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Research article

Establishing a proof of concept for the effects of low-carbohydrate, high-fat diet (LCHFD) and physical activity on body composition in type 2 diabetes



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Gerrit J. Breukelman^{a,*}, Albertus K. Basson^b, Trayana G. Djarova^b, Brandon S. Shaw^a, Cornelia J. du Preez^c, Ina Shaw^a

^a Department of Human Movement Science, University of Zululand, KwaDlangezwa, 3886, South Africa

^b Department Biochemistry and Microbiology, University of Zululand, KwaDlangezwa, 3886, South Africa

^c Department of Consumer Sciences, University of Zululand, KwaDlangezwa, 3886, South Africa

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ABSTRACT

Overweight and obesity are both a risk factor for developing and exacerbating type 2 diabetes (T2D). While the most common diet used to treat overweight and obesity focus on high-carbohydrate, low-fat, energy deficit diets, recently, low-carbohydrate, high-fat diets (LCHFD) have become popular in targeting obesity. This proof-ofconcept study attempted to determine if an LCHFD could improve body composition variables, or if a concurrent treatment of LCHFD and physical activity would create an interference effect in individuals with T2D. Overweight and obese with T2D (n = 39) were assigned into either a 16-week combined physical activity and LCHFD group (ConG), LCHFD-only group (DieG) or control group (NonG). No statistically significant (p > 0.01) changes were found in body mass in the ConG (2.0%, F = 0.039, P = 0.846) and DieG (2.5%, F = 0.188, P = 0.188, P0.669); for body mass index in the ConG (2.2%, F = 0.046, P = 0.832) and DieG (2.3%, F = 0.098, P = 0.758.); and waist-to-hip ratio in the ConG (0%, F = 0.002, P = 0.968) and DieG (0%, F = 0.023, P = 0.882). However, clinically significant changes were observed in HbA1c in the ConG male group (23% decrease); percentage body fat for the ConG (16.7%, F = 1.682, P = 0.208, g = 0.534) and DieG (13.0%, F = 0.638, P = 0.435, g = 0.361); for waist circumferences in the ConG (5.4%, F = 0.686, P = 0.416, g = 0.341) and DieG (6.3%, F = 1.327, P = 0.264, g = 0.520); and for hip circumference in the ConG (5.8%, F = 0.993, P = 0.329, g = 0.410) and DieG (7.0%, F = 0.520); 2.668, P = 0.119, g = 0.737). Results indicate that moderate clinically significant changes in body composition are achievable with LCHFD and/or daily walking in obese adults living with T2D. However, more robust research is required to determine the effects of LCHFD, with or without concurrent physical activity, on obesity and other diabetic complication markers.

1. Introduction

Overweight and obesity are global health problems of epidemic proportions and currently combined affect more than 1.1 billion adults (Elagizi et al., 2018). Obesity is a risk factor for cardiovascular disease and metabolic diseases, such as type 2 diabetes (Elagizi et al., 2018). As such, the prevalence of diabetes is estimated to be 2.8% affecting 171 million individuals (Wild et al., 2004). Correspondingly, both diabetes and obesity create enormous healthcare costs (American Diabetes Association, 2018). Obesity and diabetes continue to increase in both sexes, at all ages and all educational levels (Mokdad et al., 2003). Problematically, overweight and obesity increase the degree of association between body composition and cardiovascular and/or metabolic risk factors in individuals with type 2 diabetes (Wing et al., 2011). This association requires the essential maintenance of optimal body composition to reduce morbidity and mortality associated with type 2 diabetes (Wing et al., 2011).

Although the aetiology of obesity is complex, obesity has been believed to be due to energy intake (i.e. through diet) exceeding energy expenditure (i.e. through physical activity) (Elagizi et al., 2018). As such, improvements in body composition have focused on either energy intake or energy expenditure, or both (Elagizi et al., 2018; Soenen et al., 2012). In this regard, the most used and clinically recommended diets to improve or maintain an ideal body composition have been found to be high-carbohydrate, low-fat, energy deficit diets (Avenell et al., 2004; Foster et al., 2003). Regardless of these guidelines that fat intake should

* Corresponding author. E-mail address: BreukelmanG@unizulu.ac.za (G.J. Breukelman).

