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TITLE

How effective is weight loss in reducing cardiometabolic risk? An observational analysis of two randomised controlled trials of community weight loss programmes.

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ABSTRACT

Background: Guidelines recommend clinicians identify individuals at high cardiometabolic risk and support weight loss in those with overweight or obesity. However, individual level data quantifying the benefits of weight change for individuals, to guide these discussions in primary care, is lacking.

Aim: Examine how weight change affects cardiometabolic risk factors, to facilitate shared decision-making between patients and clinicians regarding weight loss.

Design and setting: Observational analysis using data from two trials of referral of individuals with overweight or obesity in primary care to community weight loss groups.

Method: Linear mixed effects regression modelling, examining the association between weight change and change in systolic and diastolic blood pressure (SBP, DBP), fasting glucose, HbA1c, and lipid profile across multiple timepoints (baseline to 24 months). Subgroup analyses examined changes in individuals with hypertension, diabetes and hyperlipidaemia.

Results: 2041 participants had a mean(\pm SD) age of 50 ± 13.5 years, baseline weight 90.6 ± 14.8 kg and Body Mass Index 32.7 ± 4.1 kg/m². Mean(\pm SD) weight change was -4.3 ± 6.0 kg. All outcome measures showed statistically significant improvements. Each 1kg weight loss was associated with 0.4mmHg reduction in SBP and 0.3mmHg reduction in DBP, or 0.5mmHg and 0.4mmHg/kg respectively in people with hypertension. Each 1kg weight loss was associated with 0.2mol/mol reduction in HbA1c, or 0.6mmol/mol in people with diabetes. Effects on plasma lipids were negligible.

Conclusion: Weight loss achieved through referral to community weight loss programmes, which are commonly accessible in primary care, can lead to clinically relevant reductions in blood pressure and glucose regulation, especially in those at highest risk.

HOW THIS FITS IN:

- Guidelines recommend that clinicians identify patients who are at high risk of cardiometabolic disease (e.g. people with high blood pressure) and support weight loss in those with overweight or obesity. However, information quantifying the benefits of weight change for individuals, to guide these discussions in primary care, is lacking.
- We demonstrate that weight loss achieved through referral to community weight loss programmes, which are commonly accessible in primary care in the UK, can lead to clinically relevant reductions in blood pressure and glucose regulation, especially in those at highest risk.
- An average weight loss of 5kg could be expected to reduce HbA1c by 3mmol/mol in a person with type 2 diabetes, and reduce systolic blood pressure by 2mmHg in a person with hypertension. Larger weight losses give greater benefits, and reductions of around 10 kg are likely to be needed if a patient wishes to defer or reduce medication.

KEYWORDS: Obesity, weight loss, lifestyle, diabetes, hypertension, primary care, cardiometabolic risk

INTRODUCTION

Obesity is a global public health problem. In the UK the prevalence of obesity has more than doubled in the last 30 years and today more than a quarter of adults are living with obesity.(1, 2) Obesity causes cardiovascular disease and diabetes and a holistic approach, which treats the underlying cause will improve long-term outcomes for patients. In 2019, the NHS Long-Term Plan highlighted obesity as a key focus for disease prevention, committing to providing targeted support and access to weight management services in primary care.(3) The UK government's recent 2020 Obesity Plan reinforces this call to action, placing primary care firmly at the centre of attempts to improve access for patients to effective weight management services.(4)

Guidelines recommend that clinicians identify patients who are at high cardiovascular or metabolic risk, and promote weight loss to those with overweight or obesity, giving relevant information on the associated health risks and setting realistic targets.(5, 6) This may increase patient engagement(7), but many clinicians cite insufficient confidence, knowledge and skills to give specific recommendations(8), and feel uncertain whether behavioural support will lead to weight loss with clinically significant improvements in disease risk.(9, 10)

However, there is now good evidence that behavioural weight management programmes suitable for use at scale in routine healthcare do lead to weight loss.(11-15) While population-level benefits of weight loss in reducing cardiometabolic risk are well established, estimates of the magnitude of benefits seen on an individual level vary. Previous studies have suggested reductions of 0.5-1mmHg in blood pressure, 0.1% HbA1c, and 0.02mmol/L in total cholesterol for every 1kg of weight lost(16-20); however conclusions have often been limited by short duration of follow up and small sample sizes, with changes observed at only one follow up time point, often the point of maximal weight loss.

