

CHAPPELL, A. and SIMPER, T. 2020. A case study series of the health status and key anthropometry in very large strength athletes. *IUSCA journal* [online], 1(1), article ID 2, pages 1-20. Available from: <https://doi.org/10.47206/iuscaj.v1i1.2>

# A case study series of the health status and key anthropometry in very large strength athletes.

CHAPPELL, A. and SIMPER, T.

2020

# A Case Study Series of the Health Status and Key Anthropometry in Very Large Strength Athletes

Andrew Chappell<sup>1</sup> and Trevor Simper<sup>2</sup>.

<sup>1</sup>School of Pharmacy and Life Sciences, Robert Gordon University, Garthdee Road, Aberdeen, UK; <sup>2</sup>Food and Nutrition Group, Sheffield Business School, Sheffield Hallam University, Howard Street, Sheffield, UK.

## ABSTRACT

Strongmen are characterised by their large mass which they use to perform feats of strength, the effect this large mass has on the athlete's health are unknown. The aim of this study was to: characterise a group of strongmen and to compare observed values with established parameters for good health. We measured: resting metabolic rate, body composition, skinfolds, lipid profiles, fasted glucose, blood pressure, power output, and grip strength in 6 competitive strongmen on three separate occasions over a six-month period. Blood pressure varied with 3 of the strongmen recording hypertensive values. Fasted blood glucose levels aligned with values for healthy adults, cholesterol/lipid profiles varied with the suggestion that values are generally poorer in strongmen than in healthy adults. The large body mass of the strongmen corresponded with a large amount of adipose tissue, which may incur risk for non-communicable disease. Furthermore, the risk of disease may be further compounded by anabolic steroid use. Dietary considerations are made for

protecting cardiovascular health and the potential for 'healthy' weight gain. To our knowledge this is the first such nested cohort study in the area of strongmen competitors. Larger studies are needed to confirm and further elucidate this data. Considerations for protecting the health of strongman competition is made.

**Key Words:** Strongman, Strength Athletics, Cholesterol, Blood Pressure, Diet, Heart Disease

## INTRODUCTION

The first "World's Strongest Man" (WSM) competition took place in 1977 involving strength and power athletes from different sports including: American football, Olympic weightlifting, bodybuilding, powerlifting, wrestling and track and field. Since the original WSM competition, strongman has become a sport in its own right; competitors train specifically to develop strength and muscular endurance to complete 5 to 6 events in the fastest time possible. Events are usually based upon mythical feats of strength combined

with conventional barbell tests, examples include: the 'farmers' walk' where competitors carry heavy objects (often anvils), over a set distance and the "Atlas Stones" where athletes lift 5 progressively heavier stone boulders onto platforms (1). Strongmen are characterised as having a large stature and mass, for example, WSM competitor Brian Shaw is reported to be 2.03 m tall, with a body mass of 200kg equating to a body mass index (BMI) of 48.5 kg/m<sup>2</sup>.

The large body mass of elite strongmen and their astounding strength perhaps gives credence to the conventional strongman wisdom that "mass moves mass". However, a large body mass increases an individual's body mass index (BMI), and where excess mass is fat mass (FM), this may represent a significant risk to health. Obesity, classified as a body mass index (BMI) greater than 29.99 kg/m<sup>2</sup>, or a body fat percentage (BF%) greater than 25, may lead to insulin resistance, dyslipidaemia and arterial hypertension increasing the risk of cardiovascular disease (CVD) and type 2 diabetes mellitus (T2DM) (2,3). Moreover, retrospective cohorts indicate that life expectancy is reduced between 8 to 10 years when BMI is greater than 40 kg/m<sup>2</sup> (4). Despite the high BMI, strongmen are regularly engaged in exercise and have high levels of fat free mass (FFM) which may offer some protection against CVD and T2DM. Previous research conducted on strongman has largely focussed, on physiology related to strongman training (4), prevalence of injury (5) and 'tapering' training prior to competition (6). The physiological changes associated with strongman training should be regarded as positive for health. Acute and longitudinal affects reported similar effects compared

to traditional resistance training (7), albeit some activities do place a greater emphasis on the cardiovascular system where individuals may acutely work at high heart rate intensities (>90% than age predicted) (4). There is a paucity of research however in investigate the health status of large strongmen, and if this large size might be a risk for the athlete's health. To the authors knowledge only a single cross section reporting on health parameters of strongmen (8).

The previous cross section of strongmen investigating health parameters (mean body mass: 129.0 ± 14.7 kg, mean BMI 38.0 ± 2.7 kg/m<sup>2</sup>) indicated that compared to controls and endurance athletes, strongmen had higher resting heart rates, haemoglobin and haematocrit levels, and a lower  $\dot{V}O_2$ max scaled for body size (8). Moreover, strongmen had poorer lipoprotein profiles compared to endurance athletes and controls, despite regular exercise (8). Changes in heart morphology were also reported to be similar to those seen in other strength sports, however strongmen were had impaired myocardial relaxation, and reduced left ventricle function compared to endurance athletes and controls (8). The aforementioned may increase their risk of cardiovascular disease in large strongmen and more research is warranted to investigate the health parameters in these populations, and how risk might be managed.

Studies of offensive linesman from the National Football League (NFL) are useful for making comparisons with large strongmen. For example, the mean body mass of offensive linesmen reported in literature is 140 - 143 kg, BMI 37.8 kg/m<sup>2</sup> (9,10). These athletes are also similar in

stature to strongmen competitors (9). Amongst 109 offensive linemen recruited by Tucker et al. (10) nearly all (n - 104) had a waist circumference (WC) over the 102 cm World Health Organisation (WHO) CVD high risk cut off, n - 26 were hypertensive, n - 70 pre-hypertensive, and n - 25 had a total cholesterol (TC) greater than 200 mg/dL. Interestingly, despite their high BMI's just over half (n - 61) had a body fat % over 25, only n - 7 a waist to hip ratio (WHR) over 1.0, and all players had normal fasting blood glucose, suggesting a possible protective effect associated with lean body mass (LBM) and exercise (9). The authors conclude that CVD risk for the linemen was similar to that of the similar to the general population, and that their physical activity output seemed to mitigate the effect of their large size. Post retirement however, the NFL lineman appears to be susceptible to developing metabolic syndrome (11), which should also be a consideration for strongman populations.

Finally, competitive strongman does not adhere to the World Anti-Doping Agency Code, and like other non-Olympic sports, the use of androgenic anabolic steroids (AAS) and other prohibited substances is in the first authors experience (a performance nutritionist who works with international competitive strongmen) widespread. Despite a lack of documented accounts of steroid use in the scientific literature, the lay media have speculated several high-profile deaths in strongmen were caused by AAS use (12-15), as supraphysiological AAS use is generally associated with myocardial dysfunction (16). The large size of these athletes and propensity for AAS to negatively impact blood lipid profiles and cardiac function is likely to increase the risk of disease in

these populations and warrants research. In the present investigation anthropometric measurements, cardiometabolic measurements and performance outcome markers are measured in six strongmen at three time points over a six-month period.

