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Diet Behavior Change Techniques in Type 2 Diabetes: A Systematic Review and Meta-analysis

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Abstract

BACKGROUND Dietary behavior is closely connected to type 2 diabetes.

PURPOSE To identify behavior change techniques (BCTs) and specific components of dietary interventions for patients with type 2 diabetes associated with changes in HbA_{1c} and body weight.

DATA SOURCES The databases: Cochrane Library, CINAHL, EMBASE, PubMed, PsycINFO, and SCOPUS were searched.

STUDY SELECTION RCTs published between 1975-2017 that focused on changing dietary behavior.

DATA EXTRACTION Methodological rigor, use of BCTs, fidelity and intervention features were evaluated.

RESULTS In total, 54 studies were included, with 42 different BCTs applied using an average of 7 BCTs per study. Four BCTs, 'problem solving', 'feedback on behavior' 'adding objects to the environment', 'social comparison', and the intervention feature, 'use of theory', were associated with >0.3% (3.3 mmol/mol) reduction in HbA_{1c}. Meta-analyses revealed that studies that aimed to control or change the environment showed a greater reduction in HbA_{1c} of 0.5% (5.5 mmol/mol [95% CI -0.65, -0.34]) compared to 0.32% (3.5 mmol/mol [95% CI -0.40, -0.23]) for studies that aimed to change behavior.

LIMITATIONS Heterogeneity of dietary interventions and poor quality of reporting of BCTs.

CONCLUSIONS This study provides evidence that changing the dietary environment may have more of an effect on HbA_{1c} in adults with type 2 diabetes than changing dietary behavior. Diet interventions achieved clinically significant reductions in HbA_{1c}, while initial reductions in body weight diminished over time. If appropriate BCTs and theory are applied, dietary interventions may result in better glucose control.

Introduction

Dietary behavior is intricately linked to type 2 diabetes and has become an increasingly complex phenomenon to understand and change. There is a long association between diet and the pathogenesis of type 2 diabetes. A recent study suggested that reduced risk of type 2 diabetes was strongly associated with dietary factors such as: greater intake of fruit, vegetables, legumes, nuts, whole grains, long-chain fats and a lower intake of sugar sweetened beverages (1), trans fat, processed/red meats, sodium and a moderate alcohol intake (2). Dietary factors have also been linked to the highest proportion of deaths in type 2 diabetes, stroke and heart disease (3). There is a need to identify factors associated with effective clinical outcomes in dietary interventions (4-6). Identifying effective behavior change techniques (BCTs) in successful dietary approaches to type 2 diabetes may help to refine and improve the scalability of successful approaches to changing dietary behavior. A BCT is an observable, replicable, and irreducible component of an intervention designed to alter or redirect causal processes regulating behavior, such as ‘feedback’ or ‘self–monitoring’ (7). The objective of this systematic review and meta-analyses was to identify dietary BCTs, intervention features and specific diets associated with changes in HbA_{1c} and body weight in type 2 diabetes.

Methods

This systematic review and meta-analysis followed a registered protocol, (http://www.crd.york.ac.uk/PROSPERO/display_record.asp?ID=CRD42016042466). A PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) checklist was created and PRISMA review guidelines were followed (Supplementary file S1) .

Inclusion Criteria

(i) Randomized controlled trials (RCTs) of any duration employing a dietary intervention published in peer-reviewed journals between 1/1/1975 and 12/4/2017.

(ii) RCTs with a comparison arm or control group that constituted usual care. Usual care could include typical diabetes dietary treatment such as ADA (American Diabetes Association Diet) or carbohydrate exchange type diets.

(iii) Human participants older than 18 years of age with clinically confirmed type 2 diabetes, at time of recruitment.

(iv) Primary clinical outcome measure was HbA_{1c}. However studies reporting HbA_{1c} results as a secondary outcome measure were also included.

(v) Randomized cross-over trials were included if relevant outcome data were reported for both intervention and control groups prior to subjects crossing over to the other diet.

Exclusion criteria

(i) RCTs of diabetes prevention or RCTs in populations at risk of type 2 diabetes.

(ii) RCTs that used pharmacological agents exclusively to treat type 2 diabetes.

(iii) RCTs that included supervised physical activity.

(iv) RCTs that targeted multiple chronic diseases, gestational diabetes or type 1 diabetes.

(v) Studies not reported in English.

(vi) Studies that focused exclusively on supplement or micronutrient use.

Information sources and search strategy

The following databases were searched: Cochrane Library, CINAHL, EMBASE, PubMed, PsycINFO, and SCOPUS using a Boolean combination of key words and MeSH headings (Supplementary file S2). Search terms were developed following the protocol of an earlier review (8) and a series of sensitivity analysese of terms, cross checking results against identified reference criteria. Additional records identified from other sources such as reference lists of relevant reviews, studies with multiple intervention arms in an earlier review and all included studies were searched for additional sources. The original search was conducted on 22 February 2016 and repeated on 12 April 2017.

Article Screening

Articles were initially screened by two research team members based on titles and abstracts and then full texts of the remaining articles (KC and RM), see Figure 1. A third member of the review team (HG) oversaw any disagreements of search results and had the final say on included studies. Inter-rater agreement by Cohen's Kappa for the full text search results was 0.82.

Article Classification

Studies that aimed to control or change environment were classified as studies where all food or the majority of food was provided to participants. Studies in this category could also be described as studies with high internal validity. Studies that aimed to change behavior were classified as studies where participants were instructed or educated about diet changes by dietitians or health care professionals, included a variety of diets and no food was provided. Studies in this category could also be described as having high external validity.

Low carbohydrate diets were classified as studies where carbohydrate intake of <130g/ day was recommended (9). Low fat diets were classified as studies where dietary fat intake of <30% was recommended (10). High protein diets were classified as studies where protein intake of >2g/kg/day was recommended (11).

Data extraction process

Data extraction was carried out by one member of the team (KC) and relevant information was stored in Excel file templates. All data regarding HbA_{1c}, weight loss, intervention features, BCTs, fidelity coding and risk of bias was checked by another member of the research team (RM). All corresponding authors were contacted by email (where contact details were available, n = 50 of 54) using a standardized template to request additional information. The response rate was 34% (over a period of six weeks).

Risk of bias and fidelity assessment

The Cochrane Collaboration risk of bias tool was used to assess methodological quality (12), where assessment criteria are applied to seven aspects of trials to yield an appraisal of 'low risk', 'high risk' or 'unclear risk' of bias. Studies were independently assessed by two members of the review team for risk of bias and methodological quality (KC and RM). Assessment results were discussed and agreed after the first ten studies and again after the first 20 studies. Inter-rater reliability was calculated and discrepancies were discussed after each round. Treatment fidelity was assessed according to the five categories proposed by Bellg *et al.* (13). Each category and subcategory were assigned a score of yes, no, or unclear. RCTs were independently assessed by two members of the review team (KC and HG) and results were discussed and assessments repeated following discussion of the first 10 and first 20 articles. Inter-rater reliability is based on the final 34 studies.

