table 1 may often reflect pension. In addition to being older, our group was educated, predominantly Caucasian, and had researched the diet themselves, and followed it without support or approval from their healthcare practitioners. This may be reflective of the type of person with T2D who adopts an LCHF diet. However, it is also possible that recruitment avenues (e.g. LCHF-Facebook groups, university-associated mailing lists and doctors' offices in more affluent areas of Cape Town) selected this type of participant. Researchers anticipated a greater interest from and recruitment of males than females, as anecdotes on social media purport that diets, particularly an LCHF diet, are more effective in males. However, our sample fortuitously, but unintentionally, included equal numbers of males and females, possibly reflective of the greater LCHF diet-following population's sex-distribution or an aspect of the study which made females more likely to enrol (e.g. perhaps females are more willing to be interviewed). The sample size was too small to determine whether either males or females experienced significantly greater changes in any health-parameter measured after adopting an LCHF diet, but our results give no indication of significant differences between the sexes.

4.8. Limitations

Sampling. The purpose of this study was to describe the diet and health history of T2D patients who had adapted to a long-term LCHF diet. As such, recruitment would likely have favoured participants for whom the diet worked, and would likely exclude participants who had difficulty sustaining the diet, or did not experience notable positive effects. As such, reports regarding positive and negative aspects of the diet are likely over and under representations respectively.

Convenient sampling within the more affluent areas in Cape Town, South Africa, and over social media and SSISA and ESSM mailing lists, may have biased recruitment of a more educated, higher socioeconomic sample. This represents a population that would have been more likely to receive and be receptive to information about an LCHF diet and had the means to research it and adopt it. Future research is required to investigate the feasibility of an LCHF

diet in lower socioeconomic groups.

Small sample size makes getting statistically significant associations and showing differences between females and males difficult.

Dietary analysis. SAFOODS is the only database that is validated for research in South Africa. However, this study discovered that it is not suitable for a population following an LCHF diet. Multiple food items eaten by our participants, were missing from SAFOODS. The missing nutritional information had to be compiled by the researchers from alternative non-validated sources, reducing the validity and reliability of the data, and thus the accuracy of the final diet composition results. For the majority of foods, these sources also exclude micronutrient data, which resulted in underestimation of micronutrient intake (with the exception of sodium). Unfortunately, SAFOODS did not include analysis of selenium intake, so the sufficiency of this micronutrient is not known. Selenium intake is of interest, as strict KDs used for refractory epilepsy are often insufficient in this mineral. An error during data collection meant that nutritional supplementation information and sodium intake from added table salt was not captured, which would have compounded the underestimation in micronutrient status in this study.

As with all studies that assess diet, further dietary intake estimation inaccuracies were introduced by participants' and researchers' likely erroneous estimations of food quantity or mass. In the many instances when the MRC food photo manual didn't specify the food-mass-conversion for normal household measurements of various foods, the researchers (TM and KL) estimated food mass from participants' dietary records using Google (non-validated sources). This compounded the errors that were already intrinsic in participants' (non-weighted) 3-day food records' food quantity estimations. Future studies should consider using weighted food records, for longer periods, to reduce these estimation errors. Additionally, there is concern over the potential overestimation of carbohydrate content of meals listed in SAFOODS, for our LCHF population (e.g. SAFOODS may assume that sauces are thickened with wheat or maize flour, when those following LCHF may rather use cream or coconut cream as a 'pre-thickened' sauce-base).

Other limitations of dietary capture, included lack of capturing non-macro- and micro-nutrient dietary factors, which may have an effect on physiology associated with insulin resistance and T2D supplements, including fermented foods and beverages or probiotics, and

non-nutritive sweeteners. However, apart from not capturing information regarding supplement use, this research team attempted to improve diet capture accuracy by including three diet analysis methods, and capturing diet related information qualitatively as well.

Other. The outcomes at the (current) time of the study were compared to self-reported weight, and blood parameters (e.g. Hba1c) from different laboratories, for diagnosis and pre-LCHF timepoints. This may have affected the accuracy of the reported changes between the three timepoints.

This study did not investigate the effects of confounding factors, such as exercise and other lifestyle and diet changes (e.g. fasting and food quality), on health outcomes. Instead any potential confounding factors that changed while following an LCHF diet were observed and noted (i.e. other factors that may have played a role in participants' health besides an LCHF diet). One of the purposes of this study was to identify potential confounders to inform what to control for in future studies.

While triglycerides are relatively predictive of CVD risk, LDL and HDL particle numbers and densities (rather than cholesterol concentrations), as well as other biomarkers and imaging techniques (specifically the CAC scan) would have offered better risk prediction for atherosclerotic CVD [94]. Therefore, these should be investigated in future LCHF studies; particularly in light of the persisting concerns amongst many over potential CVD risk associated with a high fat diet.

4.8. Summary and conclusion

To the researchers' knowledge this is the first study to describe the composition of and perceptions around an *ad libitum*, real-world, self -selected and -administered version of a long-term (2.1 ± 1.5 y) LCHF diet in T2D, and investigate coexisting health parameters, including T2D status, health and HRQoL.

The study group's diet was adopted and adapted based on self-identified actual life requirements, challenges, and changes in health and T2D status, to create, what to them was a workable and sustainable real-world version of an LCHF diet, consisting of: 67% fat (143.2 ± 67.9 g, mainly SFA from added fat, eggs, meat, poultry, seafood and full cream dairy), 10% carbohydrate *(*64.0 ± 27.9 g, primarily full cream dairy, nuts and seeds), 20 % protein (96.0 ±

37.4 g, primarily animal origin) and 3% alcohol (primarily wine). Cholesterol intake (616.3 (402.8–804.2) mg) was higher, and fibre (14.7 ± 7.5 g), calcium and folate lower than recommendations. Added sugar (0.5% of energy intake) and processed foods were seldom consumed. Daily energy intake was 1946.3 ± 807.2 kcal. (436.7 ± 728.3 kcal. d^{-1} calculated energy deficit). IF (≥ 16 h. d^{-1}) was followed by 61% of the group.

This study's results support mounting evidence indicating that an LCHF diet can be sustainable in individuals with T2D and may be linked to improvements in glycaemic control, medication reduction, body weight, hunger and dietary control, health (including CVD and metabolic risk reductions) and HRQoL. This study did not lend support to most common concerns regarding increased health risks associated with an LCHF diet (see INTRODUCTION section 1.5 Concerns regarding LCHF diets). Interestingly, health improvements were seen despite lack of guidance, support or supervision from healthcare practitioners. However, this group did experience some dietary challenges (including initial adaptation, constipation and psychological and social aspects) and possible nutritional deficiencies (folate and calcium), which could be improved and overcome with appropriate guidance and support by health practitioners (see section 4. DISCUSSION).

When interpreting these findings, it is important to keep in mind that, while an LCHF diet was the central and common component of participants' intervention, they may have implemented other healthy lifestyle choices concurrently (e.g. more exercise). Some observed health benefits may have been a direct result of an LCHF diet (e.g. reduced HbA1c, pharmacotherapy and weight), however these and other health benefits may also have been downstream secondary health benefits mediated by the primary health improvements (e.g. LCHF-associated weight loss in turn improved pain, sleep and mobility). It is not possible to distinguish between the direct effect of an LCHF diet and the downstream health benefits (e.g. weight loss) as contributing factors to the health improvements described in this study. Additionally, the relative contribution of these factors to health improvements likely varies amongst individuals. However, it is clear that an LCHF diet, with or without IF, reduced hunger and cravings and gave this group a sense of control over eating, which likely facilitated and played an integral role in the observed weight loss, and downstream weight loss-associated health benefits, including glycaemic control and improvements in HRQoL.

This study and literature review supports the case by Feinman et al. (2015) for "use of low-carbohydrate diets as the first approach to treating type 2 diabetes" [2], and indicates

that this intervention can be self-managed, sustainable and effective for at least some individuals with T2D. This study also adds real-world context to the implementation of an LCHF diet and proposes areas for LCHF dietary support and improvement, in order to further maximise associated health improvements, minimise any risk and overcome challenges.

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APPENDICES

APPENDIX A: RECRUITMENT ADVERTISEMENT – POSTER

APPENDIX B: RECRUITMENT ADVERTISMEST – EMAIL

Do you have **Type 2 Diabetes**? Do you follow **a low carbohydrate high fat (Banting) diet**? If so, we would like you to volunteer to participate in a short study entitled:

'Descriptive study of Type 2 Diabetes Mellitus patients following a low carbohydrate high fat diet.'

This study will be conducted by researchers from the University of Cape Town. The main aim of the study is to find out more about how you follow a low carbohydrate high fat diet and how your diabetes has progressed, so that we can design future studies.

To be eligible, you must:

- have followed **a low carbohydrate high fat diet** for at least the **past 6 months,**
- be 18 years of age or older and
- have been diagnosed with type 2 diabetes

You will NOT be able to participate if you:

- have significantly changed your diet in the past 6 months

If you volunteer to participate, you will be required to:

- Complete an online health, diabetes, diet and exercise questionnaire.
- Visit the Sports Science Institute* on 1 occasion (90 min) for basic health screening.
- Provide a blood sample for glucose testing.

**The Sports Science Institute of South Africa is located in Boundary Road, Newlands, Western Cape. Willing participants who live too far away can still participate via online surveys and telephonic interviews, without coming in to the Sports Science Institute.*

What are the risks and benefits for partaking in the trial?

By taking part you will help us design future studies in this field that will potentially improve the health and well-being of patients with type 2 diabetes. You will also receive a report of all results including dietary analysis, body measurements, blood pressure, and glucose, insulin and HbA1C blood concentrations. The risks and discomfort associated with taking part are small.

For more information and to apply, please contact any of the following researchers at the Division of Exercise Science and Sports Medicine, University of Cape Town:

Chris Webster	Cell: 083 400 7206	Email: chris.webster@uct.ac.za
Tamzyn Murphy Campbell	Cell: 084 313 4103	Email: tamzynmurphycampbell@gmail.com
Salih Solomon	Cell: 072 129 9841	Email: salihsolomon@gmail.com

APPENDIX C: RECRUITMENT ADVERTISEMENT - MEDIA RELEASE

Sports Science Institute of South Africa
For immediate release
16 January 2017

PARTICIPANTS REQUIRED FOR UCT STUDY INVESTIGATING TYPE II DIABETICS (OR EX DIABETICS) FOLLOWING A LOW CARBOHYDRATE HIGH FAT DIET

Do you have or have you ever had **Type 2 Diabetes**? Do you follow **a low carbohydrate high fat (Banting) diet**? If so, we would like you to volunteer to participate in a short descriptive study.

The Division of Exercise Science and Sports Medicine at UCT based at the Sports Science Institute of SA (SSISA), is recruiting volunteers for a research study. The main aim of the study is to find out more about how you follow a low carbohydrate high fat diet and how your diabetes has responded and progressed. This will assist us in designing future studies.

If you volunteer to participate, you will be required to: complete an online health, diabetes, diet and exercise questionnaire; visit the Sports Science Institute on 1 occasion (90 min) for basic health screening; provide a blood sample for glucose testing; and keep a food diary for 3 days.

To be eligible, you must:

- have followed a low carbohydrate high fat diet for at least the **past 6 months** and made no major changes to the way you eat during this period
- be 18 years of age or older
- have been diagnosed with type 2 diabetes at some time in your life

What are the benefits and risks for partaking in the trial?

- By taking part, you will help us design future studies in this field that will potentially improve the health and well-being of patients with type 2 diabetes.
- You will receive a report of all results including dietary analysis, body measurements, blood pressure, and glucose, insulin and HbA1C blood concentrations.
- The risks and discomfort associated with taking part are small.

To apply or for more information, please contact Chris Webster before 24 February 2017.

Tel: 083 400 7206 **Email:** chris.webster@uct.ac.za

APPENDIX D: GENERAL HEALTH QUESTIONNAIRE

This questionnaire will ask for details about your type 2 diabetes and will focus around three main periods:

1) The time around your initial type 2 diabetes diagnosis.

2) The time around when you started following a LCHF diet.

3) Your current diabetes status.

The questionnaire goes into detail around your use of chronic medications for the management of diabetes and other related conditions. In the event that you do not know exact dates, try to give as accurate an answer as possible.

The basic structure of the questionnaire is as follows:

Demographics and socioeconomic status Family history and your health history Details about the time when you were diagnosed with diabetes Details about the time when you started following a LCHF diet Details of your diabetic medication use Details of your non-diabetic medication use Current exercise Medical professionals and future study

It should take 30 min to 1 hour to complete. You are able to save the questionnaire and resume from where you left off by clicking the 'Resume Later' button.

Do not hesitate to contact a researcher if you experience problems with the questionnaire.

Section A: code

Participant code and eligibilty criteria.

A1. Enter the code provided to you by the researchers.

A2. Have you previously been diagnosed with type 2 diabetes by a doctor?

Yes ☐

No ☐

A3. Have you been following a low carbohydrate high fat (LCHF) diet for at least the past 6 months?

Yes ☐

No ☐

A4. Are you 18 years of age or older?

Yes ☐

No ☐

A5. You are not eligible for this study due to your type 2 diabetes status.

Please contact a researcher if you feel a mistake has been made.

End survey ☐

A6. You are not eligible for this study due to your diet.

Please contact a researcher if you feel a mistake has been made.

End survey ☐

A7. You are not eligible for this study due to your age.

Please contact a researcher if you feel a mistake has been made.

End survey ☐

Section B: personal

This section contains questions relating to your demographics and socioeconomic status status.

B1. Date of birth:

B2. What is your height?

Answer in centimetres (cm)

B3. What is your current weight?

Answer in kilograms (kg)

B4. Biological sex:

Male ☐

Female ☐

Intersex ☐

B5. **Ethnicity:**

Black ☐
White ☐
Mixed ☐
Indian ☐
Other ☐

Other

B6. **Highest level of education:**

Primary school ☐
High school ☐
Tertiary undergraduate ☐
Tertiary postgraduate ☐
Other ☐

Other

B7. **Usual monthly income:**

R 0 - R 9 999 ☐
R 10 000 - R 24 999 ☐
R 25 000 or more ☐

B8. **How many people currently live in your household?**

B9. **How many rooms in total are there in your household?**

Count an open plan area as the number of functional rooms.

Section C: family

This section cotains questions about your family history.

C1. **Has your <u>mother or sister</u> had heart disease (e.g. heart attack / angina / heart failure)?**

	Yes	No	Unsure
Before the age of 65?	☐	☐	☐

	Yes	No	Unsure
After the age of 65?	☐	☐	☐

C2. **Has your father or brother had heart disease (e.g. heart attack / angina / heart failure)?**

	Yes	No	Unsure
Before the age of 55?	☐	☐	☐
After the age of 55?	☐	☐	☐

C3. **Have any of your close family members had any of the following conditions?**

Close family members refer to siblings, parents and grandparents.

	No family history	Unsure	Father and/or mother	Brother(s) and/or sister(s)	Grandparent(s)	A combination of the previous options
Overweight or obesity	☐	☐	☐	☐	☐	☐
Pre-diabetes	☐	☐	☐	☐	☐	☐
Type 2 diabetes	☐	☐	☐	☐	☐	☐
High blood pressure	☐	☐	☐	☐	☐	☐
High cholesterol	☐	☐	☐	☐	☐	☐

Section D: health

This section contains questions on your health status, smoking history and alcohol intake.