## METHODS

### *Presentation of the Athletes*

The present observation was performed on 6 active male strongmen. One of the athletes competed international and had previously placed in the top 3 of the UKs Strongest Man and World Team Strongman Championships. Another competed as a master's strongman and was an international level IPF powerlifter, the remaining participants had all competed at national or regional strongman level. Participants visited the laboratory on three occasions separated by 12 weeks (baseline, 12 wks, and 24 wks). At each visit the participant's metabolic health, exercise performance, anthropometric characteristics and dietary intake was assessed. Three of the six strongmen attended all three laboratory visits, two visited the laboratory on two occasions and one participant visited the laboratory only once. Four of the strongmen reported using AAS throughout the observation, while another professed to having previously using AAS. All participants provided written informed consent before participation, and provided approval for publication of the data. This research project was approved by the university ethical review board.

### *Indirect calorimetry and RMR prediction equations*

Resting metabolic rate (RMR) was established using the conventions for good practice (17). Participants reported to the laboratory between 08:00 and 10:00 a.m following an overnight fast from 10 pm. On the morning of testing, participants were asked to refrain from physical activity (cycling, brisk walking, taking the stairs). Participants were asked to abstain from alcohol 24 h preceding each visit and from caffeine on the morning of their visit. Participants were also asked to consume 500 ml of water on the morning of the trial to encourage hydration. Participants rested in the supine position for 20 mins, followed by 10 mins wearing a facemask for the measurement of RMR (AS Instruments, Oxford, United Kingdom). RMR was captured by breath-by-breath measurements of oxygen consumption ( $\dot{V}O_2$ ) and carbon dioxide ( $\dot{V}CO_2$ ) collected via Douglas bag and analysed immediately for volume of O<sub>2</sub> and CO<sub>2</sub> fractions using an electronic gas analyser (GIR250, MTL, Luton, United Kingdom). The total volume of gas expired was then measured including the fraction extracted for the gas analyser. The gas analyser was calibrated prior to use by testing a known gas concentrations (4 % CO<sub>2</sub>; 16 % O<sub>2</sub>, 100 % N<sub>2</sub>, MTL, Luton, United Kingdom). Two five-minute gas samples were collected via Douglas bags and analysed. If a respiratory coefficient (RQ) of < 0.7 or > than 1.0 was recorded this was considered a protocol violation. A Coefficient of variation (CV) of < 10 % was considered acceptable for  $\dot{V}O_2$  and  $\dot{V}CO_2$ . Environmental conditions during testing were: humidity  $27.9 \pm 7.2$  %; temperature  $22.1 \pm 2.6$  °C.

Two prediction equations were used to estimate BMR: Schofield (18) and Cunningham (19). The Schofield (18) estimates BMR based on body mass, age and sex, and is the preferred method for estimating BMR by WHO (20). The Cunningham (19) equation estimates BMR based on the FFM, estimated using bioelectrical impedance (BIA). RMR was then compared to prediction estimates and the difference calculated.

### *Body composition/anthropometry and ultrasound*

Height and weight were measured using a stadiometer (Holtain, Crymych, United Kingdom) and a Tanita multi-frequency bioelectrical impedance analyser (InBody 720, Biospace, Urbandale, Iowa, USA). The Tanita was also used to assess fat mass (FM), fat free mass (FFM) and total body water. BMI and the fat free mass index (FFMI) were calculated as kg/m<sup>2</sup> and FFM kg/m<sup>2</sup>. Ultrasound (Bodymetrix, Professional Ultrasound, Cartwright fitness, Chester, UK) was used to perform a 7-site skin fold assessment. The sum of these skinfolds was used to calculate a BF% (21). Briefly, a thin layer of ultrasound gel was applied to the wand head and then wiped across the skin at the skin fold site. The ultrasound was then held perpendicular to the point of skin contact at each of the observable sites: pectoralis minor, subscapular, axilla, triceps, suprailliac, rectus abdominis, and quadriceps. Differentiation between tissue interfaces (muscle mass, body fat and bone) was determined based on the thickness of tissues and length of time for ultrasonic waves to pass through and reflect back to the sound head of the transducer. Local averaging of each signal at each site was performed. Ultrasound

signals were interpreted using the Bodymetrix ultrasound analysis software (IntelaMetrix, Brentwood, California, United States).

#### *Blood pressure and resting heart rate*

Blood pressure and heart rate were measured in a supine position following RMR measurements. Resting heart rate, and blood pressure, was determined using a digital blood pressure monitor (UA-787 Plus, A & D Instruments Ltd, Oxfordshire, UK). Cuff size (22-32 cm or 32-45 cm) was selected on a case-by-case basis. Measurements were made in triplicate and mean values calculated.

#### *Blood parameters*

A single-use lancing device (Accu-chek Softclix Pro, Roche Diagnostics Ltd, West Sussex, UK) was used to obtain capillary blood samples. To determine TC, whole blood triglycerides (TAG) and creatine kinase (CK), two 30- $\mu$ L samples were collected in Microsafe collection and dispensing tubes (Inverness Medical, Cheshire, UK) and applied immediately to: Reflotron TC test strips (measurement range, 2.59 - 12.9 mmol/L); Reflotron TAG test strips (measurement range, 0.80 - 6.86 mmol/L); and Reflotron CK test strips (measurement range, 25 - 1900 U/l) (all Inverness Medical, Cheshire, UK). For HDL whole blood was collected in 300  $\mu$ L EDTA dipotassium salt-coated centrifuge tubes (Microvette CB 300, Hematology/Potassium EDTA; SARSTEDT Ltd, Leicestershire, UK), spun at room temperature for 2 mins (Centrifuge MC6; SARSTEDT Ltd) and 30  $\mu$ L of plasma applied to a Reflotron HDL test strip (measurement range, 0.26-2.59 mmol/L). The low-density lipoprotein (LDL) fraction was estimated by subtracting the sum of total TAG and HDL

divided by 2.19, from TC. The Reflotron Plus (Inverness Medical), a reflectance photometer, was then used to analyse each sample. Fasted blood glucose was measured via a 20  $\mu$ L whole blood samples added to an aliquot containing heparin and saline before being read using a Biosen C-line (EKF diagnostics, Ebendorfer, Germany).

#### *Testosterone and C-reactive protein (CRP)*

Salivary testosterone, and CRP was measured for participants 1, 2, and 3 using a commercially available enzyme immunoassay (Salimetrics, Carlsbad, California, USA). The range of sensitivity was 6.1 to 600 pg/ml, and 0.0 to 3000 pg/ml for testosterone, and CRP respectively. Participants expectorated 5 ml of saliva into a collection tube which was centrifuged (10,000 g  $\times$  10 mins) before the supernatant was removed for long term storage (-80  $^{\circ}$ C). Analysis was performed as per manufactures instructions and samples were analysed in duplicate. The procedure was the same for both kits with the exception of the antibody enzyme conjugate and enzyme coated 96 well plate. Briefly, standards, controls and samples were added to a 96 well followed by the antibody enzyme conjugate and mixed on a plate rotator at 500 rpm for 2 h, at room temperature. The plate was then washed with a buffer containing phosphate and dH<sub>2</sub>O, before the addition of Tetramethylbenzidine solution (which produces a light reaction), followed by mixing at 500 rpm, for 30 min at room temperature. A stop solution (Sulfuric acid) was then added and mixed at 500 rpm for a further 3 mins at room temperature. Samples were then read on a plate reader within 5 minutes at 450 nm (Synergy HT, BioTek Instruments, Winoski,

Vermont, USA), and Gen5 microplate reader software (BioTek Instruments, Winooski, Vermont, USA).