Coding of Behavior Change Techniques

Michie's v1 BCT taxonomy (7) was used to identify and code BCTs related to diet only that were identified in each study. A list of all 93 BCTs and their descriptions is available (http://www.bct-taxonomy.com) (7). A BCT was only coded when it was explicitly mentioned in the intervention methodology. BCTs were coded separately for intervention and control groups. A coding rubric was developed by three authors (KC, RM and HG) to guide the coding process (Supplementary file S3). All included studies were coded independently by two authors (KC and RM), immediately after training in the use of Michie's taxonomy. A third master coder (HG) independently assessed the coding results and arbitrated any disagreements. Cohen's kappa and PABAK calculations were used to establish inter-coder reliability of BCTs present and absent. A BCT must have been used in at least three studies for inclusion in the moderator analysis (14). Both coders discussed coding practices and results after coding the first study and again after independently coding the remaining 53 studies. All available information including study manuals, protocols and earlier methodology papers were also used to code each study. Rationale for classification of intervention features such as mode of delivery, provider, intensity / frequency of intervention and other variables included is documented in an earlier review carried out by the review team (8). Intensity was defined as the number of total or face-to-face contacts with intervention personnel and frequency was defined as the average number of weeks between contacts.

Analysis

We defined a HbA_{1c} reduction of $\geq$0.3% (3.3 mmol/mol) as clinically significant, which follows the precedent set by others (14; 15). Meta-analyses were conducted using RevMan (v5.3) on the primary outcome measure of HbA_{1c} and the secondary outcome of body weight, where sufficient data were available. We recorded changes in outcomes at 0-3, 3-6, 6-12, and

12-24 months. The use of these time points allowed a greater number of studies to be included. In studies that reported data from multiple time-points, we used the time point closest to the end of the intervention for analysis. Mean differences and SDs in the differences for HbA_{1c} and weight loss between baseline and data reported at the different timepoints were calculated. SDs for missing data were calculated from SE, *p* and *t* values where reported, using the Cochrane guidelines or were calculated using a correlation (method documented in an earlier review by this research team) (8). Correlations of 0.75 (HbA_{1c}) and 0.98 (weight loss) were used to calculate missing standard deviations following a sensitivity analysis of correlations and reported standard deviations. Statistical significance of the moderator and meta-analyses was set at $p \leq 0.05$. In all cases the meta-analyses used the raw mean difference (RMD) and a random effects model to calculate results. Meta-analyses were carried out on both interventions that aimed to change behavior only and interventions that aimed to change environment only. Further meta-analyses were carried out on HbA_{1c} and weight loss at 0-3, 3-6, 6-12, 12-24 months. Meta-analyses were also carried out on different diet types.

Moderator analyses

A series of moderator analyses were carried out to investigate the association between BCTs/intervention features and clinical outcome results (HbA_{1c}) where the presence or absence of a certain BCT or intervention feature in certain studies was associated with changes in HbA_{1c}. The moderator analyses reported the standardized mean difference (SMD) in outcomes, using Comprehensive Meta-Analysis software (CMA). The random effects analysis was used to conduct all moderator analyses. For every moderator variable (BCT and intervention feature), we calculated the point estimate, 95% confidence intervals, Q statistic and p-value.

As a result of the large effect size observed in the control group, a series of subgroup analyses were carried out in an attempt to elucidate the true effect size of intervention groups compared to control groups. Further subgroup sensitivity analyses were carried out on true control groups where studies with >1 contact with a dietitian in control group were excluded, and additional subgroup analysis of studies where studies with a control group change in HbA_{1c} of $\geq$-0.3% (3.3 mmol/mol) were removed. Moderator analyses of BCTs associated with reductions in HbA_{1c} were also carried out on studies using interventions that aimed to change behavior primarily, environment primarily, and studies at three months only.

Results

Study selection and study characteristics

The inclusion criteria were met by 54 studies (16-69). Summary characteristics of included studies are outlined in Supplementary File S4. Average age of participants was 57.7 ($\pm$4.7) years for intervention groups and 58.1 ($\pm$5.1) years for controls. For intervention and control groups respectively, mean duration of diabetes, where reported, was 7.6 ($\pm$3.3) and 7.3 years ($\pm$3.1), mean baseline HbA_{1c} 8.1% ($\pm$1%) (69.8 mmol/mol) and 8.12% ($\pm$1%) (69.9 mmol/mol), BMI 32.1 kg/m^2 ($\pm$4.1) and 31.8 kg/m^2 ($\pm$4). Six of the included studies were carried out in a community center setting (12 studies did not report the setting), one online study, one study in a hotel setting, and all remaining studies (n = 34) were carried out in a clinical or academic setting. All participants in the 54 studies were classified as having type 2 diabetes, however diagnostic criteria for HbA_{1c} varied among included studies from a minimum of 5.5% (47.4 mmol/mol) (34) to a maximum of 12% (103.4 mmol/mol) (23; 57). The mean percentage of dropouts per study was lower in the intervention (12.6%) groups than control groups (16.4%). Studies with low glycaemic index and high protein diets (mean percentage number of dropouts 1% and 1.8% respectively) reported the lowest number of dropouts, with meal replacement studies reporting the highest dropout rate (mean percentage number of dropouts 28%) (Supplementary file S5).

Risk of bias and treatment fidelity

In the assessments of risk of bias, 63% were ‘unclear’ while 34% were ‘low’ across all seven variables with only 2% classified as ‘high’ risk of bias (Supplementary file S6a, S6b). Raters agreed on 78% of risk of bias assessments following initial assessment and came to agreement on the remainder through discussion. Treatment fidelity results are documented in Supplementary file S7. Overall, the reported use of treatment fidelity strategies was very low with 78% assessed as having ‘not’ used a treatment fidelity strategy. The most widely used treatment fidelity strategy was in the subcategory of ‘monitoring and improving enactment of treatment skills’ where 68.5% of all studies reported use of ‘ensure participant use of behavioral skills’. Raters agreed on 76.5% of assessments, and resolved remaining disagreements through discussion and arbitration.

Meta-analyses of diet interventions

Meta-analysis of interventions that aimed to change behavior (n = 37) showed an overall reduction in HbA_{1c} of 0.32% (3.5 mmol/mol [95% CI -0.40, -0.23 $p < 0.00001$]) while interventions that aimed to change or control the environment showed an overall reduction in HbA_{1c} of 0.5% (5.5 mmol/mol [95% CI -0.65, -0.34 $p < 0.00001$])(Figure 2). Sensitivity analysis removing studies with a $\geq$0.3% (3.3 mmol/mol) reduction in HbA_{1c} in the control group (n = 21) increased further the observed effect size on HbA_{1c} with behavioral interventions showing a reduction of 0.32% (3.5 mmol/mol [95% CI -0.41, -0.24 $p < 0.00001$]) compared to 0.66% (7.3 mmol/mol [95% CI -0.88, -0.45 $p < 0.00001$]) with environment controlled studies (n = 11) (Supplementary file S8).

Studies included in this review focused on different dietary approaches: low carbohydrate (n = 9), low fat (n = 16), high protein (n = 5), meal replacements (n = 4), low glycemic index (n = 3), medical nutritional therapy (n = 2), Mediterranean (n = 2) and others (n = 13). There was considerable heterogeneity in the diets used in control groups. There was a “true” control group in 28 studies, where no additional intervention support or contact was provided. In 16 studies, ADA or AHA guidelines were applied to control groups with varying degrees of intervention support provided (Supplementary file S9). The duration of interventions carried out ranged from four weeks to two years. In 21 studies, there was an additional minor physical activity component.

Studies using meal replacements (0.56% / 6.2 mmol/mol) and high protein diets (0.5% / 5.5 mmol/mol) were associated with the greatest reductions in HbA_{1c}. Low carbohydrate diets of 0.44% (4.8 mmol/mol) showed a greater reduction in HbA_{1c} than low fat diets 0.40% (4.4 mmol/mol), or low glycemic index diets of 0.09% (1 mmol/mol) (Supplementary file S10).