Here, we aim to examine the association between weight loss and changes in cardiometabolic risk profile (blood pressure, glycaemia and lipids), at any time after referral to a weight loss intervention, using data from an international cohort of participants in two large weight loss trials with body weight measured over 24 months. This analysis incorporates the variations in individuals' own weight loss trajectories to provide a realistic assessment of the changes in markers of cardiometabolic risk as a result of intentional weight loss achieved through interventions available in routine practice.

METHODS

Study Characteristics

We pooled data from two trials that tested the effectiveness of GP referral to community weight loss programmes, in an observational analysis including all trial arms. Both trials and protocols have been published in full.(12, 21) The first(21), a parallel group, non-blinded, multicentre randomised controlled trial recruited 772 adults with overweight or obesity from 115 primary care practices across Australia, Germany, and the UK. Participants were randomly assigned to either 12 months of standard care, or 12 months free membership to a weekly weight-management programme (WW, previously known as WeightWatchers), and were followed up with assessments at baseline, 2, 4, 6, 9 and 12 months. The second parallel-group, non-blinded, multicentre randomised trial(12), known as the WRAP trial, recruited 1269 adults with overweight or obesity from 23 primary care practices in England. Participants were randomly assigned to brief advice and self-help materials, a weight-management programme (WW) for 12 weeks, or the same weight-management programme for 52 weeks, and were followed up with assessments at baseline, 3, 12 and 24 months.

It has been difficult to draw conclusions about changes in cardiometabolic risk factors relevant to individual patients in UK primary care from previous systematic review evidence because of the heterogeneity and intensity of included interventions, and the lack of individual patient data with repeated measures over time(17, 19, 22). These two studies were selected to address these limitations, being as representative as possible of patients referred in routine primary care settings, with use of community weight loss programmes that are currently widely available in UK practice, and comparable pragmatic trial designs justifying pooling data from both studies. Our access to individual patient data, with multiple follow up measurements at different timepoints, enabled us to account for the variability of individuals' weight loss trajectories.

Statistical analyses

Analyses were performed using Stata v16 SE (StataCorp LP, College Station, TX). The primary analyses assessed the association between the extent of weight change and changes in cardiovascular disease risk factors. Data from all participants in both studies were pooled, and all analyses adjusted for baseline values, time, gender, age, ethnicity, trial and treatment group. We used a linear mixed effects modelling approach, with observations nested within participant, to account for repeated measurements over time from the same participant.

Separate linear mixed effects regression models were fitted for changes in systolic blood pressure (SBP), diastolic blood pressure (DBP), glucose, glycated haemoglobin (HbA1c), total cholesterol, high-

and low-density lipoprotein (HDL, LDL), and triglycerides. For each individual participant, repeated measurements were available at different time points (any of: 2, 3, 4, 6, 9, 12, 24 months) for each outcome. Random slope models with an unstructured covariance structure were fitted to the data, as time (visit in months) was included as both a fixed and random effect, to account for individual variability in the outcome over time. For all analyses, calculated weight change from baseline was used as the weight change value for each time-point, and the coefficient of weight change (at any time) yielded the primary outcome for each model. Change was calculated as observation 2 – observation 1 (e.g. weight at follow up minus weight at baseline). Model outputs are reported to 1 significant figure, or to the nearest integer for clinical examples.