### *Anaerobic power and hand grip strength*

Anaerobic power was measured using a Wingate tests on a Velotron cycle (Racer-Mate, Seattle, WA). Participants pedalled at maximum effort, attempting to attain a maximum number of pedal revolutions against a load equal to 7.5% of their bodyweight. The protocol was as follows: participants performed a 5 min warm-up at a rate equivalent to 50 watts, before completing a familiarisation phase. The familiarisation involved a 20 s lead in, before a 10 second acceleration phase which included a countdown, at the end of which the load was automatically added to the flywheel for 3 s. After this familiarisation phase the load was removed and participants returned to the warm up pace for a further 2 min. The protocol was then repeated with the 20 s lead in, 10 s second acceleration phase, before the load was added and participants attempted a maximal effort for 20 s with verbal encouragement. The peak power output, mean power output, and output scaled for bodyweight were all obtained from the Velotron software (RaceMate One V.4.1.0.6). Hand grip strength (HGS) was measured using a Hand Grip Dynamometer (Takei, Nigata City, Nigata Pref, Japan). Strength was measured in both hands, and participants adjusted the handle position to suit their own preference repeated at each visit. The test arm was positioned parallel to the floor with the elbow flexed at 180°, the participant then performed a maximal isometric contraction by squeezing the dynamometer while bringing their arm parallel to their lower body. Each

participant performed three maximum strength attempts per hand allowing for 30 s rest between attempts.

### *Dietary intake*

A 24 h dietary recall interview was carried out by a registered nutritionist (AC) for all participants. Participants documented all food items and beverages consumed in the previous 24 h, along with weights and/or portion sizes. Diets were then analysed using dietary analysis software (Nutritics Research Edition v5.092, Dublin, Ireland). Total energy intake, macronutrient and caffeine intake are expressed as (kcal), (g) and (mg), and scaled for bodyweight as kcal/kg, g/kg and mg/kg of bodyweight (BW). Energy intake and protein intake from dietary supplements was determined based on nutritional information obtained from manufacturers websites. Energy availability was calculated based on the total energy intake, minus the measured RMR. Finally, the mean number of food items consumed was counted. The percentage of the diet made up of specific food groups was based on the European Food Safety Agency food classification system for dietary reporting (22). Any food group making up less than 1 % of the dietary intake was placed in the 'others' category.

## **RESULTS**

### *Participant characteristics and indirect calorimetry*

The mean age and height of participants was  $33.5 \pm 5.8$  yrs and  $1.85 \pm 0.07$  m. Individual anthropometric characteristics at baseline, 12 and 24 wks are reported in table 1. The body mass and BMI of the cohort was between 100.7 to 178.7 kg and

33.1 to 48.0 kg/m<sup>2</sup>. Measurement of BF% by ultrasound were consistently reported 53-66% lower than the BIA method resulting in higher estimates of FFM between 4.6 to 19.5 % using the ultrasound (table 1). Mean salivary testosterone was: baseline, 928 ± 587 pg/ml; 12 wks, 469 ± 112 pg/ml; 24 wks, 732 ± 343 pg/ml. Mean CRP concentrations were: baseline, 383 ± 259 pg/ml; 12 wks, 999 ± 557 pg/ml; 24 wks, 126 ± 83 pg/ml. RMR, predicted BMR, blood pressure, heart rate and metabolic markers (fasting blood glucose, blood lipids and CK) are reported in table 2. Measured RMR ranged between 1179 kcal in the smallest participant to 4988 kcal in the largest. BMR prediction equations estimated energy requirements between 53 to 64 % lower and higher than RMR, with no discernible pattern trend (table 2).

#### *Dietary intake of strongmen populations*

Participant's 24 h nutrient intake is reported in table 3. Macronutrient and energy intake ranged from: CHO 213 to 1183 g, PRO 200 to 464 g, fat 67 to 162 g, energy 3014 to 9427 kcal. Participants consumed a mean of 15.4 individual food items per day. Dairy as full fat yoghurt, whey and milk, cereal as oats, fruits and vegetables and confectionary were popular food items. The contribution each food group made to the cohort's diet is presented in figure 1. A sample diet for participant 3 is provided as a supplement (S1). Competitors reported consuming between 1 to 4 dietary supplements throughout the study. Whey protein was the most commonly consumed supplement, maltodextrin, multi ingredient pre-exercise formulas and branch chain amino acids were also consumed.

#### *Performance metrics of strongmen competitors*

Results of the Wingate testing and HGS are presented in table 4. Power output relative to LBM is presented as a supplement (S2). Peak power output and anaerobic power scaled for BW ranged 979 to 1971 W, and 6.9 to 12.3 W/kg BW. The mean watts achieved over the Wingate and the anaerobic capacity scaled for BW was between 574 to 1230 W and 4.0 to 9.0 W/kg BW. HGS in the left hand was between 37 and 76 kgf, while strength in the right was between 47 and 87 kgf. The difference in strength between the left and the right reflect more right handed dominant participants in the study. Wingate performance scaled for LBM indicated that the highest power outputs were highest in athletes with the highest LBM.



Participant	Baseline						12 weeks					24 weeks		
	1	2	3	4	5	6	1	2	3	4	5	1	2	3
Height (cm)	1.80	1.93	1.91	1.90	1.75	1.82	1.80	1.93	1.91	1.90	1.75	1.8	1.93	1.91
Weight (kg)	141.9	178.7	140.8	140.9	100.7	129.9	138	172.7	137.8	134.7	101.5	143.1	170.1	133.2
BMI (kg/m <sup>2</sup> )	43.8	48.0	38.6	39.0	33.1	39.4	42.6	46.4	37.8	37.3	33.3	44.2	45.7	36.5
Σ 7 Skin Folds (cm)	81.4	108.6	59.7	56.2	84.5	107.3	95.2	103.4	63.3	52.1	77	95.5	98.6	64.5
Left Bicep Muscle Thickness (mm)	53.2	55.8	55.1	60.1	44.0	40.0	-	58.2	65.1	58.6	46.0	52.7	56.6	52.3
Right Bicep Muscle Thickness (mm)	52.2	59.0	59.4	57.6	40.6	40.4	-	55.8	60.7	64.8	41.0	53.0	56.9	57.8
Fat Free Mass: Ultrasound (kg)	113.0	132.8	116.0	119.6	77.9	96.1	106.1	129.4	113.3	116.0	79.9	109.5	128.1	108.7
Body Fat: Ultrasound (%)	20.3	25.7	17.6	15.1	22.6	26.0	23.1	25.1	17.8	13.9	21.3	23.5	24.7	18.4
Lean Body Mass: BIA (kg)	91.0	108.5	97.1	101.0	72.4	84.7	86.5	108.8	100.9	98.6	76.1	91.7	107.8	99.2
Body Fat: BIA (%)	35.9	39.3	31.0	28.3	28.1	34.8	37.3	37.0	26.8	26.8	25.0	35.9	36.6	25.5
Total Body Water (%)	51.3	46.6	51.4	55.6	52.4	49.7	47.0	50.4	56.9	60.0	56.4	51.3	50.7	57.2
FFMI: Ultrasound (kg/m <sup>2</sup> )	34.9	35.6	31.8	33.1	25.6	29.2	32.7	34.7	31.0	32.1	26.2	33.8	34.4	29.8
FFMI: BIA (kg/m <sup>2</sup> )	28.1	29.1	26.6	28.0	23.8	25.7	26.7	29.2	27.6	27.3	25.0	28.3	29.0	27.2
FFMI: Difference Ultrasound to BIA (%)	19.5	18.3	16.4	15.4	7.1	12.0	16.9	15.9	11.0	15.0	4.6	16.3	15.7	8.7
Waist Circumference (cm)	125.4	138.5	116.0	116.0	105.0	116.0	124.5	136.0	114.5	112	103.5	127.5	138	112
Hip Circumference (cm)	133.6	141.0	125.5	117.0	110.0	129.0	128.0	139.0	119.0	112.5	111	132.5	137	118.5
Waist To Hip Ratio	0.94	0.98	0.92	1.0	0.95	0.90	0.97	0.98	0.96	1.00	0.93	0.96	1.01	0.95

**Table 1.** Anthropometric Observations of Strongmen Competitors  
 BMI, Body Mass Index, BIA, Bio-electrical impedance, FFMI, fat free mass index, - measurement not recorded.