Meta-analysis of changes in HbA_{1c} and body weight at different time points

Meta-analyses showed differences in HbA_{1c} between intervention and control groups at different time points, presented graphically in Figure 3. Combining all studies and all time points in one overall meta-analysis, (n = 59, 54 studies) showed a reduction in HbA_{1c} of 0.35% (3.8 mmol/mol [95% CI -0.43, -0.28 p < 0.00001]) (Supplementary file S11). Heterogeneity as measured by I^2 was 62%, 44%, 38% and 68% at 0-3, 3-6, 6-12 and 12-24 months respectively. Sensitivity analysis comparing data at exactly 3, 6, 12 and 24 months to that of data at 0-3, 3-6, 6-12 and 12 to 24 months using a larger dataset, n = 54, showed no significant differences.

The difference in body weight loss between intervention and control groups was 2.34kg (95% CI -2.99, -1.69 p <0.00001), 2.94kg (95% CI -3.92, -1.97 p < 0.00001), 2.27kg (95% CI -3.32, -1.21 p < 0.0001) and 2.14kg (95% CI -3.34, -0.93 p = 0.0005), at 0-3, 3-6, 6-12 and 12-24 months respectively (Supplementary file S12). Combining all studies and time points revealed a reduction in body weight of 2.41kg (95% CI -2.96, -1.86 p < 0.00001), (Supplementary file S13). Heterogeneity as measured by I^2 was 84%, 93%, 88% and 27% at 0-3, 3-6, 6-12 and 12-24 months respectively.

BCTs used

A total of 42 distinct BCTs were applied in the intervention groups, seven of which were reported only once. The number of BCTs used in a single RCT ranged from three (25; 35; 45) to 17 (41). The five most frequently occurring BCTs were: ‘instruction on how to perform a behavior’ (n = 54), ‘credible source’ (n = 45), ‘self-monitoring of behavior’ (n = 37), ‘monitoring of behavior by others without feedback’ (n = 32) and ‘social support (unspecified)’ (n = 24) (Table 1).

Control group BCTs were coded separately, 28 different BCTs were identified with the number of BCTs used in a single study ranging from 0 (49; 60) to 15 (44) (Supplementary file S14). Inter-rater agreement as determined by Cohen’s kappa was 0.7 after coding 44 studies, and 0.68 after all 54 studies were coded. A breakdown of BCTs by category (Supplementary file S15) and BCTs ‘not’ used was also carried out (Supplementary file S16).

Moderator analysis of BCTs

The original moderator analysis showed no BCTs coded for diet behavior were associated with $\geq$ 0.3% (3.3 mmol/mol) reduction in HbA_{1c} (Supplementary file S17). Subgroup analysis of interventions using only true control groups showed that the BCTs ‘social comparison’ (0.52%, [5.7 mmol/mol] $p = 0.012$) and ‘feedback on behavior’ (0.365% [4 mmol/mol] p = 0.046) were associated with clinically and statistically significant reductions in HbA_{1c} (Supplementary file S18). Subgroup analysis of interventions excluding studies with control group pre-post change of $\geq$ 0.3% (3.3 mmol/mol) in HbA_{1c} also showed the BCT ‘feedback on behavior’ (0.34% [3.7 mmol/mol]) associated with clinically significant reductions in HbA_{1c} (Supplementary file S19). Subgroup analysis of BCTs reporting outcome changes at three months showed that the BCT ‘problem solving’ (0.63% [6.9 mmol/mol]) was associated with clinically significant reductions in HbA_{1c} (Supplementary file S20). Moderator analysis was not carried out at 12 months because insufficient data were available.

Subgroup analysis of interventions aimed at changing behavior showed that the BCTs ‘feedback on behaviour’ (0.52% [5.7 mmol/mol] $p = 0.007$) and ‘adding objects to the environment’ (0.39% [4.3 mmol/mol]) were associated with a clinically significant reduction in HbA_{1c} (Supplementary file S21). Subgroup analysis of interventions aimed at changing the dietary environment showed that the BCT ‘problem solving’ (0.5% [5.5 mmol/mol]) was associated with a clinically significant reductions in HbA_{1c} (Supplementary file S22).

Moderator analysis of intervention features

The original moderator analysis showed no intervention feature was associated with a clinically significant reduction in HbA_{1c} (Supplementary file 23). Subgroup moderator analysis excluding studies with control group pre-post change of ≥ 0.3% (3.3 mmol/mol) in HbA_{1c} showed that the only intervention feature associated with a clinically significant reduction in HbA_{1c} was the use of a theoretical model or framework (0.33% (3.6 mmol/mol). Other intervention features associated with reductions in HbA_{1c} were studies with a higher frequency and number of both total and face-to-face contacts with intervention personnel (Supplementary files S23).

Discussion

These findings suggest that changing or controlling dietary environmental factors may be more effective than strategies to change dietary behavior in attempting to reduce HbA_{1c} in adults with type 2 diabetes. High protein diets and meal replacement programmes produced the greatest reductions in HbA_{1c}. A clinically significant difference in HbA_{1c} at 0-3, 3-6, and 6-12 months was reported when all dietary approaches were combined in meta-analyses. Weight loss occurred, but diminished over time. Moderator analyses identified four BCTs--‘problem solving’, ‘feedback on behavior’ ‘adding objects to the environment’, ‘social comparison’ and the intervention feature ‘use of theory’--that were associated with clinically significant reductions in HbA_{1c}.

Diets where the environment was changed or controlled (e.g., where all food was provided) were more than twice as effective in reducing HbA_{1c} than diets using behavioral change interventions. This observation was consistent when a range of different foods were provided, including high protein diets (29; 30; 48), meal replacement diets (21; 27; 50; 57), low carbohydrate (51; 62), low fat (51; 55; 63), Mediterranean (33), Korean traditional (35), vegetarian (36) and partial formula or partial low calorie diets (52). These studies represent a more internally valid approach compared to studies aimed at changing behavior; however, successful externally valid interventions are required in order to change diet in a real-world setting. It has been suggested that environmental changes to social, built and food environments in addition to individual behavioral changes are required in order to adopt a healthy diet and lifestyle (70). Changing the environment has been identified as one of the overall theoretical themes associated with changing behavior particularly in the longer term (71).

In regard to the type of diet, our finding of a modest but statistically significant reduction in HbA_{1c} at three, six and twelve months, needs to be interpreted with caution as a range of different diets were combined in an effort to elucidate the most effective BCTs and intervention features. The observed reduction in HbA_{1c} is greater than in previous reports with fewer (n = 20) studies of high protein, low carbohydrate and low glycemic index diets. Our data also indicate that the use of meal replacements and high protein diets result in the greatest reduction in HbA_{1c}, with low carbohydrate diets showing a greater reduction in HbA_{1c} than low fat diets. However, meal replacement interventions also had the highest dropout rate, suggesting these interventions may not be externally valid or the most acceptable approach for participants. The average number of dropouts per study was the lowest for the high protein diets at 1.8% suggesting that it was the diet most acceptable to participants. The overall meta-analysis showed an overall weight loss of 2.41kg with the greatest decrease observed at six months (2.94kg) but diminishing over time—a pattern consistent with previous work (4).

Beyond the BCT ‘adding objects to the environment’ the three other BCTs ‘social comparison’, ‘feedback on behavior’ and ‘problem solving’ have all strong theoretical foundations and have been shown to be efficacious in other studies (5; 8; 72; 73). Given that the use of more BCTs was not associated with greater effectiveness, the pattern of application or fidelity of use of BCTs may be of greater importance. Of the 93 BCTs in Michie’s taxonomy (7), 51 BCTs were not found in any of the 54 reviewed studies. BCTs most frequently used came from the categories: ‘feedback and monitoring’, ‘shaping knowledge’, ‘goals and planning’, ‘comparison of behavior’ and ‘social support’. The BCTs ‘behavioral contract’ and ‘commitment’ were not used in any of the included studies. The studies reviewed focused almost exclusively on reflective motivation, suggesting that deployment of a wider range of BCTs needs to be investigated in changing dietary behavior and improving HbA_{1c} and body weight in adults with type 2 diabetes.

