Long-Term Weight Loss From Lifestyle Intervention Benefits Blood Pressure? A Systematic Review

Lorna Aucott, Helen Rothnie, Linda McIntyre, Mohan Thapa, Charles Waweru, Denise Gray

Abstract—Weight gain may increase blood pressure. Weight loss may reduce this. Reviews have considered the long-term effects of weight loss but are related mainly to more obese participants often on obesity medication and/or undergoing obesity surgery. This systematic review, based on lifestyle interventions for adults (18 to 65 years) with mean baseline BMI of <35 kg/m², links weight change to blood pressure difference. A systematic review of studies reporting weight differences and blood pressure outcomes, published between 1990 and 2008 with follow-up of ≥2 years identified 8 clinical trials or controlled before and after studies (represented by 9 articles) and 8 cohort studies. Differences ranged from −11 to +4kg for weight, −7 to +2.2 mm Hg for diastolic blood pressure and −13 to +6.1 mm Hg for systolic blood pressure. For this population group, no quantifiable relationship between weight and diastolic blood pressure difference was found, possibly because of small weight losses, differing weight status responses, or because pharmacologically controlled hypertension masked weight loss influences. Systolic differences were in line with previous reviews of 1 kg:1 mm Hg relationship, but only for follow-up periods of 2 to 3 years, possibly reflecting the fact that regardless of maintained weight loss, blood pressure often reverts back to higher levels. Lifestyle interventions for weight and blood pressure are limited in this target group, and there has been no exploration of successful intervention components. An individual patient data analysis may uncover baseline and medication effects, explore differences between weight groups, and may identify successful components. Such an analysis would enable effective development of preventative interventions for both hypertension and obesity. (Hypertension. 2009;54:756-762.)

Key Words: lifestyle ■ obesity prevention ■ weight loss ■ blood pressure

Hypertension is an important risk factor for cardiovascular morbidity and mortality.1,2 In adults, hypertension often rises with increasing body weight.3 Intervention studies and reviews have reported blood pressure reductions associated with weight loss, particularly in the short-term.4–6 A recent review of long-term effects of weight loss on hypertension in overweight or obese subjects, excluding surgical weight loss interventions, concluded that blood pressure reduction was about half that found in short-term trials.7 A leading aim of contemporary public health policy is to reduce levels of obesity, although how to achieve this, or how effective population-level interventions to reduce obesity would be in reducing blood pressure, is still not evident. The aim of this research was to systematically review evidence linking long-term weight and lifestyle changes to blood pressure changes for those with a body mass index (BMI) of ≤35 kg/m².

Methods
This review considers longitudinal data rather than “between-treatment” data linking weight differences with blood pressure differences. Consequently, differences recorded for weight and blood pressure from clinical trials (CTs) (including randomized control trials), controlled before and after studies (CBAs), and cohort studies (including interrupted time series) between 1990 and 2008 were considered.

Two systematic literature searches (part of a National Prevention Research Initiative–funded economic evaluation of obesity prevention for UK adults) were conducted. The first identified CTs and CBAs with lifestyle interventions to prevent weight gain. The second identified similar interventions in cohorts as well as studies without formal interventions but where weight loss/prevention was intentional and lifestyle based.

The searches used MeSH terms and text words for “Trials,” “obesity,” “overweight,” “weight differences” appropriately combined. The first search, for CTs and CBAs from 1990 onwards, was based on key reports, systematic reviews, and primary studies indexed in Medline, Embase, PsycINFO, CINAHL, The Evidence Based Medicine Reviews Collection, CABI Nutrition Abstracts and Reviews, along with hand searching of International Journal of Obesity and Obesity Research. The last date of this search was October 2007 (full details available from the authors). Studies relevant to longitudinal measures in both weight and health outcomes were considered in this review. These included lifestyle intervention arms (even if the other arms were surgical or drug-based) provided the inclusion criteria were met. Thus, although some of these studies
were CTs, for our analysis there were no “true” control groups. The search was extended up to April 2008 using the same search strategy ensuring that all relevant studies from this phase were identified. Medline, Embase, and CINAHL were also searched from 1990 to 2008 for cohort studies. Reference lists from relevant primary and review articles were investigated.