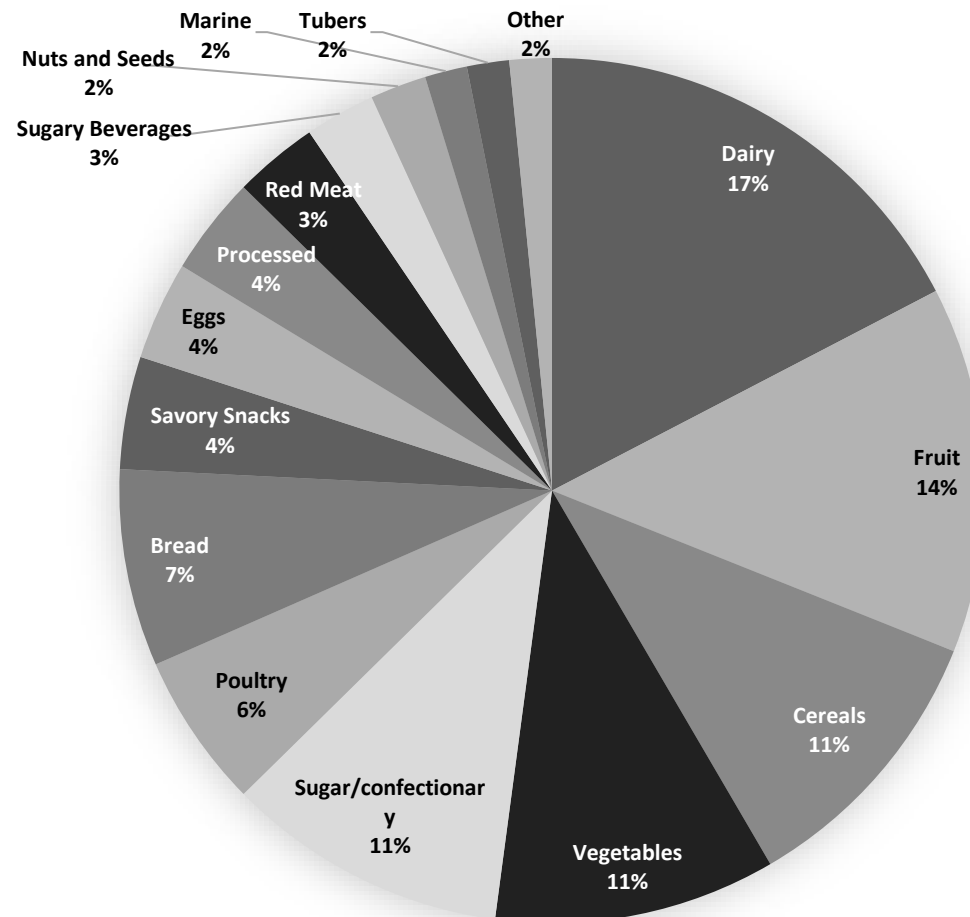
Participant no.	Baseline						12 weeks					24 weeks		
	1	2	3	4	5	6	1	2	3	4	5	1	2	3
Measured RMR (kcal/day)	2403	3719	3414	2330	1179	2416	2386	3559	3785	3337	2524	2326	4988	3375
Schofield BMR (kcal/day)	2829	3383	2812	2814	2208	2648	2770	3293	2767	2720	2221	2847	3253	2698
Cunningham BMR (kcal/day)*	2335	2713	2469	2552	1934	2199	2239	2720	2549	2450	2014	2351	2699	2514
Diff between predictive (Schofield) and RMR	+16%	-9%	-21%	+18%	+47%	+9%	+14%	-7%	-37%	-22%	-13%	+18%	-53%	-25%
Diff between predictive (Cunningham) and RMR	-3%	-27%	-28%	+10%	+64%	-9%	-6%	-24%	-33%	-27%	-20%	+1%	-46%	-26%
SYS Blood pressure (mmHg)	129	130	141	136	123	127	124	131	121	119	107	126	131	124
DIA Blood pressure (mmHg)	76	90	100	88	84	86	65	89	65	73	71	74	85	73
Resting Heart Rate (bpm)	68	82	85	70	65	72	60	79	94	72	64	69	65	59
Fasting Blood Glucose (mmol/l)	5.2	4.7	4.3	4.2	4.0	4.7	5.8	4.2	4.3	4.2	4.0	3.7	5.4	4.9
Total Cholesterol (mmol/l)	4.5	9.9	4.0	6.9	4.9	5.4	4.4	5.0	5.0	4.8	4.2	4.3	5.6	3.6
HDL (mmol/l)	1.4	-	-	-	1.3	0.6	-	0.7	0.8	0.8	1.0	1.0	0.6	1.0
LDL (mmol/l)	0.9	-	-	-	0.7	1.1	-	1.3	1.2	1.0	0.8	1.1	1.5	0.7
Triglycerides (mmol/l)	1.2	1.4	1.9	1.0	2.0	2.2	1.8	1.4	1.6	1.9	1.5	0.8	1.7	1.1
Creatine Kinase (U/l)	150	662	52	190	278	30	863	162	52	543	714	86	24	929

**Table 2.** Estimated Energy Requirements and Selected Health Indices of Strongmen Competitor

\* Lean mass based on BIA, SYS systolic, DIA diastolic, BPM beats per minute, LDL Low density lipoprotein, HDL high density lipoprotein, - measurement not recorded.