We performed pre-specified sub-group analyses according to participants' baseline hypertension status, diabetes status, and plasma lipids, for the outcomes of blood pressure, glycaemia and lipid profile, respectively. Hypertension status was defined by: GP record coding as hypertensive, taking antihypertensive medication, baseline SBP>140mmHg, or baseline DBP>90mmHg. Diabetes status was defined by: GP record coding as diabetic, taking diabetes medication, or baseline HbA1c in diabetic range (≥ 48 mmol/mol). Due to inconsistent coding for hyperlipidaemia between datasets, clinical thresholds for baseline values were used to define abnormal plasma lipids: total cholesterol >5mmol/L, HDL <1mmol/L, calculated LDL >3mmol/L, TG >1.7mmol/L. For each outcome we fitted a separate linear mixed effects model for each subgroup, as models including an interaction term for subgroup status did not converge.

RESULTS

The final dataset comprised 2041 participants with a mean \pm SD age of 50 ± 13.5 years and weight of 90.6 ± 14.8 kg (BMI 32.7 ± 4.1 kg/m²) at baseline. Three-quarters of participants were female, with just under 10% from ethnic minority groups (table 1). Mean \pm SD weight change across all time points was -4.3 ± 6.0 kg (range -51.2 to +21.5kg), equating to 4.7% total bodyweight loss (table 2). Nearly half of participants achieved 5% weight loss measured on at least one occasion and just under a quarter lost >10% (table 2).

There were statistically significant improvements in all outcome measures (SBP, DBP, HbA1c, glucose, total cholesterol, LDL- and HDL- cholesterol, and triglycerides) with weight loss (table 3).

On average across the whole population, each kilogram of weight loss was associated with a 0.4mmHg reduction in SBP and 0.3mmHg reduction in DBP. For people with hypertension, greater

reductions of 0.5mmHg and 0.4mmHg per kilogram were seen in SBP and DBP, respectively, compared with 0.3mmHg and 0.3mmHg for people without hypertension (table 4).

Each kilogram of weight loss was associated with a 0.2mmol/mol reduction in HbA1c. In people with diabetes, each kilogram of weight loss was associated with a 0.6mmol/mol reduction in HbA1c compared with 0.1mmol/mol in people without diabetes (table 4). Calculated examples of the change in BP and HbA1c that a patient might expect with different magnitudes of weight loss are presented in table 5.

The effect on plasma lipids was small, with a change of -0.02, -0.01 and 0.003mmol/L in total-, LDL- and HDL-cholesterol, and -0.02mmol/L in triglycerides for every 1kg of weight lost. A slightly greater change in HDL (0.006mmol/L) and triglycerides (-0.04mmol/L) was seen in those with abnormal plasma lipids at baseline (table 4).

DISCUSSION

Summary

In individuals with overweight or obesity, weight loss achieved through referral to community weight loss programmes does lead to clinically relevant reductions in blood pressure and glycaemic control, especially in those at highest cardiometabolic risk. These data indicate that a 5kg weight loss in people with hypertension will lead to an average reduction in SBP of 2.5mmHg, increasing to 5mmHg with 10kg weight loss (table 5). For people with diabetes, 5kg and 10kg weight loss may improve HbA1c by 3mmol/mol and 6mmol/mol, respectively.

Strengths and limitations

We analysed two large pragmatic trials of community weight loss programmes which are typical of the behavioural interventions available for referral in UK primary care. The findings are directly relevant for clinicians responding to NICE guidance that suggests clinicians should set specific goals for weight loss with individual patients – with realistic expectations for changes in associated cardiometabolic risk factors(5, 6). Previous studies of weight loss and cardiovascular risk outcomes have used single timepoints at maximal observed effect,(17) with meta-analyses based only on participants that completed the studies(22), and as such cannot give an unbiased estimate of the typical association. Our approach addresses this limitation by performing a meta-analysis of individual patient data using mixed effects regression modelling to allow incorporation of all data for individuals across available timepoints. We use multiple repeated observations over time to account for individuals' trajectories of weight loss and regain and the associated change in cardiometabolic outcomes, to provide a more realistic appraisal of the effects of weight change on an individual

patient basis. This enables estimation of the average size of effect an individual might expect per kilogram of weight loss, rather than average effect of an intervention.