Criteria for inclusion were a ≥2-year follow-up for studies with either lifestyle interventions for weight loss (dietary, exercise, behavioral, or environmental) or where weight loss was intentional (including weight cycling) along with records of long-term differences in blood pressure for adult (18 to 65 years at recruitment) participants. Studies were excluded if participants had a mean baseline BMI >35 kg/m², or had eating disorders, were pregnant, or were severely mentally or physically handicapped. Although there were no language restrictions, studies with ethnic groups not relevant were excluded. Although there were no language restrictions, studies with ethnic groups not relevant were also excluded from authors. Only once sufficient information was obtained was final inclusion possible. Of those with extractable data, suitable papers. Several articles required extra information text documents were assessed resulting in 54 potentially competent in the relevant language, or translated by a third party before assessment. All titles/abstracts and full text papers were independently assessed against the inclusion criteria by 2 reviewers, with any disagreements being arbitrated by discussion or by a third reviewer.

**Statistical Analysis**

Blood pressure (BP) changes related to weight loss in the long term were considered longitudinally for significance. Ideally, differences between follow-up and baseline were required along with associated precision. When not provided, suitable imputed estimates were made for mean differences using the change between means at follow-up and baseline. Similarly, associated standard deviations (SDs) were estimated using $SD_{diff} = \sqrt{SD_{df}^2 + SD_{Bf}^2}$, where suffixes D, f, and B represent difference, follow-up, and baseline, respectively. Theoretically, the difference variance would be $\sigma_{df}^2 = \sigma_d^2 + \sigma_f^2 - 2\sigma_{df}$, where $\sigma_{df}$ is the covariance between follow-up and baseline measures. However, where only estimates of baseline and follow-up variances are known, the proposed estimate gives a conservative measure with no covariance assumptions.

BP differences (and percentage changes) were correlated with weight differences and other variables that potentially affect BP, namely mean age, follow-up time, sex mix, and baseline variables. Meta-regression models were constructed using weighted least squares (WLS) regression predicting BP differences. Using the standard error (SE) of the mean differences of either the diastolic or systolic mean differences, model weights were defined as $1/SE^2$. The generated regression coefficients SEs then required adjustment to determine coefficient significance. Initially all subgroups were included. However, some subgroups represented different follow-up times, consequently models were also constructed separately for follow-up times of 2 to 3 years and 3+ years.

**Results**

We initially present lifestyle arms of CTs followed by cohort studies with lifestyle interventions, and finally those studies with no intervention but where weight loss was intentional. The searches identified 4977 abstracts, from which 405 full text documents were assessed resulting in 54 potentially suitable papers. Several articles required extra information from authors. Only once sufficient information was obtained was final inclusion possible. Of those with extractable data, 17 articles recorded BP measures, where 9 related to 8 CT studies11–19 and 8 were cohort studies with either lifestyle interventions or with intentional weight loss.20–27 Supplemental Tables S1 and S2 (please see http://hyper.ahajournals.org) give the basic characteristics of these CTs and cohort studies.

Studies had between 24 and 72 months follow-up with single or mixed gender groups. Most studies reported differences in weight and BP from baseline to follow-up. However, one weight cycling study reported on the risk of developing hypertension after 2 years, having classified participants into weight cycling groups based on the previous 2 years of intentional weight differences.

Longitudinal differences in weight and BP were tested for significance (Tables 1 and 2) using imputed mean differences and SDs when difference information was not available. One study provided difference data without precision, hence the average SD (from studies with SDs of differences) was used. Measures of precision were occasionally given as standard error of the mean (SEM) or as confidence intervals (CIs). These were converted into SDs for consistency. Similarly, differences reported as percentage change were converted to actual means and SDs. Studies providing only BMI were converted to estimate weight (kg) using UK tables28 to represent European or U.S. heights29 as appropriate.