D1. **Do you have, or have you ever had, any of the following?**

Every row requires an answer.

	Currently have	Previously had	No	Unsure
High blood pressure	☐	☐	☐	☐
Abdominal obesity	☐	☐	☐	☐
Dyslipidemia (high cholesterol and/or triglycerides)	☐	☐	☐	☐
Neuropathy (nerve damage leading to loss of sensation / numbness / tingling	☐	☐	☐	☐
Non-alcoholic fatty liver disease or other liver condition	☐	☐	☐	☐
Kidney conditions	☐	☐	☐	☐
Intermittent claudication (cramping pain in the legs, often caused by exercise)	☐	☐	☐	☐

	Currently have	Previously had	No	Unsure
Retinopathy (blurred or fluctuating vision)	☐	☐	☐	☐
Stroke or transient ischemic attack (TIA, temporary blockage in brain blood vessel)	☐	☐	☐	☐
Angina (chest pain)	☐	☐	☐	☐
Anemia (red blood cell deficiency)	☐	☐	☐	☐
Thyroid dysfunction	☐	☐	☐	☐
Vitamin D deficiency	☐	☐	☐	☐

D2. **Do you have or have you ever had any of the following?**

Every row requires an answer.

	Currently have	Previously had	No	Unsure
Lung disease	☐	☐	☐	☐
Pulmonary tuberculosis (TB)	☐	☐	☐	☐
Asthma	☐	☐	☐	☐
Epilepsy	☐	☐	☐	☐
Celiac disease	☐	☐	☐	☐
Crohn's disease	☐	☐	☐	☐
Depression / Anxiety	☐	☐	☐	☐
Cancer	☐	☐	☐	☐
Glandular fever	☐	☐	☐	☐
Gout	☐	☐	☐	☐
Drug (illigal or over the counter) / alcohol addiction	☐	☐	☐	☐

D3. **Do you have any medical conditions that are not covered by the above questions?**

Yes ☐

No ☐

D4. **Please specify which medical condition(s).**

1 ☐☐☐☐

2 ☐

3 ☐

4 ☐

5 ☐

D5. **Do you currently smoke cigarettes?**

Yes ☐

No ☐

D6. **How long have you been smoking (years)?**

☐

D7. **How many cigarettes do you smoke per day?**

1 - 4 per day ☐

5 - 9 per day ☐

10 - 19 per day ☐

20 - 29 per day ☐

30 or more per day ☐

D8. **Have you smoked in the past?**

Yes ☐

No ☐

D9. **When did you stop smoking?**

☐

D10. **For how many years did you previously smoke (years)?**

☐

D11. **How many cigarettes did you smoke per day?**

1 - 4 per day ☐

5 - 9 per day ☐

10 - 19 per day ☐

20 - 29 per day ☐

30 or more per day ☐

D12. Thinking back over the past 6 months, on average how many standard alcohol units per week did you usually drink?

Note: 1 standard unit is equal to 200 ml of beer, 100 ml of wine or 25 ml of spirits. In other words: 2/3 of a can of beer; a small glass of wine; and a shot of spirits are 1 unit each.

- ☐ 0 units
- ☐ 1 - 2 units per week
- ☐ 3 - 6 units per week
- ☐ 7 - 10 units per week
- ☐ 11 - 14 units per week
- ☐ 15 or more units per week

Section E: women

E1. Are you currently pregnant?

- ☐ Yes
- ☐ No
- ☐ Unsure

E2. Have you had a hysterectomy?

- ☐ Yes
- ☐ No

E3. Do you take hormones (**HRT** or birth control in the form of the pill, a patch, injection or hormone-releasing intrauterine device)?

- ☐ Yes
- ☐ No
- ☐ Unsure

E4. Do you have regular periods?

- ☐ Yes
- ☐ No
- ☐ Unsure

E5. **When was your last period?**

- 3 months ago ☐
- 6 months ago ☐
- 1 year ago ☐
- More than one year ago ☐

Section F: diagnosis

This section contains questions which relate to your diabetes diagnosis.

F1. **When were you diagnosed with diabetes?**

F2. **Did you follow a specific diet prior to your diabetes diagnosis?**

- Yes ☐
- No ☐
- Unsure ☐

F3. **What diet did you follow prior to your diagnosis?**

F4. **What was your weight at the time of your diagnosis?**

Answer in kilograms (kg)

F5. **At the time of your diagnosis, how often did you exercise per week?**

- 0 days per week ☐
- 1 - 2 days per week ☐
- 3 - 4 days per week ☐
- 5 or more days per week ☐

F6. **What types of exercise were you doing around the time when you were diagnosed?**

- Walking ☐
- Jogging / running ☐
- Cycling ☐
- Swimming ☐
- Strength training ☐
- Yoga / pilates ☐

Cardio activities at the gym ☐

Sports (i.e. soccer / hockey etc.) ☐

Other ☐

Other

F7. **How long were your exercise sessions, on average, around the time when you were diagnosed?**

Answer in minutes.

F8. **Which best describes the intensity of your average exercise session around the time when you were diagnosed?**

Light (very easy breathing / able to maintain a conversation) ☐

Moderate (heavier breathing but able to speak / building up a sweat) ☐

Hard (very heavy breathing and unable to speak) ☐

F9. **Was your fasting blood glucose measured as part of your diagnosis?**

Yes ☐

No ☐

Unsure ☐

F10. **What was your fasting blood glucose concentration at the time of diagnosis?**

Answer in mmol/l.

To convert mg/dl to mmol/l, divide your glucose value by 18.

F11. **Was your HbA1c measured as part of your diagnosis?**

Yes ☐

No ☐

Unsure ☐

F12. What was your HbA1c at the time of diagnosis?

Answer in % (DCCT).

You can covert IFCC (mmol/mol) to DCCT (%) at the following url: http://www.diabetes.co.uk/hba1c-units-converter.html

F13. Was an oral glucose tolerance test performed as part of your diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

F14. Was insulin measured as part of your diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

Section G: lchf

This section contains questions which relate to your diabetes around the time when you started eating a low carbohydrate, high fat diet.

G1. When did you start eating a LCHF diet?

G2. Did you follow a specific diet prior to starting your LCHF diet?

- Yes ☐
- No ☐
- Unsure ☐

G3. What diet did you follow prior to starting your LCHF diet?

G4. What best describes the progression of your diabetes from the time of diagnosis to the time when you started eating a LCHF diet?

- Drastic deterioration and worsening of symptoms ☐
- Some deterioration and worsening of symptoms ☐
- No change ☐
- Some improvement and reduction in symptoms ☐
- Drastic improvement and reduction in symptoms ☐

G5. **Did you have your blood glucose or HbA1c checked prior to starting your LCHF diet?**

- glucose ☐
- HbA1c ☐
- both ☐
- no ☐

G6. **When was the closet time before starting your LCHF diet that you did have your blood glucose and/or HbA1c measured?**

G7. **What was your blood glucose (mmol/l)?**

To convert mg/dl to mmol/l, divide by 18.

G8. **What was your HbA1c at the the time when you started following a LCHF diet?**

Answer in % (DCCT).

You can covert IFCC (mmol/mol) to DCCT (%) at the following url: http://www.diabetes.co.uk/hba1c-units-converter.html

G9. **What is the main reason that you started eating a LCHF diet?**

G10. **What was your weight at the time when you started following a LCHF diet?**

G11. **What best describes the progression of your diabetes since you started eating a LCHF diet?**

- Drastic deterioration and worsening of symptoms ☐
- Some deterioration and worsening of symptoms ☐
- No change ☐
- Some improvement and reduction in symptoms ☐
- Drastic improvement and reduction in symptoms ☐

G12. **Since you've been following a LCHF diet, have you experienced changes in any of the following?**

Mark 'not applicable' if you are not affected.

	Not applicable	Drastic worsening	Some worsening	No change	Some improvement	Drastic improvement
Allergies	☐	☐	☐	☐	☐	☐
Asthma	☐	☐	☐	☐	☐	☐
Eyesight	☐	☐	☐	☐	☐	☐
Skin conditions	☐	☐	☐	☐	☐	☐
Gastrointestinal issues (e.g. irritable bowel syndrome / constipation)	☐	☐	☐	☐	☐	☐
Post nasal drip	☐	☐	☐	☐	☐	☐
Concentration	☐	☐	☐	☐	☐	☐
Sleep	☐	☐	☐	☐	☐	☐
Pre-menstrual syndrome (PMS)	☐	☐	☐	☐	☐	☐
Other	☐	☐	☐	☐	☐	☐

G13. **What is 'other' referring to in the above question?**

Section H: biguanides

The next series of questions will involve details of all the diabetic medications that you currently, or have previously, taken.

H1. **Have you ever taken biguanides?**

Examples of medications in this class include metformin / glucophage.

- ☐ Yes
- ☐ No
- ☐ Unsure

H2. **What was the name of your biguanide medication?**

H3. **When did you first start taking this medication?**

H4. Were you taking this biguanide medication at the time when you started following a lchf diet?

Yes ☐
No ☐

H5. Did you ever stop taking this medication?

Yes ☐
No ☐

H6. When did you stop taking this medication?

H7. Did you ever restart taking this medication?

Yes ☐
No ☐

H8. When did you restart taking this medication?

H9. Did you re-stop taking this medication?

Yes ☐
No ☐

H10. When did you stop taking this medication?

H11. Have you since restarted taking this medication again?

Yes ☐
No ☐

H12. When did you restart this medication?

H13. Are you currently taking this medication?

Yes ☐
No ☐

H14. What is your current dose (mg daily)?

H15. What was your maximum dose of this drug (mg daily)?

Section I: insulin

Diabetic medications continued...

I1. Have you ever taken insulin?

- Yes ☐
- No ☐
- Unsure ☐

I2. When did you first start taking insulin?

I3. Were you taking insulin at the time when you started following a lchf diet?

- Yes ☐
- No ☐

I4. Did you ever stop taking this medication?

- Yes ☐
- No ☐

I5. When did you stop taking this medication?

I6. Did you ever restart taking this medication?

- Yes ☐
- No ☐

I7. When did you restart taking this medication?

I8. Did you re-stop taking this medication?

- Yes ☐
- No ☐

I9. When did you stop taking this medication?

I10. Have you since restarted taking this medication again?

- Yes ☐
- No ☐

I11. When did you restart taking this medication?

I12. Are you currently taking this medication?

Yes ☐

No ☐

I13. What is your current dose (units per day)?

I14. What was your maximum dose of this drug (units per day)?

Section J: sulphonylureas

Diabetic medications continued...

J1.

Have you ever taken sulphonylureas?

Examples of medications in this class include chlorpropamide, glibenclamide, gliclazide, glimepiride andglipizide.

Yes ☐

No ☐

Unsure ☐

J2. What was the name of your sulphonylureas medication?

J3. When did you first start taking this medication?

J4. Were you taking this sulphonylurea medication at the time when you started following a lchf diet?

Yes ☐

No ☐

J5. Did you ever stop taking this medication?

Yes ☐

No ☐

J6. When did you stop taking this medication?

J7. Did you ever restart taking this medication?

Yes ☐

No ☐

J8. When did you restart taking this medication?

J9. Did you re-stop taking this medication?

Yes ☐

No ☐

J10. When did you stop taking this medication?

J11. Have you since restarted taking this medication again?

Yes ☐

No ☐

J12. Are you currently taking this medication?

Yes ☐

No ☐

J13. What is your current dose (mg daily)?

J14. What was your maximum dose of this drug (mg daily)?

Section K: othermed

All diabetic medications that you have ever taken must be entered into this questionnaire.

If you take, or took, any diabetic medications that you have not captured in the previous 3 questions, enter them here.

You can enter medications from any class of diabetic drugs here.

K1. Apart from the medications mentioned in the previous questions:

Have you ever taken any other medications for the <u>treatment of your diabetes</u>?

Yes ☐

No ☐

Unsure ☐

K2. How many other medications have you taken for the treatment of your diabetes?

1 ☐
2 ☐
3 ☐
4 ☐
5 ☐

K3. What is the name of other medication 1

K4. When did you first start taking this medication?

K5. Were you taking this medication at the time when you started following a lchf diet?

Yes ☐
No ☐

K6. Did you ever stop taking this medication?

Yes ☐
No ☐

K7. When did you stop taking this medication?

K8. Did you ever restart taking this medication?

Yes ☐
No ☐

K9. When did you restart taking this medication?

K10. Did you re-stop taking this medication?

Yes ☐
No ☐

K11. When did you stop taking this medication?

K12. Have you since restarted taking this medication again?

Yes ☐
No ☐

K13. Are you currently taking this medication?

Yes ☐

No ☐

Section L: othermed2

Diabetic medications continued...

L1. What is the name of other medication 2

L2. When did you first start taking this medication?

L3. Were you taking this medication at the time when you started following a lchf diet?

Yes ☐

No ☐

L4. Did you ever stop taking this medication?

Yes ☐

No ☐

L5. When did you stop taking this medication?

L6. Did you ever restart taking this medication?

Yes ☐

No ☐

L7. When did you restart taking this medication?

L8. Did you re-stop taking this medication?

Yes ☐

No ☐

L9. When did you stop taking this medication?

L10. Have you since restarted taking this medication again?

Yes ☐

No ☐

L11. Are you currently taking this medication?

Yes ☐

No ☐

Section M: othermed3

Diabetic medications continued...

M1. What is the name of other medication 3

M2. When did you first start taking this medication?

M3. Were you taking this medication at the time when you started following a lchf diet?

Yes ☐

No ☐

M4. Did you ever stop taking this medication?

Yes ☐

No ☐

M5. When did you stop taking this medication?

M6. Did you ever restart taking this medication?

Yes ☐

No ☐

M7. When did you restart taking this medication?

M8. Did you re-stop taking this medication?

Yes ☐

No ☐

M9. When did you stop taking this medication?

M10. Have you since restarted taking this medication again?

Yes ☐

No ☐

M11. Are you currently taking this medication?

Yes ☐

No ☐

Section N: othermed4

Diabetic medications continued...

N1. What is the name of other medication 4

N2. When did you first start taking this medication?

N3. Were you taking this medication at the time when you started following a lchf diet?

Yes ☐

No ☐

N4. Did you ever stop taking this medication?

Yes ☐

No ☐

N5. When did you stop taking this medication?

N6. Did you ever restart taking this medication?

Yes ☐

No ☐

N7. When did you restart taking this medication?

N8. Did you re-stop taking this medication?

Yes ☐

No ☐

N9. When did you stop taking this medication?

N10. Have you since restarted taking this medication again?

Yes ☐

No ☐

N11. Are you currently taking this medication?

Yes ☐

No ☐

Section O: othermed5

Diabetic medications continued...

O1. What is the name of other medication 5

O2. When did you first start taking this medication?

O3. Were you taking this medication at the time when you started following a lchf diet?

Yes ☐

No ☐

O4. Did you ever stop taking this medication?

Yes ☐

No ☐

O5. When did you stop taking this medication?

O6. Did you ever restart taking this medication?

Yes ☐

No ☐

O7. When did you restart taking this medication?