However, there are limitations. Firstly, participants were predominately female (75%) and white (90%). This limits the extent to which our findings may be generalised to other patient groups, especially for patients of different ethnicities, as this may modify the effect of weight loss and could not be adequately assessed in these analyses.(23, 24) Secondly, the relatively small proportion of participants with diabetes at baseline (12%) and fewer HbA1c measurements available than for other outcomes may make the findings relating to glycaemia less reliable. Thirdly, it remains possible that a confounder correlated with weight loss, but not caused by it, explains the association we report between weight loss and improvements in cardiometabolic risk factors, though this seems unlikely. We could not account for changes in concomitant medication use in our model (a frequent limitation among meta-analyses describing BP/HbA1c change with weight loss(17, 18, 22)). In pragmatic trials in routine practice such as these, GPs may have added or removed medications in response to changes in blood pressure or glycaemia, thus underestimating the strength of the association, and we do not have data to assess whether this is the case.

Comparison with existing literature

Previous reviews have reported up to a 1kg:1mmHg relationship between change in weight and change in BP(17, 18, 22), approximately double that reported here. One such meta-analysis of RCTs reporting a 1kg:1mmHg ratio assessed outcome measures at the timepoint of maximal observed effect (a mean duration of 35.3 weeks), which may represent acute but not more sustained changes, whereas we use repeated measures for individuals up to 24 months from commencing weight loss.(17) Additionally, as in our findings, the effect of weight loss on BP was larger in patients with pre-existing hypertension. People with hypertension formed a larger proportion of the study populations in the meta-analysis (half the included patients, compared with 35% in our study), which may contribute to the larger effect found. The demographics of the population we studied (average age 50 years, majority female participants) is more representative of the people currently being referred to community weight loss programmes in the UK (median age 49 years, 90% female (25, 26)) than people commonly recruited to trials such as those included in Neter et al's meta-analysis (which studied a younger, predominately (64%) male population)(17).

Our findings that suggest a 10kg weight loss is associated with a 6mmol/mol (0.5%) improvement in HbA1c in patients with type 2 diabetes align with data presented from observational analyses of the Look AHEAD study of individuals with type 2 diabetes and obesity over 1 year.(27) The greater

benefit demonstrated in fasting glucose in the short term in Look AHEAD may have been observed due to the worse glycaemic control of their study population at baseline (mean \pm SD baseline HbA1c (%) 7.28 ± 1.17 , mean (\pm SD) baseline fasting glucose (mmol/L) 8.5 ± 2.5 in LookAhead; compared to the subgroup of participants with type 2 diabetes in our study: mean \pm SD baseline HbA1c (%) 6.9 ± 1.35 , mean (\pm SD) baseline fasting glucose (mmol/L) 7.4 ± 2.6).

A previous meta-analysis reported a 0.2mmol/L reduction in total cholesterol per 10kg weight loss, which is consistent with our finding.(19) This 3-5% reduction in total cholesterol observed - while statistically significant - is less than a quarter of the expected reduction from starting treatment with a statin.(28) While this alone is unlikely to significantly reduce cardiovascular risk for an individual, a higher body mass index (BMI) is associated with higher cardiovascular disease risk independent of cholesterol status, and as such any weight reduction is likely to be of benefit (29, 30). In addition, population level benefits remain; it is estimated that a reduction of only 2% in the total cholesterol level of each person in the population could lead to 42 fewer deaths per 100,000 people from coronary heart disease over 10 years(31, 32).

Implications for research and practice

The need to support weight loss in people with overweight or obesity and at high cardiometabolic risk is now widely recognised, and is a core recommendation in national guidance and the NHS Long Term Plan.(3, 5) Despite this, pharmacotherapy remains the predominant management strategy for treatment and prevention of cardiometabolic disease: in the recent Health Survey for England over 90% of people with diabetes or cardiovascular disease reported being on one or more prescribed medications(33), while only a quarter of people with overweight or obesity reported ever having received a health professional's advice to lose weight(34).