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only be on occasion and in small quantities, obesity prevalence rates continue to increase (Breukelman et al., 2018; Santos et al., 2012). This has led to the rise of numerous novel, and popular, treatments such as the low-carbohydrate, high-fat diets (LCHFD) (Foster et al., 2003). This type of diet is defined as consuming no more than 50 g (g) of carbohydrates and up to 1800 calories from saturated fats daily (Volek and Westman, 2002). LCHFD seemingly have a multitude of purported positive effects, including improvements in body mass, improved glycemic control, reduction in cardiovascular disease risk factors, increased total energy expenditure due to increased thermal effects and increased feelings of satiety (Demol et al., 2009; Noakes and Windt, 2017). Despite the numerous purported benefits of LCHFDs, a high-fat diet is considered a risk factor for the development of overweight/obesity. Although the LCHFD has become popular in targeting obesity, their efficacy in individuals with type 2 diabetes may prove ineffective, and possibly dangerous due to the potential precipitation of acidosis as a result of a high-fat intake (Salas-Salvadó et al., 2011). Furthermore, Boden et al. (2005), indicated that a LCHFD may prove ineffective in individuals with type 2 diabetes, since non-diabetic individuals release less postprandial insulin when restricting carbohydrate intake. In addition, LCHFDs have not yet unequivocally proven to mitigate weight gain and does not always improve glucose tolerance, insulin secretion (Lamont et al., 2016). While there is an undoubted desirability of omitting carbohydrates via an LCHFD in diabetics, the possible precipitation of acidosis via a high-fat intake may not warrant their use (Salas-Salvadó et al., 2011). In addition, the efficacy of a high-fat diet has limited evidence for metabolic advantages leading to improvements in body composition and we hypothesise that an LCHFD, despite its popularity, will not improve body composition variables. In addition, since the administration of more than one treatment may influence another treatment though the different molecular mechanisms mediating adaptation, a phenomenon known as multiple treatment interference, the addition of physical activity to an LCHFD may even create an interference effect that offsets the benefits of the addition of physical activity (Shaw et al., 2009). To this point, this study aimed to determine if an LCHFD could improve body composition variables, or if a concurrent treatment of LCHFD and physical activity would create an interference effect in type 2 diabetics. This would provide support for guidance to those individuals living with type 2 diabetes to help them understand the preferred strategies to prevent the progression of diabetic complications.

2. Methodology

2.1. Study design

The present study employed a small scale proof-of-concept investigation pretest-posttest design. Purposive sampling was utilised to recruit participants who volunteered to participate in this study. Participants were recruited from a Diabetic Clinic in the Zululand area, Kwazulu-Natal, South-Africa and were all diagnosed with type 2 diabetes. Participants were randomly assigned into either a concurrent physical activity and LCHFD group (ConG) (n = 13; 41–71yrs.), an LCHFD only group (DieG) (n = 13; 31–71yrs.) or a control group (NonG) (n = 13, 44–69yrs.) using an intention-to-treat analysis. Random assignment was ensured to minimise selection bias.

2.2. Study participants

Thirty-nine (28 female and 11 male) type 2 diabetics, aged 31–71 years, were recruited for the present intention-to-treat study. Inclusion criteria required that all participants be free of any absolute or relative contraindications to exercise (American College of Sports Medicine (ACSM), 2014) and were medically and clinically stable and ambulant without any aids. Eligibility criteria included a diagnosis of type 2 diabetes (i.e. oral glucose tolerance test (OGTT) levels >11.1 mmol per litre (mmol.L⁻¹) at two hours and lack of islet cell antibodies), overweight or

obese (BMI >25 and >30 kg per square meter (kg.m⁻²), respectively) (ACSM, 2014) adult with a previously sedentary lifestyle (did not participate in regular exercise more than twice a week), stable body mass (± 2 kg) over the past year, and having had no change in their regular medication usage for at least six months prior to enrolling in the study. All participants were screened and received approval from a medical doctor at the clinic for participation in the study. Participant baseline characteristics are included in Table 1.