Participant no.	Baseline						12 weeks					24 weeks		
	1	2	3	4	5	6	1	2	3	4	5	1	2	3
<b>Total Intake</b>														
<b>CHO (g)</b>	-	582	1183	528	549	213	276	768	1039	313	510	507	309	989
<b>Free sugars (g)</b>	-	1	309	62	168	46	41	104	273	7	92	107	0.23	300
<b>Fibre (g)</b>	-	32	59	36	19	26	21	65	75	29	25	38.8	6.9	92
<b>PRO (g)</b>	-	464	302	414	297	200	291	428	300	302	310	389	276	349
<b>Fat (g)</b>	-	264	388	233	67	156	83	252	400	162	115	119	75	346
<b>Saturated Fat (g)</b>	-	123	162	112	35	59	20	91	123	71	46	34	36	124
<b>Energy (kcal)</b>	-	6560	9427	5860	3991	3055	3014	7048	9427	3916	4517	4760	3018	8463
<b>Energy Availability (Kcal)</b>	-	2841	6014	3530	2812	639	628	3489	5643	579	1993	2434	-1970	5088
<b>Caffeine Intake (mg)</b>	-	176	466	224	338	0	0	473	468	288	463	276	126	396
<b>Intake Scaled for BW</b>		1.0	3.3	1.6	3.4	0	0	2.7	3.4	2.1	4.6	1.9	0.7	3.0
<b>CHO (g/kg BW)</b>	-	3.3	8.4	3.7	5.5	1.6	2.0	4.4	7.5	2.3	5.0	3.5	1.8	7.4
<b>PRO (g/kg BW)</b>	-	2.6	2.1	2.9	2.9	1.5	2.1	2.5	2.2	2.2	3.1	2.7	1.6	2.6
<b>Fat (g/kg BW)</b>	-	1.5	2.8	1.7	0.7	1.2	0.6	1.5	2.9	1.2	1.1	0.8	0.4	2.6
<b>Energy (kcal/kg BW)</b>	-	36.7	67.0	41.6	39.6	23.5	21.8	40.8	68.4	29.1	44.5	33.3	17.7	63.5
<b>Adjusted Caffeine (mg/kg BW)</b>	-	1.0	3.3	1.6	3.4	0.0	0.0	2.7	3.4	2.1	4.6	1.9	0.7	3.0
<b>Percentage of Energy</b>														
<b>CHO (%)</b>	-	33.3	47.1	33.8	51.6	26.1	34.3	40.9	41.3	30.0	42.3	39.9	38.9	43.8
<b>PRO (%)</b>	-	28.3	12.8	28.3	29.8	26.2	38.6	24.3	12.7	30.8	27.5	32.7	35.7	16.5
<b>Fat (%)</b>	-	36.2	37.0	35.8	15.1	46.0	24.8	32.2	38.2	37.2	22.9	22.5	22.7	36.8
<b>Supplement Intake</b>														
<b>PRO from Supplements (%)</b>	-	12.3	15.5	22.7	17.5	0.0	5.4	17.2	20.8	25.8	16.2	24.8	0.0	17.9
<b>Energy from Supplements (%)</b>	-	7.6	9.1	15.6	10.5	0.0	4.8	10.2	10.7	19.4	9.7	4.0	0.0	4.0

**Table 3.** Dietary Intake of Strongmen Over 24 weeks  
CHO Carbohydrate, PRO Protein, BW bodyweight, - measurement not recorded



**Figure 1.** Competitive Strongmen's Diet as a Percentage Food Group Intake. Others, includes food groups that made up less than 1 % of participants intake e.g. legumes and vegetable oils.

Participant no.	Baseline						12 weeks					24 weeks		
	1	2	3	4	5	6	1	2	3	4	5	1	2	3
<b>Hand Grip Strength</b>														
Left (kgf)	59	74	65	46	51	37	60	75	62	53	56	59	76	62
Right (kgf)	68	78	69	56	47	51	62	80	60	45	46	69	87	68
<b>Wingate</b>														
Peak Watts (W)	979	1971	1350	1733	1030	1158	1321	1904	1444	1387	1098	1363	1941	1389
An. Power (W/kg BW)	6.9	11.0	9.6	12.3	10.2	8.9	9.6	11.0	10.5	10.3	10.8	9.5	11.4	10.4
Mean Watts (W)	755	1209	1120	1175	869	910	738	1146	1230	1093	874	574	1227	1200
An. Capacity (W/kg BW)	5.3	6.8	8.0	8.3	8.6	7.0	5.3	6.6	8.9	8.1	8.6	4.0	7.2	9.0
Min Watts (W)	584	890	892	803	638	640	349	748	986	752	672	295	797	959
Fatigue Index (%)	20.4	55.9	29.1	47.2	23.8	26.2	49.1	58.9	31.8	33.9	24.2	53.9	58.7	30.1
Total Work (J)	15091	24176	22401	23494	17378	18205	14770	22910	24602	21860	16248	114475	24530	23996

**Table 4.** Performance metrics for Competitive Strongmen Over 24 Weeks  
Abbreviations. kgf kilograms of force, An. anaerobic, W watts, J joules, % percentage

## DISCUSSION

To our knowledge, this is the first longitudinal observation to report health and performance outcomes in strongmen competitors. This study is also the first to document the energy intake of strongmen competitors. The aim of this report was to characterise metabolic and performance metrics of strongman so health professionals, coaches and dieticians might better understand these populations. Analysis of the data indicates that the metabolic health of the current cohort is similar to previous reports of large athletes and strongmen (8,9). All the participants were clinically obese, a number were hypertensive, and had elevated TC and reduced HDL in some, but not all cases. Resistance training is known to have positive effects on HDL and TC (23, 24), the athlete's large size combined with AAS use (4 of the 6 participants were using AAS throughout the study) however may explain some of these findings. Confectionary, savoury snacks, processed meats and sugar sweetened beverages accounted for over 20 % of the food items consumed; contributed to dietary pattern a high saturated fat intake. Dietary manipulation rather than exercise modification may therefore likely to be a viable option to modulate CVD risk. Strongmen might therefore be encouraged to consume a diet higher in lean meats, and omega-3 fatty acid containing food items to more effectively manage their blood lipid levels. Although consideration is needed for the practicalities of making these changes where energy intake is high.

### *Anthropometrics and indirect calorimetry*

Participant's weight, BMI and FM was between 101 to 179 kg, 33 to 48 kg/m<sup>2</sup> and

28 to 39 % respectively. This large mass is consistent with reports in strongmen populations, NFL offensive linemen and sumo-wrestlers (8-10,25). To our knowledge, the 179 kg strongman is also likely one of the largest athletes ever reported in the scientific literature (26). All strongmen were clinically obese (BMI > 29.9 kg/m<sup>2</sup>), and two were morbidly obese (BMI > 40 kg/m<sup>2</sup>) significantly increasing their risk for CVD, breast, colon, prostate, endometrium, kidney and gall bladder cancer and T2DM (27). Furthermore, all participants had a WC greater than 104 cm (range 105 to 139 cm) and a WHR over 0.90 (range 0.92 to 1.01). Both measures are indicative of visceral fat, and measurements greater than 104 cm, and 0.90 are associated with an increase the risk for the aforementioned diseases (27). Blood pressure of participants and resting heart rate was also similar to offensive linemen and strongmen competitors (9, 10). Optimal blood pressure is considered to be 120 / 80 mmHg in adults of this age, however three participants were noted as hypertensive at baseline based on the WHO cut offs for CVD risk (systolic blood pressure: 140 – 159, or diastolic blood pressure: 90 – 99) (28).

Energy requirements and prediction equations were dependent on body mass as expected. Prediction equations however over or underestimate indirect calorimetry by as much as 53 to 64% in some cases. Reasons for this discrepancy may be two-fold, although RMR is largely dependent on body mass, both FFM and FM are known to be independent predictors of BMR (29). As a result, prediction equations not accounting for both FFM and FM are less reliable when compared to indirect calorimetry measurements in obese population (30). To account for body

composition the Cunningham (19) prediction equations were also used, however, they were no more effective at predicting RMR than the Schofield equations (18). Secondly, although participants were fasted and rested on the morning of the trial, elevated CK levels reflect those seen 24 to 48 h post exercise following weight training (31). On four occasions participants had CK levels greater than 500 u/l indicative of strenuous prior exercise. The effect of prior exercise on RMR is well known and has been attributed to excess post-exercise O<sub>2</sub> consumption (EPOC). EPOC may be present up to 48 h post exercise and depending on the intensity of the prior exercise may increase by as much as 24.7 % (32). It is worth noting however the practical difficulties in recruiting athletes during a competitive season, and that these athletes basal state is likely reflective of someone constantly recovering from exercise.