O8. Did you re-stop taking this medication?

Yes ☐

No ☐

O9. When did you stop taking this medication?

O10. Have you since restarted taking this medication again?

Yes ☐

No ☐

O11. Are you currently taking this medication?

Yes ☐

No ☐

Section P: chronicmeds

This section deals with chronic (long-term) medications taken for conditions other than diabetes.

P1. Have you ever taken any of the following chronic medications, or medications for any of the following conditions?

Chronic medication use means that the medication was taken over the long term.

In this case, any medication that you took continuously for a minimum of 3 months can be considered chronic.

Aspirin ☐

Blood pressure ☐

Heart disease / cardiac conditions ☐

Lipids (cholesterol / triglycerides) ☐

Allergies (excluding seasonal use) ☐

Asthma (excluding carrying a pump for occasional attacks) ☐

Post-nasal drip ☐

Lung or respiratory tract conditions ☐

Gastrointestinal conditions (stomach / abdominal pain) ☐

Skin conditions (such as psoriasis) ☐

Autoimmune diseases (such as rheumatoid arthritis / Hashimoto's thyroiditis) ☐

Muscle or joint pain (such as osteoarthritis) ☐

Depression / anxiety ☐

Sexual dysfunction or infertility ☐

Other ☐

None ☐

P2. When did you start taking aspirin?

P3. Were you taking aspirin at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P4. Were you taking aspirin at the time when you started following a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P5. Are you currently taking aspirin?

Yes ☐
No ☐
Unsure ☐

P6. When did you stop taking aspirin?

P7. What is/was the name of your blood pressure medication?

Note: if you have taken more than one blood pressure medication then just fill out the first one here.

P8. When did you start taking this blood pressure medication?

P9. Were you taking this blood pressure medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P10. Were you taking this blood pressure medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P11. Are you currently taking this blood pressure medication?

Yes ☐

No ☐

P12. When did you stop taking this blood pressure medication?

P13. Have you ever taken any other chronic (long term) medications for the management of blood pressure?

Yes ☐

No ☐

Unsure ☐

P14. What is/was the name of your blood pressure medication?

Note: only fill out the second one you have taken here.

P15. When did you start taking this blood pressure medication?

P16. Were you taking this blood pressure medication at the time of your diabetes diagnosis?

Yes ☐

No ☐

Unsure ☐

P17. Were you taking this blood pressure medication at the time when you started eating a LCHF diet?

Yes ☐

No ☐

Unsure ☐

P18. Are you currently taking this blood pressure medication?

Yes ☐

No ☐

P19. When did you stop taking this blood pressure medication?

P20. Have you ever taken any other chronic (long term) medications for the management of blood pressure?

Yes ☐
No ☐
Unsure ☐

P21. What is/was the name of your blood pressure medication?

Note: If you have taken more than three blood pressure medications then please fill out the third one here. Any additional blood pressure medications can be filled out in the 'other' section.

P22. When did you start taking this blood pressure medication?

P23. Were you taking this blood pressure medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P24. Were you taking this blood pressure medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P25. Are you currently taking this blood pressure medication?

Yes ☐
No ☐

P26. When did you stop taking this blood pressure medication?

P27. What is/was the name of your heart/cardiac medication?

Note: If you have taken more than one heart/cardiac medication then only fill out the first one here.

P28. When did you start taking this heart/cardiac medication?

P29. Were you taking this heart/cardiac medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P30. Were you taking this heart/cardiac medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P31. Are you currently taking this heart/cardiac medication?

Yes ☐
No ☐

P32. When did you stop taking this heart/cardiac medication?

P33. Have you ever taken any other chronic (long term) medications for the management of heart disease / cardiac conditions?

Yes ☐
No ☐
Unsure ☐

P34. What is/was the name of your heart/cardiac medication?

Note: only fill out the second one you have taken here.

P35. When did you start taking this heart/cardiac medication?

P36. Were you taking this heart/cardiac medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P37. Were you taking this heart/cardiac medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P38. Are you currently taking this heart/cardiac medication?

Yes ☐
No ☐

P39. When did you stop taking this heart/cardiac medication?

P40. Have you ever taken any other chronic (long term) medications for the management of heart disease / cardiac conditions?

Yes ☐
No ☐
Unsure ☐

P41. What is/was the name of your heart/cardiac medication?

Note: If you have taken more than three blood pressure medications then please fill out the third one here. Any additional blood pressure medications can be filled out in the 'other' section.

P42. When did you start taking this heart/cardiac medication?

P43. Were you taking this heart/cardiac medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P44. Were you taking this heart/cardiac medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P45. Are you currently taking this heart/cardiac medication?

Yes ☐
No ☐

P46. When did you stop taking this heart/cardiac medication?

P47. What is/was the name of your lipid/cholesterol medication?

Note: if you have taken more than one lipid medication then just fill out the first one here.

P48. When did you start taking this lipid/cholesterol medication?

P49. Were you taking this lipid/cholesterol medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P50. Were you taking this lipid/cholesterol medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P51. Are you currently taking this lipid/cholesterol medication?

Yes ☐
No ☐

P52. When did you stop taking this lipid/cholesterol medication?

P53. Have you ever taken any other chronic (long term) medications for the management lipids (cholesterol and/or triglycerides)?

Yes ☐
No ☐
Unsure ☐

P54. What is/was the name of your lipid/cholesterol medication?

Note: If you have taken more than two lipid medications then please fill out the second one here. Any additional lipid medications can be filled out in the 'other' section.

P55. When did you start taking this lipid/cholesterol medication?

P56. Were you taking this lipid/cholesterol medication at the time of your diabetes diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

P57. Were you taking this lipid/cholesterol medication at the time when you started eating a LCHF diet?

- Yes ☐
- No ☐
- Unsure ☐

P58. Are you currently taking this lipid/cholesterol medication?

- Yes ☐
- No ☐

P59. When did you stop taking this lipid/cholesterol medication?

P60. What is/was the name of your allergies medication?

If you have taken more than one chronic medication for the management of allergies, fill out one here and fill out the additional one(s) under 'other'.

P61. When did you start taking this allergies medication?

P62. Were you taking this allergies medication at the time of your diabetes diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

P63. Were you taking this allergies medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P64. Are you currently taking this allergies medication?

Yes ☐
No ☐

P65. When did you stop taking this allergies medication?

P66. What is/was the name of your asthma medication?

If you have taken more than one chronic medication for the management of asthma, fill out one here and fill out the additional one(s) under 'other'.

P67. When did you start taking this asthma medication?

P68. Were you taking this asthma medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P69. Were you taking this heart/cardiac medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P70. Are you currently taking this asthma medication?

Yes ☐
No ☐

P71. When did you stop taking this asthma medication?

P72. What is/was the name of your post-nasal drip medication?

If you have taken more than one chronic medication for the management of post-nasal drip, fill out one here and fill out the additional one(s) under 'other'.

P73. When did you start taking this post-nasal drip medication?

P74. Were you taking this blood pressure medication at the time of your diabetes diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

P75. Were you taking this post-nasal drip medication at the time when you started eating a LCHF diet?

- Yes ☐
- No ☐
- Unsure ☐

P76. Are you currently taking this post-nasal drip medication?

- Yes ☐
- No ☐

P77. When did you stop taking this post-nasal drip medication?

P78. What is/was the name of your lung/respiratory tract medication?

If you have taken more than one chronic medication for the management of these conditions, fill out one here and fill out the additional one(s) under 'other'.

P79. When did you start taking this lung/respiratory tract medication?

P80. Were you taking this lung/respiratory tract medication at the time of your diabetes diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

P81. Were you taking this lung/respiratory tract medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P82. Are you currently taking this lung/respiratory tract medication?

Yes ☐
No ☐

P83. When did you stop taking this lung/respiratory tract medication?

P84. What is/was the name of your gastrointestinal medication?

If you have taken more than one chronic medication for the management gastrointestinal conditions, fill out one here and fill out the additional one(s) under 'other'.

P85. When did you start taking this gastrointestinal medication?

P86. Were you taking this gastrointestinal medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P87. Were you taking this gastrointestinal medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P88. Are you currently taking this gastrointestinal medication?

Yes ☐
No ☐

P89. When did you stop taking this gastrointestinal medication?

P90. What is/was the name of your skin condition medication?

If you have taken more than one chronic medication for the management of skin conditions, fill out one here and fill out the additional one(s) under 'other'.

P91. When did you start taking this skin condition medication?

P92. Were you taking this skin condition medication at the time of your diabetes diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

P93. Were you taking this skin condition medication at the time when you started eating a LCHF diet?

- Yes ☐
- No ☐
- Unsure ☐

P94. Are you currently taking this skin condition medication?

- Yes ☐
- No ☐

P95. When did you stop taking this skin condition medication?

P96. What is/was the name of your autoimmune disease medication?

If you have taken more than one chronic medication for the management of autoimmune conditions, fill out one here and fill out the additional one(s) under 'other'.

P97. When did you start taking this autoimmune condition medication?

P98. Were you taking this autoimmune condition medication at the time of your diabetes diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

P99. Were you taking this autoimmune condition medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P100. Are you currently taking this autoimmune condition medication?

Yes ☐
No ☐

P101. When did you stop taking this autoimmune condition medication?

P102. What is/was the name of your joint and/or muscle pain medication?

If you have taken more than one chronic medication for the management of joint and/or muscle pain, fill out one here and fill out the additional one(s) under 'other'.

P103. When did you start taking this joint and/or muscle pain medication?

P104. Were you taking this joint and/or muscle pain medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P105. Were you taking this joint and/or muscle pain medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P106. Are you currently taking this joint and/or muscle pain medication?

Yes ☐
No ☐

P107. When did you stop taking this joint and/or muscle pain medication?

P108. What is/was the name of your depression/anxiety medication?

If you have taken more than one chronic medication for the management of depression/anxiety, fill out one here and fill out the additional one(s) under 'other'.

P109. When did you start taking this depression/anxiety medication?

P110. Were you taking this depression/anxiety medication at the time of your diabetes diagnosis?

- Yes ☐
- No ☐
- Unsure ☐

P111. Were you taking this depression/anxiety medication at the time when you started eating a LCHF diet?

- Yes ☐
- No ☐
- Unsure ☐

P112. Are you currently taking this depression/anxiety medication?

- Yes ☐
- No ☐

P113. When did you stop taking this depression/anxiety medication?

P114. What is/was the name of your sexual dysfunction or infertility medication?

If you have taken more than one chronic medication for the management of sexual dysfunction or infertility, fill out one here and fill out the additional one(s) under 'other'.

P115. When did you start taking this sexual dysfunction or infertility medication?

P116. Were you taking this sexual dysfunction or infertility medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P117. Were you taking this sexual dysfunction / infertility medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P118. Are you currently taking this sexual dysfunction / infertility medication?

Yes ☐
No ☐

P119. When did you stop taking this sexual dysfunction or infertility medication?

P120. Are there any other chronic medications that you take, or have taken, that you have not yet captured in this questionnaire?

Click 'yes' to report another medication.

Yes ☐
No ☐
Unsure ☐

P121. What is/was the name of this medication?

P122. What condition is/was treated with this medication?

P123. When did you start taking this medication?

P124. **Were you taking this medication <u>at the time of your diabetes diagnosis?</u>**

Yes ☐
No ☐
Unsure ☐

P125. **Were you taking this medication at the time <u>when you started eating a LCHF diet?</u>**

Yes ☐
No ☐
Unsure ☐

P126. **Are you <u>currently</u> taking this medication?**

Yes ☐
No ☐

P127. **When did you stop taking this medication?**

[ ]

P128. **Do you, or have you previously, taken any other chronic (long-term) medications that you have not yet entered into this questionnaire?**

Yes ☐
No ☐
Unsure ☐

P129. **What is/was the name of this medication?**

[ ]

P130. **What condition is/was treated with this medication?**

[ ]

P131. **When did you start taking this medication?**

[ ]

P132. **Were you taking this medication <u>at the time of your diabetes diagnosis?</u>**

Yes ☐
No ☐
Unsure ☐

P133. Were you taking this medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P134. Are you currently taking this medication?

Yes ☐
No ☐

P135. When did you stop taking this medication?

P136. Do you, or have you previously, taken any other chronic (long-term) medications that you have not yet entered into this questionnaire?

Yes ☐
No ☐
Unsure ☐

P137. What is/was the name of this medication?

P138. What condition is/was treated with this medication?

P139. When did you start taking this medication?

P140. Were you taking this medication at the time of your diabetes diagnosis?

Yes ☐
No ☐
Unsure ☐

P141. Were you taking this medication at the time when you started eating a LCHF diet?

Yes ☐
No ☐
Unsure ☐

P142. Are you currently taking this medication?

Yes ☐
No ☐

P143. When did you stop taking this medication?

P144. Do you, or have you previously, taken any other chronic (long-term) medications that you have not yet entered into this questionnaire?

- Yes ☐
- No ☐
- Unsure ☐

P145. What is/was the name of this medication?

P146. What condition is/was treated with this medication?

P147. When did you start taking this medication?

P148. Were you taking this medication <u>at the time of your diabetes diagnosis?</u>

- Yes ☐
- No ☐
- Unsure ☐

P149. Were you taking this medication at the time <u>when you started eating a LCHF diet?</u>

- Yes ☐
- No ☐
- Unsure ☐

P150. Are you <u>currently</u> taking this medication?

- Yes ☐
- No ☐

P151. When did you stop taking this medication?

P152. Do you, or have you previously, taken any other chronic (long-term) medications that you have not yet entered into this questionnaire?

- Yes ☐
- No ☐
- Unsure ☐

P153. What is/was the name of this medication?

P154. What condition is/was treated with this medication?

P155. When did you start taking this medication?

P156. Were you taking this medication <u>at the time of your diabetes diagnosis?</u>

- Yes ☐
- No ☐
- Unsure ☐

P157. Were you taking this medication at the time <u>when you started eating a LCHF diet?</u>

- Yes ☐
- No ☐
- Unsure ☐

P158. Are you <u>currently</u> taking this medication?

- Yes ☐
- No ☐

P159. When did you stop taking this medication?

Section Q: currentfitness

The following questions related to the amount of exercise that you do <u>currently.</u>

Q1. How physically active are you at work?

- Low (sit for most of the day) ☐
- Moderate (mostly sit but some light walking) ☐
- High (walk / stand for most of the day) ☐
- Very high (work involves a high level of physical labour) ☐

Q2. Do you routinely walk / run / cycle as a form of transport?

- Yes ☐
- No ☐

Q3. **Do you currently exercise?**

- [ ] Yes
- [ ] No

Q4. **What is the main reason that you do not currently exercise?**

Q5. **How often do you <u>currently</u> exercise per week?**

- [ ] 1 - 2 days per week
- [ ] 3 - 4 days per week
- [ ] 5 or more days per week

Q6. **What type of exercise do you do currently?**

- [ ] Walking
- [ ] Jogging / running
- [ ] Cycling
- [ ] Swimming
- [ ] Strength training
- [ ] Yoga / pilates
- [ ] Cardio activities at the gym
- [ ] Sports (i.e. soccer / hockey etc.)
- [ ] Other

Other

Q7. **How long are your <u>current</u> exercise sessions on average?**

Answer in minutes.