2.3. Ethical approval

Ethical approval was obtained from the Institutional Review Boards of the University of Zululand (UZREC 171110-030), South Africa. Participants provided written informed consent and were able to withdraw from the study at any time. Participants' confidentiality and anonymity were ensured throughout the study.

2.4. Anthropometric assessment

Anthropometric measurements were carried out according to the methods proposed by the International Society for the Advancement of Kinanthropometry (ISAK) (Norton and Olds, 1996) and measured by the same technician (registered biokineticist and level 1 ISAK-certified). All measurements were measured at least twice, and if large differences were found between measurements, a third measurement were taken (ACSM, 2014). Rotation throughout the measuring sequence were used to allow the body to regain normal texture. The results were based on the average of the different measurements. Body mass (BM) was measured in kilogrammes (kg), to the nearest 0.1kg on a calibrated medical scale (Micro RGT-200 Health Scale), whilst stature was measured in meters (m), to the nearest millimetre, using a standardised stadiometer (Marsden H-628 Free Standing Height Measure, UK). Body mass index (BMI) was calculated by dividing the participant's body mass (kg) by stature squared (m²) and expressed as kilogrammes per square meter (kg.m⁻²). Skinfolds (subscapular, tricep, suprailliac, abdominal, thigh and calf) were taken on the right side of the body using a Lange skinfold caliper in a rotation sequence (Cambridge Scientific Industries, Inc. Maryland, USA) and percentage body fat (%BF) was calculated using the equation of Jackson and Pollock (1978). Waist circumference was measured with the participant standing upright, with their arms at the sides of the body and feet together. A horizontal measurement was taken at the narrowest part between the umbilicus and the xiphoid process of the torso (ACSM, 2014). Hip circumference was measured with the participant standing with their legs slightly apart. A horizontal measurement was taken at the maximal circumference of the hip (ACSM, 2014). Waist-to-hip ratio (WHR) as a ratio measurement of the circumference of the waist to that of the hip was calculated by the following equation: WHR = waist circumference \div hip circumference (ACSM, 2014).

2.5. Intervention programme

The ConG and DieG were required to follow an LCHFD requiring participants not to eat more than 50g of carbohydrates per day for the 16week experimental period (Volek and Westman, 2002). Participants in these experimental groups were given three lists and were shown that they were able to eat any foods listed on the green list, minimal foods listed on the orange list, but not to consume any foods off of the red list (Noakes et al., 2013). The participants were provided with a single set of instructions on how participants were to document food intake to complete the self-report booklet records at the initial meeting. Every four weeks throughout the 16-weeks of the study, the self-report booklet was collected, and a new self-report document-booklet was given to each participant to complete their food consumption for that following four-week period. The participants were also contacted via phone every

Table 1. Participants baseline characteristics data.