#### *Biochemical measures*

With the exception of participant 3 and 4, all participants had a TC and LDL less than the 6.1 mM and 4.0 mM cut offs for increased CVD risk (33). Among adults in the UK a TC values over 5 mM is considered high and optimal under 4.0 mM (34). Interestingly participant 2 reported a TC of 9.9 mM at baseline, before reducing his TC to less than 6.0 by the third laboratory visit. This drop in TC coincided with a reduction in the saturated fat content of his and an 8 kg drop in bodyweight which was accompanied by a reduction in FM, as he actively attempted to improve his blood lipid profile. Furthermore, he also ceased taking AAS, which are known to negatively influence blood lipids (17). In consideration, of the overall lipid profile of

our strongmen the values are similar to those reported in Strongman and NFL populations (8,9) LDL and TAG concentrations less than 2.0 mM is considered to be healthy in people at risk of heart disease, while a HDL of 1.0 mmol/l is considered ideal (33). Unfortunately, because of a technical error HDL and LDL levels were unavailable for all time points, however in five instances HDL levels were less than 1.0 mM, LDL was less than 2.0 mM and TAG ranged between 0.8 and 2.2 mM. Blood lipids from strongmen have previously been compared to endurance athletes and healthy controls, and the profiles reported here reflect the previous observations of elevated TC and reduced HDL (8). Of most concern are the HDL levels, the effect that AAS have on HDL is well known this may account for the lower than 1.0 mM values reported here, furthermore the high saturated fat diet may have been a contributing factor (35). Moreover Venckunas (8), reported impaired myocardial relaxation in strongmen, consistent with AAS use (16).

All participants reported FBG levels of less than 5.8 mM. FBG levels are an indicator of T2DM risk and WHO classifies prediabetes as levels between 6.1 to 6.9 mM (36). Obesity significantly increases an individual's risk of impaired glucose tolerance, it's possible that the large amount of LBM strongmen possess provides some protection against T2DM (37). The LBM of the participants was between 72 to 109 kg, well beyond the 61.7 kg for the reference man (38), which might explain some of the discrepancies between the anthropometrics and biochemical markers. Similar findings were observed in the offensive lineman where participants had a high BMI but normal FBG levels (9). Salivary CRP values were

within reference ranges for a healthy population, while testosterone concentrations were elevated reflecting the participants reported using AAS (39).

#### *Power output and hand grip strength*

In consideration of power output and body mass versus LBM we observe that the person with the greatest body mass also has the greatest power output (see fig 3) and the greatest lean tissue value. It is not possible with this data to determine a strict relationship between lean tissue and power output, or overall body mass and power output. It is also worth pointing out that strongmen events are not all static movements (e.g. pushing a barbell from the chest) but often involve pulling and pushing weight over distance (pulling a bus) and overall bodyweight as well as lean tissue almost certainly play a part in the dynamics of moving large objects. The Wingate test however is useful for comparisons with other sports and peak wattages (979 to 1971 w) were greater than values reported amongst elite athletes from different sports (mean 833 w), NCAA Division 1 power athletes (mean 1084 w), and collegiate line backers (mean 1223 w) (40-42). Scaled for body mass strongmen were less powerful than track and field athletes (10.2 w/kg BW vs 12.2 w/kg BW) (43), although the ability to generate a greater absolute peak power is a more relevant metric for this population. HGS is known to correlate with upper body strength, ballistic performance and powerlifting strength (44). The values reported here are the first-time strongman HGS has been reported in the literature.

#### *Dietary intake*

A key concern amongst strongmen is gaining and maintaining a large body mass, so overall energy intakes are high. Therefore, consideration of the macronutrient breakdown and especially the quality of fatty acids in the diet is warranted; overall the intake of fat was 34.37 % and average energy intake over 5000 kcals. All athletes met the ACSM guidelines for protein intake (1.6 to 2.0 g/kg BW) and were in an energy surplus, however they may benefit from consuming a diet slightly higher in carbohydrate to meet the 4.0 to 7.0 g/kg BW recommendations for strength athletes (45). A key question for this group is, what would the effects of achieving their energy requirements if fat was reduced, or sources were switched from red meat, confectionary and high saturated fat foods to sources such as avocado, nuts and fatty fish i.e. essentially those with a lipid profile higher in mono and polyunsaturated fatty acids considered healthier and cardio-protective (46-49). Alongside the consideration of fatty acid intake, dieting for AAS use needs to be considered as evidence suggests a negative disruption to lipid profiles (35). In the present case-study lipid profiles are the metric which denotes a health risk in this group- and anecdotally we note that in participant 2 reducing TC to half the original level after he made changes to dietary intake following nutritional advice at the same time as cessation of steroid intake.

#### **PRACTICAL APPLICATIONS**

Prediction equations proved to be unreliable for estimating energy requirements in this population, however the energy intake of participants suggests



participants were likely meeting daily energy requirements. Measurement of health indices related to cardiovascular health suggest an elevated risk of CVD in this population, although the large amount of LBM they possess may offers some protection against T2DM. Continuous monitoring of body composition, body mass, blood pressure and lipid profiles is warranted and will be especially informative when athletes are using high energy density 'healthier' foods and when athletes are using AAS. Health practitioners should be aware that the diets followed by strongmen may need some revision to help mitigate some of these health risks. The evidence around altering saturated fat intake in the diet needs considering- for example: simply reducing saturated fat intake and replacing this with refined carbohydrate does not improve indices of cardiovascular health; whereas exchanging saturated fats with high fibre whole grains and unsaturated fats seem to positively effect cardiovascular risk factors (45). Examples of 'healthier food' alternatives include peanut butter, nuts and seeds in general, avocado, salmon- essentially foods high in protein and unsaturated fats compared with the saturated and trans fats obtained from fast food, chocolate, cookies etc. Future research should seek to add to this field by conducting larger studies to further determine the health parameters of strongman populations, particularly in relation to cardiovascular health. Moreover, researchers should consider strongwomen populations and weight division (Under 90kg, and Under 105kg), and whether these athletes may also be at risk of cardiometabolic diseases.

## References

1. History of the World's Strongest Man. Available at: <http://theworldstrongestman.com/history/> Accessed 07 June 2019.
2. Antonopoulos, AS, Oikonomou, EK, Antoniades, C, et al. From the BMI paradox to the obesity paradox: the obesity–mortality association in coronary heart disease. *Obes Rev* 17: 989 – 1000, 2016.
3. Prospective Studies Collaboration. Body-mass index and cause-specific mortality in 900 000 adults: collaborative analyses of 57 prospective studies. *Lancet* 373:1083 - 1096, 2009.
4. Woulfe, C, Harris, N, Keogh, J, et al. The physiology of strongman training. *Strength Cond J* 36: 84 - 95, 2014.
5. Winwood, PW, Hume, PA, Cronin, JB, et al. Retrospective injury epidemiology of strongman athletes. *J Strength Cond Res* 28: 28 - 42, 2014.
6. Winwood, PW, Dudson, MK, Wilson, D, et al. Tapering Practices of Strongman Athletes. *J Strength Cond Res* 32: 1181 - 1196. 2018.
7. Winwood, PW, Cronin, JB, Posthumus, LR, et al. Strongman vs. traditional resistance training effects on muscular function and performance. *J Strength Cond Res*, 29: 429 - 439. 2015.
8. Venckunas, T, Vasiliauskas, D, Marcinkeviciene, JE, et al. Strongmen sport is associated with larger absolute heart size and impaired cardiac relaxation. *J Strength Cond Res* 25: 2919 - 2925, 2011.
9. Kraemer, WJ, Torine, JC, Silvestre, R, et al. Body size and composition of National Football League players. *J Strength Cond Res* 19: 485 - 489, 2005.
10. Tucker, AM, Vogel, RA, Lincoln, AE, et al. Prevalence of cardiovascular disease risk factors among National Football League players. *JAMA* 301: 2111 - 2119, 2009.
11. Miller, MA, Croft, LB, Belanger, AR, et al. Prevalence of metabolic syndrome in retired National Football League players. *Am J Cardiol* 101: 1281 - 1284, 2008.
12. Titans Of Testosterone The (mostly) juiced-up athletes competing for the title of World's Strongest Man tote stones, toss kegs and tow trucks for puny paychecks and the glory