Q8. **Which best describes the intensity of your <u>current</u> average exericise session?**

- [ ] Light (very easy breathing / able to maintain a conversation)
- [ ] Moderate (heavier breathing but able to speak / building up a sweat)
- [ ] Hard (very heavy breathing and unable to speak)

Q9. **How would you categorize your current fitness level?**

- ☐ Very unfit
- ☐ Unfit
- ☐ Moderately fit
- ☐ Fit
- ☐ Very fit

Q10. **Did the way that you used to exercise (type/time/intensity) <u>at the time just prior to starting a LCHF diet</u> differ from how you currently exercise?**

- ☐ Yes
- ☐ No

Q11. **How often did you exercise <u>at the time just prior to starting a LCHF diet?</u>**

Answer in session per week.

- ☐ 0 days per week (do not exercise)
- ☐ 1 - 2 days per week
- ☐ 3 - 4 days per week
- ☐ 5 or more days per week

Q12. **What type of exercise did you do <u>at the time just prior to starting your LCHF diet?</u>**

- ☐ Walking
- ☐ Jogging / running
- ☐ Cycling
- ☐ Swimming
- ☐ Strength training
- ☐ Yoga / pilates
- ☐ Cardio activities at the gym
- ☐ Sports (i.e. soccer / hockey etc.)
- ☐ Other

Other

Q13. How long were your exercise sessions, on average, at the time just prior to starting your LCHF diet?

Answer in minutes.

Q14. Which best describes the intensity of your average exericise sessions at the time just prior to starting your LCHF diet?

- ☐ Light (very easy breathing / able to maintain a conversation)
- ☐ Moderate (heavier breathing but able to speak / building up a sweat)
- ☐ Hard (very heavy breathing and unable to speak)

Section R: dietweighthistory

The following questions relate to your doctor and dietitian use.

R1. Are you currently seeing a doctor and/or dietitian to help manage your diabetes?

- ☐ No
- ☐ Doctor
- ☐ Dietitian
- ☐ Both a doctor and a dietitian

R2. Was a LCHF diet initially prescribed for you by a doctor and/or dietitian?

- ☐ No
- ☐ Doctor
- ☐ Dietitian
- ☐ Both a doctor and dietitian

R3. Is your decision to follow a LCHF diet supported by your doctors / dietian?

- ☐ No
- ☐ Doctor
- ☐ Dietitian
- ☐ Both a doctor and a dietitian

R4. Did a dietitian initially help you develop and follow your LCHF diet?

- ☐ Yes
- ☐ No

Section S: future

Involvement in future research.

S1. **Would you consider being involved in more detailed future studies investigating LCHF diets in diabetics?**

- Yes ☐
- No ☐

S2. **Would you consider stopping all medication for a period of 1 month prior to being tested in a research trial?**

This would only happen if your doctor and our medical team deemed it safe for you to do so and you would be carefully monitored by our medical staff during this time.

- Yes ☐
- No ☐

S3. **Would you consider participating in an intervention where you are required to eat a 'best practice' mixed diet which contains carbohydrate for a period of 8 weeks?**

- Yes ☐
- I would consider a 2 - 4 week intervention ☐
- No ☐

APPENDIX E: FOOD FREQUENCY QUESTIONNAIRE (FFQ)

You will be presented with a list of foods and asked to select how often you have eaten those foods on average during the past 6 months.

NOTE:

You are answering how often you have eaten the portion size (in brackets) of a food in past 6 months.

You are answering how often you have eaten that portion size on average. For example: if you eat a portion 7 times a week, that works out at 1 portion per day on average (even if you ate all 7 portions on the same day).

An answer is required for each item.

Be sure to include the foods that you have eaten during training and competition.

Section A: Food

A1. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Regular breakfast cereals / granola e.g. All Bran (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Low carb cereals / granola (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Porridge / oats (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
FutureLife (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Added sugar to cereals / oats (1 teaspoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A2. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
White bread (1 slice / half roll)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Brown bread (1 slice / half roll)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Whole wheat bread (1 slice / half roll)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rye bread (1 slice / half roll)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Low carb bread (1 slice / half roll)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Regular crackers / savoury biscuits (3 crackers)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Seed crackers (3 crackers)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A3. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Pasta / noodles (1 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rice (1 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Pap / mieliepap (1 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Heba pap (1 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Millet (1 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Cous cous (1 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Quinoa (1 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A4. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Potatoes (1 medium or 1/2 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Starchy veg - sweet potato / butternut / carrots / corn (1/2 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Non-starchy veg - broccoli / spinach / cabbage / salad greens etc. (1/2 cup cooked or 1 cup raw)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Olives (small hand full: 10 small / 5 large)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Avocado (1/2 fruit)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Banana / grapes (1 small banana / 12 grapes)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Berries (1 cup)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fresh fruit excluding the above - e.g. apples etc (1 tennis ball sized / 2 golf ball sized fruit)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Dried fruit (3 pieces)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Jam (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A5. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Fatty red meat - beef / lamb / pork etc (1 palm-sized portion)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lean red meat - trimmed of fat (1 palm-sized portion)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fatty poultry - with skin (1 palm-sized portion)	☐	☐	☐	☐	☐	☐	☐	☐	☐

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Lean poultry - chicken breast / skin removed (1 palm-sized portion)	☐	☐	☐	☐	☐	☐	☐	☐	☐
How often was the poultry crumbed?	☐	☐	☐	☐	☐	☐	☐	☐	☐
Bacon / sausages (3 rashers / 1 sausage)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Organ meats (e.g. liver, kidneys) (1/2 cup)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fish (1 palm-sized portion)	☐	☐	☐	☐	☐	☐	☐	☐	☐
How often was fish breaded / battered?	☐	☐	☐	☐	☐	☐	☐	☐	☐
Shellfish (1 palm-sized portion)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Eggs (1 egg)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Biltong (hand full)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lunch meats - salami / sandwich ham / chicken roll (1 slice)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Protein shake/ powder (e.g. whey, casein, soy proteins) (1 serving)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A6. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Full cream milk - including in tea / coffee (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Low fat milk - including in tea / coffee (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fat free milk - including in tea / coffee (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Cream / sour cream (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Full fat unsweetened yoghurt (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Fat free / low fat plain yoghurt (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Flavoured / sweetened low fat yoghurt (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hard cheese (1 matchbox sized portion)	☐	☐	☐	☐	☐	☐	☐	☐	☐

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Soft cheese (2 tablespoons)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Cottage cheese (2 tablespoons)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A7. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Butter (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Margarine (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Ghee (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lard (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Medium Chain Triglyceride (MCT) oil (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Coconut oil (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Coconut milk / cream (1/4 tin or 5 tablespoons)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Coconut flakes / desiccated coconut (1 small hand full)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Olive oil (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sunflower oil (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Canola oil (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Other oil (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Salad dressing (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Mayonnaise (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A8. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Baked beans (1/2 cup)	☐	☐	☐	☐	☐	☐	☐	☐	☐

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Soya beans (1/2 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Other beans - kidney etc (1/2 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Soya milk (1 cup / 250ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Soya meat substitutes (palm-sized)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Peas (1/2 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lentils (1/2 cup cooked)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Peanuts (1 small handful / 30 g)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Cashews (1 small handful / 30 g)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Macadamia nuts / pecans / walnuts (1 small handful / 30 g)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Other nuts - e.g. almonds (1 small handful / 30 g)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sweetened nut butter (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Unsweetened nut butter (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Seeds (small handful / 30 g)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A9. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Fruit juice (1 glass / 300 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Regular soft drink (1 can / 340 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Diet soft drink (1 can / 340 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Energy drink (1 can / 375 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sports energy drink (1 bottle / 500 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Regular beer (1 can / 340 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Lite beer (1 can / 340 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Wine (1 glass / 200 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Alcoholic coolers - e.g. Savannah (bottle / 300 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Hot chocolate / Milo (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Added sugar to hot beverages (1 teaspoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A10. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

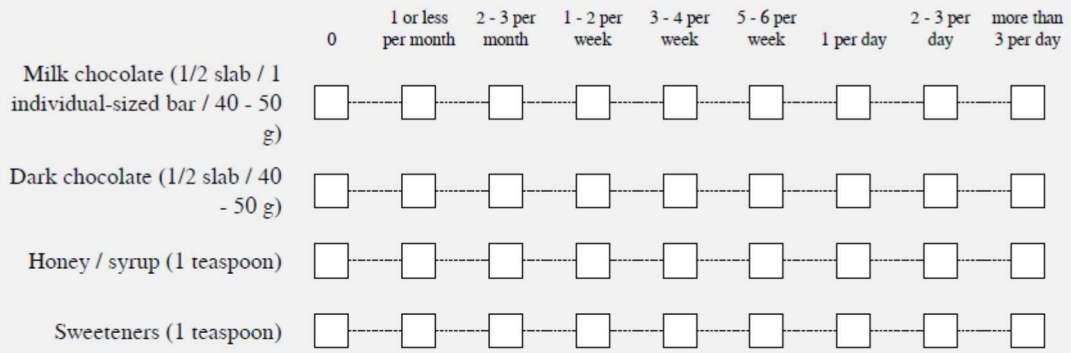
	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Regular pizza (1 medium 30 cm pizza)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Low carb pizza (1 medium 30 cm pizza)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Burger (1 medium)	☐	☐	☐	☐	☐	☐	☐	☐	☐
French fries (1 cup / 250 ml)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Pies (1 pie)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sauces e.g. tomato, BBQ (1 tablespoon)	☐	☐	☐	☐	☐	☐	☐	☐	☐

A11. How often have you eaten the following foods on average in the past 6 months?

Remember to select the average of how often you have eaten the portion of the food in brackets over the past 6 months.

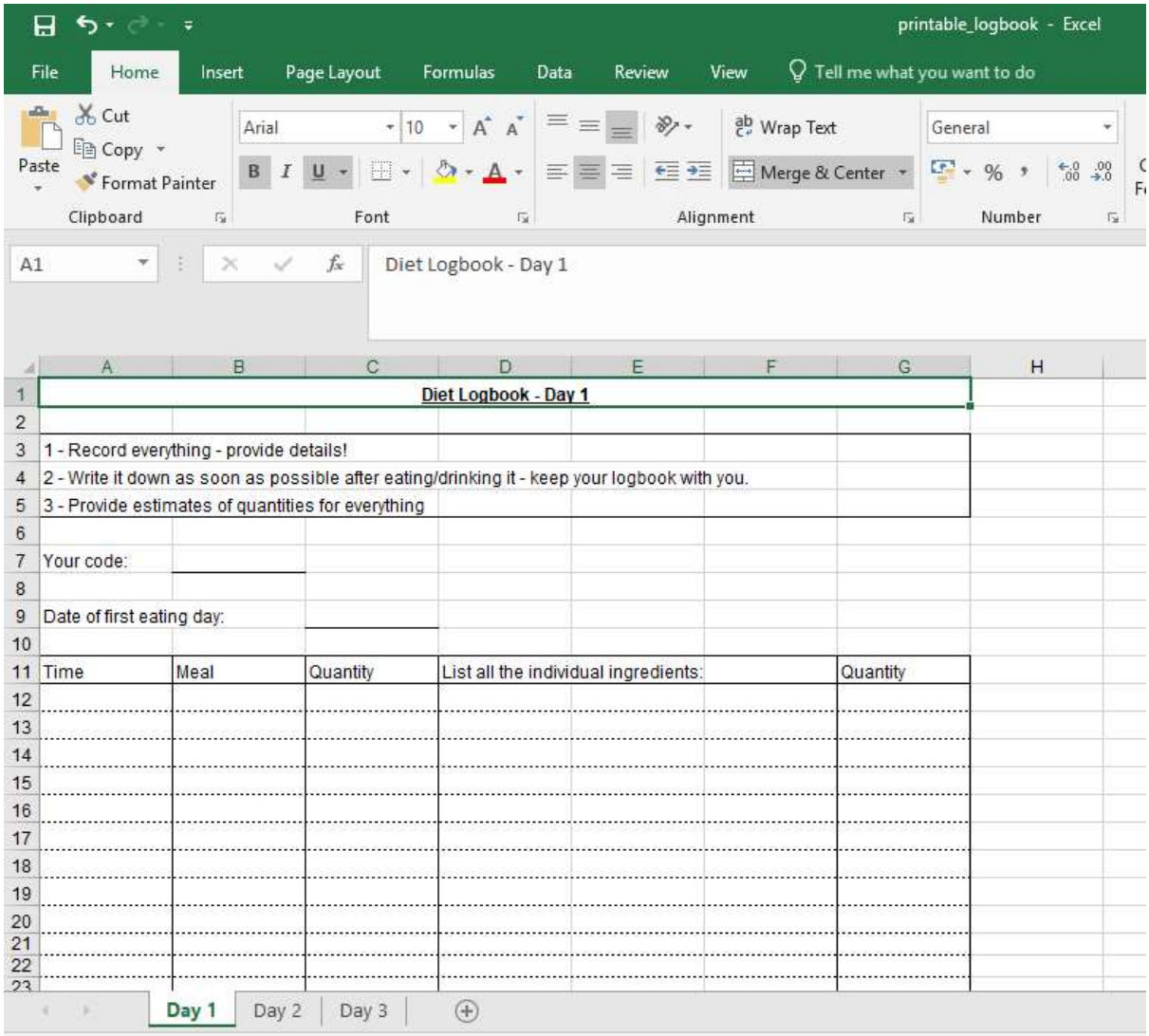
e.g. You eat 7 portion of chocolate but only on the weekend: select 1 per day.

	0	1 or less per month	2 - 3 per month	1 - 2 per week	3 - 4 per week	5 - 6 per week	1 per day	2 - 3 per day	more than 3 per day
Dessert - e.g. ice cream / pudding (1/2 cup)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Pastries / croissants etc. (1 item)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Slice of cake / muffin / brownie / waffle (1 item)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sweet biscuits (1 biscuit)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Rusks (1 rusk)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Sweets (1 packet / 50g)	☐	☐	☐	☐	☐	☐	☐	☐	☐
Crisps (1 small packet)	☐	☐	☐	☐	☐	☐	☐	☐	☐



Thank you for completing the questionnaire. You may close this window.

APPENDIX F: 24 HOUR RECALL (24HR) AND 3-DAY FOOD RECORD SPREADSHEET TEMPLATE



printable_logbook - Excel

File | Home | Insert | Page Layout | Formulas | Data | Review | View | Tell me what you want to do

Cut | Copy | Format Painter | Paste | Clipboard | Arial | 10 | Font | Wrap Text | Merge & Center | Alignment | General | Number

A1 | Diet Logbook - Day 1

	A	B	C	D	E	F	G	H
1	Diet Logbook - Day 1							
2								
3	1 - Record everything - provide details!							
4	2 - Write it down as soon as possible after eating/drinking it - keep your logbook with you.							
5	3 - Provide estimates of quantities for everything							
6								
7	Your code:							
8								
9	Date of first eating day:							
10								
11	Time	Meal	Quantity	List all the individual ingredients:			Quantity	
12								
13								
14								
15								
16								
17								
18								
19								
20								
21								
22								
23								

Day 1 | Day 2 | Day 3

APPENDIX G: OPEN-ENDED STRUCTURED INTERVIEW QUESTIONS

Say to participant:

- Please speak freely and honestly. Your identity will be protected and we are not here to judge anything you say
- Try to talk about your personal experiences rather than what you think we may want to hear, or what you have heard in the media and from other people.