	Groups Females/Males	Females	Males	Baseline	Post test			
Age per group	ConG F (n = 10)	42–73yr						
	ConG M $(n = 2)$		55–67yr					
	DieG F ($n = 6$)	33–61yr						
	DieG M $(n = 4)$		37–72yr					
	NonG F $(n = 9)$	49–70yr						
	NonG M $(n = 4)$		59–65yr					
Age of diagnosis of type 2 diabetes	ConG F (n = 10)	30–57yr						
	ConG M $(n = 2)$		25–62yr					
	DieG F ($n = 6$)	31–58yr						
	DieG M $(n = 4)$		30–70yr					
	NonG F $(n = 9)$	25–62yr						
	NonG M $(n = 4)$		32–50yr					
Duration of type 2 diabetes	ConG F (n = 10)	3–24yr						
	ConG M $(n = 2)$		5–30yr					
	DieG F ($n = 6$)	2–15yr						
	DieG M $(n = 4)$		2–7yr					
	NonG F ($n = 9$)	2–31yr						
	NonG M $(n = 4)$		15–27yr					
Medication	ConG F (n = 10)	Glucophage (500–1	Glucophage (500–1000mg); Januvia (50mg)					
used at baseline	ConG M $(n = 2)$	Glucophage (500–1	Glucophage (500–1000mg)					
	DieG F $(n = 6)$	Glucophage (500–1	Glucophage (500–1000mg); Mengen (850mg); Gliclazide (80mg)					
	DieG M $(n = 4)$	Glucophage (500–1	Glucophage (500–1000mg); Diaglucide (60mg)					
	NonG F $(n = 9)$	Glucophage (500–1	000mg); Diaglucide (60mg) Bigs	ens (1000mg); Glucovance (50	Omg)			
	NonG M $(n = 4)$	Glucophage (500–1	Glucophage (500–1000mg); Diaglucide (60mg); Insulin glargine (100ulml); Januvia (50mg)					
Co-morbidities	ConG F $(n = 10)$	Asthma; High bloo	Asthma; High blood pressure; High Cholesterol					
	ConG M $(n = 2)$	Asthma; High bloo	d pressure; High Cholesterol					
	DieG F ($n = 6$)	High blood pressur	e; High Cholesterol					
	DieG M $(n = 4)$	High blood pressur	e; High Cholesterol					
	NonG F ($n = 9$)	High blood pressur	e; High Cholesterol					
	NonG M $(n = 4)$	Asthma; High bloo	d pressure; High Cholesterol					
HbA1c	ConG F $(n = 10)$			$\textbf{6.90} \pm \textbf{1.592}$	$\textbf{6.57} \pm \textbf{1.477}$			
	ConG M $(n = 2)$			9.35 ± 0.250	$\textbf{7.10} \pm \textbf{0.20}$			
	DieG F ($n = 6$)			5.93 ± 0.774	5.92 ± 0.437			
	DieG M $(n = 4)$			5.68 ± 0.303	5.73 ± 0.349			
	NonG F $(n = 9)$			$\textbf{8.06} \pm \textbf{1.949}$	8.21 ± 1.982			
	NonG M $(n = 4)$			7.23 ± 1.465	$\textbf{7.75} \pm \textbf{0.923}$			
Glucose levels	ConG F (n = 10)			$\textbf{7.45} \pm \textbf{2.263}$	$\textbf{7.06} \pm \textbf{1.250}$			
	ConG M $(n = 2)$			10.78 ± 0.62	7.71 ± 0.210			
	DieG F ($n = 6$)			5.93 ± 0.467	5.86 ± 0.672			
	DieG M $(n = 4)$			$\textbf{6.83} \pm \textbf{0.689}$	5.50 ± 0.579			
	NonG F ($n = 9$)			$\textbf{7.93} \pm \textbf{1.723}$	$\textbf{8.34} \pm \textbf{1.820}$			
	NonG M $(n = 4)$			7.08 ± 1.465	7.39 ± 1.481			

week to insure compliance and to remind them to complete the self-report booklet. The self-report booklet records were reviewed in detail after each meeting with the participants. In addition to the LCHFD, the ConG engaged in a 16-week physical activity programme entailing walking a minimum of 10 000 steps daily (Tudor-Locke et al., 2011) (measured using a pedometer wristband). All the participants in each group received a personal physical activity logbook to record their number of steps daily. With every assessment, the ConG were required to bring their physical activity logbook to the assessment, where it was reviewed to confirm that they were still complying to the 10 000 steps daily. The participants were also contacted via phone every week to ensure compliance and to remind them to complete their physical activity logbook. The NonG were required to continue their normal activities and not to follow any diets or structured exercise programme throughout the 16-week experimental period.

2.6. Data analysis

Variables were reported as means±standard deviations (SD). For all measured variables in this study, the small sample size necessitated that the normality assumption could not be met and use of a more conservative p-value (0.01 rather than 0.05) was utilised for conducting significance tests. Paired-samples t-tests were utilized to examine the differences between pre-test and post-test variables. A two-way (group × time) repeated measures analysis of variance (ANOVA) were utilized to examine the differences between pre-test and post-test variables. Hedges g was used to determinate the effect size (the value of 0.2 was considered for small effect, 0.5 for moderate effect, and 0.8 for large effect) (Brydges, 2019). Version 25.0 of the IBM Statistical Package for the Social Sciences (SPSS) for Windows (IBM Corporation, Armonk, NY) was used for all data analysis.