Our findings show that modest weight loss is associated with improved cardiometabolic risk factors, and that more weight loss is associated with greater changes; so how much weight should patients aim to lose? A threshold of 5% body weight loss is often quoted as a target from which clinically significant results may be expected(6). This was close to the average weight loss seen in this study (4.3kg, 4.7% average percentage body weight change) and was associated with small but some clinically relevant benefits; for example, a 2.5mmol/mol (95% CI 1.5 to 3.4mmol/mol) reduction in HbA1c in patients with diabetes. With over 40% of people referred to a 12-week community weight loss programme maintaining 5% weight loss at 1 year, this presents an appropriate and reasonable initial goal(12). However, greater weight loss brings bigger benefits. For an individual patient hoping to avoid starting or escalating medication usage in response to a diagnosis of raised BP or impaired glycaemia, weight loss of at least 10% (equating to 9-10kg weight loss for the average patient in our

study population, approximately one standard deviation above the average weight loss seen) may be required to give a realistic possibility that it will reduce their need for medication. Metformin monotherapy can be expected to reduce HbA1c by around 12mmol/mol(35), while starting an antihypertensive agent reduces average SBP by 9.1mmHg, and DBP by 5.5mmHg(36). In the studies analysed here, 26% of participants referred to community weight loss services lost $\geq 10\%$ of their body weight (≥ 9 -10kg weight loss). Our results suggest that weight loss of this magnitude could confer benefits in BP and HbA1c equivalent to approximately half that of commencing drug monotherapy with antihypertensive agents or metformin. This is not an insignificant amount of weight for a patient to lose, and is far greater than the average weight loss of 1kg observed when patients are just given brief advice to lose weight by their GP without referral for additional support(11). This reinforces the message that clinicians should focus on directing patients towards effective support if they are aiming to achieve these greater weight losses. It is hard to predict which patients will have most success from weight management programmes, except based on early weight loss achievements once attempting the programme(37). Around 1 in 7 people following this kind of intervention will achieve $\geq 10\%$ body weight reduction at 1 year in the 12 week programmes that are commonly commissioned.(11, 12) However, this increases to 1 in 3 people referred to a 52 week programme, suggesting that repeat (or extended) referrals for people who are achieving weight loss are clinically beneficial, and have been shown to be cost effective(12).

Here we have focussed on cardiometabolic risk, but the individual and population benefits of this achievable weight loss are likely to be much wider, from improvements in comorbidities (such as reduction of pain and improvements in mobility in osteoarthritis(38)) to reductions in primary care resource utilisation.(39) A recent study demonstrated that excess weight accounts for an estimated 11% (£229 million) of all primary care consultation costs and 20% (£384 million) of prescription medication costs, with each BMI increase of 2kg/m² (in women with BMI >20kg/m²) associated with 5.2% (4.8–5.6) and 9.9% (9.2–10.6) higher mean annual consultation and prescription medication cost, respectively.(39) Helping patients to reduce their weight could not only improve their health but also reduce pressures on primary care. But to realise this potential, and that of population level prevention of CVD, there is a need to actively offer referrals to weight loss programmes and engage far more widely than current figures suggest. The type of interventions in the trials in this study are widely available for GP referral and use in the UK NHS.

In summary, moderate weight loss of 5-10kg can achieve clinically meaningful reductions in markers of cardiometabolic risk and may offer the opportunity for patients to defer or reduce medication. These data provide clinicians with the information they need to discuss this option in their consultations.

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CONFLICT OF INTEREST

PA, SAJ, HH and IC have received research grant funding but no personal remuneration from commercial weight loss companies. ALA is Principal Investigator on two publicly funded trials where the intervention is provided by WW.

AUTHOR CONTRIBUTIONS

EM, PA and SAJ developed the concept for the study. SAJ, PA, AA, JH, EB, IC, HH were involved in data collection. EM, PA, JO and AN developed the statistical analysis plan and model. EM drafted the manuscript for publication, with input from PA and SAJ. All of the authors were involved in review and revision of the manuscript and approved the final manuscript.

DISCLAIMER

This report is independent research funded by the National Institute for Health Research. The views expressed in this publication are those of the authors and not necessarily those of the NHS, the University of Oxford, the NIHR or the Department of Health and Social Care.

Table 1: Baseline characteristics.