1. Tell me a bit about your how and when you were diagnosed with diabetes?

2. Describe how healthy your lifestyle was, leading up to your diagnosis.
   - Diet,
   - Exercise,
   - Smoking, alcohol, stress, sleep

3. When did you start following an LCHF diet?
   - Relate to diabetes diagnosis

4. Have you made any other lifestyle changes since you were diagnosed with diabetes?
   - Diet
   - Exercise changes
   - Any other lifestyle changes? Smoking, sleep, stress etc

5. Tell me about how your diabetes has progressed.
   - Have there been times where it got worse?
   - Have there been any times where it has improved?
   - Was there anything that might have caused these changes?

6. Describe your current LCHF diet.
   - What foods do you eat?
   - What foods do you avoid?
   - Do you count calories?
   - Do you restrict calories?
   - How many meals do you eat in a day?
   - How long do you usually fast for between meals?

7. Describe how your LCHF diet has progressed from when you first started till now.

8. Has your LCHF diet influenced your diabetes?
   - How? – related to blood glucose.
   - Positive?
   - Negative?
   - Medication?

9. Has your LCHF diet influenced other aspects of your life?
   - Which ones?

- General health and well-being?
- Exercise capacity/ performance
- Other Positive?
- Other Negative?

10. Have you tried following any other diets to manage your diabetes?
- What diets were they?
- How did they impact your diabetes?
- How did you feel about these diets?

11. [Questions tailored to individual based on their questionnaire, if any]

12. As a diabetic, is there anything else you would like to share about diet, particularly an LCHF diet?

C13	58 y/o M. HbA1c reduced from 12.9 to 5.5 %, with cessation of insulin and a biguanide, and 8 kg weight loss, over 4 years on an LCHF diet.
C17	52 y/o M. Hba1c decreased from 10.8 to 5.4 %, with cessation of a biguanide and 7.7 kg weight loss, over 10 months on an LCHF diet.
C19	46 y/o M. HbA1c reduced from 14.7 to 5.3 %, with cessation of a biguanide, and 23 kg weight loss, over 7 months on an LCHF diet.
C29	46 y/o M. HbA1c decreased from 8.2 to 5.3 % (not on T2D pharmacotherapy), with 60 kg weight loss, over 1 year on an LCHF diet.
C31	53 y/o M. Hba1c decreased from 10.2 to 5.9 %, with insulin and biguanide cessation, and 25 kg weight loss, over 1 year 3 months on an LCHF diet.
C37	65 y/o M. HbA1c reduced from 6.9 to 6.1 %, with no change in biguanide dose and increased insulin (26 to 88 u. d-1), and 15 kg weight loss, over 7 months on an LCHF diet.
C57	64 y/o F. HbA1c decreased from 6.6 to 5.7 %, with no change in biguanide dose, and 7 kg weight loss, over 6 months on an LCHF diet.
C59	55 y/o F. HbA1c decreased from 9 to 6.7 %, with no change in biguanide dose and reduction in insulin (from 40 to 32 u. d^{-1}), and a 2kg weight loss, during one year on an LCHF diet.
C73	67 y/o M. HbA1c reduced from 6.1 to 5.5 %, with no change in bigunaide dose, and 13 kg weight loss, over 6 years on an LCHF diet.
C79	41 y/o F. HbA1c decreased from 8.1 to 5.3 %, with a reduction in biguanide dose, and 63.3 kg weight loss, over 3 years 2 months on an LCHF diet.
C83	59 y/o M. HbA1c decreased from 7 to 6.2 %, with cessation of a biguanide, and 17.8 kg weight loss, over 1 year on an LCHF diet.
C89	62 y/o M. Hba1c decreased from 6.5 to 6.1 %, with no change in biguanide dose, and 12 kg weight loss, over 2 years on an LCHF diet.
C97	59 y/o F. HbA1c decreased from 10.4 to 6.1 % (not on T2D pharmacotherapy), with 6 kg weight loss, over 7 months on an LCHF diet.
C311	67 y/o F. Hba1c reduced from 7.1 to 5.3 %, with no change biguanide dose, and 10 kg weight loss, over 2 years 2 months on an LCHF diet.
C319	67 y/o F. Hba1c reduced from 6.5 to 7.2 %, with cessation of all T2D pharmacotherapy (insulin and two T2D medications), and 28.5 kg weight loss, over 2 years 2 months on an LCHF diet.
C343	52 y/o F. HbA1C reduced from 6.7 to 4.8 % (not on T2D pharmacotherapy), with 4 kg weight loss, over 3 years 2 months on an LCHF diet.
T101	51 y/o F. HbA1c decreased from 9.6 to 5.4 %, with no change in biguanide dose and insulin cessation, and 32 kg weight loss, over 1 year on an LCHF diet.
T103	49 y/o M. HbA1c remained unchanged at 5.4 %, with cessation of biguanide, and 5.4 kg weight loss, over 8 months on an LCHF diet.

T107	49 y/o F. Hba1c reduced from 5.4 to 5.3 %, with no change in biguanide dose, and 6.5 kg weight loss, over 7 months on an LCHF diet
T109	48 y/o M. HbA1c reduced from 7.3 to 4.8 %, with cessation of 3 classes of T2D medications, and 54 kg weight loss, over 4.5 years on an LCHF diet.
T127	63 y/o F. Hba1c reduced from 7.8 to 5.6 %, with increased sulphonylurea dose and insulin cessation, and 41 kg weight loss, over 10 months on an LCHF diet.
T131	67 y/o F. Current HbA1c is 8.5 % (previous records are unavailable). No change in T2D pharmacotherapy (insulin, and biguanides, Metformin and Glucophage), and 5.8 kg weight loss, over 10 months on an LCHF diet.
T137	65 y/o M. HbA1c decreased from 10.5 to 7.7 %, with insulin cessation and biguanide continuation, and 62.5 kg weight loss, over 2.5 years on an LCHF diet.
T139	63 y/o F. HbA1c and body weight remained normal and relatively constant (only minor loses) over 3 years on an LCHF diet.
T149	67 y/o F. Hba1c at the time of the study was 6.1 % (not available for other time points). Biguanide continuation and sulphonylurea initiation, and 30.5 kg weight loss, over 3 years 2 months on an LCHF diet.
T151	72 y/o M. HbA1c decreased from 6.3 to 6.1 %, with insulin cessation and biguanide initiation, and 14 kg weight loss, over 4 years on an LCHF diet.
T157	33 y/o F. HbA1c decreased from 8.7 to 5.5 %, with meglitinide initiation, and 7 kg weight loss, over 9 months on an LCHF diet.
T163	67 y/o M. HbA1c increased from 6.1 to 6.5 %, with insulin cessation and biguanide continuation, and 26 kg weight loss, over 2 years 3 months on an LCHF diet.

APPENDIX I: CODE BOOK

Parent Node	Child Node	Definition	Quote examples
EATING BEHAVIOUR	Control	Addiction, over-indulgence, portion control, binging, calorie counting	Hunger ▪ *one of the things I noticed before I was diagnosed was for example, when I got hungry, it wasn't kind of like, 'oh, I think I would like to eat something now', it was, 'if you don't eat something now, you'll die!' kind-of-hunger. It was a really sharp pain in the stomach, like 'I just have to eat something!'. I haven't experienced any of those hunger pangs really ever since I started this diet. And there have been a couple of days where I just decided when I woke up, and I've not been hungry, I decided not to bother. And I got through most of the day and not bothered and then maybe had an early meal in the evening… And obviously one of the other things I have measured, is my ketones and clearly as soon as you're in a proper ketone state, you stop feeling hungry. It's not hard then to not eat.* (C97) ▪ *I had noticed that I was putting on weight around my tummy and the only symptom that I actually had was, I was hungry all the time. And then when I started the low carb diet, I found that due to the sort of high fat, it was easier. I was not quite as hungry…* (T139) ▪ *The only time I feel hungry is after a weekend when I've sort of been a bit bad. When I've eaten maybe more carbs than I normally do. It's funny because often, if ever I feel hungry, I would think, "what have I eaten?", and it will always be carbohydrates.* (C343) ▪ *if I misbehave over a weekend, I say, "ja I am going to have bread with my soup" or whatever, I find that I feel yuck after that, you can actually feel the rise in blood sugar, just from bread and that kind of thing…that's why I don't drink alcohol either anymore, because alcohol tends to have that effect. You have a couple of glasses of wine and the next morning, it's like you're starving.* (C79) ▪ *I tend to do the fasting kind of thing, so I eat once a day probably. The rest of the time I'll drink coffee, I drink lots and lots of coffee… I don't get hungry very often.* (C79) ▪ *the beauty of the whole Banting scenario in terms of the lifestyle situation is you don't get the hunger cravings, so it's not that you have to go somewhere and you feel well I have to eat. You don't have to eat.* (C89) ▪ *I just eat what I want and when I am hungry. In fact, sometimes I don't feel hungry at all. I can go out for a five-hour bike ride and get home and I don't really feel like eating which is a bit odd. It might be two or three hours later I sort of force myself to eat something.* (T103) ▪ *I am not hungry and I am actually not missing carbs, which I cannot believe.* (T107) ▪ *So, if I stick to the low carb thing properly, I'm never ever hungry. I am more bored, or need food because of boredom, than I am actually feeling hungry. I feel completely like I can just read how I am feeling, as to what I should eat and what not, or if I've eaten too much.* (C343)

			▪ *I struggle with hunger, but then it's all in the mind. I read something that says decide if you are hungry, or if you are just bored. So, most of the time you're bored, you just want to nibble, your mouth must just move.* (C59) Portion and calorie control ▪ *to me, if I've got to measure each and every little portion it's not practical, you can't live your life like that, so I've got to teach myself to be able to judge what I'm allowed to eat and what I'm not allowed to eat…* [I decide how much to eat] *by looking at it.* (C37) ▪ *I never counted calories I never weighed, measured or counted anything.*(C13) ▪ *I'm conscious of calories, but I generally don't restrict them. I generally eat until I've had enough… My weight is fairly stable. It is not going up, it is not going down terribly fast, so I presume I am basically eating as much as I'm using.* (C17) ▪ *My weight is stable so that tells me my macros' are probably about right. I don't measure anything, I don't weigh anything… I just eat what I want and when I am hungry.* (T103) ▪ *I go with the principle of eat when you're hungry and when you're full, stop and that's it.* (C79) ▪ *I eat until I have had enough.* (T149) ▪ *I just eat until I feel full.* (T151) ▪ *I don't have any cravings or any hunger. I used to snack a lot as well, because I was always hungry before I was diagnosed. I just eat until I'm not hungry. I don't count calories, I don't weigh the food, I just cook a 250 gram steak, which is enough for me, because there is about 60 grams of protein in there. So, I just eat it until I am full really. If I'm hungry still, I will probably have some berries with a bit of cream afterwards, but that's all. It satiates me. Then I last all night and all the next day until the following evening meal. The portion of vegetables I have is quite small. When I do have vegetables, it is only a small portion with the meat...* (C83) ▪ *I eat until I'm basically full… there's no calorie count at all…But in terms of what we do today, as I say, it's very, very, monitored and controlled. So, we don't eat between meals at all and I drink black coffee, that is all I drink…* (C89) ▪ *I haven't* [purposefully controlled what I eat] *but what is interesting, what happened since I have been Banting, I will not get hungry. But my meals are smaller. One of the things I have learned through the Banting Coach, is you eat only when you are hungry. And your body will tell you when you're hungry… I find that I am not hungry, and so easily, I can have a breakfast at twelve o'clock...You know so and I am full.* (T131) ▪ *At the first, when I started off, I was under the impression you had to eat three times a day. And so, I did. I assumed that cakes were nice, provided it qualified as Banting and so I would do that as well. And once I hit a plateau and I read some books and particularly the book by this one doctor who pushes fasting. But I then realised that hang on, I, I am getting it wrong, I am eating, because it is a meal time, I am not eating because I am hungry and I am really following,*

			what, the same bad patterns. I am just changing what is on the plate. That is it. I need to reorganize my life and so then I began to say, well why don't I eat when I am hungry? And learn to stop eating when I am full. (T137) ▪ *I don't restrict my calories but I do do Intermittent fasting.* (T157) ▪ *… I can't over eat on protein because that gets converted to glucose too. So not bigger than the fist of my hand. So, I watch it. And I have to eat lower fat, you know? I can still fry in coconut oil or add some to my eggs... But you know, it's self-limiting, you eat till you are full. Which is surprisingly far less than I did before on Banting.* (C311) ▪ *I suppose in a way I do restrict, it is not like weighed or whatever but it's like this sort of size meat* [palm sized], *I stick to probably a hundred and fifty and two hundred grams sort of meat size. You know that sort of size meat. But the vegetables I mean are on a normal plate.* (T101) ▪ *I do limit how much protein I eat…* (T149) ▪ *I am not aware of restricting calories in any way. Eat what I feel like and I am afraid I am still gluttonous and I accept that. But generally, it doesn't affect my weight. Except when my carb intake is slightly higher in the form of eating too many seed crackers or too many nuts, nuts are my nemesis… My portion size has increased* [since first adopting LCHF]. *I am aware that I am having too much protein but I am having it anyway and that is kind of what I am living with. So, I think when I started I was more vigilant about limiting my protein size as well and wanting it to be more sort of perfect diet candidate…It's just nice to eat more. I'm certainly not hungry. I never have to eat out of hunger. I mean I don't have to eat that much out of hunger. It's just lekker. Which worries me actually because I kind of think if I pick up like you know, a kilo every few months in twenty years' time I am going to have picked up twenty kilo's. So, I need to pull back there. I am vigilant about it.* (T109) ▪ *I do know like for instance, your protein should be the size of the palm of your hand…* (T139) ▪ *In a day I want to try and get my carbs and proteins down to less than 100. My carbs at 30… keep the fat content up to about the 60%... My goal would be, in my ideal sort of nice day, I would have eaten twelve hundred and fiftyish calories.* (C97) ▪ *…now the situation is if anything is on the red list, I don't have it at all. Anything on the orange list, we have, as it says very little and then primarily, we eat from the green list.* (C89) ▪ *I don't count calories… For probably 3 years, I'm eating out of a side plate, so that is my portion and I don't go for seconds.* (C59) ▪ *But now, it's 1 meal and that meal itself, is actually quite small, so yesterday for breakfast, we went for breakfast and I had a triple omelette, full of cheese, some bacon and sausages and that was all I ate for the whole day, apart from some cheese for the rest of the day*. (C73) ▪ *… I always use a small plate. I don't use a large plate. I don't weigh anything and I am not over aware of the portion size.* (T139)