3. Results

Of the 39 participants recruited, 35 completed the study (ConG n = 12, DieG n = 10 and NonG n = 13). Reasons for the dropout of the four participants include: two participant's unavailability to be tested throughout the 16 weeks; one participant's general practitioner advising leaving the study without explanation; and the other participant had a family incident resulting in withdrawal from the study. No adverse responses to the physical activity were observed or reported. Table 2 demonstrates the findings regarding body mass, body mass index, %BF, waist circumferences, hip circumference and waist-to-hip ratio following the 16-week experimental period.

Following the experimental period, t-tests indicated that no significant (p > 0.01) changes were found for body mass in the ConG (p = 0.423; 2.0% decrease) and DieG (p = 0.335; 2.5% decrease); for body mass index in the ConG (p = 0.415; 2.2% decrease) and DieG (p = 0.379; 2.3% decrease); for %BF in the ConG (p = 0.104; 16.7% decrease) and DieG (p = 0.217; 13.0% decrease); for waist circumferences in the ConG (p = 0.208; 5.4% decrease) and DieG (p = 0.132; 6.3% decrease); for hip circumference in the ConG (p = 0.165; 5.8% decrease) and DieG (p = 0.059; 7.0% decrease) and waist-to-hip ratio in the ConG (p = 0.484; 0% difference) and DieG (p = 0.441; 0% difference).

Following the experimental period, ANOVA demonstrated that no significant (p > 0.01) changes occurred for body mass in the ConG (2.0%, F = 0.039, P = 0.846) and DieG (2.5%, F = 0.188, P = 0.669); for body mass index in the ConG (2.2%, F = 0.046, P = 0.832) and DieG (2.3%, F = 0.098, P = 0.758.); for %BF in the ConG (16.7%, F = 1.682, P = 0.208) and DieG (13.0%, F = 0.638, P = 0435.); for waist circumferences in the ConG (5.4%, F = 0.686, P = 0.416) and DieG (6.3%, F = 1.327, P = 0.264); for hip circumference in the ConG (5.8%, F = 0.993, P = 0.329) and DieG (7.0%, F = 2.668, P = 0.119) and waist-to-hip ratio in the ConG (0%, F = 0.002, P = 0.968) and DieG (0%, F = 0.023, P = 0.882).

In the ConG, small effect sizes were found for waist circumference (g = 0.341) and hip circumference (g = 0.410), with a moderate effect size being found for %BF (g = 0.534). In the DieG, small effect sizes were observed for %BF (g = 0.361), while moderate effect sizes were found for waist circumference (g = 0.520), and hip circumference (g = 0.737). In the NonG, all effect sizes were established to be below 0.2.

4. Discussion

The present study aimed to established if LCHFD would be beneficial to overweight or obese type 2 diabetic participants, and not comparing nutritional states between the LCHFD and traditional diabetic diets. In this regard, the present study failed to elicit a change in body mass and this finding is in contrast to Katan (2006) and Krieger et al. (2006) who found that an LCHFD decreased body mass in their non-diabetic samples. However, even a reduction in body mass by 5-10% could significantly improve health in patients with cardiovascular and metabolic disease risk factors associated with obesity (Krousel-Wood et al., 2008). In this regard, the minimal 2.2% and 2.3% decreases in body mass in the ConG (Table 2 and Figure 1), DieG (Figure 2) and NonG (Figure 3) respectively, failed to demonstrate even clinically significant improvements. In this regard, Stevens et al. (2006) defined weight maintenance as < 3% body mass change, and any change >5% as clinically significant. Clinical significance can also be described as any changes observed which could be considered as important or worthwhile by the researcher or participant (Page, 2014) and Page (2014) has proposed effect size as one of the most important pointers to predict clinical significance. This minimal change in body mass in the ConG could be explained by Breukelman et al. (2013), which stated that an increase in lean body mass through exercise could alter body mass, especially when a decrease of fat percentage (Figure 4) is noted, as this group was required to walk daily.