**Clinical Trials**

Two of the 8 CTs were diet-based, whereas the rest had diet and physical activity components; most had clinical or academic settings. The follow-up periods were 2 to 4.5 years. Two articles reported on the same study after 27 months14 and 51 months.15

Although the main analysis for this review will be longitudinal, it is worth noting that of the 8 CTs, only 4 had “true” controls and even then only 1 specified their control to have “no dietary advice.” Otherwise, the control groups were under “usual care” for participants who had coronary artery disease,12 hypertension,13 or diabetes mellitus.18 This probably meant that they received diet/physical activity advice and maybe even relevant medication. Therefore, adjustment for available “control group BP change” would only be truly appropriate for 1 study.11

The weight and BP differences are given in Table 1. Average weight losses of 4 to 10 kg were observed for the meal replacement study,14,15 being largest for the “2 replacement meal” arm (Group B) at 27 months. Other significant average weight losses were approximately 3 kg.12,17,18 A walking group19 lost on average nearly 4 kg, although this was not significant, probably because of the large estimated SD. The largest and significant average drop in systolic blood pressure (SBP), 15 mm Hg, was reported by the meal replacement study.14,15 Other relevant SBP reductions were approximately 4 mm Hg. Reductions in diastolic blood pressure (DBP) of 3 to 5 mm Hg were reported by the hypertension trials,11,13 the meal replacement trial14,15 (significance suffers from small sample sizes), and the Finnish diabetes prevention study.18 Interestingly, results from the hypertension studies11,13 suggest better BP reductions using weight-reducing diets rather than sodium restriction.

**Cohort Studies**

Seven of the 8 included cohorts (Table 2) had dietary or behavioral interventions, with some incorporating exercise or combinations of regimes. Interventions were delivered in different settings, ranging from residential clinics to free-living work places with varying follow-up periods of 2 to 6 years. In addition, the frequency and duration of contact for each intervention differed; the residential intervention25 was
Other studies had inconsistent relationships between weight loss and BP reduction. The Spanish workplace study\textsuperscript{23} had a positive correlation between weight loss and BP reduction, whereas the TOHP\textsuperscript{13} study did not. The best predictors of difference in DBP used baseline weight and BP changes. Correlations of mean weight differences were used to develop regression models. Comparing BP differences with other variables indicate high correlations between baseline and final BPs and weight changes. Correlations of mean weight differences were used to develop regression models.

Table 1. Weight Differences With Diastolic and Systolic Blood Pressure Differences - CTs

<table>
<thead>
<tr>
<th>Study</th>
<th>Setting</th>
<th>Subgroup Description</th>
<th>Follow-Up Sample, n</th>
<th>Weight Difference, kg (SD)</th>
<th>SBP Difference, mm Hg (SD)</th>
<th>DBP Difference, mm Hg (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HPT\textsuperscript{11}</td>
<td>Clinical centers</td>
<td>Calorie restricted (36)</td>
<td>117</td>
<td>-1.63 (4.44)*</td>
<td>-5.00 (9.74)*</td>
<td>-4.20 (8.65)*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Calorie+Na restricted (36)</td>
<td>114§</td>
<td>-0.14 (4.38)</td>
<td>-3.60 (6.65)*</td>
<td>-3.70 (8.58)*</td>
</tr>
<tr>
<td>Haskel\textsuperscript{12}</td>
<td>Hospital</td>
<td>Risk reduction (48)</td>
<td>118</td>
<td>-3.00 (4.00)*</td>
<td>-0.60 (11.10)</td>
<td>-1.30 (7.10)**</td>
</tr>
<tr>
<td>TOHP\textsuperscript{13}</td>
<td>Academic medical centers</td>
<td>Diet+PA (36)</td>
<td>547††</td>
<td>-0.20 (5.90)</td>
<td>-0.80 (8.70)*</td>
<td>-3.20 (6.50)*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diet+PA+Na (36)</td>
<td>552‡‡</td>
<td>-0.30 (5.50)</td>
<td>-0.50 (9.00)</td>
<td>-2.90 (6.70)*</td>
</tr>
<tr>
<td>Ditschuniet\textsuperscript{14}</td>
<td>University hospital clinic</td>
<td>Group A (27)</td>
<td>31</td>
<td>-7.70 (16.00)*</td>
<td>-2.00 (19.11)</td>
<td>-3.00 (8.49)†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Group B (27)</td>
<td>32</td>
<td>-10.4 (19.16)*</td>
<td>-15.00 (19.21)*</td>
<td>-4.00 (7.81)§</td>
</tr>
<tr>
<td>Flechtner-Mors\textsuperscript{15}</td>
<td>Clinic</td>
<td>Group A (51)</td>
<td>38</td>
<td>-4.10 (15.42)</td>
<td>-1.00 (20.52)</td>
<td>-3.00 (9.22)†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Group B (51)</td>
<td>37</td>
<td>-9.5 (19.09)*</td>
<td>-13.00 (19.85)*</td>
<td>-4.00 (8.49)*§</td>
</tr>
<tr>
<td>Kuller\textsuperscript{16}</td>
<td>Clinic</td>
<td>Intervention Group (30)</td>
<td>245¶</td>
<td>-2.13 (5.58)*</td>
<td>-4.1 (14.26)*</td>
<td>+2.20 (9.45)*†</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intervention Group (42)</td>
<td>245¶</td>
<td>-1.00 (5.58)*</td>
<td>-3.90 (14.26)*</td>
<td>+0.49 (9.45)*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Intervention Group (54)</td>
<td>245¶</td>
<td>-0.08 (5.58)</td>
<td>-0.12 (14.26)</td>
<td>+1.50 (9.45)*</td>
</tr>
<tr>
<td>Heshka\textsuperscript{17}</td>
<td>Multi-center academic</td>
<td>Self-help (24)</td>
<td>159</td>
<td>-0.10 (7.57)</td>
<td>-2.40 (12.60)*</td>
<td>0.00 (7.57)</td>
</tr>
<tr>
<td>Lindstrom\textsuperscript{18}</td>
<td>Multi-center study</td>
<td>Commercial (mainly diet) (24)</td>
<td>148</td>
<td>-3.00 (7.30)*</td>
<td>-2.20 (13.38)*</td>
<td>-0.06 (8.52)†</td>
</tr>
<tr>
<td>Kukkonen-Harjula\textsuperscript{19}</td>
<td>Research institute clinic</td>
<td>Walking group (31)</td>
<td>20</td>
<td>-3.96 (15.12)</td>
<td>+1.00 (23.60)</td>
<td>+2.00 (15.62)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Resistance training (31)</td>
<td>26</td>
<td>-1.22 (12.43)</td>
<td>+4.00 (19.85)</td>
<td>+2 (13.45)</td>
</tr>
</tbody>
</table>