The only intervention feature in the moderator analysis that was associated with clinically significant reductions in HbA_{1c} was the ‘use of theory/model’ to inform interventions. Similar findings have been reported where dietary behavior interventions in cancer prevention were more effective when informed by theory (74). However, fidelity of the use of theory was not reported in the studies included in our review or other reviews (74) and descriptions of use of theory varied considerably from “integrated concepts from different theories” (46) to “behavior modification treatment used principles from the modern learning theory” (37), and “group educational classes were based on the social cognitive theory” (21). The social cognitive theory (75) was the only theory reported more than once (21; 46; 64).

Our findings might suggest that higher frequency and greater number of contacts are associated with greater reductions in HbA_{1c} which is consistent with our previous systematic review of combined diet and physical activity interventions (8), although, this may arise from more intervention content being delivered. However, we cannot be sure because fidelity was so poorly reported in almost all categories and in all studies apart from one subcategory of ‘monitoring and improving enactment of treatment skills’ where 68.5% of studies reported use of fidelity. This subcategory was coded ‘yes’ when the intervention description reported that subjects carried out a 3 or 7-day food record and this was reviewed by the dietician. The criteria (13) for intervention fidelity assessment do not take account of the extent of use of each category, which is particularly relevant in assessing participant adherence to dietary programmes, i.e. enactment. Low levels of enactment make it difficult to assess the efficacy of interventions.

These findings from large, well controlled dietary interventions have potentially important implications for type 2 diabetes management and suggest that interventions aimed at changing the dietary environment warrant further scrutiny. It is impractical from a treatment perspective to provide food and control the food environment as a scaleable solution to type 2 diabetes treatment in the community. However, this finding does provide evidence that changing or altering the food environment or using high internally valid interventions are efficacious. Providing foods at the beginning of a programme or intervention might be an effective strategy to help people manage their diabetes, followed by instruction on how to choose, shop for and prepare these foods, gradually weaning them off of reliance on foods provided. We would suggest that future studies look at the economic ramifications of changing the food environment from policy, marketing and farming perspectives. Individual behavior change efforts might benefit from increased awareness of the dietary environment and exerting greater control over one’s dietary environment. For example, the individual could be guided through an audit of their current home food environment (stored food supplies) and inappropriate food would be removed to eliminate potential impulse food consumption of inappropriate foods. If we really want to change diet in real world settings, we also need to find BCTs associated with successful externally valid dietary interventions.

Future studies ought to quantify intervention fidelity, which would allow the identification of more effective BCTs. The use of video or audio recordings of consultations with dietitians and other healthcare professionals may help to better define the range of BCTs being deployed in any given intervention.

A strength of our study is the use of the most recent and comprehensive taxonomy of behavior change available (7). We have provided a comprehensive analysis of fidelity categories and subcategories as well as detailed subgroup meta- and moderator analyses. A limitation of our study is the heterogeneity in the dietary interventions which included different diagnostic criteria for type 2 diabetes that are likely to have variable effects on HbA_{1c} and weight in patients with type 2 diabetes. However we think that this heterogeneity in the interventions and in their efficacy is likely to have increased random error rather than bias, making our findings even more compelling. The quality of reporting of BCTs in different studies varied considerably and was poor overall. This is particularly true of fidelity measures and any conclusions must be tempered with the recognition of significant inter- and intra-study variability in adherence to intervention protocols. As study protocols do not always stipulate each BCT used, BCTs are likely to have been under-reported.

In conclusion, this systematic review and meta-analysis provide evidence that changing dietary environment may be more important than focusing on dietary behavior in type 2 diabetes treatment. More robust reporting of content, fidelity, and frequency of BCTs and intervention fidelity as well as better alignment of intervention design with behavior change theory would be helpful in refining interventions so that they are more efficacious.

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Abbreviations

BCT: Behavior change technique; ADA: American diabetes association; AHA: American Heart Association; CCE: Conventional Carbohydrate Exchange; FPG: Fasting plasma glucose; PRISMA: Preferred reporting items for systematic reviews and meta-analyses; RCT: Randomized controlled trial; TidieR: Template for intervention description and replication checklist and guide; SD: Standard deviation.

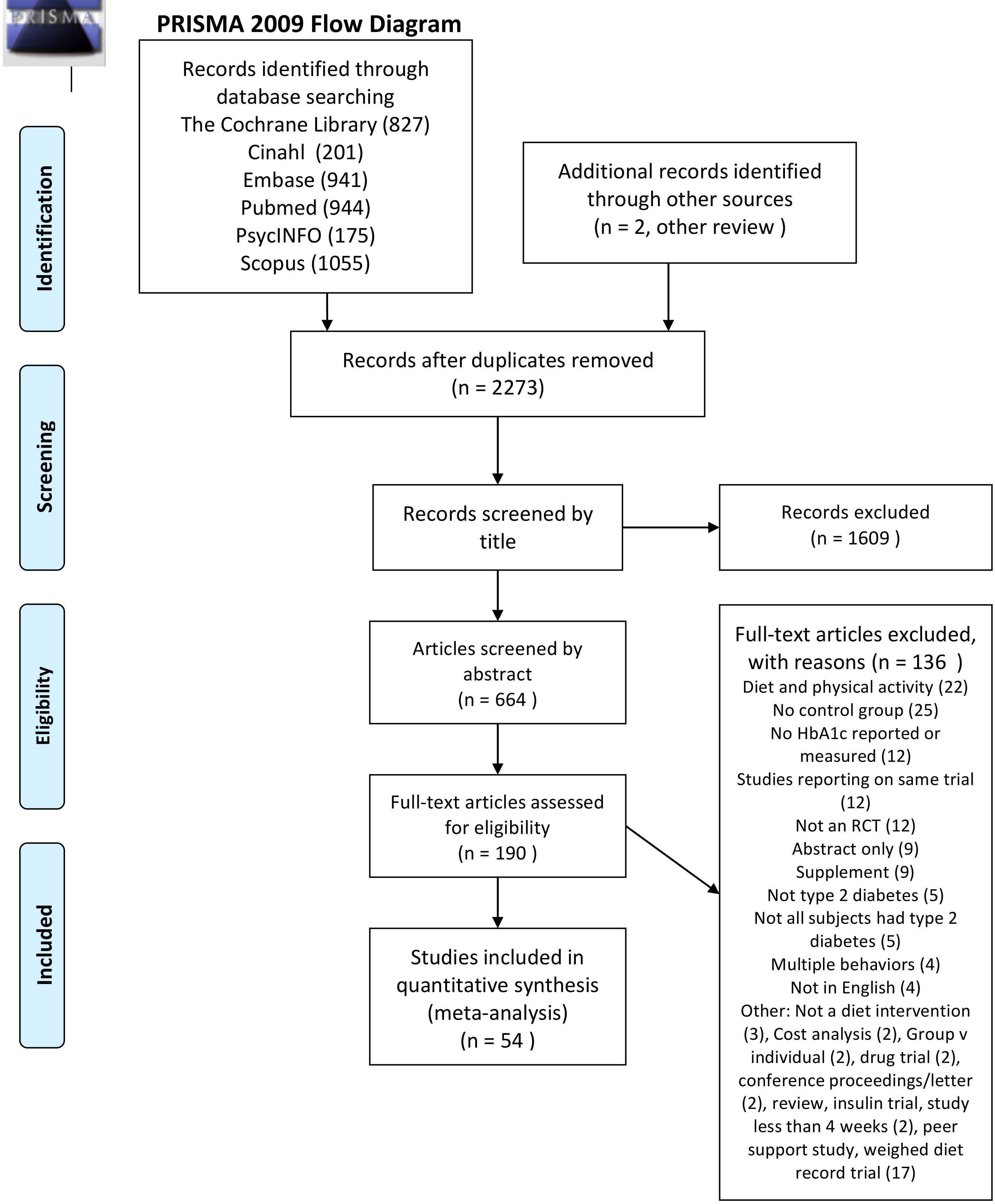
Disclosure statement

There was no conflict of interest declared by any of the authors.