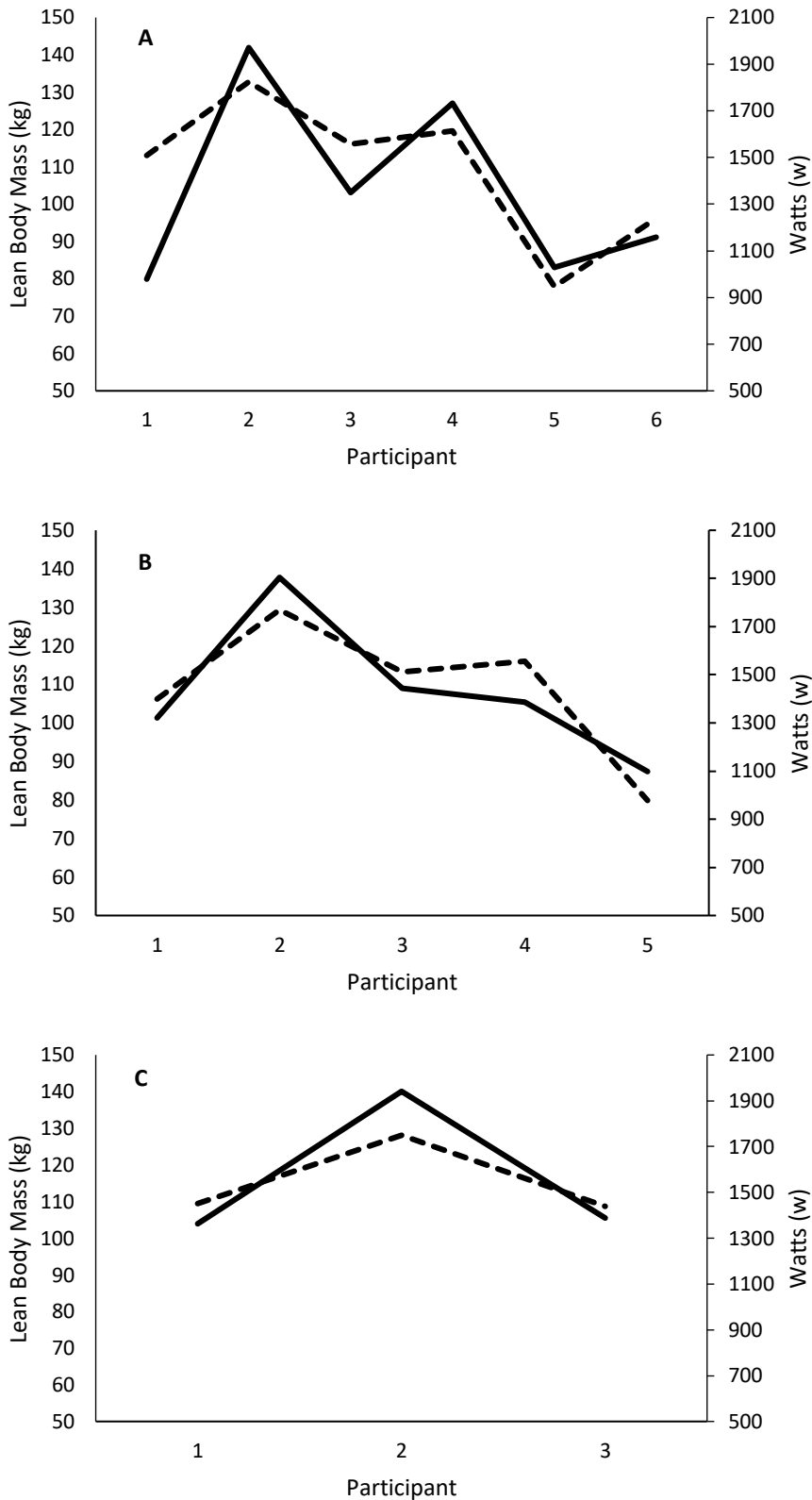
- of exposure on late-night cable TV. Available at:  
<https://www.si.com/vault/1999/10/18/268369/titans-of-testosterone-the-mostly-juiced-up-athletes-competing-for-the-title-of-worlds-strongest-man-tote-stones-toss-kegs-and-tow-trucks-for-puny-paychecks-and-the-glory-of-exposure-on-late-night-cable-tv>  
Accessed 07 June 2019.
13. Strongest Man deaths place BBC under fire. Available at:  
<https://www.scotsman.com/sport/strongest-man-deaths-place-bbc-under-fire-1-1380839> Accessed 07 June 2019.
  14. Heavy Hearts: Death of a Strongman. Available at:  
<https://www.seattletimes.com/sports/other-sports/heavy-hearts-death-of-a-strongman/>  
Accessed 07 June 2019.
  15. Long-time steroid use helped kill famed strongman Mike Jenkins, prompts coroner's warning. Available at:  
<https://www.pennlive.com/midstate/2014/06/long-time-steroid-use-helped-k-1.html>  
Accessed 07 June 2019.
  16. Baggish, AL, Weiner, RB, Kanayama, G, et al. Cardiovascular toxicity of illicit anabolic-androgenic steroid use. *Circ* 135: 1991 - 2002, 2017.
  17. Compher, C, Frankenfield, D, Keim, N, et al. Best practice methods to apply to measurement of resting metabolic rate in adults: A systematic review. *J Am Diet Assoc* 106: 881 - 901, 2006.
  18. Schofield, WN. Predicting basal metabolic rate, new standards and review of previous work. *Hum Nutr Clin Nutr* 39: 5 - 41, 1985.
  19. Cunningham, JJ. A Reanalysis of the factors influencing basal metabolic rate in normal adults. *Am J Clin Nutr* 33: 2372 - 2374, 1980.
  20. FAO/WHO/UNU Expert consultation. Energy and protein requirements. *World Health Organ Tech Rep Ser* 724: 71 - 112, 1985.
  21. Jackson, AS, Pollock, ML. Generalized equations for predicting body density of men. *Br J Nutr* 40: 497 - 504, 1978.
  22. European Food Safety Authority (EFSA). The food classification and description system FoodEx 2 (revision 2). *EFSA Supporting Publications* 12: 804E, 2015
  23. Schrauwen-Hinderling, VB, Hesselink, MK, Meex, R, et al. Improved ejection fraction after exercise training in obesity is accompanied by reduced cardiac lipid content. *J Clin Endo Metab* 95:1932 - 1938, 2010.
  24. Tambalis, K, Panagiotakos, DB, Kavouras, SA, et al. Responses of blood lipids to aerobic, resistance, and combined aerobic with resistance exercise training: a systematic review of current evidence. *Angiology* 60: 614 - 632, 2009.
  25. Yamauchi, T, Abe, T, Midorikawa, T, et al. Body composition and resting metabolic rate of Japanese college Sumo wrestlers and non-athlete students: are Sumo wrestlers obese? *Anthropol Sci* 112: 179 - 185, 2004.
  26. Sedeaud, A, Marc, A, Schipman, J, et al. Secular trend: morphology and performance. *J Sports Sci* 32: 1146 - 1154, 2014.
  27. James, WPT, Jackson-Leach, R, Mhurchu, et al. Overweight and obesity (high body mass index). *Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors* 1: 497 - 596, 2004.
  28. World Health Organization and International Society of Hypertension Writing Group, 2003, World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. *J Hypertens* 21: 1983 - 1992, 2003.
  29. Sabounchi NS, Rahmandad H, Ammerman A. Best-fitting prediction equations for basal metabolic rate: informing obesity interventions in diverse populations. *Int J Obes* 37:1364 - 1370, 2013.
  30. Sun, MX, Zhao, S, Mao, H, et al. Increased BMR in overweight and obese patients with type 2 diabetes may result from an increased fat-free mass. *J Huazhong U Sci Med* 36: 59 - 63, 2016.
  31. Baird, MF, Graham, SM, Baker, JS et al. Creatine-kinase-and exercise-related muscle damage implications for muscle performance and recovery. *J Nutr Metab* 11: 2012.
  32. Speakman JR, Selman C. Physical activity and resting metabolic rate. *Proceedings of the Nutrition Society* 62(3): 621 - 634, 2003.
  33. National Institute for Health and Clinical Excellence (2008); Familial hypercholesterolemia: identification and management Clinical guidance CG71.