*Paired t test (follow-up-baseline) significant at P<0.05.
†Marginally not significant.
‡Imputed mean differences from follow-up–baseline values; Imputed SD for mean difference = \(\sqrt{SD^2_{\text{follow-up}} + SD^2_{\text{base}}}\).
§n=115 for SBP and DBP follow-up differences.
¶Imputed SD values based on average of 15 subgroups where SDs of mean differences were given.
††Sample size not presented. The 54-month follow-up sample size used as a conservative estimate (drop off increases with time).
**Marginally significant.
†††Diet group n=527 for DBP and SBP.
‡‡‡Diet Na group n=537 for DBP and SBP.
PA indicates physical activity; Na, sodium; SD, standard deviation.

The Figure shows mean differences, with 95% confidence intervals, for weight and BP. These data, combined using random effects models, demonstrate overall average differences of -2.8 kg (95% CI, -13.2, 7.5) for weight, -1.9 mm Hg for DBP (95% CI, -9.5, 5.6) and -2.9 mm Hg for SBP (95% CI, -9.2, 3.3). These nonsignificant differences have wide confidence intervals. Formal associations were further examined by meta-regression.

**Regression Analysis**

In total there were g=26 subgroups with information on weight and BP changes. Correlations of mean weight differences and percent weight changes between mean BP differences were similar. Hence, for easier interpretation, only mean weight differences were used to develop regression models. Comparing BP differences with other variables indicate high correlations between baseline and final BPs and hence are considered in the models. The reported models reflect all data and then are split into follow-up times of 2 to 3 years and 3+ years. In addition, sensitivity analysis was conducted to assess the impact of the Spanish workplace study\textsuperscript{23} given the gender confounding concern. This latter sensitivity analysis indicated little difference and has not been further reported. The final prediction models for BP differences are presented in Table 3.

The best predictors of difference in DBP used baseline DBP along with weight difference as independent variables.
However, this may reflect pharmacological treatment of hypertension rather than any weight loss effect. The model based only on weight differences accounted for 3% of the overall variation, suggesting the strength of relationship reduces with time (Table 3).

### Discussion

This review determined weight loss effects on BP at ≥2 years from studies with lifestyle interventions for weight loss (or with intentional weight loss recorded). The target population was set for those of normal weight up to BMI ≤35.