Author contributions

KC, LQ, GOL, FF, HG and KMG formulated the research question, defined the search terms. KC carried out the electronic searches. KC and RM carried out the search process, the methodological assessment and the BCT coding, HG guided the BCT coding process and acted as a master coder. KC and HG carried out the fidelity assessment. KC carried out the moderator analysis and the meta-analysis. All authors were involved in writing and reviewing the final manuscript.

Figure 1: PRISMA 2009 Flow diagram of search process and results



Study or Subgroup	Intervention Mean	Intervention SD	Intervention Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Random, 95% CI
Al-Shookri et al 2012	−0.8	1.191	85	−0.4	1.587	85	2.6%	−0.40 [−0.82, 0.02]
Anderson-Loftin et al 2005	−0.5	1.063	38	−0.3	1.979	27	1.0%	−0.20 [−1.02, 0.62]
Andrews et al 2011	−0.09	0.665	246	0.09	0.69	93	5.4%	−0.18 [−0.34, −0.02]
Azadbakht et al 2011	−1.7	0.39	31	−0.5	0.79	31	3.6%	−1.20 [−1.51, −0.89]
Barnard et al 2009	−0.34	0.94	49	−0.14	0.85	50	3.2%	−0.20 [−0.55, 0.15]
Bowen et al 2016 A	−0.45	1.53	50	−0.25	1.847	25	0.9%	−0.20 [−1.04, 0.64]
Carter et al 2016	−0.6	0.8	31	−0.8	1	32	2.4%	0.20 [−0.25, 0.65]
Coppell et al 2010	−0.5	0.927	45	0	0.888	48	3.0%	−0.50 [−0.87, −0.13]
Daly et al 2006	−0.55	0.86	51	−0.23	0.66	51	3.7%	−0.32 [−0.62, −0.02]
Dyson et al 2007	−0.4	0.919	6	−0.2	0.919	6	0.6%	−0.20 [−1.24, 0.84]
Elhayany et al 2010 A	−2	0.927	61	−1.6	0.608	28	3.4%	−0.40 [−0.72, −0.08]
Elhayany et al 2010 B	−1.8	0.663	63	−1.6	0.608	27	3.9%	−0.20 [−0.48, 0.08]
Evangelista et al 2009 A	−0.7	0.2	5	−0.5	0.1	2	4.6%	−0.20 [−0.42, 0.02]
Evangelista et al 2009 B	−0.8	0.2	5	−0.5	0.1	2	4.6%	−0.30 [−0.52, −0.08]
Franz et al 1995	−1.1	1.2	88	−0.8	1.269	85	3.0%	−0.30 [−0.67, 0.07]
Goldstein et al 2011	−1	1.5	14	−1	1.1	16	0.7%	0.00 [−0.95, 0.95]
Imai et al 2011	−1.5	1.269	65	−0.9	1.217	27	1.8%	−0.60 [−1.15, −0.05]
Johnsson et al 2009	−1.1	0.4	13	−0.7	0.6	13	2.8%	−0.40 [−0.79, −0.01]
Kaplan et al 1987	−0.46	1.994	18	0.36	1.088	18	0.6%	−0.82 [−1.87, 0.23]
Kattelmann et al 2010	−0.3	1.515	51	−0.2	1.03	53	2.1%	−0.10 [−0.60, 0.40]
Kondo et al 2014	0	0.283	11	0	0.283	12	4.5%	0.00 [−0.23, 0.23]
Koo et al 2010	−0.59	0.61	19	−0.43	1	18	1.9%	−0.16 [−0.70, 0.38]
Laitinen et al 1993	−0.6	1.234	40	−0.3	1.32	46	1.9%	−0.30 [−0.84, 0.24]
Liu et al 2015	−0.49	0.772	58	−0.01	0.89	59	3.7%	−0.48 [−0.78, −0.18]
Luger et al 2013	−0.3	0.989	21	−0.2	0.678	21	2.0%	−0.10 [−0.61, 0.41]
Ma et al 2008	−0.35	0.91	19	−0.43	0.905	21	1.8%	0.08 [−0.48, 0.64]
Mesci et al 2010	−1.22	1.63	38	−0.59	1.56	35	1.2%	−0.63 [−1.36, 0.10]
Miller et al 2002	−0.5	0.474	45	0	0.48	47	5.0%	−0.50 [−0.69, −0.31]
Muchiri et al 2016	−1	1.32	41	−1	1.48	41	1.6%	0.00 [−0.61, 0.61]
Nicholson et al 1999	−1.4	1.138	7	−1	0.762	4	0.5%	−0.40 [−1.53, 0.73]
O' Neill et al 2016	−0.35	0.933	223	0.12	1	250	5.2%	−0.47 [−0.64, −0.30]
Saslow et al 2017	−0.8	0.372	11	−0.3	0.359	8	3.4%	−0.50 [−0.83, −0.17]
Sato et al 2017	−0.65	1.44	30	0	0.349	32	1.9%	−0.65 [−1.18, −0.12]
Talib et al 1997	−0.3	1.52	58	0.4	2.036	13	0.5%	−0.70 [−1.87, 0.47]
Trico et al 2016	−0.3	0.4	8	−0.2	0.212	9	3.6%	−0.10 [−0.41, 0.21]
Visek et al 2014	−0.415	2.036	20	−0.503	2.036	20	0.4%	0.09 [−1.17, 1.35]
Yamada et al 2014	−0.6	0.48	12	−0.2	0.678	12	2.3%	−0.40 [−0.87, 0.07]
Yang et al 2017	−0.3	0.707	33	0	0.99	23	2.2%	−0.30 [−0.77, 0.17]
Ziemer et al 2003	−1.9	1.606	94	−1.9	1.584	126	2.6%	0.00 [−0.43, 0.43]
Total (95% CI)			**1803**			**1516**	**100.0%**	**−0.32 [−0.40, −0.23]**

Heterogeneity: $Tau^2 = 0.03$; $Chi^2 = 72.90$, df = 38 (P = 0.0006); $I^2 = 48\%$
Test for overall effect: Z = 7.14 (P < 0.00001)