	Study 1 (21)	Study 2 (12)	Total sample
N, total participants	772	1269	2041
Observations, n			
Weight	4033	3952	7985
BP	3853	3939	7792
HbA1c	1265	1314	2579
Blood test, other (glucose, lipid profile – n, each)	2251	1329	3580
Sex, n			
Male	104	408	512
Female	668	861	1529
Ethnicity, n			
White	482	1138	1620
Asian	23	26	49
Black	4	22	26
Other	68	41	109
Data not available	195	42	237
Age (years) *	47 ± 12.7	53 ± 13.7	50 ± 13.5
Baseline BMI (kg/m ²)*	31.4 ± 2.6	34.5 ± 5.1	32.7 ± 4.1
Baseline Weight (kg)*	86.7 ± 11.5	96.1 ± 17	90.6 ± 14.8
Baseline HbA1c (mmol/mol)*	38.2 ± 5.3	41.3 ± 10.2	39.2 ± 7.4
Baseline Glucose (mmol/L)*	5.0 ± 0.8	5.7 ± 1.7	5.2 ± 1.2
Baseline Systolic BP (mmHg)*	124 ± 16	133 ± 17	128 ± 17
Baseline Diastolic BP (mmHg)*	79 ± 9	80 ± 10	79 ± 10
Baseline Total cholesterol (mmol/L)*	5.3 ± 1	5.3 ± 1.1	5.3 ± 1
Baseline HDL cholesterol (mmol/L)*	1.4 ± 0.4	1.7 ± 0.6	1.5 ± 0.5
Baseline LDL cholesterol (mmol/L)*	3.2 ± 0.9	3.0 ± 1.0	3.1 ± 0.9
Baseline Triglycerides (mmol/L)*	1.5 ± 0.9	1.5 ± 0.8	1.5 ± 0.9

Diabetes at baseline, n (%)	51 (7%)	190 (15%)	241 (12%)
Hypertension at baseline, n (%)	92 (12%)	631 (50%)	723 (35%)

*mean \pm SD

Table 2: Mean weight loss

	Observations, n	Mean weight change, kg \pm SD	Mean % change from baseline, % +/- SD	Number of people achieving \geq 5% weight loss, n (% with recorded weight)	Number of people achieving \geq 10% weight loss, n (% with recorded weight)
Across all data points	5944	-4.3 \pm 6.0	-4.7 \pm 6.4	973 (48)	444 (22)
At 12 months	1267	-5.6 \pm 7.2	-6.1 \pm 7.6	596 (47)	317 (25)
At 24 months	1197	-3.6 \pm 7.7	-3.8 \pm 8.1	419 (35)	213 (18)

Table 3: Association of clinical variables with weight change

Model	Units	Association with weight change (Change in outcome per kg weight change (95% C.I))	p value
Systolic BP [#]	mmHg	0.4 (0.32 to 0.45)	<0.001
Diastolic BP [#]	mmHg	0.3 (0.29 to 0.37)	<0.001
Total Cholesterol [#]	Mmol/L	0.02 (0.01 to 0.02)	<0.001

HDL Cholesterol[#]	Mmol/L	-0.003 (-0.006 to 0.0006)	0.015
LDL Cholesterol[#]	Mmol/L	0.01 (0.01 to 0.02)	<0.001
Triglycerides[#]	Mmol/L	0.02 (0.02 to 0.03)	<0.001
HbA1c[#]	Mmol/mol	0.2 (0.13 to 0.22)	<0.001
Glucose[#]	Mmol/L	0.02 (0.01 to 0.03)	<0.001

statistically significant change at p<0.05;

Table 4: Sub-group analysis according to baseline diabetes, hypertension and lipid status