			▪ *One of the things I have done is, I have reduced the size of the plate that I serve in and infact my plate is more like a side plate now… why don't I not worry that the plate is half empty by getting a smaller plate (T137)* Food addiction ▪ *I went through a phase in this winter where I broke down and my sugar addiction kicked back in and I started raiding the biscuit cupboard at work, and if I have 1 biscuit it becomes 3 biscuits, it becomes the rest of the packet… And then also, on my way home, I would stop and buy chocolate and then 1 was not enough, buy 2, and with that, my weight went back from …77, it jumped up to 88, and fortunately I've been able to get control of that, although it's a little bit difficult. The sugar addiction is extremely real and I have managed to get all that back into control again and it's* [the weight is] *basically coming off slowly.* (C13) ▪ [I ate a lot of sugar]. *A lot. At one stage, I thought I was totally addicted to chocolates, totally addicted. Totally addicted.* (C59) ▪ *… I was a Coke-alcoholic, soda drinker. Being diabetic, I would on a weekend, go and buy four 2 litres of cold drink because I was going to be thirsty over that weekend and I polished that stuff.* (C13) ▪ *…I believe I was one of these people who were addicted to whatever is in the breads and carbohydrates because one piece of bread was never enough. Not having bread at all I could completely cope with, but having one piece of bread was just infuriating for me and difficult for me, unsustainable for me.* (T109) ▪ *If I smoke I will smoke, kind of thing, and I used to control it, a lot of the time I would have 1 or 2 smokes in the evening, but I would always land up going back to smoking about 20 a day kind of thing. And that is what I realised from Banting actually, is carbs are probably something that I'm probably never going to be able to eat for the rest of my life, certainly not on a regular basis. Maybe I will have a cheat once a month, have a cheat like go out and have a really nice pasta, something like that. But I guess my days of carbing are over, and my days of smoking were over in the year 2000.* (C37) ▪ *I indulged a lot in sugar* [before adopting LCHF]. *I mean I daily had pastries and cake and chocolate. I was a chocolate addict. I am not talking about dark chocolate. Although I miss the sweet stuff, the less you have of it the less you want. But I didn't know it then...* [When I first started Banting] *I still got in a lot of sugar. You know and as we know, that triggers the craving for more sugar. So, it wasn't until I said nothing, no more, that I really got rid of the trigger.* (C311) ▪ *… the desire for sweetness has died by and large.* (C37) ▪ *I had a very sweet tooth* [before LCHF], *sweeties and chocolates and all those kinds of things* [Laughing]. (T127) ▪ *my husband eats cookies and chocolates and things in our home… I thought it would drive me, but it doesn't bother me at all. You know I am so surprised to be able to walk passed all of them in the shopping centre and it is no trigger. Whereas before* [LCHF] *I would never be able to do that.* (C311)

			▪ *I mean sometimes I still crave, I feel I crave carbohydrates sometimes. So sometimes I feel like it's not as easy as other times.* (C319) ▪ *I still struggle sometimes with chips, but I tell myself no it tastes bitter, don't even think of eating it.* (C59) ▪ *Although I was very weary about the sweet taste of things* [when I first started LCHF]. *If we would have supper outside our house or in a restaurant or with friends, when something tastes sweet I wouldn't eat it. That was the one thing I tried to wean myself off, was being careful that I wouldn't get addicted to that sweet taste. That was the one thing that I was really pertinent on and I really watched myself.* (C19) ▪ *I think it took time to get rid of the desire to eat sweet sugary stuff… the other change that has definitely happened is, I will not touch anything sweetened, because it makes me want to eat sweet things again. So, I don't know if other people have reported this to you, like the other* day [my wife] *bought some coconut, you know it says coconut and all this, and I took one bite and it was just full of Xylitol and very, very sweet and immediately I wanted to eat the whole thing, immediately and I would have had that and another one, that was the immediate response. And I had to say no hold on, you must keep that, I'm not eating that and then after about 5 minutes, the desire went... But the point is the addiction remains and it's very powerful, but it has taken 6 years now. I don't think I've ever given in to the addiction again… I haven't given in, but the power is very, very powerful.* (C73) ▪ *I'll eat a lemon now and coffee and 90% chocolate tastes sweet. So, my sweet taste has completely changed.* (C73) ▪ *At that time* [when diagnosed with T2D], *I dropped the sugar, everything that contained sugar, I just wouldn't eat anymore, but I wasn't aware about bread and potatoes and rice, that was something I didn't know at the time, so I continued to eat potatoes rice and carbs, but the added sugar was out of the question, I stopped it. I didn't drink any sodas anymore.* (C31) ▪ *even then* [when diagnosed with T2D], *I definitely cut sugar... but not necessarily carbohydrates… I went more for the low GI's and I was still eating pasta… if there was cake I would have a very small slice, as opposed to none, which I sort of have now...* (C343) ▪ *what I know about myself is I am quite extreme and it is easier for me to make extreme choices and eliminating alcohol is easier than trying to be balanced about and having one glass or…because I was never a one glass kind of boy.* (T109) ▪ *I'm an all or nothing person, either you're having no carbs at all, consciously, or if you're having some, it creeps back.* (C89) ▪ *It's like there is so many choices that in the end it's too hard. You know I enjoy having fewer choices. Like the rule of no gluten that takes care of a lot but if I can have two* ▪ *slices of bread per day then that is not out of the equation, you know? I have to cope with that. I find that more difficult.* (C311) ▪ *My type of way is if I buy a packet of sweets I have to eat it all. It's not take two and put the rest away.* (T107)

			▪ *my cupboards are still chock full of bags of crisps, chocolates, all sorts of things that you shouldn't eat and which my guests tuck into, and I'm fine with that. And I just use different cupboards. It doesn't bother me. I don't feel like I need to eat the sugars in any shape. I don't feel as if I'm going to binge on them. If I binge on things, it would be something like pork scratchings which are on the top shelf, so I would have to get a ladder out.* (C97) ▪ *I find it difficult to resist like a hot French loaf… So, I think it is a two or three a month thing, but it's just like what I would do. If someone's served up hot French loaf or a garlic bread I'll have a slice of it. That kind of thing.* (C319)
	Frequency	Only code when related to timing, gaps, number of meals, fasting etc.	
LCHF DIET DESCRIPTION	Foods (choose, avoid)	Must mention specific foods or food types. Include things like: starchy foods, sweet tasting foods etc. Include beverages and alcohol.	High fat foods ▪ *I have increased my intake of butter, I have increased my intake of cream, I have increased avocado oil, avocados and olive oil.* (T151) ▪ *I changed from having oats for breakfast to eggs for breakfast and increased my olive oil, I think I was on one teaspoon of olive oil per meal and just started to have that more liberally and I remember noting that that's when my weight loss increased.* (T109) Carbohydrate containing foods ▪ *I used to eat a lot of cashew nuts, but after knowing just how many carbohydrates there are actually in them… I switched from eating cashew nuts, to macadamia nuts, which I never ate before. Now I have gotten used to it. I still miss my cashew nuts. So, I have done those kind of changes.* (T157) ▪ *I was still eating things like pumpkin, but… I realised* [that] *was too high* [in carbohydrate] *for me. That's what actually shot my sugars up... And eventually I decided, various foods, even though they were Banting foods, were still pushing my sugars up, and I decided I need to go into ketosis. So, I have been in ketosis, for quite some time now, I think it was one and a half years.* (T139) ▪ *I would say I have gone off dairy, not altogether, if I go out and have a cup of coffee and I can't get any coconut milk, then I will have a little bit of milk, but generally speaking, I use coconut milk and coconut yoghurt* (T139) ▪ *I've learnt a lot since I started* [an LCHF diet]. *So, I have modified quite a few things. Lately it was dairy. I had no clue I was eating too much and that was the problem with my blood glucose.* (C29) ▪ *I've learnt that dextrose that is contained in the sweeteners is not a very good thing. It will spike my blood sugar. So, a few things like that I learnt, that I changed over the months.* (C29)

			Sweet taste ▪ *Initially I thought, oh well this was wonderful, that I was being able to have the erythritol, but then I realised, actually, I need to cut down on that sweetness, because my body is still craving sweetness. So, I have gone down on that as well, but there are occasions that I need to have something sweet. Celebrations or whatever. More and more I have learnt to adapt… Before diagnosis, it was cakes and all sorts of things. I went from that to xylitol and then from xylitol to erythritol. So, when we do it, I try to have cake, but made with erythritol and not eat a lot.* (T139) ▪ *…we know that* [sugar] *triggers the craving for more sugar. So, it wasn't until I said nothing, no more, that I really got rid of the trigger.* (C311) ▪ *And you ask what's modified, I think it took time to get rid of the desire to eat sweet sugary stuff. And then sorry the other change that has definitely happened, is I will not touch anything sweetened, because it makes me want to eat sweet things again.* (C73) ▪ *I have just had a HbA1c and I think I was 6.5. That is the highest I have been since I left CDE* [diabetes care programme]. *It is because I am cheating a bit more. I am pushing the envelope a little bit, I do treat myself maybe once a week, or maybe a little bit more than that, when we go out and somebody has got a nice pudding and I will have a small amount of ice cream and things like that... I cheat more and more now, because I am not as worried as I used to be about my blood glucose… but I don't cheat often… maybe once a week.* (T151) Food quality ▪ *as the things progressed, I got more and more conscious of what I was eating and where it was coming from… your choices start changing.* (C79) ▪ *Well when I first started low carb I didn't understand the distinction between, or the benefit of removing processed food. I was still eating some packet goods. Some ready meals that I thought well they're relatively low carb. But I hadn't understood the benefit of that. So that certainly changed.* (T103)
	Progression or evolution of diet	Include info on how they started LCHF.	For example: "When I started LCHF I just focused on cutting out sugar" Fats ▪ *I think I was better when I first started. I think I ate more fat, but at the same time I think my fat's cut down because I don't need it all because I very seldom feel short of energy.* (C343) ▪ *…probably I aim to eat more fat, so I look for the fat* (C73) ▪ *Personally, having looked at the research, it seems quite clear that if you want your HDL to go up, exercise helps, but so does eating saturated fats. So, I've been specifically adding in a bit of saturated fat into my meals, so for example, I never used to drink coffee, now I've started drinking coffee and I put a tablespoon full of cream in it. And I'm kind of hopeful that the end result of that should be that it increases my HDL.* (C97)

			▪ *Whilst I was losing weight, initially on the low carb approach I guess I didn't really need the fat because I was burning my body fat. Now that I'm like ten percent body fat I actually need the fats and there is a difference between low carb for weight loss, low carb high fat for maintenance and I guess instinctively, I came to realise that If I wanted to stabilise my weight I needed to start eating more calories because of all the training I was doing.* (T103) ▪ *I changed from having oats for breakfast to eggs for breakfast and increased my olive oil, I think I was on one teaspoon of olive oil per meal and just started to have that more liberally and I remember noting that that's when my weight loss increased.* (T109) ▪ *I started to go lower on my diet, as with regards to lower carb and higher fat, and then eventually moving towards ketosis*. (T139) Protein ▪ *…what I'm trying to do currently, is to keep my carbs at 30 grams per day or under, or my carbs plus proteins at less than 100.* (C97) ▪ *I am aware that I am having too much protein but I am having it anyway and that is kind of what I am living with* (T109) Carbohydrate ▪ *I started to go lower on my diet, as with regards to lower carb and higher fat, and then eventually moving towards ketosis*. (T139) ▪ *Although I had cut the carbs down dramatically, it was creeping back… then… I've dropped, I think without measuring it directly, but I think that my carb intake is around about under 25-30 grams a day. And as a result of that, everything came back into line, so my weight dropped off again and my blood sugar readings are good* (C89) ▪ *I used to eat a lot of cashew nuts, but after knowing just how many carbohydrates there are actually in them… I switched from eating cashew nuts, to macadamia nuts, which I never ate before. Now I have gotten used to it. I still miss my cashew nuts. So, I have done those kind of changes.* (T157) ▪ *I was still eating things like pumpkin, but… I realised* [that] *was too high* [in carbohydrate] *for me. That's what actually shot my sugars up... And eventually I decided, various foods, even though they were Banting foods, were still pushing my sugars up, and I decided I need to go into ketosis. So, I have been in ketosis, for quite some time now, I think it was one and a half years.* (T139) Modifications in response to a weight loss plateau. ▪ *The biggest difference that I remember when introducing the fat was that the hunger was sated…and I remember noting that that's when my weight loss increased.* (T109)

			▪ *I think I had lost 30 kg's at that point and I could see the weight loss slowing down, so I thought maybe I actually need to start looking at not only just the carb count but also the calorie amount, because high fat as you know, has lots of calories in it and you can still overeat hugely if you don't watch out, so that is when I started to pay more attention.* (C79) ▪ *I brought in the fasting after Christmas this year because* [my wife and my] *weight plateaued. That's why I did it. I decided to accelerate my weight loss, because I'm trying to get below 100 down to about 85 kilograms and I was about halfway through my weight loss after a year or so* (C83)
	Sustainability		▪ *I think for me it's sustainable, I feel like it's become a lifestyle* (C343) ▪ *Forever* [describing how long she will follow LCHF]. *I've been doing it since 2014, it is 2017 now and I don't intend changing it ever.* (C79) ▪ *I think it is actually perfect for me. I am going to stay on it for life.* (C319) ▪ *I've never been able to lose weight for more than a few months and I've been at it for almost a year now, so I would say I'm pretty much going to eat that way for the rest of my life.* (C29) ▪ *I simply came back to it saying this is sustainable, it is sustainable, my … doctor asked me if I'm going to eat like this for the rest of my life and I said well why not*. (C13) ▪ *Knowing how I feel and how good I feel health wise and weight wise, I would say, I would never want to go back to the other way, because I wouldn't want the battles that I had with the excess weight and with the diabetes particularly, and all the other health problems, which have gone and it is being like a miracle. And so, I would say, for me it would be totally sustainable.* (T127) ▪ *I am going to stay on it for life. You know with the weight problems I've had all my life… Yes, I won't go back to eating potatoes and crisps and noodles and things like that.* (C319) ▪ *It's very easy. I've been doing it essentially for 6 years now*. (C73) ▪ *Oh, it is easy. It's a way of life. It's a lifestyle…I am not going to go back.* (T151) ▪ *I think you realise it is not difficult once you're in*. (T163) ▪ *It's really sustainable and really filling and tasty and great food. I love it.* (T103) ▪ *I think it takes out much of the joy for me at this stage. Maybe it's just early days. …* [Since eat LCHF] *I couldn't freely partake in everything anymore.* (C311)
OTHER DIETS		Which 'diet' and the impact of	▪ *I was trying, but I couldn't* [keep the weight off]*, every year or every other year, I would start a diet, lose some weight and gain it back.* (C29)