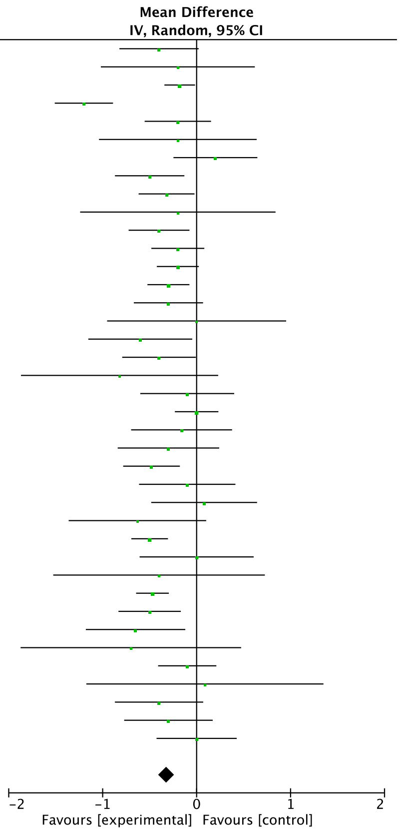


Figure 2A: Meta analysis of studies of dietary behavioral intervention (n = 37)

* Pedersen 2007 was not included as the intervention provided a portion control plate to subjects rather than a specific diet or food group.
** Yusof 2009 was not included as it did not specify the amount of food provided to subjects

	Intervention			Control				Mean Difference
Study or Subgroup	**Mean**	**SD**	**Total**	**Mean**	**SD**	**Total**	**Weight**	**IV, Random, 95% CI**
Cheskin et al 2008	−0.29	0.884	31	0.32	1.523	17	3.0%	−0.61 [−1.40, 0.18]
Foster et al 2009	−0.9	0.723	34	0.03	0.723	34	8.2%	−0.93 [−1.27, −0.59]
Gannon et al 2003	−0.8	0.692	12	−0.3	0.692	12	5.0%	−0.50 [−1.05, 0.05]
Gannon et al 2004	−2.2	0.827	6	0	0.502	2	2.2%	−2.20 [−3.16, −1.24]
Goday et al 2016	−0.9	0.435	45	−0.4	0.663	40	10.3%	−0.50 [−0.74, −0.26]
Itsiopoulos et al 2011	−0.3	1.012	27	0	1.123	27	4.8%	−0.30 [−0.87, 0.27]
Jung et al 2014	−0.72	0.324	21	−0.25	0.32	20	11.3%	−0.47 [−0.67, −0.27]
Kahleova et al 2011	−0.68	0.86	37	−0.59	0.89	37	7.2%	−0.09 [−0.49, 0.31]
Li et al 2016	−0.61	1.652	79	0.1	1.703	60	4.9%	−0.71 [−1.27, −0.15]
Nuttall et al 2008	−1.7	0.81	6	−1.65	0.471	2	2.3%	−0.05 [−0.97, 0.87]
Pi-Sunyer et al 1999	−1	1.1	92	−0.6	1.131	100	8.8%	−0.40 [−0.72, −0.08]
Rock et al 2014 A	−0.7	0.927	66	0.1	0.992	33	7.1%	−0.80 [−1.21, −0.39]
Rock et al 2014 B	−0.3	0.995	67	0.1	0.992	32	6.9%	−0.40 [−0.82, 0.02]
Shirai et al 2013	−0.6	1.1	119	−0.4	0.8	110	10.2%	−0.20 [−0.45, 0.05]
Williams et al 1998 A	−0.71	1.59	16	−0.23	1.04	8	1.8%	−0.48 [−1.54, 0.58]
Williams et al 1998 B	−0.97	1.7	15	−0.23	1.04	7	1.6%	−0.74 [−1.89, 0.41]
Yip et al 2001	−0.8	0.959	41	−0.6	1.1	16	4.4%	−0.20 [−0.81, 0.41]
Total (95% CI)			**714**			**557**	**100.0%**	**−0.50 [−0.65, −0.34]**

Heterogeneity: $Tau^2 = 0.04$; $Chi^2 = 33.22$, $df = 16$ ($P = 0.007$); $I^2 = 52\%$
Test for overall effect: $Z = 6.32$ ($P < 0.00001$)

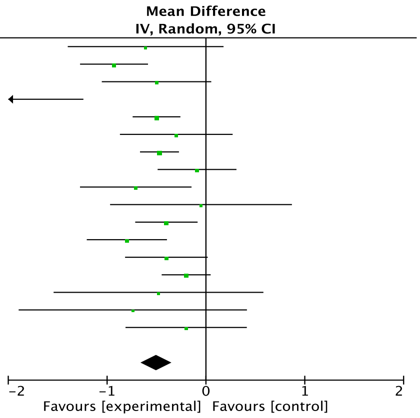


Figure 2B: Meta analysis of studies changing or controlling environment (n = 17)

Meta analyses of studies aimed at changing dietary behavior (2A) and studies aimed at changing dietary environment (2B).
Values reported in meta analyses represent mean difference and SD of the difference in HbA_{1c} from baseline to specific timepoint for intervention and control groups. Total figure provides the combined weighted difference of all studies between intervention and control groups. 95% CI are also reported.

	Intervention			Control				Mean Difference
Study or Subgroup	**Mean**	**SD**	**Total**	**Mean**	**SD**	**Total**	**Weight**	**IV, Random, 95% CI**
Al-Shookri et al 2012	−1.3	1.273	85	−0.8	1.6	85	3.1%	−0.50 [−0.93, −0.07]
Azadbakht et al 2011	−1.7	0.39	31	−0.5	0.79	31	4.0%	−1.20 [−1.51, −0.89]
Bowen et al 2016 A	−0.99	1.51	50	−0.98	1.44	25	1.7%	−0.01 [−0.71, 0.69]
Bowen et al 2016 B	−1.04	1.689	50	−0.98	1.44	25	1.6%	−0.06 [−0.79, 0.67]
Carter et al 2016	−0.6	0.8	31	−0.8	1	32	3.0%	0.20 [−0.25, 0.65]
Daly et al 2006	−0.55	0.86	51	−0.23	0.66	51	4.2%	−0.32 [−0.62, −0.02]
Dyson et al 2007	−0.4	0.919	6	−0.2	0.919	6	0.9%	−0.20 [−1.24, 0.84]
Evangelista et al 2009 A	−0.7	0.2	5	−0.5	0.1	2	4.8%	−0.20 [−0.42, 0.02]
Evangelista et al 2009 B	−0.8	0.2	5	−0.5	0.1	2	4.8%	−0.30 [−0.52, −0.08]
Foster et al 2009	−0.9	0.723	34	0.03	0.723	34	3.8%	−0.93 [−1.27, −0.59]
Franz et al 1995	−1.1	1.2	88	−0.8	1.269	85	3.6%	−0.30 [−0.67, 0.07]
Gannon et al 2003	−0.8	0.692	12	−0.3	0.692	12	2.4%	−0.50 [−1.05, 0.05]
Gannon et al 2004	−2.2	0.827	6	0	0.502	2	1.1%	−2.20 [−3.16, −1.24]
Goldstein et al 2011	−1.9	1.8	20	−1.2	1	17	1.2%	−0.70 [−1.62, 0.22]
Itsiopoulos et al 2011	−0.3	1.012	27	0	1.123	27	2.3%	−0.30 [−0.87, 0.27]
Johnsson et al 2009	−1.1	0.4	13	−0.7	0.6	13	3.4%	−0.40 [−0.79, −0.01]
Jung et al 2014	−0.72	0.324	21	−0.25	0.32	20	5.0%	−0.47 [−0.67, −0.27]
Kahleova et al 2011	−0.68	0.86	37	−0.59	0.89	37	3.3%	−0.09 [−0.49, 0.31]
Kondo et al 2014	0	0.283	11	0	0.283	12	4.7%	0.00 [−0.23, 0.23]
Koo et al 2010	−0.59	0.61	19	−0.43	1	18	2.5%	−0.16 [−0.70, 0.38]
Li et al 2016	−0.61	1.652	79	0.1	1.703	60	2.3%	−0.71 [−1.27, −0.15]
Luger et al 2013	−0.3	0.989	21	−0.2	0.678	21	2.6%	−0.10 [−0.61, 0.41]
Mesci et al 2010	−1.22	1.63	38	−0.59	1.56	35	1.6%	−0.63 [−1.36, 0.10]
Miller et al 2002	−0.5	0.474	45	0	0.48	47	5.0%	−0.50 [−0.69, −0.31]
Nicholson et al 1999	−1.4	1.138	7	−1	0.762	4	0.8%	−0.40 [−1.53, 0.73]
Nuttall et al 2008	−1.7	0.81	6	−1.65	0.471	2	1.2%	−0.05 [−0.97, 0.87]
O' Neill et al 2016	−0.62	0.837	251	−0.16	0.811	257	5.4%	−0.46 [−0.60, −0.32]
Pi-Sunyer et al 1999	−1	1.1	92	−0.6	1.131	100	4.0%	−0.40 [−0.72, −0.08]
Trico et al 2016	−0.3	0.4	8	−0.2	0.212	9	4.0%	−0.10 [−0.41, 0.21]
Visek et al 2014	−0.415	2.036	20	−0.503	2.036	20	0.7%	0.09 [−1.17, 1.35]
Williams et al 1998 A	−0.65	1.35	16	−0.03	1.03	7	1.0%	−0.62 [−1.63, 0.39]
Williams et al 1998 B	−0.4	1.14	16	−0.03	1.03	7	1.1%	−0.37 [−1.32, 0.58]
Yang et al 2017	−0.3	0.707	33	0	0.99	23	2.9%	−0.30 [−0.77, 0.17]
Yip et al 2001	−0.8	0.959	41	−0.6	1.1	16	2.1%	−0.20 [−0.81, 0.41]
Yusof et al 2009	−0.48	0.76	51	−0.31	0.945	49	3.8%	−0.17 [−0.51, 0.17]
Total (95% CI)			**1326**			**1193**	**100.0%**	**−0.37 [−0.48, −0.26]**