Considered overall, a 3-kg weight loss may reduce BP. The hypertension trials suggest that sodium-restricted diets even without weight loss are beneficial, but that weight loss from calorie control gave better BP reduction. Patients under chronic disease management, particularly those with diabetes, had better weight losses with consequent BP improvement.

Prediction of the impact of weight loss was not possible for DBP given its high dependency on baseline DBP, possibly reflecting the fact that high initial DBP would be medically treated. In comparison, prediction of SBP for this target group suggests that an individual with a 5 kg weight loss may expect on average a 5.6 mm Hg drop in SBP.

A review by Netter et al4 suggests that, for every kilogram of weight lost, a 1-mm Hg reduction is possible for both DBP and SBP. The findings here are similar for SBP but less predictable for DBP. Less responsive changes in DBP were also reported in our previous review on obese populations for all interventions of 2 or more years follow-up times.7,31

Clearly there is substantial heterogeneity between the different studies. The impact of the actual lifestyle interven-
tions without “true” control groups is difficult to assess, given that BP tends to increase over time and ideally this would be adjusted for in the analysis.

The effect of medication is difficult to disentangle, especially for DBP. Even for studies with no reported hypertension medication there is no guarantee that physicians did not prescribe antihypertensives. The TOHP study suggests that BP measurements would be censored after initiation of antihypertensive drug treatment. However, studies reported patients with reduced medication, and Basler showed that the greater the weight loss the greater the reduction of medication. Conducting an individual patient data (IPD) analysis may be useful, encompassing weight and BP differences along with other patient variables including medication.

It has been shown that initial reductions of BP seen with weight loss may be short lived. Even when weight loss is partially maintained in the long-term, BP tends to revert back to initial levels. The lack of predictability for studies of longer than 3 years for SBP differences seen in this review may reflect this, suggesting that although weight loss is important, other aspects of lifestyle difference (such as increased physical activity, or improved diet) may also be critical for hypertension, as suggested for other target groups in our previous review.7

Two of the studies, although included descriptively, were not combined analytically. Kauffmann et al suggested that BP improved with weight loss in their adherent group. Field et al concluded that there were no adverse effects of weight

<table>
<thead>
<tr>
<th>Table 3. Meta Regression to Predict Blood Pressure Differences With Weight Differences Using Weighted Least Squares</th>
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<tbody>
<tr>
<td></td>
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<tr>
<td>DBP&lt;sub&gt;ALL&lt;/sub&gt;*</td>
</tr>
<tr>
<td>SBP&lt;sub&gt;ALL&lt;/sub&gt;*</td>
</tr>
<tr>
<td>Follow-up ≤36 months</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Follow-up &gt;36 months</td>
</tr>
</tbody>
</table>

*aAll: all subgroups.
†P<0.01.
‡P<0.001.

Diastolic blood pressure differences (DBP), weight by 1/(mean diastolic difference variance).
Systolic blood pressure differences (SBP), weight by 1/(mean systolic difference variance).
cycling on hypertension. However, definitions of weight cycling have yet to be standardized. This review was not able to adequately account for confounders such as medication, salt reduction, duration of contact, or even weight category itself. Interventions often included environmental, behavioral, and attitudinal changes. However, attributing success to these components has not been possible.

Perspectives

This review suggests that lifestyle interventions are effective in reducing SBP, although the evidence for DBP was less convincing in this target group of normal to obese participants. This is an important group for obesity treatment but almost more importantly for obesity prevention and hence needs further more detailed investigation. A better understanding of treatment and prevention might be possible using individual patient data analysis, particularly on studies whose primary objective was weight reduction. This would allow for more flexible subgroup analysis and adjustments for covariates and confounding factors and may facilitate evaluation of interventions by taking their differing components into account.

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Disclosures

None.

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ONLINE SUPPLEMENT

Long-term weight loss from lifestyle intervention benefits blood pressure? - A systematic review

Short title: Long-term lifestyle weight loss benefits BP?