		the diet (which, positive, negative). Can include lack of a diet. Can just refer to how they used to eat.	▪ *I tried* [Weigh Less] *a number of times… I had their booklet and I would follow it and I would lose like point four of a kg and I mean okay, I need to lose like 50 kg's, I am going to be here like for the next 500 years.* (C79) ▪ *I went to Weight Watchers and I went to Weigh Less and I did the Herbalife and I went to dietitians…I would lose weight but it was never sustainable… I would lose the weight and then I would be ten times worse than I was before.* (T101) ▪ *I was always hungry* [on Weigh Less] (T149) ▪ *… to me, if I've got to measure each and every little portion it's not practical, you can't live your life like that* (C37) ▪ *I tried just about everything under the sun… the low-fat, the fat-free… all of that. Of course it doesn't work, partly because you end up emotionally feeling deprived, or for me that was a major issue.* (T137) ▪ *The weight that I lost at the beginning of this year, during the very low calorie* [diet shake] *2 months,* [and] *my HbA1c, remained normal at normal levels since then.* (C17)
POST LCHF	Emotions, Reactions, Motivations, Social	Any emotionally stated changes in the impact of reduced diseases, symptoms, conditions, states etc. Includes states on social impact or interacting with society. General statements referring to mental health. i.e. coping better. Must refer	▪ *I was still having sweeteners in my coffee and tea at the time, I found that habit very hard to break. I eventually broke through that…* (C319) ▪ *But I have always been somebody who has taken comfort in food and I can remember whenever it got stressed, I would go and of course carbs are the cheapest things to buy, so I would eat. So, I certainly had a very unhealthy lifestyle… I don't any longer get tempted by cake and I am not one of those who has to have masses of ice-cream and things like that. That doesn't bug me anymore, at all... And when I do feel that right now I am stressed, all I have got to do, is to do what I would do with any 5-year old child, say, "Here is a new toy, let us play with it", and 2 minutes later, the tantrum is gone. And I have got to realise, I am like that too. So, if I am sitting down panicking, I am going to want to eat. If I could break that cycle for 60 seconds, the desire to feed my face will go away.* *…I grew up …* [in] *an Afrikaans family, and food was a sign of love. So, your mother would overcook and you would have to eat, because if the plate was not empty, she thought you didn't love her… So, you had to start breaking the link between love and food and that was not all that easy. It was tougher than I thought. But now it is a matter of you eating until you're full and then you stop.* (T137)

		to post-LCHF.	
	Negatives and Challenges	Includes all negatives including lack of negatives, emotional, non-emotional or logistical challenges. It MUST BE CLEAR from their answer that they are perceiving it as a negative or challenge. For example: if the say "I can't get drunk anymore" it is not clear that this is perceived negatively whereas "I really miss getting drunk" has a negative associated with it.	▪ *I can't think of any. I mean it's only been beneficial.* (C73) ▪ *Do you know what? I can't think of one thing that has not improved.* (T103) ▪ *No, I've only had positive.* (C89) ▪ *No, absolutely no negative.* (T109) ▪ *I was going to mention that* [social impact] *as well, as a negative, but it is actually not that negative. But you do, you do get people who go, "that's a fad!" or, "you are not doing that!", mocking it and criticising it, and got quite angry with it, saying like, "Tim Noakes is whatever"...but slowly a lot more of them are starting to do the same thing… even my boss is slowly coming around to it.* (C343) ▪ *As far as the wider public is concerned, sometimes, boy oh boy, the first question is, "what are the long-term effects?" And I say, "the long-term effects are maybe I won't have my feet amputated and I won't go onto dialysis and I will keep my eyesight". But as far as that is concerned, people haven't got a clue, they don't know what they're talking about and there is a tremendous negativity out there.* (C89) ▪ *I think it is difficult for me at times, in a social atmosphere. Whereas other people don't understand that I am trying to do it without medication and I am trying to lower my sugars with diet and you know. They will say things like, "oh, just have a piece of cake. It won't kill you," and inside I am saying, "well actually it may just"* [laughing]. *Because they don't understand. So that is really the only sort of the negative side of it.* (T139) ▪ *I don't want to call it a battle but it is a battle with regards to general public, because they don't understand why I am doing the low carb. And then you get those sometimes, the frowns, the "why are you not eating fruit anymore", "why are you not eating sugar anymore?" So, I think that, that has been my main battle.* (T157) ▪ *I am trying to fit in with everybody. Socially it's been difficult.* (T101) ▪ *…there are negatives and the negatives come, I think, mainly out of your social life. Because you're the person who is different, you are the person who is not doing it. You are the person who is not going out.* [My wife] *doesn't drink, so that makes it quite a lot easier, but you just don't do it like everybody else.* (T163) ▪ *The only time it becomes a little awkward, is if you get invited around to somebody's house for a dinner party or something or other like that, and now you're kind of caught between the world where they're dishing up food that you don't want to eat, or you actually might want to eat, but you shouldn't eat. You*

			don't want to offend them and you don't want to be picky. I mean we don't eat out every night it's something that happens once every 3 months or something, so when it happens, deal with it. (C37) ▪ *…maybe I haven't got my head right, but* [LCHF] *does take the joy out of things. My husband wants to go eat out all the time, and we do. We regularly eat out, three, four times a week, which is difficult for me, you know? And he gets a bottle of wine, and one of his great joys in life is to have me share it with him. Which I can't do anymore because I am not allowed to drink alcohol. So, I only have sparkling water and it's hard. …But you know it puts food into proper perspective:* [it] *is not what you need to enhance your life. You need it for fuel for your body. That is how I feel now… my husband loves food and going to all these fairs and places, and that's his life you know? So, I am not joyfully partaking in that anymore.* (C311) ▪ *… I miss participating in the social side of drinking champagne and being silly and drunk. As I don't drink any alcohol anymore. I kind of miss that. I feel like I am less fun, less fun at parties and I don't really want to go… you know I've withdrawn from that kind of social interaction.* (T109) ▪ *Because of the changes in my diet, I do find it slightly difficult, because obviously temptation is always there. Go to confirmation parties and braai's and those kind of things, the temptations are there …but …90 percent of the time, I would say no.* (T157) ▪ *I do find it difficult, sometimes that, I get peckish and then there is nothing to snack on. It is inconvenient but it is nothing major life-changing.* (T149) ▪ *Eating out is not as easy. You've got to sort of think where you are going, what they serve and what's on the menu and things like that.* (C343) ▪ *The only problem is that, if you are on the road traveling as we do, if you feel hungry, you can't just walk into a take-aways and find something Banting-friendly, because it is not yet everywhere.* (C319) ▪ *I don't find it at all difficult to eat out. The difficulty is cheap, you know you can't eat out fast foods, but you can eat out.* (C97) *We are going on a month's holiday. We are going to tour the countries for* [my husband's] *seventieth birthday… I don't know if am setting myself up, because I won't be able to keep this up then. And if I keep this going until then, then I am going to be sick the first time I eat something that I shouldn't. Because that is what I've experienced in the past. You know, you grow accustomed to not having…your system adjusts... Then when you have it, it's a big spanner in the works. Especially with my sensitive digestive system… I have to be so careful.* (C311) *…the only negative that I can find, especially with your breads and things, it is a bit expensive.* (T131) *So, in the beginning it was very expensive. Now it is much cheaper, because I can do one week with half of the things I bought earlier on.* (C31) *What I found, for example, is because fat is so vilified, practically all the meat that you get in a supermarket is priced inversely proportional to how good it is for you. So, for example, my meat bill has gone down dramatically, because now what I do is I buy the cheapest cuts, because those are the ones that have got fat in them.* (C97)

			It's just expensive, that is all… to buy a new wardrobe [Laughing]. (T163) *the first two weeks is not so nice, but thereafter, just amazing and I will never go back.* (T127) ▪ *It hurt for a couple of weeks, I missed my spuds, the potatoes, rice, pasta etcetera, but after a couple of weeks, I settled into it and was fine with it.* (C17) ▪ *It doesn't worry me anymore. In the beginning, of course it was hard. If I walked through the bakery section and they've just brought out new bread rolls with garlic butter smeared all over and all you could smell was the fresh bread and the garlic etcetera. That used to get me a little bit, but no longer. I can walk through the cake* ▪ *department, the sweets department, the bread section, it doesn't turn me on anymore.* (C319) ▪ *I could live on fruit, it's something that I really miss but I've gone cold turkey.* (T107) ▪ *I don't particularly like rich or fatty food… it took me a while to get used to… But, you know, after a week or two it's fine.* (C19) ▪ *I guess initially it is quite a big step mentally…there is all the things that are drummed into you about high fat's going to give you high cholesterol that's going to give you a heart attack. And there is a lot of momentum in the opposite direction that needs to be tackled. And even I found some of that quite difficult to get my head around. So… that's the problem I guess, for some people.* (T103) ▪ *…it's very difficult to, if I can use the word, cold turkey. It's just impossible. Interestingly enough, only in retrospect did I realise … what happened to me when I went onto the LCHF… About 2 or 3 weeks after we started the LCHF, I got extreme vertigo and it lasted for about 2 weeks, but on 2 of the days, I couldn't even lift my head. And I think that was a carb flu type symptom. I think it was a withdrawal that took place.* (C89) ▪ *I wasn't drinking enough water and it was hurting me when I was weeing and nothing came out you know. The urine looked very dense and it was like giving me a burning sensation and that's because I suspect I should have been drinking more water and that's my biggest mistake, I don't drink enough water.* (C83) ▪ *I was quite constipated for the first year or so. I would get periods of quite severe constipation and it has disappeared completely in the last 2 years.* (C73) ▪ *I think the only thing that I struggle with time to time is constipation.* (T107) ▪ *Oh, the one thing that I do have a problem with is extreme constipation. To a point where I feel uncomfortable and then it goes for a week. It is really uncomfortable and I don't know what to do about it. I have had a problem* [before LCHF]*, but it become worse.* (T149) ▪ *The only thing…that I am having problems with is my cholesterol.* (C319) ▪ *I have developed some gout and that could be diet.* (T127)

			Well the ketosis, I think I used to have more ketosis, so my wife would complain about my breath. Which is interesting actually now because I have to run to get ketosis. It doesn't matter what I eat. (C73)
	Soft Conditions	Must be referring to a specific condition or state but not with reference to a diagnosed condition. For example: Sleep (not sleep apnoea), Energy, Exercise, Pain, Concentration, cognition, cognitive capacity. Includes broad statements of general health or physical well-being.	Symptoms associated with diabetic complications ▪ [just before my T2D diagnosis my eyesight] *was bad to a point where I couldn't read my emails. I had my old glasses and I had reading glasses over that. My eyes took …about three months* [after adopting LCHF] *to average out. Now it's fine.* (C19) ▪ *I have no sense of the neuropathy anymore* [after LCHF]. *It was so incredibly painful it was just like hurting in my legs. Even having the air-conditioning on in our bedroom, the wind current from the air conditioner would cause me pain. It's bizarre to think that. In any sense, it was awful. Horrible horrible horrible. So, that is completely gone.* (T109) ▪ *I used to have* [before LCHF] *… a lower back or a tingling in the leg or… low level symptoms or something… I feel that those are more a less gone.* (T151) Body weight and mental health ▪ *I was so overweight [before LCHF], I was just a fat slug.* (T163) ▪ *I was like a beached whale [before LCHF]. The first time I saw myself in a mirror after 5 or 6 months and I said to my husband you can divorce me if you want.* (C53) Energy and exercise ▪ *…before* [LCHF]*, I was always sleepy…and tired and just had no energy, and now I am sort of up and clearing the house and spring cleaning and my husband is very amused. Because I am always active, like I used to be in my twenties. Okay, so I have got a lot more energy. And before, you know, going for a walk was a chore. Now I really enjoy it.* (T127) ▪ *Now* [on LCHF] *all of a sudden I… had so much energy. I mean things that I can do that other okes* [South African slang for 'guys' or 'people']*, younger than me can't do, or okes the same age as me, they can't even come close. I mean when an oke sees me on a ladder, they freak, "what you doing on a ladder?! You're not 25 years or 40 years old!" and da da da da. But I just got the energy levels.* (T163) ▪ *Energy levels definitely are better. I used to get real slumps, especially in the afternoon. Like real, just like thinking, "Woah, there is something wrong!" or, you know? I don't have that anymore. So definitely, energy levels are much better.* (C343) ▪ *Workwise I am a little better* [since adopting LCHF]*, I can concentrate longer and work longer. I think, I'm more alert because with teaching, you have to be more tuned in, and I've got more energy.* (C83) ▪ [Before LCHF] *I kind of felt like everyday tasks would just make me really tired very easily… I kind of realised that at home, you're just trying to cut everything back to the bare minimum. It's a difficult one. Now* [after adopting LCHF] *I find I'm more active during the day.* (C97)

			▪ [LCHF] *brought back the desire to move. As I say, I was lethargic, I was a couch potato. The following year, I bought a bicycle I have done the Argus now, done a number of events… and I'm enjoying it and I want to do it, so that desire has come back.* (C13) ▪ *immediately* [after adopting LCHF] *I felt more energy and a new sort of enthusiasm. And then I was running, it really got me back into running.* (C73) ▪ *I'm not a gym bunny, I'm never going to be like a hard-core, but just in terms of walking around the block with my child every day that kind of thing, 3 or 4 years ago, you must be joking. I'd be like, do what!?* (C79) ▪ *I've got more energy, I'm fitter than I've ever been. My health's great, my energy is great. I sleep well. My athletic performance has improved. The other thing I've noticed is after exercise, where ordinarily you'd feel sore for three or four days, I recover very quickly now... It's quite remarkable.* (T103) ▪ *I dropped 30 minutes off the run within 5 weeks. And what was interesting was I was coming down the hill and I hadn't really noticed what time it was because I only checked that at the end and suddenly I started sprinting, I hadn't felt like that for 20 years. My running times went back to where they were 20 years before. They weren't spectacular, but at least they are a lot better than what they had been. So, I completely reversed that running, the running performance was just amazing.* (C73) ▪ *And interestingly enough, having lost the 10 or 15 kilos in the last 2 years, since I went on the LCHF, I'm sad to say that my cycling speed has not increased.* (C89) ▪ *I am not able to distinguish* [whether weight loss or diet is responsible for my improved exercise capacity and performance]. *The function of the diet is the weight loss and the function of the weight loss is that I am not carrying seventy-three extra kilo's… because of* [the weight loss] *I am able to exercise without the pain that came before and that is why I am fitter.* (T109) Pain ▪ before my T2D diagnosis] *I started to develop sore joints. Which actually, when I look back, I had it even when my children were babies, so in my thirties, slightly sore joints, which this* [LCHF] *diet has reversed completely.* (C343) ▪ [after adopting LCHF, avoiding added sugar and dairy] *… suddenly, more or less, the pain* [from arthritis and recurrent urinary tract infections] *is better, it's under control.* (C311) ▪ *I am now able to walk bare-feet in the house, which I could never before, it was too painful. I really couldn't have done that. I used to have to wear Mohair socks, 24 hours a day, I have just changed them three times a day, because my feet were permanently cold and that is not the case, anymore.* (T137) ▪ *The pain in my knees and my back, have disappeared.* (C17) ▪ *…my feet still hurt* [idiopathic neuropathy] *and I can't walk 2 or 3 miles anymore, that's impossible.* (C31) Mobility and activities of daily living