Heterogeneity: $Tau^2 = 0.05$; $Chi^2 = 88.94$, df = 34 ($P < 0.00001$); $I^2 = 62\%$
Test for overall effect: $Z = 6.58$ ($P < 0.00001$)

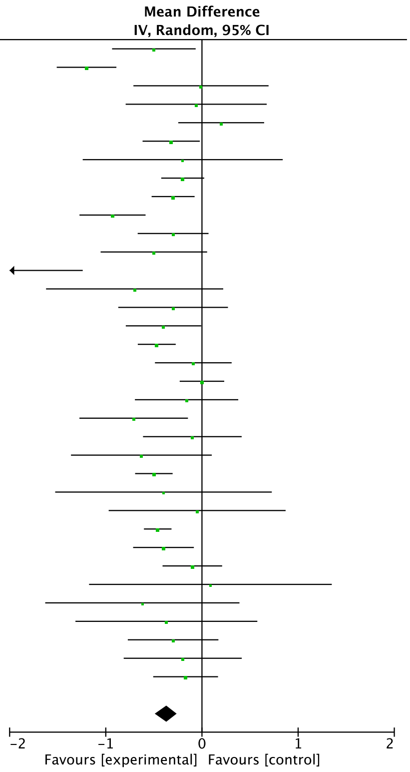


Figure 3A: Meta-Analysis of HbA_{1c} changes at 0-3 months (n = 35)

	Intervention			Control				Mean Difference	Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 95% CI	IV, Random, 95% CI
Al-Shookri et al 2012	−0.8	1.192	85	−0.4	1.587	85	4.3%	−0.40 [−0.82, 0.02]	
Anderson-Loftin et al 2005	−0.5	1.063	38	−0.3	1.98	27	1.6%	−0.20 [−1.02, 0.62]	
Andrews et al 2011	−0.07	0.714	247	0.14	0.721	97	8.7%	−0.21 [−0.38, −0.04]	
Barnard et al 2009	−1	1.2	49	−0.6	1.1	50	3.9%	−0.40 [−0.85, 0.05]	
Bowen et al 2016 A	−0.45	1.53	50	−0.25	1.847	25	1.5%	−0.20 [−1.04, 0.64]	
Bowen et al 2016 B	−1.13	1.847	50	−0.25	1.847	25	1.4%	−0.88 [−1.77, 0.01]	
Coppell et al 2010	−0.5	0.927	45	0	0.889	48	4.9%	−0.50 [−0.87, −0.13]	
Franz et al 1995	−0.9	1.192	88	−0.7	1.269	85	5.0%	−0.20 [−0.57, 0.17]	
Goday et al 2016	−0.9	0.435	45	−0.4	0.663	40	7.2%	−0.50 [−0.74, −0.26]	
Goldstein et al 2011	−1.6	1.8	20	−1	1.2	18	1.2%	−0.60 [−1.56, 0.36]	
Kattelmann et al 2010	−0.3	1.515	51	−0.2	1.03	53	3.4%	−0.10 [−0.60, 0.40]	
Laitinen et al 1993	−0.6	1.217	40	−0.1	1.414	46	2.9%	−0.50 [−1.06, 0.06]	
Ma et al 2008	−0.75	0.891	19	−0.67	0.905	21	2.9%	−0.08 [−0.64, 0.48]	
Muchiri et al 2016	−1.13	1.294	41	−1.1	1.47	41	2.6%	−0.03 [−0.63, 0.57]	
O' Neill et al 2016	−0.63	0.92	235	0.02	0.963	247	8.7%	−0.65 [−0.82, −0.48]	
Pedersen et al 2007	0.26	1.148	56	0.04	0.946	61	4.8%	0.22 [−0.16, 0.60]	
Rock et al 2014 A	−0.8	0.8	71	−0.2	0.992	30	4.5%	−0.60 [−1.00, −0.20]	
Rock et al 2014 B	−1.1	0.959	73	−0.2	0.992	30	4.3%	−0.90 [−1.32, −0.48]	
Saslow et al 2017	−0.9	0.394	12	−0.5	0.39	9	5.4%	−0.40 [−0.74, −0.06]	
Sato et al 2017	−0.65	1.44	30	0	0.349	32	3.2%	−0.65 [−1.18, −0.12]	
Shirai et al 2013	−0.6	1.1	119	−0.4	0.8	110	7.0%	−0.20 [−0.45, 0.05]	
Talib et al 1997	−0.3	1.52	58	0.4	2.036	13	0.8%	−0.70 [−1.87, 0.47]	
Williams et al 1998 A	−0.71	1.59	16	−0.23	1.04	8	1.0%	−0.48 [−1.54, 0.58]	
Williams et al 1998 B	−0.97	1.7	15	−0.23	1.04	7	0.9%	−0.74 [−1.89, 0.41]	
Yamada et al 2014	−0.6	0.48	12	−0.2	0.678	12	3.7%	−0.40 [−0.87, 0.07]	
Ziemer et al 2003	−1.9	1.606	94	−1.9	1.584	126	4.2%	0.00 [−0.43, 0.43]	
Total (95% CI)			**1659**			**1346**	**100.0%**	**−0.37 [−0.48, −0.26]**	

Heterogeneity: $Tau^2 = 0.03$; $Chi^2 = 45.01$, df = 25 (P = 0.008); $I^2 = 44\%$

Test for overall effect: Z = 6.45 (P < 0.00001)