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Email: laucott@abdn.ac.uk
<table>
<thead>
<tr>
<th>Author, Year, Country</th>
<th>Study description</th>
<th>Intervention</th>
<th>Proportion of Females</th>
<th>Follow up proportion</th>
<th>n</th>
<th>Age</th>
<th>BMI</th>
<th>DBP</th>
<th>SBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>HPT (1990) US (11)</td>
<td>Hypertension Prevention Trial</td>
<td>Calorie and/or sodium restricted diet (excluded if on HT medication)</td>
<td>0.35</td>
<td>&gt;0.88</td>
<td>841</td>
<td>38.6</td>
<td>27</td>
<td>83</td>
<td>125</td>
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<tr>
<td>Haskell (1994) US (12)</td>
<td>SCRIP study investigating effect of intensive multiple risk factor reduction programme in men and women with CAD</td>
<td>Diet, physical activity and medications (lipid and HT)</td>
<td>0.14</td>
<td>0.39</td>
<td>300</td>
<td>56 (7.4)</td>
<td>27 (4)</td>
<td>71 (9)</td>
<td>121 (17)</td>
</tr>
<tr>
<td>TOHP (1997) US (13)</td>
<td>Trials of Hypertension Prevention, Phase II. 2x2 factorial clinical trial to provide a firmer basis for preventing high BP</td>
<td>Diet (reduced sodium) &amp; exercise (PA). Not hypertensive patients</td>
<td>0.34</td>
<td>0.61</td>
<td>2382</td>
<td>Range 30-54</td>
<td>*32 (5)</td>
<td>86 (2)</td>
<td>128 (6)</td>
</tr>
<tr>
<td>Ditschuneit (1999) Germany (14) and...</td>
<td>Prospective dietary, two-arm, parallel 12-week intervention followed by prospective, single-arm, (Group A): Diet then Diet + 1 meal and snack replaced. Follow up at 27 and 51 months</td>
<td></td>
<td>0.82</td>
<td>0.63 &amp; 0.76</td>
<td>50</td>
<td>46.5 (11.6)</td>
<td>34 (4)</td>
<td>82 (5)</td>
<td>140 (14)</td>
</tr>
<tr>
<td>Study</td>
<td>Country/Region</td>
<td>Study Design/Intervention</td>
<td>Group B</td>
<td>n</td>
<td>12-Month Change in Weight (Mean ± SD)</td>
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<td>Flechtner-Mors (2000)</td>
<td>Germany (15)</td>
<td>4-year trial on Slim Fast replacements for meals &amp; snacks</td>
<td>0.76</td>
<td>0.64 &amp; 0.74</td>
<td>50</td>
<td>44.8 (9.7)</td>
<td>33 (4)</td>
<td>82 (5)</td>
<td>139 (15)</td>
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<td>Kuller (2001)</td>
<td>US (16)</td>
<td>To prevent rise of LDL cholesterol and weight gain in premenopausal women</td>
<td>1.00</td>
<td>0.46</td>
<td>535</td>
<td>47 (2)</td>
<td>25 (3)</td>
<td>68 (8)</td>
<td>110 (12)</td>
</tr>
<tr>
<td>Heshka (2003)</td>
<td>US (17)</td>
<td>Multi-centre trial comparing self-help weight loss versus a structured commercial weight loss programme</td>
<td>0.85</td>
<td>0.73</td>
<td>423</td>
<td>44.5 (10)</td>
<td>34 (4)</td>
<td>79 (9)</td>
<td>122 (13)</td>
</tr>
<tr>
<td>Lindstrom (2003)</td>
<td>Finland (18)</td>
<td>Finnish Diabetes Prevention Study Clinical trial investigating the effects of an intervention programme designed to prevent or delay the onset of T2DM in IGT’s</td>
<td>0.67</td>
<td>0.49</td>
<td>522</td>
<td>55 (7)</td>
<td>31 (5)</td>
<td>86 (9)</td>
<td>140 (18)</td>
</tr>
</tbody>
</table>
Kukkonen-Harjula (2005) Finland (19)

Investigating effect phased diet and exercise programmes on occurrence of Metabolic Syndrome in Men

Diet & Physical Exercise (walking or resistance).
Those on regular medication excluded

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| Diet & Physical Exercise | 0.00 | 0.51 | 90 | 42 Range 34-50 | 33 (3) | 83 (10) | 131 (13) |

* Average baseline weights (kg) converted into BMI using average height of American People.(29)