In addition, the lack of change in BMI in this study is in line with Naude et al. (2014), who found that there were little or no differences in weight loss and BMI following in their meta-analysis examining the effects of LCHFD's on some indices of body composition. However, the present study and that of Naude et al. (2014) are in contrast to Ruth et al. (2013), who found that an LCHFD led to a decrease in body mass (-7.1 \pm 4.6% of body mass), body fat (-2.5 \pm 2.9%) and BMI (-2.5 \pm 1.5 kg.m⁻²). While the study of Ruth et al. (2013) and the present study had a similar cohort (i.e. age: 21–62 years vs. 31–71 years and n = 18 vs. DieG: n = 10, respectively) and a similar study design (i.e. 12 weeks vs. 16 weeks, respectively), the study of Ruth et al. (2013) utilised obese only participants while, the present study utilised overweight and obese participants. These higher initial values in adiposity could then possibly explain the

	Groups	Pre-test	Post-test	p-value	% Difference
Body mass (kg)	ConG (n = 12)	89.40 ± 21.649	87.591 ± 21.531	0.846	↓2.0
	DieG (n = 10)	104.70 ± 13.436	102.07 ± 12.273	0.669	↓2.5
	NonG (n = 13)	104.877 ± 31.642	105.546 ± 31.595	0.959	↑0.6
Body mass index (kg \cdot m ⁻²)	ConG (n = 12)	32.417 ± 7.577	31.725 ± 7.531	0.832	↓2.2
	DieG (n = 10)	38.860 ± 5.753	38.0 ± 5.897	0.758	↓2.3
	NonG $(n = 13)$	38.231 ± 10.244	38.292 ± 10.079	0.988	↑0.3
Percentage body fat (%)	ConG (n = 12)	37.661 ± 13.167	31.438 ± 8.936	0.208	↓16.7
	DieG (n = 10)	36.202 ± 14.554	31.526 ± 9.836	0.435	↓13.0
	NonG (n = 13)	34.812 ± 15.418	35.315 ± 13.252	0.932	↑1.4
Waist circumference (cm)	ConG (n = 12)	98.458 ± 15.489	93.167 ± 14.456	0.416	↓5.4
	DieG (n = 10)	110.0 ± 14.029	103.1 ± 11.229	0.264	↓6.3
	NonG $(n = 13)$	112.231 ± 15.719	112.538 ± 17.332	0.964	↑0.3
Hip circumference (cm)	ConG (n = 12)	115.858 ± 16.747	108.917 ± 15.908	0.329	↓5.8
	DieG (n = 10)	125.350 ± 11.807	116.70 ± 10.631	0.119	↓7.0
	NonG $(n = 13)$	121.385 ± 20.439	120.308 ± 24.524	0.908	↓0.9
Waist-to-hip ratio	ConG (n = 12)	$\textbf{0.850} \pm \textbf{0.104}$	0.852 ± 0.091	0.968	0
	DieG (n = 10)	0.876 ± 0.073	0.881 ± 0.068	0.882	0
	NonG $(n = 13)$	0.940 ± 0.105	0.942 ± 0.103	0.966	0

Table 2. Body composition in individuals with type 2 diabetes following low-carbohydrate, high-fat diets with/without physical activity.

Data reported as means±standard deviations (SD).

ConG: combined physical activity and low-carbohydrate; high-fat diet group; DieG: low-carbohydrate; high-fat diet group; NonG: control group; kg: kilogrammes; kg.m⁻²: kilogrammes per square meter; %: percent; cm: centimeters.



Figure 1. Body Mass changes in the 16-week combined physical activity and low-carbohydrate, high-fat diet (LCHFD) group (ConG).



Figure 2. Body Mass changes in the 16-week low-carbohydrate, high-fat diet (LCHFD)-only group (DieG).



Figure 3. Body Mass changes in the 16-week control group (NonG).



Figure 4. Fat percentage changes in the 16-week combined physical activity and low-carbohydrate, high-fat diet (LCHFD) group (ConG).

reason for improvements in the study of Ruth et al. (2013), but not that of the present study.

Despite the present study not eliciting changes in body mass and BMI, clinically significant findings were found for %BF in both the ConG (16.7%, g = 0.534) and DieG (Figure 5) (13%, g = 0.361), compared to the NonG (Figure 6) (1.4%, g = -0.034). These changes in %BF could have health benefits for a type 2 diabetic as described by Wing et al.