- Available at:  
<https://www.nice.org.uk/guidance/cg71/info/maionforpublic> Accessed 07 June 2019.
34. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circ* 106: 3143 – 3421, 2002.
  35. Achar, S, Rostamian, A, Narayan, SM. Cardiac and metabolic effects of anabolic-androgenic steroid abuse on lipids, blood pressure, left ventricular dimensions, and rhythm. *Am J Cardiol* 106: 893 - 901, 2010.
  36. Huang, Y, Cai, X, Mai, W, et al. Association between prediabetes and risk of cardiovascular disease and all cause mortality: systematic review and meta-analysis. *BMJ* 355: i5953, 2016.
  37. Kahn, SE, Hull, RL, Utzschneider, KM. Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nat* 444(7121): 840 - 846, 2006.
  38. Snyder, WS. *Report of the task group on reference man*. Pergamon Press; Oxford. 1974.
  39. Clifton, S, Macdowall, W, Copas, AJ, et al. Salivary testosterone levels and health status in men and women in the British general population: findings from the Third National Survey of Sexual Attitudes and Lifestyles (Natsal-3). *J Clin Endocrinol Metab* 101: 3939 – 3951, 2016.
  40. Seiler S, Taylor, M, Diana, R, et al. Assessing anaerobic power in collegiate football players. *JASSR* 4: 9 – 15, 1990.
  41. Gacesa, JZP, Barak, OF, Grujic, NG. Maximal anaerobic power test in athletes of different sport disciplines. *J Strength Cond Res* 23:751 – 755, 2009.
  42. Coppin, E, Heath, EM, Bressel, E, et al. Wingate anaerobic test reference values for male power athletes. *Int J Sports Physiol Perform* 7: 232 – 236, 2012.
  43. Zajac, A, Jarzabek, R, Waskiewicz, Z. The Diagnostic Value of the 10- and 30-Second Wingate Test for Competitive Athletes. *J Strength Cond Res* 13: 16 - 19, 1999.
  44. Cronin, J, Lawton, T, Harris, N, et al. A brief review of handgrip strength and sport performance. *J Strength Cond Res* 31: 3187 – 3217, 2017.
  45. Thomas, DT, Erdman, KA, Burke, LM. American College of Sports Medicine Joint Position Statement. Nutrition and Athletic Performance. *Med Sci Sports Exerc* 48: 543 – 568, 2016.
  46. Scientific Advisory Committee on Nutrition (2019): Saturated fats and health. Public Health England and UK Health Departments. Available at:  
<https://www.gov.uk/government/publications/saturated-fats-and-health-sacn-report>  
Accessed 12 December 2019.
  47. Kris-Etherton, PM, Petersen, K, Van Horn, L. Convincing evidence supports reducing saturated fat to decrease cardiovascular disease risk. *BMJNPH* 0: 1-4, 2018.
  48. Clifton P, Keogh J. Dietary Fats and Cardiovascular Disease: An Evidence Check Rapid Review Brokered by the Sax Institute for the National Heart Foundation of Australia. The National Heart Foundation of Australia: Adelaide, Australia. 2017.
  49. Farvid MS, Ding M, Pan A, et al. Dietary linoleic acid and risk of coronary heart disease: a systematic review and meta-analysis of prospective cohort studies. *Circulation* 130:1568-1578. 2014.

## Supplementary Data

Item/description	Amount (g)	Item/description	Amount (g)
<b>Meal 1</b>		<b>Meal 5</b>	
Nestle, Cheerios	80 g	English Cheddar Cheese	45 g (1 x average portion)
Whole Milk Cows	515 ml (1 pint)	Stilton Blue Cheese	35 g (1 x average portion)
Brown Bread, toasted	64 g (2 x medium slices)	Brie Soft Cheese	40 g (1 x average portion)
Seedless Grapes	250 g (Half a punnet)	Low Fat Yogurt	125 ml (1 x average pot)
Low Fat Yoghurt	250 g (2 x average pots)		
Banana & Strawberry Smoothie (with whole milk)	400 g (1 large portion)		
<b>Meal 2 – Pre Exercise</b>		<b>Meal 6</b>	
Preworkout (Brand: PhD)	15 g (1 serving)	Spaghetti Bolognese	
Maltodextrin	60 g	White Pasta	400 g (extra-large portion)
BCAA Powder	15 g (1 tablespoon)	Minced Beef	300 g (extra-large portion)
Coca-Cola	500 ml (1 bottle)	Tomato Passata	200 ml (half a jar)
<b>Meal 3 – Post Exercise</b>		Garlic Bread	240 g (1 baguette)
Whey Protein (Brand: NRG)	30 g (one serving)	<b>Meal 7</b>	
Whole Milk	250 ml (half pint)	Walkers, Ready Salted Crisps	90 g (2 x packet)
Banana	200g (2 x average banana)	McVities, Mini Cheddars	50 g (1 x packet)
Cadburys Twirl	160 g (4 x small bars)	Cadburys Twirl	160 g (4 x small bars)
<b>Meal 4</b>		Haribo, Super Mix	215 g (1 large packet)
McVities, Mini Cheddars	50 g (1 x packet)		
Walkers, Ready Salted Crisps	90 g (2 x packet)		
Egg Mayonnaise Sandwich White Bread	290g (2 x sandwich)		

**Supplement S1.** Participant 3 Baseline Sample Strongman Menu



**Supplement S2a-c.** Anaerobic Power Output Assessed by Wingate Relative to the Lean Body Mass of Strongmen. **A** baseline, **B** 12 weeks, **C** 24 weeks, Solid line (—) indicates power output (W), broken line (- - -) indicates lean body mass (estimated by bioelectrical impedance) relative to power output.