CAD: Coronary artery disease; SCRIP: The Stanford coronary risk intervention project; HRT: hormone replacement therapy; BP: blood pressure; HT: hypertension; IGT: impaired glucose tolerance; LDL: low density lipoprotein; PA: physical exercise; T2DM: type 2 diabetes mellitus.
<table>
<thead>
<tr>
<th>Author, Year, Country</th>
<th>Study description</th>
<th>Intervention</th>
<th>Proportion of Females</th>
<th>Follow up proportion</th>
<th>Baseline Mean (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Basler (1991) Germany (20)</td>
<td>Prospective hypertension study to reduce body weight, cholesterol levels, and need for medication. Patients from primary care units.</td>
<td>Group Behaviour counselling on lifestyle differences – 12 weekly 90 min sessions. Encouraged to reduce HT medication</td>
<td>0.80</td>
<td>0.06</td>
<td>965 57.4 (9.8) * 30 (5) 94 (12) 153 (20)</td>
</tr>
<tr>
<td>Eriksson (1991) Sweden (21)</td>
<td>Malmö Prospective Study feasibility of long-term intervention to reduce risk factors for newly diagnosed type 2 diabetes melitus</td>
<td>Dietary advice and physical activity program. 15% on HT medication at baseline reduced to 7.4% by 6th year</td>
<td>0.00</td>
<td>0.89</td>
<td>181 48.1 (0.7) 27 (3) 92 (10) 137 (16)</td>
</tr>
<tr>
<td>Kaufmann (1992) Chile (22)</td>
<td>Prospective workplace study—2 year follow-up of a cardiovascular risk control programs</td>
<td>Diet + exercise advice using several programs</td>
<td>0.15</td>
<td>1.00</td>
<td>836 45 (8.67) 25 (0)</td>
</tr>
<tr>
<td>Author, Year, Country</td>
<td>Study description</td>
<td>Intervention</td>
<td>Proportion of Females</td>
<td>Follow up proportion</td>
<td>n</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>-----------------------</td>
<td>----------------------</td>
<td>---</td>
</tr>
<tr>
<td>Martinez-Gonzalez (1998) Spain (23)</td>
<td>Prospective workplace study—3 year follow up of a multifactorial workplace program aimed at preventing cardiovascular disease</td>
<td>Diet (sodium and alcohol reduction and how to reduce weight and cholesterol) plus physical activity advice</td>
<td>† 0.23</td>
<td>0.63</td>
<td>1555</td>
</tr>
<tr>
<td>Field (1999) USA (24)</td>
<td>The Nurses Health Study II Prospective observational study data</td>
<td>Intentional weight loss (weight cycling). Monitored up to HT development so not on medication</td>
<td>1.00</td>
<td>1.00%</td>
<td>46,224</td>
</tr>
<tr>
<td>Sjostrom (1999) Sweden (Raw data available) (25)</td>
<td>Prospective clinical study designed to test the short- and long-term effectiveness of a 4-week residential program to control obesity and related CVD risk factors</td>
<td>Four week residential diet + exercise program. HT was a referral risk factor, will have HT medication</td>
<td>0.56</td>
<td>0.33</td>
<td>2493</td>
</tr>
<tr>
<td>Author, Year, Country</td>
<td>Study description</td>
<td>Intervention</td>
<td>Proportion of Females</td>
<td>Follow up proportion</td>
<td>n</td>
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<tr>
<td>-----------------------</td>
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</tr>
<tr>
<td>Welty (2007) USA (26)</td>
<td>Examine effect of diet &amp; exercise on weight loss and lipid levels</td>
<td>Diet &amp; Exercise counselling program</td>
<td>NA</td>
<td>0.75-1.00</td>
<td>80</td>
</tr>
<tr>
<td>Schillaci (2003) Italy (27)</td>
<td>PIUMA Prospective observational study - long-term effect of weight differences on average 24-h blood pressure</td>
<td>Dietary advice to reduce salt as well as for weight loss. Those on HT medication excluded for this paper</td>
<td>0.34</td>
<td>1.00</td>
<td>181</td>
</tr>
</tbody>
</table>

* Weight (kg) converted to BMI using European height tables. (28)

† Refers to the 980 individuals in the final analysis

‡ Raw data for total sample of 2493 available from author (Sjostrom 1999 reports data from a sub sample of 100). (25)

§ Only 9 primary care units with n=54 patients participated in 2nd year follow-up

PIUMA: Progetto Ipertensione Monitoraggio Ambulatoriale; HT: hypertension; CVD: cardiovascular disease