			▪ *… I couldn't walk even 50 meters by then* [at his most obese] *and so I needed to use a wheelchair. I also needed to buy a business class seat, because I could no longer fit in the tourist class seats, and even in the business class seat, I needed an extension to the seatbelt. And when I got into the, well when I got out of the car, waiting for the wheelchair to arrive, they sent the smallest woman on the planet to push the wheelchair and when she saw me, I realised what her face was saying to me. I got onto the chair and she couldn't move the wheelchair. So, she had to get help and they sent another small woman and so they each grabbed one side of the wheelchair and began to push, but because one was stronger than the other one, the wheelchair wobbled, it didn't move forward in the same direction and I heard people say to me, "Oh God, that aircraft is going to have a puncture when it lands."* *I had to be taken into the aircraft, via a lift on the outside through the galley and inevitably when you got to the airport, they made you take your shoes off and I was too big to put them back on again, so I would then stand there helpless until somebody would now realise I couldn't get my shoes back on. So, and when they said take your belt off, I had braces, because belts didn't fit me anymore. So, once you lose your braces, you keep your trousers up and grab your hand luggage and walk without shoes, it is not, it is not possible. By then I had also had to stop driving, because I would fall asleep behind the wheel anyway. And my world began to shrink. And I just wanted to do something, but I really didn't know what to do.* *And at my worst, I could walk around 50 to 100 metres, then I will have to sit down. So, even if I wanted to go shopping, the car had to be driven to the entrance, to one shop, I would go in, come back, sit in the car and will then drive to the next entrance. So, I could only go to certain shopping centres, where the car could park within a short distance. One of the celebrations I got to, when I got to 60 kilo loss, was to go Kirstenbosch and on that day I put the timer on my mobile phone, so I could measure how far I have walked and I was on that day able to do a 12 kilometre walk. I haven't been able to do that in 20, 25 odd years. So, for me that was a major thing. I can drive again. I can, my quality of life has been improved. I can walk if I really want to. I don't have to worry that I am going to lose my breath and there is not going to be a place to sit. Because that's the nightmare in a shopping centre. They don't have enough places for obese people to sit and rest and you can't. I mean even going to a restaurant, its fine but most of those chairs can't cope with 165 kilo. So, if you sit down, you are going to break the chair and that has happened. So, you know, in that sense the quality of my life improved.* (T137) ▪ *I feel a lot better and I'm 100% convinced it's because I'm a lot lighter. My knees are not sore. I'm up and about.* (C19) ▪ *I had to walk a distance of one and a half block* [to the clinic] *and* [previously, before LCHF] *I couldn't, because my ankles got so sore… I had to stop every 5 minutes… To let it relax and I couldn't walk. Now, I walk down to the clinic and back and it is a gravel road and it's a very steep uphill.* (T149) ▪ *…in 2012, the doctor said, "you are not allowed to drive a car, it's too dangerous you can have hypos or hypers". So, I wasn't allowed to drive. And now, a few weeks ago, I renewed my driver's license.* (C31) ▪ *The first time I was able to go to Woolies* [clothing shop] *and buy a shirt, I was completely, I was so traumatised, I couldn't do it. Because for how much of your life, you would go to a shop and you would say, "this is my size," and they say, "these are the three shirts, that will fit you". That is it. You choose from three. Now you are going to Woolies and suddenly there are 30 or 40 shirts that could fit you. And I didn't know how to make a decision. I actually had to get help, I had to get my friends to come with me and say, "it is too traumatic, I can't, I can't choose. I don't know how to do it anymore, I haven't learned how to choose clothes"…and*

			even now, this past weekend was the first time I bought a shirt, without help. I am finally now able to do it. So, in that sense the quality of my life… has changed on so many levels, ja [Afrikaans for "yes"]. (T137) Mental and psychological ▪ *I think at work I'm back to my normal self. As in mood, as in tolerance levels although occasionally my temper is a little bit short, shorter than it used to be.* (C13) ▪ *I'm more focused and I am able to concentrate more on a particular task or whatever. I don't find myself drifting off into a haze of daydreams or whatever, if that is the right description. Clarity of thinking.* (C17) ▪ *I haven't had that when I get home from work I'm sort of ratty because I am so hungry. I haven't had that at all so it definitely helped the mood swings relating to sugar.* (C343) ▪ *Just generally also like more alert, more awake, more alive, if you want to call it that. I don't have any of those other weird issues like sleep apnoea and all that kind of stuff. I've never had those kinds of problems either, but just generally you feel better.* (C79) ▪ *I think I'd become quite a bit depressed without ever really noticing it… and it's actually helped that.* (C79) ▪ *I am aware of being able to cope with stress better having lost weight. Things that used to stress me don't so much. Not that I am immune from stress but I am better able to cope with it. I kind of think my cognition is better, I think it's improved. I haven't tested it and I don't know, but I feel smarter. I also feel braver. Yes, having succeeded to lose so much weight it's given me a real confidence in myself.* (T109) Sleep ▪ *I sleep much better now, I sleep very well. I think the sleep only really improved once I'd lost a lot of weight.* (T109) ▪ *I noticed in the last couple of months, and it is only in the last couple of months, pretty much since my weight stabilised and my blood sugars have stabilised in the sort of 4 and 5 ranges, that I'm also sleeping a lot better. And I tend to have a good record of that because I've got the monitor… I can see how many times I must have woken up during the course of the night, and these days, I've quite often got 4 and 5 hours gaps, which had never happened 3 months ago.* (C97) ▪ [sleep] *started to improve, probably within 3 weeks of starting the lower carbs seriously. And the trigger was, I had to rush off to PE* [Port Elizabeth, a town in the Eastern Cape of South Africa] *and I left my machine behind. The machine that kept me breathing at night. Because I would fall asleep, even in meetings, I would fall asleep. And at night, I would wake up and realise that I had stopped breathing. And it was really very difficult to start breathing again, because I knew what I had to do: in my case, I had to jump out the bed, I had to run towards the cupboard and then I had to try and cough, so I could start the breathing process. But by then you know, you have wet the floor and, you know, your body has just done what it is supposed to do. And then you have got to go and shower. And this happened every night. So, later you become afraid of going to bed, because you know what is going to happen. And this machine, then*

			stopped that process… Every single night you had to plug it in and when there was a power failure, you couldn't sleep... But when I got to PE, I suddenly realised that I had left my machine behind and I didn't choke and that would have been a couple of weeks [of sleeping unassisted by the apnoea machine]. *I have kept the machine, just as a souvenir.* (T137) Skin ▪ *… look I always had a good skin, but my skin is getting better.* (T131) ▪ *My skin has improved. I do have sun damage from growing up at the sea as a youngster… but my doctor keeps telling me that I have far less wrinkles than I should for a sixty-six year old.* (C319) ▪ [Skin tags are] *suddenly not coming up as much as they used to.* (T107) Gastrointestinal ▪ *What's that!?… flatulence.* [Laughing] *I so seldom do that anymore and I used to do it all day, every day.* (T163) ▪ *Well I find that my bowels are much better, I don't get as much wind as I used to and I don't have a number 2 that often now, I have it about once every 3 or 4 days which is nice as well. And so that's changed for me. That's a definite plus I think. Especially at night-time, because I used to give off a lot of wind at night, so that has stopped.* (C83) *Also see quotes relating to worsening of constipation in *POST-LCHF* node, *Negatives and Challenges* child node above. Fertility ▪ *I've never actually been tested for polycystic ovarian syndrome. My husband and I have been married since I was 34, all of that weight and various other things meant that we never had the child we wanted. I lost all of the weight in 2014. September of 2015, I went to my GP and said, "okay, I've gone into menopause. I'm 40…" and this and this and this. So, she said well it could be that, or it could be that, or you're pregnant. I said don't be ridiculous, we've been waiting for like 6 years for a child and it's not going to happen… and my daughter is now a month old.* (C79) General health ▪ *I then got the benefit and spin of weight loss and all my blood numbers improving and normalising and I'm saying but hang on, my life has normalised.* (C13) ▪ *No, nothing* [no issues] – *all back to normal.* (C19) ▪ *You feel so much better and you know, I am amazed. It is so amazing. I always think being healthy and in a good condition, peak condition, that is our birth right. Because I am also always amazed at how quickly you forget in what state you were and you completely forget all the aches and pains. It feels like it's always been like that, you know? But it wasn't. I remember very well before, you know, I have to force myself to remember that. Because the natural wellness feels so, the healthy wellness feels so natural.* (C311)

			▪ *It's taken me three years but my body has healed astronomically. It's been amazing* (C319) ▪ *So really my health has improved so much. If you knew me then and you know me now, you'd be amazed.* (C319) ▪ *Knowing how I feel and how good I feel health-wise and weight-wise, I would say, I would never want to go back to the other way, because I wouldn't want the battles that I had with the excess weight and with the diabetes particularly, and all the other health problems, which have gone, and it has been like a miracle… I think I have got my life back and for me that is really, that is really important. You know, I have got my life back.* (T127)
WEIGHT		Any mention. Must refer to their weight. Exclude: "my family is overweight" etc.	▪ *All of my adult life, I was too heavy and I always wanted to find a way to fix it, to change it to make myself better, but I couldn't find a way until I found about LCHF. And since then, like I said, before I've lost about 130 pounds which is about 60 kilos of weight loss…I've never been able to lose weight for more than a few months and I've been at it for almost a year now, so I would say I'm pretty much going to eat that way for the rest of my life.* (C29) ▪ *…stuck to it* [LCHF] *for like 10 days and lost 6.4 kg's. So that kind of like sold me, forget about blood sugar results or anything else, it was like 6.4 kg's, I mean when I was on the Weigh Less diet for a year, I lost like 4 kg's.*(C79) ▪ *I think that the weight loss also motivated me because previously I couldn't lose weight.* (C19) ▪ *I was a size 16, I am a size 14 now. So, even if it is in a year, I only got down one size, quite frankly, I don't care.* (T131) ▪ *I added the fat and from that, end of November to my next medical of call it March, is where I dropped probably about 10 kgs. I'm not too sure what the weight was, but in that time, all my blood markers just improved…* (C13) ▪ *I was always an overweight child and no matter what sport I did I would firm up but I will never lose weight. You know I'd be one of these real like muscular, chunky type sports people, you know?* (T101) ▪ *I was trying, but I couldn't* [keep the weight off]*, every year or every other year, I would start a diet, lose some weight and gain it back.* (C29) ▪ *The weight that I lost at the beginning of this year, during the very low calorie* [diet shake] *2 months,* [and] *my HbA1c, remained normal at normal levels since then.* (C17) ▪ *And I really believe that that was one of the triggers that allowed me to lose weight so fast. The fact that I stopped, I reduced the Insulin, I really believe that, that was one of factors.* (T137)

APPENDIX J: MISSING FOOD ITEM LIST

SHORTNAME	kjoul	prot_tot	pl_prot	an_prot	fat_tot	satfat	mufat	pufat	choles	cho_tot	sug_tot	sug_add	fib_tot	ca	fe_tot	mg	p	k	na	zn	cu	mn	vita_re	thiamin	ribofl	niacin	vit_b6	folate	vit_b12
Samphire	45	3.1	3.1	0	0.7	0.1	0.1	0.2	0	9.1	0.6		0.5	150	2.2	110	80	50	870	0.4	0.3		0	0	0		0		
Capers	96.3	2.4	2.4	0	0.9	0.2	0.1	0.3	0	4.9	0.4		3.2	40	1.7	33	10	40	2964	0.3	0.4		138	0	0.1	0.7	0	23	0
Seaweed, roasted	1247	31.84	31.84	0	4.01	1.356	1.091	1.091	0	52.39	3.04		5.6					1244	575										
Hummus	1105	6.6	6.6	0	22	3.4			0	9.8	0		2.7						342										
Yoghurt, double cream, plain	370	3.6			7.3	4.8			114	2	2		0						57										
Yoghurt, double cream, Greek	405	3.9			4.4	2.9	0.2	0.2	68	12	10		0.7						47										
Yoghurt, Greek	490	4.05			9.19	5.716	0.32	0.32	35	5.04	4.62		0					156	55										
Yoghurt, full cream, plain	268	3.3			3.4	2.4			11	5	4.6		0.2						57										
Quiche,with Meat, Poultry or Fish	1238	8.61			22.83	10.518	2.892	2.892	126	14.24	0.35		0.4					139	362										
Quiche, vegetable, crustless	662	9.1			12	7	1	1	112	3	0		2.6						392										
Cheese grillers	1295	16			28.5	12	4.7	4.7	10	1	0.3		1						750										
Droewors, beef	1920	38.3	0	38.3	34.3	18.3			111	0	0		0						1901										
Droewors, game	1234	62		62	4.3	2			124	1	0		0.3						2408										
Pie, Pepper Steak, commercial, baked	448.3	2.6			8.14	5	0.63	0.63		4	1.2		2.7						230.6										
Pate, duck liver	1492.43	10.7			33	10	16	5	93.3	4	3.3		0					190	556.7										
Sushi, california roll	527	5.29			0.47	0.085	0.166	0.166	14	24.27	4.99		0.9					128	223										
Mayonnaise, LCHF / Banting / creamy	1851	2.2			52	6.5			0	1	0.2		1						1055										
Ice Cream, LCHF / Banting	515	3.8			7.6	5.3			22	9	4		2.5						48										
Avocado oil	3699	0			100	11.56	13.486	13.486		0	0		0					0	0										
Sesame oil	3699	0			100	14.2	41.7	41.7	0	0	0		0					0	0										
Chocolate, dark, 90% cacao	2510.4	10		0	55	32.5	22.5	22.5	0	30	7.5		12.5																
Chocolate, dark, 85% cacao	2197.5	10			45	27.5			0	30	12.5	12.5	7.5						50										

Chocolate, dark, 70% cacao	2615	7.5			47.5	30			0	42.5	30	30	7.5						25										
Cinnamon	1034	4	4		1.2	0.3	0.2	0.1	0	81	2.2		53.1	1002	8.3	60	60	431	10	1.8	0.3	17.5	88.5	0	0	1.3	0.2	6	0
Banting tomato sauce	293	2.5			4	0.5	0.6	0.6		4	3.4								750										
Periperi sauce	280	0			4	0				8	8		0						2480										
Pesto	837	4.6	4.6		0.5	0.06	0.07	0.07	0	77.3	0		77.3	180	0.36			0	125										
Almond Milk	71.128	0.62	0.62		1.46	0.112	0.93	0.353	0	0.57	0.14		0	10	0			21	2										
Psyllium husk	837	4.6			0.5	0.06	0.07	0.07	0	77.3	0		77.3	333.3	2.22			0	125										
Konjac products	126	0.39			0.04	0.009	0.019	0.019	0	7.06	0.13		1					207	4										
Erythritol	17	0			0	0			0	100	0		0						0										
Equal, artifical sweetener	600	38			0	0				42									60										
Xylitol	1092	0			0	0	0	0	0	70.84	63.25		0					3	1										
Sucralose, artifical sweetener	200	20			20	20				200	20		20						200										
Natreen, artifical sweetener	1400	0								80																			
Seed crackers	2470	29			34	6	1	1	90	42	3		9						160										
Banting / LCHF bread	1586	14.62			31.9	4.769	12.474	12.474	85	14.15	2.32		8.3					448	356										
Banting / LCHF granola	2129	17			48	21				4	3		4						230										
Heba Pap	1589	21.7			21.9	7.3				40.8	8		32.8						72										
Banting / LCHF pizza base	577	14.54			7.22	3.243	0.697	0.697	77	3.56	1		1.5					144	535										
Snack Bars LCHF	1672.5	35.5			35	12.5	5	0	25	37.5	2.5		22.5	80	1.6			125	475										
Banting/LCHF wraps	865.45	18.2			10.55	2.37			42.09	2.73	2.55		2.73						556.92										
Kombucha	55.7	0			0	0	0	0	0	3.1	0.9		0	0	0	0	0	0	4.4	0	0		0	0.1	0.1	0.9	0.1	23	0.3