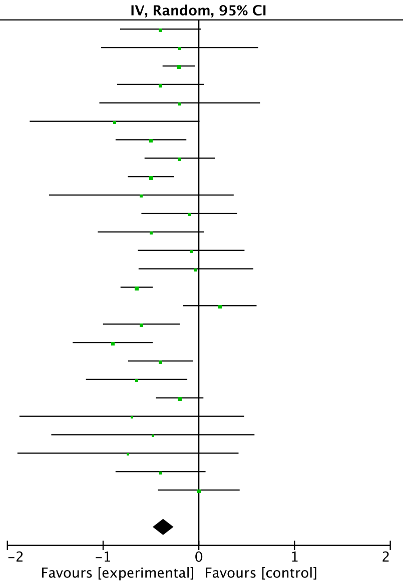


Figure 3B: Meta-Analysis of HbA_{1c} changes at 3-6 months (n = 26)

Study or Subgroup	Intervention Mean	Intervention SD	Intervention Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Random, 95% CI
Andrews et al 2011	−0.09	0.665	246	0.09	0.69	93	14.5%	−0.18 [−0.34, −0.02]
Cheskin et al 2008	−0.29	0.885	31	0.32	1.523	17	2.1%	−0.61 [−1.40, 0.18]
Elhayany et al 2010 A	−2	0.927	61	−1.6	0.608	28	8.1%	−0.40 [−0.72, −0.08]
Elhayany et al 2010 B	−1.8	0.663	63	−1.6	0.608	27	9.5%	−0.20 [−0.48, 0.08]
Goldstein et al 2011	−1	1.5	14	−1	1.1	16	1.5%	0.00 [−0.95, 0.95]
Imai et al 2011	−1.6	1.221	68	−1	1.217	30	4.2%	−0.60 [−1.12, −0.08]
Laitinen et al 1993	−0.6	1.234	40	−0.3	1.32	46	4.0%	−0.30 [−0.84, 0.24]
Li et al 2016	−0.42	1.597	76	0.35	1.382	59	4.4%	−0.77 [−1.27, −0.27]
Liu et al 2015	−0.49	0.772	58	−0.01	0.89	59	8.8%	−0.48 [−0.78, −0.18]
Ma et al 2008	−0.35	0.91	19	−0.43	0.905	21	3.7%	0.08 [−0.48, 0.64]
Muchiri et al 2016	−1	1.32	41	−1	1.48	41	3.3%	0.00 [−0.61, 0.61]
O' Neill et al 2016	−0.35	0.933	223	0.12	1	250	14.0%	−0.47 [−0.64, −0.30]
Rock et al 2014 A	−0.7	0.927	66	0.1	0.992	33	6.1%	−0.80 [−1.21, −0.39]
Rock et al 2014 B	−0.3	0.995	67	0.1	0.992	32	5.8%	−0.40 [−0.82, 0.02]
Saslow et al 2017	−0.8	0.372	11	−0.3	0.359	8	7.9%	−0.50 [−0.83, −0.17]
Talib et al 1997	−1.5	1.506	57	−0.5	1.287	15	2.2%	−1.00 [−1.76, −0.24]
Total (95% CI)			**1141**			**775**	**100.0%**	**−0.40 [−0.52, −0.28]**

Heterogeneity: $Tau^2 = 0.02$; $Chi^2 = 24.04$, df = 15 (P = 0.06); $I^2 = 38\%$

Test for overall effect: Z = 6.53 (P < 0.00001)

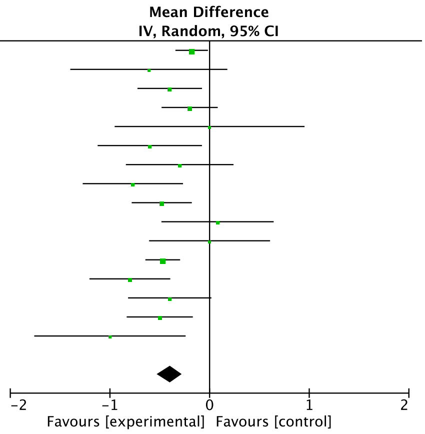


Figure 3C: Meta-Analysis of HbA_{1c} changes at 6-12 months (n = 16)

Study or Subgroup	Intervention Mean	Intervention SD	Intervention Total	Control Mean	Control SD	Control Total	Weight	Mean Difference IV, Random, 95% CI
Barnard et al 2009	−0.34	0.94	49	−0.14	0.85	50	27.3%	−0.20 [−0.55, 0.15]
Cheskin et al 2008	0	1	16	−1.2	1.125	8	14.9%	1.20 [0.28, 2.12]
Imai et al 2011	−1.5	1.263	65	−0.9	1.217	27	22.7%	−0.60 [−1.15, −0.05]
Kaplan et al 1987	−0.46	1.994	18	0.36	1.089	18	12.9%	−0.82 [−1.87, 0.23]
Uusitupa et al 1993	0	1.311	38	0.1	1.327	44	22.2%	−0.10 [−0.67, 0.47]
Total (95% CI)			**186**			**147**	**100.0%**	**−0.14 [−0.63, 0.35]**

Heterogeneity: $Tau^2 = 0.19$; $Chi^2 = 12.35$, df = 4 (P = 0.01); $I^2 = 68\%$
Test for overall effect: Z = 0.56 (P = 0.58)

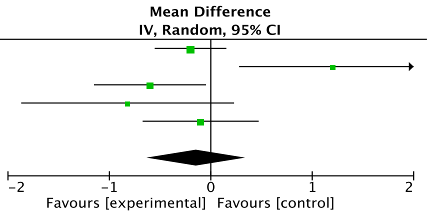


Figure 3D: Meta-Analysis of HbA_{1c} changes at 12-24 months (n = 5)

Values reported in meta analyses represent mean difference and SD of the difference in HbA_{1c} from baseline to specific timepoint for intervention and control groups. Total figure provides the combined weighted difference of all studies between intervention and control groups. 95% CI are also reported.

BCT no.	BCT Label	No of studies that reported BCT
4.1	Instruction on how to perform a behaviour	54
9.1	Credible source	45
2.3	Self-monitoring of behaviour	37
2.1	Monitoring of behaviour by others without feedback	32
3.1	Social support (unspecified)	24
1.1	Goal setting (behaviour)	23
12.5	Adding objects to the environment	22
2.4	Self-monitoring of outcome(s) of behaviour	15
2.5	Monitoring outcome(s) of behaviour by others without feeback	12
2.6	Biofeedback	12
6.1	Demonstration of the behaviour	12
1.3	Goal setting (outcome)	10
1.2	Problem solving	9
8.1	Behavioural practice/rehearsal	9
2.2	Feedback on behaviour	7
1.5	Review behaviour goal(s)	6
3.3	Social support (emotional)	6
13.2	Framing/reframing	5
1.7	Review outcome goal(s)	4
6.2	Social comparison	4
4.2	Information about antecedents	3
12.1	Restructuring the physical environment	3
1.4	Action planning	2
1.6	Discrepancy between current behaviour and goal	2
2.7	Feedback on outcome(s) of behaviour	2
5.1	Information about health consequences	2
7.1	Prompts/cues	2
8.2	Behaviour substitution	2
8.3	Habit formation	2

10.3	Non-specific reward	2
10.4	Social reward	2
11.2	Reduce negative emotions	2
11.3	Conserving mental resources	2
12.2	Restructuring the social environment	2
15.3	Focus on past success	2
3.2	Social support (practical)	1
5.4	Monitoring of emotional consequences	1
8.6	Generalisation of a target behaviour	1
8.7	Graded tasks	1
9.2	Pros and cons	1
10.9	Self-reward	1
12.3	Avoidance/reducing exposure to cues for the behaviour	1

Table 1: BCTs most frequently occurring

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