(2011), who stated that an optimal body composition will reduce morbidity and mortality associated with type 2 diabetes. This decrease in fat percentage could also possibly elucidate the 23% decrease in HbA1c and 28% decrease in glucose for the ConG male group. This supposition is further supported by Avenell et al. (2004), who stated that diets resulting in weight loss, can decrease and even prevent type 2 diabetes. These findings of clinically improved %BF are in contrast to the supposition of



Figure 5. Fat percentage changes in the 16-week low-carbohydrate, high-fat diet (LCHFD)-only group (DieG).



Figure 6. Fat Percentage changes in the 16-week control group (NonG).

Hall et al. (2015), who proposes that a restriction of dietary fat leads to greater body fat loss than restriction of dietary carbohydrates in overweight and obese adults.

The lack of improvement in WHR in the present study, as a proxy for intra-abdominal fat (Gadekar et al., 2017) could demonstrate a lack of improvement in insulin resistance (Alfred et al., 1995) in this study's sample of overweight and obese type 2 diabetics. However, it must be noted that WHR may not reflect visceral fat, which is the more important indicator of insulin resistance (Alfred et al., 1995). This study's finding regarding a lack of change in WHR is in line with Gardner et al. (2007) who found no significant change in WHR following a 12-month high-fat diet.

Although no statistical, or clinically, significant changes were found for WHR, clinically significant changes were observed in ConG (Figure 7 and Figure 8) and DieG's (Figure 9 and Figure 10) individual waist and hip circumferences, especially when compared to the lack of change in the NonG (Figure 11 and Figure 12). While Hu et al. (2012) found no changes in waist circumference in their meta-analysis on LCHFD's, McAuley et al. (2005) found that a 24-week high-fat diet statistically significantly improved waist circumference more than high-carbohydrate diet. In the present study, the results for both the ConG (Table 2 and Figure 7) and DieG (Figure 8) groups indicate a clinically significant improvement in waist circumference, especially when considering the effect sizes of these experimental groups (g = 0.341and g = 0.520, respectively). This is especially important in that waist circumference alone can be utilised as a health risk indicator (ACSM, 2014).

It was supposed that the addition of a physical activity programme to the LCHFD could provide additional potential benefits for a type 2 diabetic's body composition. Some of the specific benefits that exercise has proposed to have on body composition include weight loss, weight control, prevention of weight gain and regain (Swift et al., 2018). It is well documented that with weight loss and an increase in physical activity, there is an improvement in metabolic control, a reduced risk of developing diabetes and a decrease in cardiovascular conditions (ACSM, 2014). The results of the present study indicate that moderate clinically significant changes in body composition are achievable with LCHFD and/or daily walking in overweight and obese adults living with type 2 diabetes. These changes in body composition may exert a positive impact on many cardiometabolic risk factors in type 2 diabetics (ACSM, 2014).

The moderate improvement of body composition in the DieG could be as a result of this group being motivated to alter the effects of diabetes as they were the group with the shortest duration living with type 2 diabetes (Table 1) (female: 2–15 years and male: 2–7 years). In this regard, Turner et al. (1999) found that less than 25% of type 2 diabetics are able to control their blood glucose with only one medication, once they have lived with diabetes for nine years. Furthermore, Nathan (2002), stated that more than 50% of diabetic patients will require insulin following 10–15 years of diagnosis.

Furthermore, the study of Hall et al. (2015) found that only their carbohydrate-restricted diet led to decreased insulin secretion and a substantial sustained increase in net fat oxidation. However, this positive effect of a decreased insulin secretion would prove ineffective in individuals with type 2 diabetes since this condition is marked by insulin resistance and a progressive lack of insulin (Taylor, 2012). In terms of the concurrent group, the administration of physical activity may have influenced the dietary effects (if any) through different molecular mechanisms mediating adaptation (Shaw et al., 2009). Further, increasing evidence is demonstrating that freely-paced walking, as in the case of self-selected intensity for 10000 steps, does not meet minimum intensity guidelines for health improvement and/or changes in body composition (Carstensen et al., 2008). The aerobic mode of exercise selected in the present study may also not have been ideal to optimise body composition changes. Furthermore, Shaw et al. (2010) found that resistance modes of exercise increase metabolically active tissue, and not aerobic modalities as utilised in the present study. This increase in lean/muscle tissue then increases fuel, and specifically fat, oxidation (Shaw et al., 2010, 2015). Future studies should investigate the effect of dietary interventions with hypertrophic-specific resistance training as they may prove particularly beneficial for individuals with type 2 diabetes since resistance-trained muscle is also more receptive to lower amounts of insulin, resulting in an enhanced uptake of glucose (Shaw and