Subgroup	Variable	Units	Association with weight change (Change in outcome per kg weight change (95% C.I))	p value
Hypertension	Systolic BP[#]	mmHg	0.5 (0.36-0.60)	<0.001
	Diastolic BP[#]	mmHg	0.4 (0.29-0.43)	<0.001
No hypertension	Systolic BP[#]	mmHg	0.3 (0.27-0.42)	<0.001
	Diastolic BP[#]	mmHg	0.3 (0.26-0.36)	<0.001
Diabetes	HbA1c[#]	Mmol/mol	0.6 (0.35-0.79)	<0.001
	Glucose[#]	Mmol/L	0.07 (0.03-0.12)	0.002
No Diabetes	HbA1c[#]	Mmol/mol	0.1 (0.09-0.14)	<0.001
	Glucose[#]	Mmol/L	0.02 (0.01-0.02)	<0.001
Abnormal plasma lipids	Total cholesterol[#]	Mmol/L	0.02 (0.01 – 0.02)	<0.001
	HDL[#]	Mmol/L	-0.006 (-0.01 - -0.0003)	0.037
	LDL[#]	Mmol/L	0.01 (0.005 – 0.02)	0.002
	Triglycerides[#]	Mmol/L	0.04 (0.03 – 0.05)	<0.001
Normal plasma lipids	Total cholesterol[#]	Mmol/L	0.02 (0.01 – 0.03)	<0.001
	HDL[#]	Mmol/L	-0.003 (-0.006 – -0.0003)	0.028
	LDL[#]	Mmol/L	0.01 (0.002 – 0.02)	0.011
	Triglycerides[#]	Mmol/L	0.02 (0.01 – 0.02)	<0.001

statistically significant change at p<0.05;

Table 5: Examples of average predicted changes in blood pressure and glycaemic control with weight change”

Variable	Example: variable change for X kg weight change	Variable change for every 5kg weight change	Variable change for every 10kg weight change
Systolic BP <i>(all patients)</i>	2mmHg (1.6 to 2.2mmHg) SBP change for 5kg weight change	2mmHg (1.6 to 2.2mmHg) SBP change for 5kg weight change	4mmHg (3.2 to 4.5mmHg) SBP change for 10kg weight change
Systolic BP <i>(hypertensive subgroup)</i>	1mmHg (0.8 to 1.2 mmHg) SBP change for every 2kg weight change	2mmHg (1.8 to 3.0mmHg) SBP change for 5kg weight change	5mmHg (3.6 to 6.0 mmHg) SBP change for 10kg weight change
Diastolic BP <i>(all patients)</i>	3mmHg (2.9 to 3.7mmHg) DBP change for 10kg weight change	1.5mmHg (1.4 to 1.8mmHg) DBP change for 5kg weight change	3mmHg (2.9 to 3.7mmHg) DBP change for 10kg weight change
Diastolic BP <i>(hypertensive subgroup)</i>	2mmHg (1.4 to 2.2 mmHg) DBP change for 5kg weight change	2mmHg (1.4 to 2.2mmHg) DBP change for 5kg weight change	4mmHg (2.9 to 4.3mmHg) DBP change for 10kg weight change
HbA1c <i>(all patients)</i>	1mmol/mol (0.7 to 1.1mmol/mol) HbA1c change for 5kg weight change	1mmol/mol (0.7 to 1.1mmol/mol) HbA1c change for 5kg weight change	2mmol/mol (1.3 to 2.2 mmol/mol) HbA1c change for 10kg weight change
HbA1c <i>(diabetic subgroup)</i>	3mmol/mol (1.8 to 4.0 mmol/mol) HbA1c change for 5kg weight change	3mmol/mol (1.8 to 4.0 mmol/mol) HbA1c change for 5kg weight change	6mmol/mol (3.5 to 8.0 mmol/mol) HbA1c change for 10kg weight change
Glucose <i>(all patients)</i>	0.2mmol (0.1 to 0.2mmol) glucose change for 10kg weight change	0.1mmol (0.1 to 0.1mmol) glucose change for 5kg weight change	0.2mmol (0.1 to 0.3mmol) glucose change for 10kg weight change
Glucose <i>(diabetic subgroup)</i>	0.6mmol (0.2 to 1.0 mmol) glucose change for 8kg weight change	0.4mmol (0.1 to 0.6mmol) glucose change for 5kg weight change	0.7mmol(0.3 to 1.2mmol) glucose change for 10kg weight change

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