Figure 7. Waist circumference changes in the 16-week combined physical activity and low-carbohydrate, high-fat diet (LCHFD) group (ConG).



Figure 8. Hip circumference changes in the 16-week combined physical activity and low-carbohydrate, high-fat diet (LCHFD) group (ConG).



Figure 9. Waist circumference changes in the 16-week low-carbohydrate, high-fat diet (LCHFD)-only group (DieG).



Figure 10. Hip circumference changes in the 16-week low-carbohydrate, high-fat diet (LCHFD)-only group (DieG).

Shaw, 2008). It should also be noted that the small and moderate changes in body composition found in the present study may be due to a lack of adherence to the LCHFD or concurrent regimes. While the present study made use of food records and physical activity logbooks, such outpatient studies are difficult to interpret mechanistically because it is not currently possible to accurately measure adherence to the recommended diets and physical activity programmes since such instruments rely on self-report and have been demonstrated to be biased (Winkler, 2005). However, although there are many problems with using self-report measures, they will continue to be a popular methodology until alternative, cost-effective and sensitive objective means of assessing outpatient dietary intake and physical activity become available (Carstensen et al., 2008; Winkler, 2005).

The findings of this study should not be universally directed to all type 2 diabetes due to the small sample. While this study made use of a small proof-of-concept sample, and detected small or moderate



Figure 11. Waist circumference changes in the 16-week control group (NonG).



Figure 12. Hip circumference changes in the 16-week control group (NonG).

improvements in body composition, these findings are still meaningful in that the findings provide support for guidance to those individuals living with type 2 diabetes to help them understand the preferred strategies to prevent the progression of diabetic complications. Further, while the present study made use of simple anthropometric measurements and indices, these measures are the most commonly used tools for assessing body composition (Sarrõa et al., 2001), especially in low-resource settings (Shaw et al., 2016). In addition, this study utilised male and female participants and improvements may prove variable in a single gender population due to gender differences in exercise responses and gender-specific adaptations to exercise (Brown et al., 2008; Fourie et al., 2012).

5. Conclusion

While the efficacy of a healthy lifestyle, including physical activity and dietary intervention remain undisputed, many important questions remain regarding the effectiveness of LCHFDs in improving health outcomes (and body composition as demonstrated in this study), especially in type 2 diabetics. A randomized trial of an LCHFD compared to a traditional diabetic diet focused on balancing total energy intake with expenditure (Hu et al., 2001) with/without concurrent exercise modalities, such as resistance training, may be warranted to evaluate their potential in preventing and/or avoiding overweight and obesity in individuals with type 2 diabetes. The results of this study provide support for guidance to those individuals living with type 2 diabetes to help them understand the preferred strategies to prevent the deterioration of body adiposity markers. The results indicate that small or moderate changes in body composition are achievable with LCHFD and/or daily walking in obese adults living with type 2 diabetes. However, more robust research is required to determine the effects of LCHFD, with or without concurrent physical activity, on obesity and other diabetic complication markers.

Declarations

Author contribution statement

Gerrit J. Breukelman: Conceived and designed the experiments; Performed the experiments; Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

Cornelia J. du Preez, Albertus K. Basson and Trayana G. Djarova: Conceived and designed the experiments; Contributed reagents, materials, analysis tools or data.

Brandon S. Shaw and Ina Shaw: Analyzed and interpreted the data; Contributed reagents, materials, analysis tools or data; Wrote the paper.

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Data availability statement

Data included in article/supplementary material/referenced in article.

Declaration of interests statement

The authors declare no conflict of interest.

Additional information

No additional information is available for this paper.

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